DETERMINANTS OF LATERALITY IN MAN

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For Diana

"Ease and leisure were given thee for
thy retired thoughts out of the sweat
of other men"

Milton

Summary

The thesis is divided into two fairly distinct parts. In the first part I describe a genetic model of cerebral dominance. This discussion commences with an account of the structure and description of handedness. This is followed by three chapters in which I suggest that left-handedness is not caused by birth-stress, and neither is there an unusually high incidence of sinistrality in epileptics or twins; there then follows a discussion of the whole concept of pathological left handedness, for which I suggest there is not adequate empirical evidence. Having thereby circumvented several major obstacles to the fitting of genetic models, I then describe a simple genetic model which will account for family and twin data on handedness. This is followed by the application of the model to cerebral speechdominance and aphasia. Finally the model is fitted to data on the inheritance of hand-clasping and arm-folding. This is followed by a brief consideration of the possible advantages of left-handedness. In the last chapter of the first part are considered several miscellaneous aspects of left-handedness and the inheritance of asymmetries.

The second part of the thesis considers aspects of the symbolism of laterality. The first chapter considers a conventional symbolism which derives at least from the

doctrine of Pythagorean opposites, and which influenced Greek sculpture and thought. The next chapter describes the "epidemiology" of a more unusual asymmetry, that of the portrayal of the left cheek in Western portraiture, and it is concluded that this is not a simple symbolism in the usual sense, but that it does have iconographical importance. The final chapter in this section describes two experiments on the particular meaning of the left and right cheek.

Contents

Page numbering is in the decimal system, as also is the Chapter numbering. Tables, figures and plates for each chapter are located at the end of that chapter, as are any appendices. References are all at the back of the thesis, and acknowledgements are all at the front of the thesis.

Part II: The Symbolism of Laterality

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Part I: The Biology of Laterality

"... many things are philosophically
delivered concerning right and left,
which admit of some suspension"

Browne, Pseudodoxia Epidemica, IV, V

CHAPTER 1: INTRODUCTION

"In the matter of left-handedness I should be rather inclined to regard the organic factor as the primary one, not the psychical, but I have no proper judgment about it all"

Sigmund Freud (see Jones, 1955)

The subject of this thesis, and in particular the reconciliation of the apparent disparity between the two parts, may be construed, like so many exercises in Western thought, as yet further 'footnotes to Plato', for it is in Plato that we find the first clear statements of our problem. In the Republic, Socrates tells of how Er had been taken up into heaven and seen two gateways,

> "and that judges were sitting between these, and that after every judgment they bade the righteous journey to the right and upward through the heaven ... and the unjust to take the road to the left and downward ....." (614.c).

In this quasi-Biblical passage Plato automatically assumes the symbolism of right representing good and left representing evil. This symbolism may be traced back further, and it was probably formalised by the Pythagoreans, and extant well before then. The unravelling of some of the ramifications of this symbolism will occupy the whole of Part II of this thesis.

Part I of the thesis discusses the biological aspects of laterality and in particular of handedness and as so often we may find evidence of a tension between the purely intellectual Plato and the more empiricist Aristotle. To Plato, as to most modern physicists up to the year 1957, it was offensive, and indeed incredible, that at bottom the universe could be anything but symmetric. In

the face of the greatest of human functional asymmetries, Plato could only suggest that,

> "it is only the folly of nurses and mothers to whom we owe it that we are all, so to say, lame of one hand. Nature, in fact, makes the members on both sides broadly correspondent; we have introduced the difference between them for ourselves, by our improper habits" (Laws, 794.d).

The theory was repeated by Plutarch, and reached Renaissance England through a translation by Sir Thomas Elyot (ca. 1535), who referred to the

> "... great folly or madness that where we do accustom our children to take meat with right hand, or if they do put forthe the left hand, anon we correct them"
>
> (Moralia, The Education of Infants, 4)

Thus was formed the environmentalist hypothesis for the origin of handedness; in its strong form it is little held today, but its weak form manifests in many guises.

Aristotle, the biologist, was too influenced by the great morphological asymmetries to be able to subscribe to the Platonic theory. The asymmetries of the heart and viscera led Aristotle to propose:-

> "And it is a universal law that, as regards above and below, front and back, right and left, the nobler part and more honourable part invariably is placed uppermost, in front, and on the right ....

unless some more important object stands in the way" (De Partibus Animalium, 665 a.21)

The latter proviso is, of course, to account for the heart's position, the heart being on the left, "so that it may counter-balance the chilliness of that side" (ibid, 666.b.8). Aristotle of course could not make the left superior since he also accepted the Pythagorean left-right symbolism (see his Metaphysics).

As a result of his theories, Aristotle felt that the predominance of right-handedness was a biological fact, and not just a cultural bias. Thus:-

> "I mean, for instance, if we were all to practice always throwing with the left hand, we should become ambidextrous. But still by nature left is left, and the right is none the less naturally superior to the left hand, even if we do everything with the left as we do with the right. Nor because things change does it follow that they are not by nature. But if for the most part and for the greater length of time the left continues thus to be left and the right right, this is by nature"
>
> (Magna Moralia, 1194.b.32)

The dichotomy between Plato and Aristotle thus typifies the modern dispute between those who profess a biological or a cultural origin of handedness.

A further historical complexity, perhaps also derivative from the Greeks, is a change in the status of symmetry. George Herbert, the metaphysical poet, could

write that, "Man is all symmetrie", and thus be, in essence, a Platonist. Sir Isaac Newton echoed Herbert when he said:-

> "Can it be by accident that all birds, beasts and men have their right side and left side alike shaped (except in their bowels)... Whence arises this uniformity in all their outward shapes but from the counsel and contrivance of an author?"

Two centuries later, with the advent of the Romantic interest in Nature, Goethe would prefer the more Aristotelian position that "The absence of symmetry seems to be evidence of the progress of evolution" (see Riese and Goldstein, 1950). The association of symmetry with theology was later to be reflected in Pauli's comment that:

> "I do <u>not</u> believe that the Lord is a weak left-hander ..."

Half a century after Goethe and only eleven years after the publication of Darwin's <u>Origin of Species</u>, Pye-Smith published the first truly evolutionary theory of handedness[1]:

> "If a hundred of our ambidextrous ancestors made the step in civilisation of inventing a shield, we may suppose that half would carry it on the right

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*[1] Often attributed to Thomas Carlyle (eg. Fritsch, 1968 who says that he discusses it in his unpublished diaries of 1871).

> arm and fight with the left, the other half on the left and fight with the right. The latter would certainly, in the long run, escape mortal wounds better than the former, and thus a race of men who fought with the right hand would gradually be developed by a process of natural selection" (Pye-Smith, 1870).

In this thesis I shall develop several of Pye-Smith's ideas, notably his emphasis upon the base-line for asymmetry being a fluctuating asymmetry, and the concept that there must be advantages to being right- or left-handed. And overall I will come down heavily on the side of Aristotle, although the symmetry of the Platonic view, and its modern variants in the expositions by Kant and Mach, will help me to arrive at that position.

CHAPTER 2: THE STRUCTURE OF HANDEDNESS

> "To the scientific mind there is no inner difference, no polarity between right and left as there is for instance in the contrast between male and female, or between the front and rear of an animal"
>
> Hermann Weyl (1952)

Summary

The structure of handedness questionnaires is shown to be compatible with a bimodal distribution of handedness. Handedness category correlates with many other items, but degree of handedness within a handedness category does not relate to other variables, and indeed may be artefactual. The existence of a third category of 'Mixed' handedness seems highly dubious and it is suggested that the use of this classification should be discounted.

2:1 Introduction

Common sense suggests that there are two types of handedness i.e. right and left: that is, two qualitatively different categories (see Figure 2.1a). Some might argue that there is a third category of persons, the ambidextrous, who are qualitatively different from both right and left handers (Figure 2.1b). And it is in principle possible to argue that there is a whole range of qualitatively different categories of handedness (Figure 2.1c). A different approach might be to suggest that there is actually a continuum between complete right-handedness and complete left-handedness, and that division of this continuum is purely arbitrary, reflecting only quantitative differences; if so, the particular distribution chosen is clearly of crucial importance, and if it is unimodal we could imagine it being arbitrarily divided into two (Figure 2.1d), three (Figure 2.1e) or more (Figure 2.1f) categories. Alternatively this unimodal normal distribution might be anchored by a significant point, such as zero, so that left handers have R-L > 0, and right-handers have R-L < 0 (Figure 2.1g). Clearly such a continuum could be yet further divided. An important compromise between the categorical structures (Figure 2.1a-c) and the continuous structures (Figures 2.1d-g) would be a bimodal or multimodal distribution, such as Figures 2.1h and 2.1i, in which, although the distributions are continuous, there are clearly 'natural boundaries' between the portions

of the distribution.

This chapter will concern itself with which of the above types of model can best accommodate the various details of handedness, particularly as assessed by questionnaire. To consider such a question is not as sterile or pedantic as it might at first seem. No reasonable genetic model may be constructed without knowing the structure of the phenotypes. Neither is it otherwise possible to answer questions of the form: Are right-handers more right-handed than left-handers are left handed?

Throughout the Chapter I will use a single main principle to decide between models; the best model is that which most simply describes the available data. Occam's razor thus gives an a priori precedence to a model with two categories rather than three. And hence also, data will not be arbitrarily sub-divided unless it is possible to demonstrate significant differences between sub-groups.

The possibility that handedness might be more than just two simple categories was first posited at the end of the last century (although of course there is a classical literature on rare cases of ambidexterity). In the 1860's Broca demonstrated that the speech centre was usually in the left cerebral hemisphere. There were however exceptions to this general rule; Dax's law, that the phenomenon was mirror-imaged in left-handers, accounted for some of the

cases, but there were still an uncomfortable number of anomalies. Various explanatory concepts were introduced to help with these difficult cases. It became acceptable to discuss handedness for different tasks as if there were a set of handednesses, each logically independent of the other; and thus it was usually possible to find at least one 'handedness' which was contralateral to the aphasic hemisphere. Similarly it became accepted that there were different <u>degrees</u> of handedness, which could be affected by heredity; thus a right-handed man with left-handed ancestors was felt to be less right-handed than a pure right-hander (i.e. he had left 'stock-brainedness', to use Foster Kennedy's (1916) expression). Similarly it was felt that early brain damage might well either shift dominance from one side to the other ('pathological left-handedness'), or alternatively might shift the degree of dominance without changing its direction. Woo and Pearson (1927) seriously complicated matters, by confusing handedness with asymmetry of grip strength, and eye-dominance, with visual acuity asymmetry, and hence concluding, on the basis of a very sophisticated statistical analysis, that "lateralism, whether ocular or manual, is a continuous variate, and dextrality and sinistrality are not opposed alternatives, but quantities capable of taking values of continuous intensity, and varying one into the other". This may well be true for grip strength asymmetry and visual acuity asymmetry, but it is not necessarily true for handedness and eye dominance.

Early studies of handedness *per se* were those of Jasper (1932), Durost (1934), Hull (1936), and Humphrey (1950). These studies made few theoretical statements, and are thus of less importance for the present purpose than later studies (see Hildreth 1949 for a description of early work). Later studies, in the sixties and seventies were rather more influential, two results being discernable. First, it became a common-place to assume that handedness must be more complex than being simply two categories (e.g. " ... the fact that handedness is not simply a two-valued variable, but must be measured along a continuum" (Shankweiler and Studdert-Kennedy 1975); or, " ... the complexities involved in the classification of handedness" (Satz *et al*, 1967)). Secondly, it also became common for large scale surveys to describe not just right and left-handedness, but right, *mixed* and left-handedness (e.g. Sand and Taylor, 1973; Thompson and Marsch, 1976; Boucher, 1977; Flemminger *et al*, 1977b).

The present work analyses two large scale questionnaires in some detail, in an attempt to provide empirical solutions to the problem of the structure of handedness.

2:2 Method

Survey 1

This questionnaire was distributed in May 1977, and October 1977 to Cambridge undergraduates (mostly in the

sciences, hence the male predominance). Thq questionnaires on these two occasions (Survey 1a and 1b) had minor differences in their supplementary questions. Questionnaires were usually distributed at the beginning of a lecture and collected at the end of that lecture, and students were also reminded after the following three lectures to return the questionnaire. A number of questionnaires were also distributed to undergraduates by a postal survey. Response rates were difficult to assess accurately but were of the order of 50%; 948 questionnaires were returned satisfactorily. It is probable that there was a preferential response of left-handers, but this is of no consequence for the purposes of the present study.

Survey 2

This was carried out in June 1977 and consisted of a six page questionnaire distributed to all 1977 graduates of the University of Cambridge. Questionnaires were delivered to individual pigeon-holes on the evening before the graduation ceremony, and asked for the assistance not only of the student but also of his or her parents in completing the questionnaire. Graduation represented the most convenient time at which a large number of students were in the presence of their parents. The questionnaire asked the students, and also the parents, to complete the 12-item handedness questionnaire of Briggs and Nebes (1975). Further questions were also asked about the handedness of other members of

the family, and it was thus possible to get reasonably reliable information on the student's aunts, uncles, grand-parents, great-grand-parents and siblings. Once again the response rate was difficult to determine accurately but was of the order of 15-20%. The same questionnaire was also distributed in September 1977 to all freshmen medical students of the University of Birmingham, in the week before their first arrival at medical school. For this questionnaire, a response rate of about 80% was achieved. For the two studies combined 511 questionnaires were completed.

All data from questionnaires were punched on to 80-column punched cards, and analysed by means of the Statistical Package for the Social Sciences (Nie et al, 1975). This statistical package was used also for the multivariate analyses.

In order to simplify the analysis, subjects were asked, in both surveys, if they had ever been forced to change their writing hand. Any subject with a history of forcing (irrespective of whether it was successful) was removed from the analysis. This removed 23 (2.42%) students from Survey 1 and 10 (1.95%) students from Survey 2.

2.3 Results

The questionnaire for Survey 1 was constructed so that it had 28 separate questions on handedness for particular tasks (see Appendix A2:1). By combining some or

all of these questions it was possible to replicate other handedness questionnaires in the literature. Thus by using only questions 1 to 12, the handedness scale of Briggs and Nebes (1975) was simulated. Details of the questions themselves, and their inter-combination for forming the other handedness scales, are given in Appendix A2:1.

Annett (1967) used only eight questions, each of which could be answered only with the categories Right, Either or Left. By combining the Usually and Always categories of the present questionnaire it was possible to simulate the Annett handedness scale (AHS). Figure 2.2 shows the number of times that a subject answered that the right-hand was used for a particular task. The J-shaped distribution is similar to that of Annett (1972). The groups are also divided according to Annett's categorisation into Right, Mixed and Left handers. A right-hander is defined as someone who uses the right-hand for all eight tasks; by symmetry, a left-hander is a person who uses the left-hand for all eight tasks, and the remainder of the subjects are classified as Mixed. The proportions of Right, Mixed and Left (0.6936, 0.2664 and 0.0399 respectively) are similar to binomial proportions, although the difference is just significant statistically ($X^2 = 4.46$, 1 df, $p < 0.05$).

Figure 2.3 shows data from the same subjects as Figure 2.2, but the subjects are now classified as if on the Oldfield handedness scale (OHS) (Oldfield, 1971). This scale uses 10

questions, each on a five-point scale (Always Right, Usually Right, Either, Usually Left, Always Left), and the results are calculated simply by adding together all of the individual items (5 for Always Right, 4 for Usually Right, through to 1 for Always Left), and then applying a standardisation such that the maximum possible right-handed score was +100, and the maximum possible left-handed score was -100. Oldfield presented his results divided up into deciles, and this has been done for the present study, both for the OHS, and for all subsequent scales.

Comparison of Figures 2.2 and 2.3 shows little significant difference between the distributions.

Figure 2.4 shows the distribution of the subjects on the Briggs and Nebes (1975) handedness scale (BNHS), in which 12 questions were asked, each of which had five possible answers. The distribution is similar to that of the OHS, but now there is a tendency for extreme categories to be used less (note the change in the ordinate scale); nevertheless the maximima are still at the extremes.

Figure 2.5 shows the results using the Crovitz and Zener (1962) handedness scale (CZHS), which has 14 questions, also on a five-point scale. Notice that now the distribution is changing; the maxima are no longer at the extremes, either for right or for left-handers.

Figure 2.6 shows the scores for the same subjects on a scale composed of all 28 items of the original questionnaire (HS1). Now it is clear that the maxima are well removed from the extremes of the distribution, and indeed the curve appears similar to that of a bimodal normal distribution. The distribution is however asymmetric about the zero line. The dotted lines of Figure 2.6 show the expected distribution if the left-handed categories (i.e. less than zero) were present in the same relative proportions as the right-handed categories; there is a surfeit of weakly left-handed subjects over strongly left-handed subjects. This bears upon the old question of whether left-handers are less extremely lateralised than right-handers, and whether there is a greater variability in their distribution (i.e. taking each half of Figure 2.6 separately, is the absolute mean of the left-handers different from that of the right-handers, and is the variance of the left-handers different from that of the right-handers?).

Before answering this question it must be realised that there are certain possibilities that would render any answer to questions such as this, quite meaningless. Right-handers and left-handers both live in a right-handed world, and thus inevitably left-handers have to confirm in their daily activities by actually using the right hand for tasks for which, all other things being equal, the left hand would be preferable. Cultural pressures alone force most left-handers to use their right-hand for holding

a knife at the dinner table; and mechanical constraints produce a tendency for left-handers to use their right-hand for using a screw-driver, or for winding the right-hand thread of a clock (supination being far stronger than pronation of the forearm). To include questions about such activities within the questionnaire is to prejudice any answer to the question as to whether the degree and variance of right-handers is different to that of left-handers. If we were to ask a person to put his right hand in the air, and found that both right and left handers did so, we would not conclude that left-handers were less well lateralised than right-handers. Similarly with handedness questionnaires; the cultural biasses must be removed as far as possible.

Figure 2.7 shows, for each of the individual questions, the mean score of both right and of left-handers, 5 being given for a reply of 'Always Right', through to a 1 for a reply of 'Always Left'. Handedness in this figure is defined in terms of the response to question 1, the hand used for writing (subjects with a history of changing writing hands being excluded). Tasks for which there is no cultural bias should, if lateralisation is equal, lie along the descending diagonal (that is left-handers and right-handers should be mirror-image symmetrical for the task). It may be readily seen that question 17 (holding a knife at table), is grossly non-symmetric, and that questions 3, 7, 22, 23, 25 and 26 all have a strong cultural asymmetry. Of the remaining 20 questions, 11 have a weak degree of cultural

bias which is statistically significant, and for one of the questions (number 4), the difference is significant in the opposite direction to the majority.

It is now possible to construct a scale using just the less biassed questions and ask whether there is still a greater variance amongst left-handers. The fact that we have chosen individual questions for which there is no asymmetry does not mean that the combination of these questions will necessarily be symmetrical; the absolute means will necessarily be symmetric, but the question as to the similarity of the variance needs to be examined empirically.

Figure 2.8 shows a modified scale (HS2) constructed by adding together the 21 questions without a strong cultural bias (see Appendix A2:1 for a list of the questions used). The distribution is far more symmetric than the distribution of Figure 2.6, as may be seen by the dotted lines in the left-handed half of the distribution which show the right-handed distribution reflected in proportion to the left-handers total occurrence[1].

Table 2.1 expresses the results more formally, showing the absolute means for right and left-handers, and the

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1. Note also the remarkable similarity of this figure to that of Figure 1 of Goodglass and Quadfasel (1954).

variances, and variance ratio for right and left-handers (i.e. the F-statistic), as well as the kurtosis and skewness. As the number of questions in a questionnaire increases the variance ratio of the distributions decreases until for HS2 it is only 1.36 (which is however significantly different from unity, $p = 0.0108$ with 114 and 971 dfs). If one constructs a scale of just the nine questions which show no statistical difference in mean value between right and left-handers (HS3) then the resultant absolute mean for right-handers is 62.49, and 62.29 for left-handers, the difference not being significant. For HS3 the variance ratio is 1.258, which is only just significant with $P = 0.05$. Due to the smaller number of questions in HS3 there is a slightly greater skewness than in the larger HS2.

Scrutiny of Table 2.1 shows that almost all the distributions, for both right and left-handers, show significant degrees of skewness and/or kurtosis. These are partly due to ceiling effects, extreme degrees of right or left-handedness not being differentiated within the relatively small numbers of questions asked. Skewness and kurtosis might perhaps also reflect a tendency for a minority of subjects not to read and to consider questions carefully, but instead to simply tick the extreme categories for all questions.

In view of the presence of these significant non-normalities, the validity of parametric tests is somewhat

suspect, skewness in particular tending to produce an artefactually high proportion of significant results (false positives) (Snedecor and Cochran, 1967, p 325). In consequence Table 2.1 also shows the significance values of the non-parametric Kolmogorov-Smirnov two-sample test for differences between two distributions (the left-handers' data being mirror-reflected about the abscissa's zero axis before carrying out the test). There are now no significant differences between left and right-handers on the HS3 scale, or even on the HS2 scale.

Table 2.1 also shows the percentage of the questions, for each of the scales, which were counted as 'strongly culturally biassed' and as 'non-culturally biassed' (see Figure 2.7). As the proportion of culturally biassed questions falls, so does the difference between the absolute means of left and right-handers.

To summarise so far, handedness may be regarded as either a J-shaped distribution, or as a bimodal normal distribution, according to the choice of questions asked. Similarly the distribution of responses of left handers and right handers are different or the same according to the particular question asked. On the basis of handedness questionnaires it is therefore not possible to say that left-handers are more variable in their handedness than right-handers; the answer obtained depends upon the questions asked. Parsimony therefore demands that in the

first instance we reject the possibility of such a difference.

For convenience of interpretation of work which has used Annett's three categories of right-mixed and left handers, Figure 2.9 shows the scores of subjects on the HS2 divided according to their Annett category. Left and right-handers on the Annett criteria are certainly of their stated handedness. Mixed handers are more problematic; instead of being truly mixed, they are rather a mixture of right and left-handers, the distribution reflecting the bi-modality of Figure 2.8.

So far distributions have been divided into left and right-handers according to whether the score on a scale is greater than or less than zero. This step must be justified; also, a far simpler criterion will be used, if possible.

For each of the 28 questions the number of usages of 'either' responses was found. For question 1 (hand used for writing) nobody answered 'Either', and for question 13 (hand used for drawing a picture) only 2 (0.21%) persons answered 'Either'. For all other questions there were far higher proportions of 'Either' responses. Since writing hand is the most lateralised of the questions, it is the most profitable to examine it as a single criterion for dividing the distribution of Figure 2.8 into two parts.

The strength of lateralisation is also shown in the fact that only 13 subjects (1.37%) answered 'Usually Right', and 5 (0.52%) answered 'Usually Left', the remaining 98.10% answering in the 'Always' category.

For HS1 (Figure 2.6) only two right hand writers (0.25%) had scores of less than zero, and 18 (13.36%) left-hand writers had scores of greater then zero; an overall inconsistency of classification of 2.16%. For HS2 (Figure 2.8) we find that 1 right hand writer (0.12%) and 10 left-hand writers (7.57%) were mis-categorised, an overall error rate of 1.18%. If the criterion is made +10 instead of zero, then 3 right-handers (0.41%) and 7 left hand writers (5.3%) are mis-classified, an overall error-rate of 1.08%. Whilst a criterion of 10 is slightly better at dividing the distribution, a criterion of zero has been retained for convenience (the criterion of 10 is presumably slightly better due to its taking account of the small cultural bias remaining in the HS2 scale.)

For the rest of this ~~paper~~ chapter a right-hander is defined as a person who both writes with his right hand and has a score of greater than zero on the HS2, and a left-hander is defined as both writing with his left-hand and having a score of less than zero on HS2. 10 subjects have therefore been excluded from the analysis. Scrutiny of the individual results of these 10 subjects revealed nothing unusual except perhaps a greater tendency than usual to use the

'either' column.

Since all persons who had been forced to change their writing hand had been eliminated from the analysis at an earlier stage, we may conclude that in the first instance, and in the absence of evidence of forcing to change hands, a person's handedness is best assessed by means of the hand uses for writing. This agrees with the conclusion reached by Annett (1970).

In the rest of the present study all degrees of handedness will be measured in terms of the 21-item scale (HS2).

Having discussed the overall distribution of HS2 we must look at the details of its structure. A normal distribution is necessarily produced if a character is influenced by a large number of statistically independent events. If therefore many of the items used for constructing the handedness scale were correlated then a normal distribution would result. Hand-clasping and arm-folding are good examples of items which might be thought to correlate with handedness, and hence have been included in handedness inventories (e.g. Berman, 1971) but neither hand-clasping nor arm-folding actually correlates with handedness at all (see Chapter 9).

The structure of a scale may be best assessed by intercorrelating all of the individual test-scores, and then factor-

analysing the resultant inter-correlation matrix. When this is done for the entire data of HS2 all of the items positively inter-correlate with each other, and the resultant factor analysis produces a large first factor accounting for 55.2% of the total variance (Principal Axis Analysis, Nie et al, 1975). Other factors are not significant (see Figure 2.10 for a plot of eigen-values against factor number). This result is similar to that of White and Ashton (1976), and of Bryden (1977). However it is inevitable that this particular form of factor analysis will produce such a result. The problem is that, in the same way as the overall distribution of scores is bimodal (Figure 2.8), so the distribution of the scores on each individual test is also bimodal. The correlation coefficient of such scores will thus only reflect the large variance due to the distance between the two peaks, and will hardly be influenced at all by the smaller variance within each handedness peak; hence a large common factor is almost inevitable. The question of far greater importance concerns the factor structure when one considers only one half of the distribution of Figure 2.8, for then it is not necessarily true that there will be a common factor.

I have therefore factor-analysed all of the items of the HS2 distribution, separately for each handedness group. The question on hand-writing was excluded from this analysis because almost all of the subjects answered in the

same category. For the right-handers all of the 190 possible inter-correlations between items were positive; for the left-handers, with a rather smaller sample size, 166 (7.3%) of the 190 inter-correlations were positive. Figure 2.10 shows that, in each case, the first factor is highly significant, accounting for 24.5% of the total variance in the right-handers, and 22.4% of the total variance in the left-handers. Using a 'scree-slope criterion' (Child, 1970) only the first factor is significant for the right-handers, and only the first two factors are significant for the left-handers. The first factor in each case loaded strongly on each of the component tests. The second factor in left-handers seemed to relate to those questions which were reversed (i.e. the correct answer for a pure left-hander would be the <u>right</u> hand), and is thus similar to the second factors found by White and Ashton (1976), and Bryden (1977).

The factor analyses are therefore justification for thinking not only that there is an overall dimension called handedness, but that within each handedness sub-group there is also a dimension called degree of handedness, and that, on the basis of evidence from questionnaires, there is only <u>one</u> dimension within each handedness category.

It might seem inefficient , in view of the uni-dimensionality of each handedness group, to use a 21-item

questionnaire in order to quantify degrees of handedness. Appendix A2:2 describes an attempt to produce a shorter, more efficient scale which retains most of the variance of the larger questionnaire.

So far in the analysis of the 28-item handedness questionnaire, I have only considered the differences between total scores of individuals, or else differences between individual items across all subjects. It is however possible that within subject variance is also of importance. Thus it would be possible to produce a score of exactly zero on, say, HS1, by ticking 'Either' for all of the questions, or alternatively by ticking 'Always Right' and 'Always Left' for alternate questions. Each strategy results in the same total score, but has very different implications. It is possible that left-handers may produce similar scores to right-handers overall, but that this may be a result of a greater variability within their responding. This may be assessed by calculating, for each individual's responses, the mean response, and the standard deviation of that subject's responses. Table 2.2 shows, for HS1, HS2 and HS3 the mean and standard deviation of the individual standard deviations. For HS3, the most interesting test of the hypothesis, left-handers show no more variability in their responding to the questions than do right-handers. This may be seen graphically in Figure 2.11.

Our justifications for regarding the major groupings

of left and right handers as separate categories, are several fold. The first thing is that individuals are consistent. If a laterality is not statistically consistent within individuals it is of little interest; one example of an inconsistent asymmetry is wing-folding in Drosphila melanogaster (see Appendix A9:1). We know from simple observation that gross handedness category is highly consistent within individuals. A second reason for regarding handedness categories as different is that they behave differently. Thus the incidence of a family history of sinistrality is different in the left-handers and the right-handers (77.5% versus 35.8% in the present sample, $X^2 = 73.3$, 1 df, $p < 0.001$). Similarly, cerebral dominance for speech is different in the two handedness categories (e.g. Zangwill, 1960).

The question thus arises as to whether the differences in the degree of handedness within each handedness category are also meaningful. Without directly testing the possibility we may presume, from the work of Raczkowski et al (1974), that since all of their questions showed at least a 74% test-retest agreement, and that half of them showed a more than 93% agreement, that the differencesin degrees of handedness are also consistent within individuals. This possibility is also being tested directly at the present.

The next question is whether degree of handedness

correlates with any other behaviours. The most important aspect of this question concerns genetic correlates. If degrees of handedness is related to genetic factors then it must be taken into account by any adequate genetic model. Hecaen and Sauguet (1971) have suggested that left-handers without a family history of sinistrality are more strongly left-handed than those with a family history. Conversely it has been suggested, e.g. Briggs et al (1976) that both left and right handers might be sub-divided into two separate groups, weak handers being the result of early brain damage, whilst strong handers are genetically determined.

Consider the question of family sinistrality. In survey 1 subjects were asked to give the handedness of their parents, grand-parents and siblings; they were also cautioned not to guess, but only to answer if they were certain. The mean number of relatives reported by left and right-handers did not differ significantly (right-handers, mean = 4.93; left-handers, mean = 4.84, $F(1,885) = 0.0208$, NS); neither did number of relatives reported relate to degree of handedness within either of the handedness categories. (right-handers, $r = 0.028$, NS; left-handers, $r = 0.018$, NS). A positive family history of sinistrality was defined as any relative being described as either left-handed or ambidextrous. As mentioned earlier there was a highly significant difference between left and right-handed categories. Figure 2.12 shows the percentage of individuals

within portions of each handedness category, with a positive family history. Within left-handers there is no difference between weak and strong left-handers, and within right-handers there is no difference between weak and strong right-handers (Left-handers, $X^2 = 3.88$, 3 df, NS; right-handers, $X^2 = 7.43$, 7 df, NS). To express this same result in a different way, the mean degree of handedness within a handedness category is not related to a family history of sinistrality (Table 2.2); this result also shows the lack of significance of the apparent trend in Figure 2.11). This is evidence against the degree of sinistrality or dextrality being genetically determined.

The degree of propositus handedness as a function of maternal and paternal handedness is also shown in Table 2.3. In right-handers there is a significant effect of maternal handedness, and an almost significant effect for paternal handedness. However given that these significance levels are small, and that a large number of tests have been carried out in Table 2.3, they are probably not truly significant; combined with the fact that the results are in opposite directions to one another, and that the trends in the left-handers data are in the opposite direction, this probably means that the results may be disregarded.

A more direct test of the inheritance of degree of handedness is possible using the data of Survey 2, in which there are measures of degree of handedness in parents and

their children. Figure 2.13 shows the mean and median degree of right-handedness of the right-handed children of two right-handed parents, parental handedness being expressed as mid-parental handedness. There is no evidence of a correlation ($F(7,300) = 0.69$, NS). This same result may be expressed in a different fashion by saying that in the same cases the multiple correlation of student handedness upon maternal and paternal handedness is not significant. Table 2.4 shows, for each handedness combination of propositus and parents, the multiple correlations of children upon parents; none are significant. The simple correlation between each parent and child is also shown; none are significant. Neither is there evidence of assortative mating, inter-parental correlations being non-significant. There were insufficient families with two left-handed parents to allow analysis.

In summary it would appear that there is no evidence for <u>degree</u> of handedness being inherited, only <u>direction</u> of handedness.

Cerebral trauma has been proposed to account for differences in direction and degree of handedness. Effects upon direction will be discussed later. Table 2.2 shows that persons who report a history of either head trauma or of neurological disease do not show differences in degree of handedness from those without such a history.

Bakan (1971) has proposed that acute cerebral anoxia of the infant during childbirth results in left-handedness, and hence, since birth complications are related to birth order, left-handedness should be more common in the first-born, and also in the fourth-born and later than in second and third-born children. Table 2.3 shows that degree of handedness does not relate to birth-rank, and that those reporting a history of birth complications or difficulties do not have a lesser degree of lateralisation.

In combination with the genetic data these results provide no evidence for the suggestion that weak and strong handers have different aetiologies.

Table 2.3 also shows several other factors which have been considered to be related to degree of handedness. There is no significant difference between the sexes; nor is degree of handedness related to hand-clasping type. A history of reading or writing difficulties is not related to degree of lateralisation, although it must be remembered that the parents sample is of well above average intelligence. Hair-parting side correlates with the direction of whorl of the head hair, another lateral asymmetry which has on occasion been claimed to relate to handedness; Table 2.3 shows there to be no relation with degree of handedness. It may also be added, without providing detailed evidence, that degree of handedness does not relate to ABO or Rhesus blood groups, eye-colour,

or colour-blindness.

Levy and Reid (1976, 1978) claimed that there were significant differences within handedness groups according to the posture of the hand during writing. A number of each handedness group write with the pen pointing towards the writer, rather than the more common way with the nib of the pen pointing away from the writer. An attempt was made to measure the posture of the hand during writing, by asking the questionnaire respondents to tick one of four figures, to indicate which best illustrated their own hand-writing position. These figures were taken from Levy and Reid (1976) as illustrating "typical writing postures". This question was not included in Survey 1a.

On the basis of tachistoscopic evidence, Levy and Reid said that right handers with a normal writing position, and left-handers with an inverted (or 'hooked') writing position were left hemisphere dominant for language, and that the remaining groups were right hemisphere dominant for language. Table 2.5 shows the proportions of the inverted and normal types in right and left handers in Survey 1b and 2. The inverted position is apparently more common in right-handers than Levy and Reid would suggest, at about 12%. The 'inverted' position in left-handers is far less common than would be expected if indeed it related to left hemisphere language dominance, for we now believe that the majority of left-handers have language

in the left hemisphere. From the present data it would seem far more likely that the inverted writing position corresponds to right-hemisphere language, and indeed the relative proportions, about 13% and 30%, (which are similar to those of Lawson (1978)) bear ~~bare~~ a remarkable similarity to the estimated proportions of right cerebral dominance obtained from dichotic listening tests (e.g. Lishman and McMeeken, 1977), unilateral ECT studies (e.g. Geffen et al, 1978), and clinical studies (Annett, 1975). Table 2.3 shows that within handedness groups the degree of handedness is unrelated to writing posture.

For the above data on hand writing position it must be emphasised that the data have not been verified by direct observation, and hence the results must be regarded as only tentative.

2:4 Discussion and Performance Asymmetries

Degree of handedness, as assessed by questionnaire, is an internally consistent concept. However it cannot be shown to relate to any genetic factors, nor to be related to a number of other independent variables. The question therefore arises as to whether it is perhaps just an artefact of the method of measurement. One possibility is that the extremity of responses used by a subject is less a function of his actual hand behaviour but more a result of personality differences between subjects. This

possibility would be supported by the finding of Hicks and Pellegrini (1978) that right and left handers showed no difference on Rotter's 'locus of control' scale, but that 'mixed' handers showed a significant difference from either right or left handers. The possibility of a personality difference is being directly tested at the present. The finding of a curvilinear relation between degree of handedness and a perceptual task (Thomas and Campos, 1978) may perhaps also be explained in such terms.

Our initial question, of the structure of handedness, therefore receives two possible answers. Handedness may have a bimodal normal distribution, with an anchoring point at the anti-mode of zero between the two halves of the distribution; alternatively the degree of handedness within each half of the distribution may yet be shown to be artefactual, in which case handedness may most economically be described as being two categories, left and right, the single best criterion for distinguishing them being the writing hand (assuming that the person has not been forced to change writing hand). The latter position would be consistent with that suggested by Colbourn (1978).

In either case, bimodal or bicategorial distribution, responses to handedness questionnaires must be reconciled with the unimodal distribution found in certain motor tasks such as finger-tapping. The discrepancy cannot be explained away by saying that handedness questionnaire items

are unreliable, since they seem to correlate moderately well with observation of the actual behaviours (Raczkowski et al, 1974; Coren and Porac, 1978). Annett (1970; 1972) has pointed out that for many tasks, e.g. peg-moving and grip-strength (Woo and Pearson, 1927; Woo, 1928), the difference between right and left hand proficiency is distributed normally, with a mean displaced towards the right-hand side (i.e. the majority are better with the right hand). On the basis of this observation Annett claims that the underlying structure of handedness is a continuous unimodal normal distribution, and that hand performance categories are derived from it in an almost arbitrary fashion, the criterion varying, and differences in criteria accounting for differences in handedness incidence found in different studies (Annett, 1975). In later studies Annett (1978) suggests that the distribution of manual skill asymmetry also shows a significant degree of negative skewness, although she presents no evidence for this, and indeed, the skewness is notably absent in the data of Woo and Pearson (1927).

It is tempting to assume, ~~that~~ since tasks such as grip-strength, peg-moving and finger-tapping are objective, whilst questionnaires are subjective (and perhaps are ultimately only measuring some form of preference), that the results of the motor test must be superior. This however need not be the case. Provins and Cunliffe (1972) measured test-retest correlations over a period of 3 days, for seven

different unimanual skilled tasks and for only two of the tasks, one of which was writing ability, was the right-left difference significantly correlated between the two occasions. Annett et al (1974) found that right-left differences on a peg-moving task showed a statistically significant test-retest correlation over a period of 7 weeks to 18 months; nevertheless the coefficient of determination showed that only 47% of the variance was retained between the two occasions. Within a group of right-handers, Shankweiler and Studdert-Kennedy (1975) found that only one of five unimanual tasks showed a significant test-retest correlation over a period of a week or two. In contrast to such results, handedness category is almost certainly highly consistent between occasions, and one might expect almost 100% of the variance to be retained. Thus in data derived from the National Child Development Study (see Chapter 3:2 for further details) 99.6% of 9153 children described by their mothers as right-handed at age 7, wrote with their right hand at age 11, and 94.4% of 1119 children described by their mothers as left handed at age 7 wrote with their left hand at age 11; the tetrachoric correlation coefficient is 0.947, indicating that 89.8% of the variance has been retained over a 4 year period, despite cultural and other pressures, and the possibility of developmental changes in degree of laterality. In adults the correlation would probably be even higher.

For peg-moving and finger-tapping there is at least

some evidence that the tasks are correlated, albeit not very well, with handedness category. But for many tasks for which one might expect a correlation with handedness, there is simply no correlation. Arm-folding and hand-clasping have already been mentioned as examples of this. Kimura and Vanderwolfe (1970) found that accuracy of fine movements of the fingers was unrelated to handedness, and was actually superior in the left hand of both left and right-handers. Wolff et al (1977) found that rhythmic tapping on a key (without a metronome as a guide) was better in the right hand in both right and left-handed subjects, there being no difference between the two handedness categories. Similarly, hand differences need not be in favour of the right hand; Ingram (1975) found that two out of five manual tasks in young children showed a significant overall left hand superiority.

Far from being typical or necessary, peg-moving and finger-tapping would seem unusual in correlating with handedness. Since even in these cases the correlation with handedness is poor, one may speculate as to the underlying handedness structure. If indeed the real structure of handedness is a bimodal normal distribution then if a test were perfectly correlated with handedness both in degree and direction then we would expect that test to have a bimodal normal distribution. As, however, the correlation between handedness and a motor test became less, so we would expect that, assuming the variance of each

half of the distribution stayed similar, ~~then~~ the means of the two distributions would come closer together. The increased height of the right-handed distribution would mean that when the tests reached a certain low degree of correlation that the mode of the left-hand distribution would be lost in the left-hand tail of the right-hand distribution. The resulting distribution would thus appear to approximate that of a unimodal normal distribution, with its mean shifted to the right. It would however be significantly skewed, in the manner pointed out for skull asymmetry by Annett (1975).

This process may be seen occurring in two sets of data from the National Child Development Study. Figure 2.14 shows the overall population distribution of over 12,000 11-year old children on a task in which they were asked to mark as many squares as possible on a piece of graph paper, using either the right or the left hand. The distribution just shows a bimodality. Figure 2.15 shows the same data broken down by handedness (as defined in Chapter 3.3). It is now clear that each hump on the curve is a result of one handedness category. As the symmetry of a task becomes greater (and the learned skill becomes relatively less) so the two modes merge into one. The same children in the NCDS were also asked to pick up 20 match-sticks as quickly as possible, one hand at a time. Figure 2.16 shows the distribution for this task, broken down by hand. Although statistically different

these distributions are clearly very similar in absolute terms, and it must be clear that the overall population distribution is unimodal. (The clear kurtosis is probably a function of number preferences, and of recording the times in integer numbers of seconds)

The overall argument is of course similar if handedness is regarded as bicategorical rather than bimodal.

2:5 Conclusions

The structure of handedness questionnaires implies a bimodal normal distribution within an anchoring point at zero. Whilst subjects appear to show internal consistency in their degree of handedness within either handedness category, degree of handedness has not been shown to correlate in any useful way with any of a number of other variables. Handedness might therefore actually be bi-categorical, the variations in degree being artefactually a function of personality variables. For genetic purposes handedness may almost certainly be regarded as bi-categorical, left and right, the single best criterion for distinguishing the two categories being the writing hand. There is no evidence that left-handers are more variable or less extreme in their handedness than are right-handers.

APPENDIX A2:1 THE HANDEDNESS QUESTIONS USED IN SURVEY 1.

Which hand do you use for the following tasks:-

1. To write a letter legibly.
2. To throw a ball to hit a target
3. To play a game requiring the use of a racket
4. At the top of a broom to sweep dust from the floor
5. At the top of a shovel to move sand
6. To hold a match when striking it
7. To hold scissors when cutting paper
8. To hold thread to guide through the eye of a needle
*9. To hold the pack whilst dealing cards.
*10. To hold the nail whilst hammering it into wood
11. To hold a toothbrush whilst cleaning one's teeth
*12. To hold the jar whilst unscrewing the lid
13. To draw a picture
*14. To hold potatoes whilst peeling
15. To hold a jug when pouring water from it
16. To hold a knife when cutting a slice of bread
17. To hold a knife when also eating with a fork
18. To hold a glass for drinking
*19. To hold a dish whilst drying it
20. To hold a comb to combone's hair
*21. To hold a deck of cards whilst sorting them
22. To hold the body of a penknife whilst opening the blade
23. To hold a clock whilst winding it
24. To reach for a book on a high shelf

25. To strum the strings of a guitar

26. To pull the trigger of a rifle

27. To hold a scrubbing brush to the floor

28. To rub writing off a blackboard with an eraser

*Indicates questions for which the correct answer for a pure right hander would be the left, hand: the data for all such questions has been reversed before any further analysis has taken place.

The above questions have been used to simulate other handedness scales (see Chapter 2:3). (Note that substitutions are not always exact, particularly in the details of the wording).

APPENDIX A2:1

Scale	Reference	Questions used
AHS	Annett (1967)	1,2,3,4,5,6,7,8.
OHS	Oldfield (1971)	1,2,4,6,7,10*,11,13,16 x 2**
BHNS	Briggs and Nebes (1975)	1,2,3,4,5,6,7,8,9,10,11,12.
CZHS	Crovitz and Zener (1962)	1,2,3,7,8,10,11,12,13,14,15, 16,18,19.
HS1	This study	1,2,3,4,5,6,7,8,9,10,11,12, 13,14,15,16,17,18,19,20,21, 22,23,24,25,26,27,28.
HS2	This study	1,2,4,5,6,8,9,10,11,12,13, 14,15,16,18,19,20,21,24, 27,28.
HS3	This study	1,5,8,9,12,13,19,21,27,28.
HS4	This study	1,5,8,9,12,21.

Notes

*Amongst my own set of questions there was no adequate substitute for Oldfield's question 10, and therefore question 10 of my own study has been arbitrarily substituted.

**Question 16 has been used twice in constructing the OHS, once as a substitute for question 6, and once as a substitute for question 7 of Oldfield's study.

APPENDIX A2:2 THE CONSTRUCTION OF AN OPTIMAL HANDEDNESS QUESTIONNAIRE

A questionnaire with 28 items is cumbersome and tedious for subjects to complete; particularly in lower intelligence subjects it is probable that the magnitude of the task prevents each question being considered as deeply as it requires. A shorter questionnaire is therefore desirable.

Ideally a shorter questionnaire should retain as much of the variance of a larger questionnaire as is possible. The ideal that has been taken is the 21 item handedness scale, HS2. The variance to be retained is that within each handedness category, and the two halves of the distribution must therefore be assessed separately. Table A2:1 shows the rank ordering of each of the 20 constituent items (excluding hand-writing) in a hierarchial multiple regression of the individual questions upon HS2, the individual question with the highest partial correlation being entered at each step. The two handedness categories are shown separately. Table A2:1 also shows the proportion of accounted variance (i.e. the square of the multiple correlation coefficient) after the introduction of each new question. Thus, for right-handers question 28 alone will account for 34.9% of the variance of HS2, whilst adding question 12 will then account for 51.9% of the variance of HS2.

It will be noted that the order of questions is different in the two handedness categories; this does not mean that the structure of handedness is necessarily different within right and left-handers, but rather reflects the particular order of correlations with each particular data, an order which chance factors can make moderately idiosynchratic. Note that the simple correlations of each question with HS2, are in 82% of cases, greater than 0.4 and all are positive.

One way of producing a shorter questionnaire would be to take the top N questions of Table A2:1 and to then use them in association with their appropriate regression coefficients, to produce a scale. This however has two inconveniences. Firstly, it requires a complex regression; and secondly, the coefficients of that regression equation would differ for right and left handers. Nevertheless if, say, a 3 item questionnaire was required for just left-handers, this would be the optimal approach.

More desirable would be a questionnaire in which the scale was produced by simple addition of the individual items, and for which the items were the same for right and for left handers. There is no simple statistical approach to such a problem, with the exception of a combinatorial multiple regression. This has not been carried out due to its unwieldiness. Instead an approach was adopted in which firstly the nine questions were taken for which there was

no cultural bias (i.e. those of HS3), thus ensuring a reasonable degree of symmetry for the final scale. These nine questions were then run in a hierarchial multiple regression against HS2, separately for each handedness category. Of the top six questions in the analysis, five were the same for each handedness group. A scale (HS4) was therefore constructed by simple addition of these five questions (and also of question 1, writing hand). Table 2.1 shows that the left and right-handed means of HS4 are not significantly different, and that the variances are only just significantly different with p less than 0.05. It must be pointed out however that the small number of questions means that there is a small degree of skewness, although this is not as great for right-handers as on the AHS, OHS or BNHS, or for left-handers on the AHS or OHS. Kurtosis is negligible for right-handers, and smaller for left handers than on the AHS or OHS.

Table A2.2 shows, separately for right and left-handers, the correlations between the HS2, HS3 and HS4 scales. The correlation for right-handers between the 6 item HS4, and the 21 item HS2 is 0.811, indicating that HS4 retains 65.7% of the variance of HS2; for left-handers the variance retention is 42.83%.

The selection of an appropriate questionnaire for different purposes is not easy. For detailed research the maximum number of questions is desirable, and HS1 or

or HS2 would be recommended. For larger scale surveys of both right and left-handers, HS3 or HS4 would be appropriate, depending upon the desired number of questions. For research on just one handedness category, an optimal n-category questionnaire may be constructed by asking the top n questions shown in Table A2.1.

It must be remembered that the analysis given in this appendix assumes that individuals will make the same response to a single question on its own, or in the context of another 4, 8, 20 or 27 questions. This is of course not necessarily true and should be investigated empirically. Nevertheless it is hoped that the approach of this appendix provides a more rational approach to questionnaire construction.

TABLE 2.1 Mean and Variance of Left and Right handers on each of the scales

Scale	Number of questions	Percent strongly biassed q.s.	Percent unbiassed questions	Absolute Means Right	Left	R - L	P	Standard deviation Right	Left	Variance Ratio
AHS	8	25%	37.5%	93.67	68.07	25.60	$< 10^{-5}$	16.31	35.18	4.65
OHS	10	10%	20%	85.57	74.94	10.63	$< 10^{-5}$	13.60	25.02	3.38
BNHS	2	16.6%	41.6%	76.32	66.92	9.40	$< 10^{-5}$	19.70	26.04	1.74
CZHS	14	14.2%	35.7%	74.92	63.33	11.59	$< 10^{-5}$	16.64	26.77	2.58
HS1	28	25%	35.7%	70.09	49.19	20.90	$< 10^{-5}$	17.36	22.86	1.73
HS2	21	0	47.6%	66.45	60.71	5.74	0.0036	19.24	22.47	1.36
HS3	9	0	100%	62.49	62.29	0.19	NS	23.48	26.33	1.258
HS4	6	0	100%	61.88	62.63	-0.75	NS	29.76	33.53	1.269

Note: Significance of variance ratios is calculated with (114,791) degrees of freedom

	Skewness				Kurtosis				Kolomogorov-Smirnov two-sample test for difference between Right and Left distributions
	Right	sig	Left	sig	Right	sig	Left	sig	
AHS	-3.45	< 0.01	1.07	<0.01	15.69	<0.01	1.04	<0.05	P < 0.0001
OHS	-1.58	< 0.01	1.22	<0.01	3.14	<0.01	1.19	<0.05	P < 0.0001
BNHS	-1.06	<0.01	0.66	<0.01	1.88	< 0.01	-0.26	NS	P = 0.003
CZHS	-0.73	<0.01	0.85	<0.01	0.71	<0.01	0.50	NS	P <0.0001
HS1	-0.65	<0.01	0.24	NS	0.34	<0.05	-0.91	<0.05	P < 0.0001
HS2	-0.53	<0.01	0.34	NS	0.14	NS	-0.69	NS	P = 0.056
HS3	-0.47	<0.01	0.81	<0.01	-0.14	NS	0.35	NS	P = 0.977
HS4	-0.75	<0.01	0.98	<0.01	0.06	NS	0.57	NS	P = 0.980

TABLE 2.2 Shows the mean and standard deviation of the individual subject's standard deviation on the items comprising the three scales HS1, HS2 and HS3.

Scale	Handedness	$\overline{SD}$	σ_{SD}	$\overline{SD}_R$ vs $\overline{SD}_L$	σ_{SD_R} vs σ_{SD_L}	Kelmogrovov-Smirnov test SD_R vs SD_L
HS1	R	0.821	0.292	$F(1,793) = 186.5$ $P \ll 0.001$	NS	K-Sz = 5.53 $P \ll 0.001$
	L	1.246	0.284			
HS2	R	0.824	0.296	$F(1,835) = 13.51$ $P = 0.083$	$P < 0.05$	K-Sz = 2.02
	L	0.943	0.380			
HS3	R	0.876	0.394	$F(1,850) = 0.004$ NS	NS	K-Sz = 0.88 NS
	L	0.878	0.457			

TABLE 2.3

		Right-handers			Left-handers		
	Group	Mean	N	Sig	Mean	N	Sig
Family History	-	66.49	503	F(1,772) = 0.206 NS	55.92	26	F(1,111) = 1.591 NS
	+	65.83	271		66.26	87	
Number of Left-handed relatives	0	66.75	520	F(3,787) = 0.966 NS linear trend P = 0.1951	55.70	27	F(3,110) = 2.375 P = 0.074 linear trend P = 0.685
	1	66.90	201		65.17	46	
	2	63.14	55		64.24	25	
	3+	61.40	15		50.81	16	
Maternal Handedness	R	66.74	700	F(1,1748) = 4.769 P = 0.0293	59.11	85	F(1,106) = 2.60 P = 0.1092
	L	60.66	50		67.69	23	
Paternal Handedness	R	65.94	676	F(1,746) = 3.026 P = 0.823	61.14	88	F(1,106) = 0.215 NS
	L	70.08	72		58.50	20	
Sex	Male	66.14	541	F(1,707) = 0.172 NS	58.96	84	F(1,98) = 2.634 NS
	Female	66.85	168		68.75	16	
Birth Rank	1	65.80	390	F(4,736) = 0.252 NS	59.96	60	F(3,99) = 1.297 NS
	2	66.79	229		66.44	27	
	3	66.32	80		52.00	13	
	4	68.85	28		58.33	3	
	5+	64.71	14		-	0	
Birth Complications	-	66.44	591	F(1,779) = 0.126 NS	59.92	102	F(1,111) = 1.618 NS
	+	65.67	90		69.00	11	
Head Trauma or Neurological disease	-	66.60	735	F(1,777) = 1.523 NS	61.39	109	F(1,111) = 2.12 NS
	+	61.88	26		44.75	4	

Head Trauma or Neurological disease	-	66.60	735	F(1,777) = 1.523 NS	61.39	109	F(1,111) = 2.12 NS
	+	61.88	26		44.75	4	
Hand position whilst writing	Normal	65.80	334	F(1,393) - 0.008 NS	60.04	41	F(1,51) = 0.089 NS
	Inverted	66.04	61		62.41	12	
Hand-clasping	Right	67.55	179	F(1,398) = 2.84 P = 0.0594	57.16	25	F(1,52) = 0.830 NS
	Left	64.89	221		63.10	29	
Reading or Writing difficulty	No	66.47	721	F(1,781) = 0.353 NS	61.43	102	F(1,111) = 0.806 NS
	Yes	64.96	62		55.00	11	
Hair parting	Right	67.77	167	F(2,745) = 1.510 NS	62.63	19	F(2,104) = 0.375 NS
	Left	66.60	485		59.88	77	
	Middle or 'Others'	63.54	96		65.63	11	

TABLE 2.4

Handedness						Simple corr's on propositus		Simple correlation of maternal and paternal handedness
Propositus	Mother	Father	N	Multiple correlation on propositus	Sig of multiple correlation	Mother	Father	
R	R	R	308	0.0743	$F(2,305) = 0.847$ NS	0.0182	0.0718	-0.0142
L	R	R	43	0.2144	$F(2,40) = 0.963$ NS	-0.166	0.132	0.0159
R	R	L	26	0.1979	$F(2,23) = 0.469$ NS	0.0232	0.1979	0.0994
L	R	L	10	0.2944	$F(2,7) = 0.332$ NS	0.1386	-0.1366	0.562
R	L	R	28	0.228	$F(2,25)_{NS} = 0.691$	-0.1198	-0.1859	-0.142
L	L	R	15	0.2696	$F(2,12)_{NS} = 0.470$	0.1718	-0.1940	0.0763

Table 2.5

Survey 1b		Hand posture whilst writing: Normal	Inverted	N	% Inverted
Handedness	Right	348	632	410	15.12%
	Left	57	19	76	24.99%

$X^2 = 4.50$, 1df, $P < 0.05$

Survey 2		Hand posture whilst writing: Normal	Inverted	N	% Inverted
Handedness	Right	380	48	428	11.21%
	Left	53	25	78	32.05%

$X^2 = 27.70$, 1 df, $P < 0.001$

TABLE A2:1

Shows, for left and right-handers separately, the order of entering each of the component items of HS2 into the regression equation, and the cumulative percentage of the variance accounted for.

	Right-handers		Left-handers	
Order of entry	Question	Variance	Question	Variance
1	28	34.8	27	31.7
2	12	51.9	19	53.7
3	8	61.9	11	67.2
4	21	70.0	9	74.4
5	4	76.3	18	79.4
6	14	81.7	12	83.2
7	15	85.4	8	85.8
8	9	88.8	20	87.9
9	19	91.1	5	90.3
10	20	92.8	14	92.3
11	10	94.2	2	94.2
12	24	95.5	24	95.7
13	5	96.6	21	96.6
14	11	97.7	10	97.7
15	18	98.3	6	98.5
16	6	98.9	4	99.0
17	27	99.4	15	99.3
18	2	99.6	16	99.6
19	16	99.8	28	99.9
20	13	99.9	13	99.9

TABLE A2:2

Correlations, for right and left-handers separately, between HS2, HS3 and HS4. Right-handers are placed in the triangle above the diagonal, and left-handers in the triangle below the diagonal.

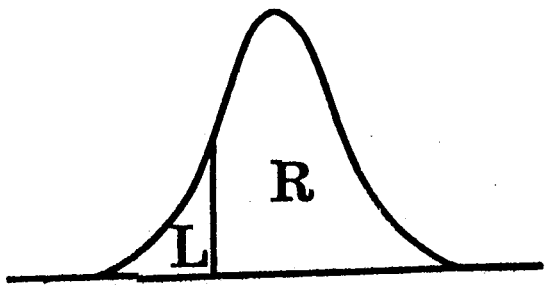
	HS2	HS3	HS4
HS2	-	0.918	0.811
HS3	0.793	-	0.925
HS4	0.654	0.946	-

Figure 2.1 Shows, schematically, various possible models of the structure of handedness.

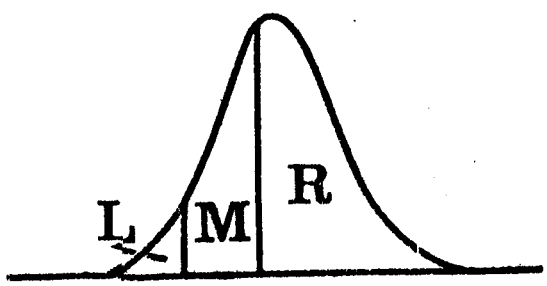
b

c

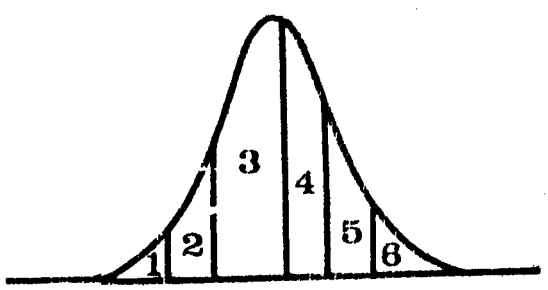
d



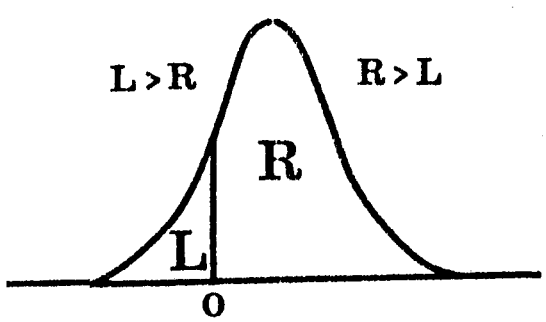
e



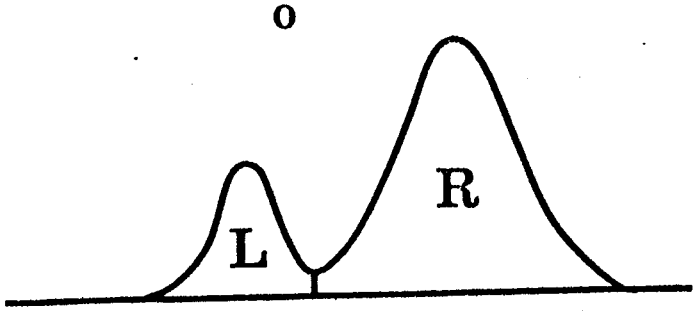
f



g



h



i

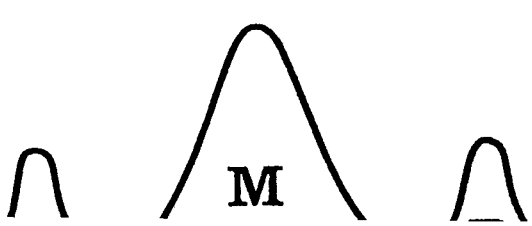


Figure 2.2 Shows the distribution of scores of the subjects of Survey 1 on the eight-item handedness scale of Annett (1967). Abscissa shows the number of questions for which the subject indicated that the right hand was used.

Annett, 1967

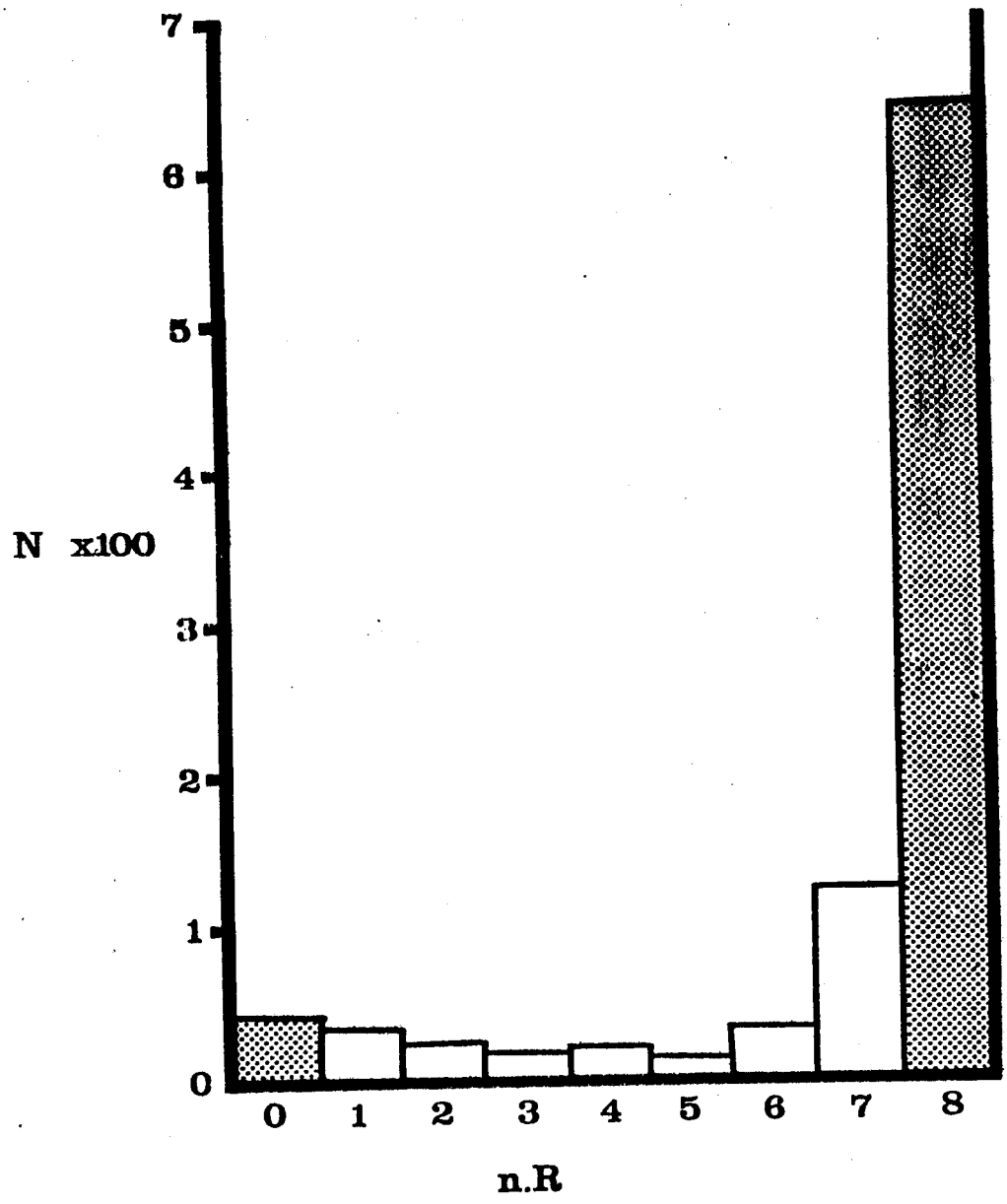


Figure 2.3 Shows the scores of the subjects of Survey 1 on the Oldfield handedness scale (OHS). Abscissa shows scores standardised as described in text. Each half of the distribution (left and right) is divided into deciles. A score of +100 indicates complete right-handedness and a score of -100 indicates complete left-handedness

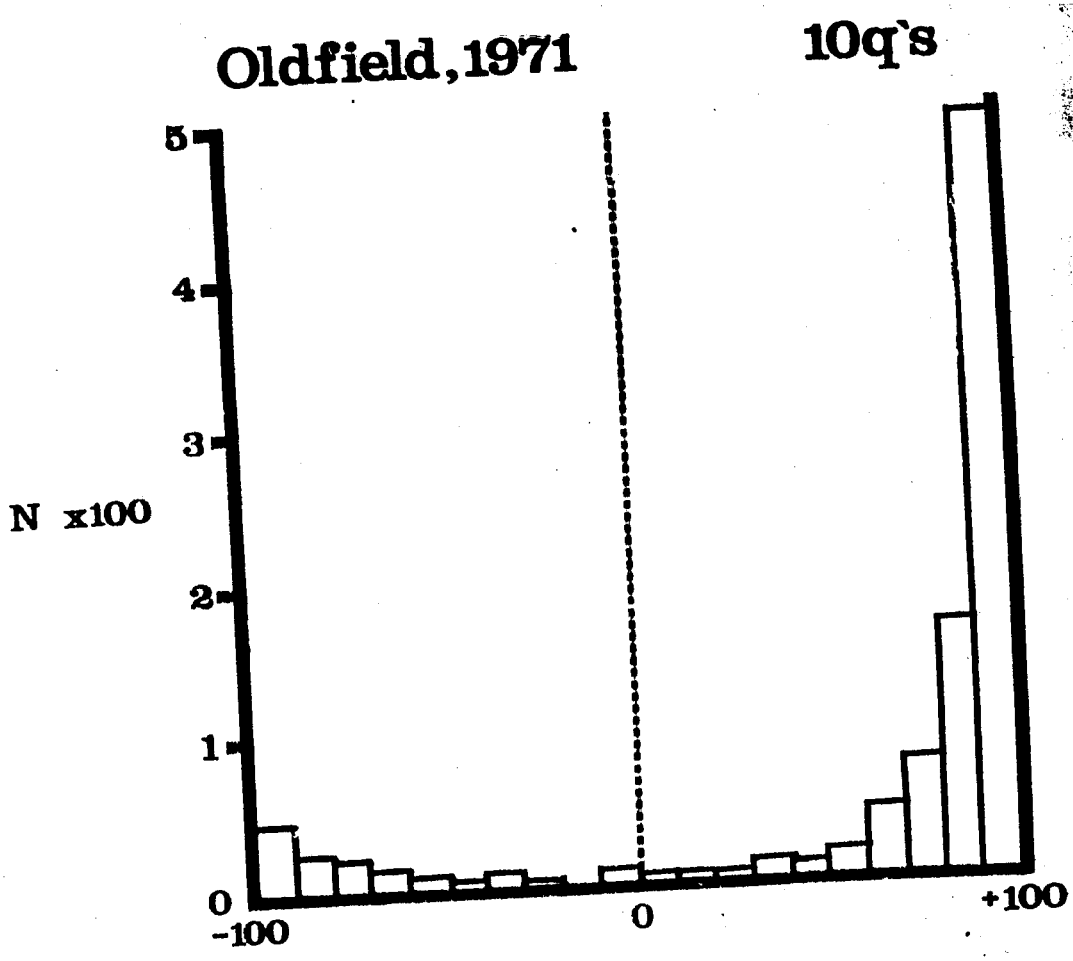

Oldfield, 1971
10q's
N x100
5
4
3
2
1
0
-100
0
+100

Figure 2.4 Shows the scores of the subjects on the Briggs-Nebes handedness scale (BNHS). Otherwise as for Figure 2.3

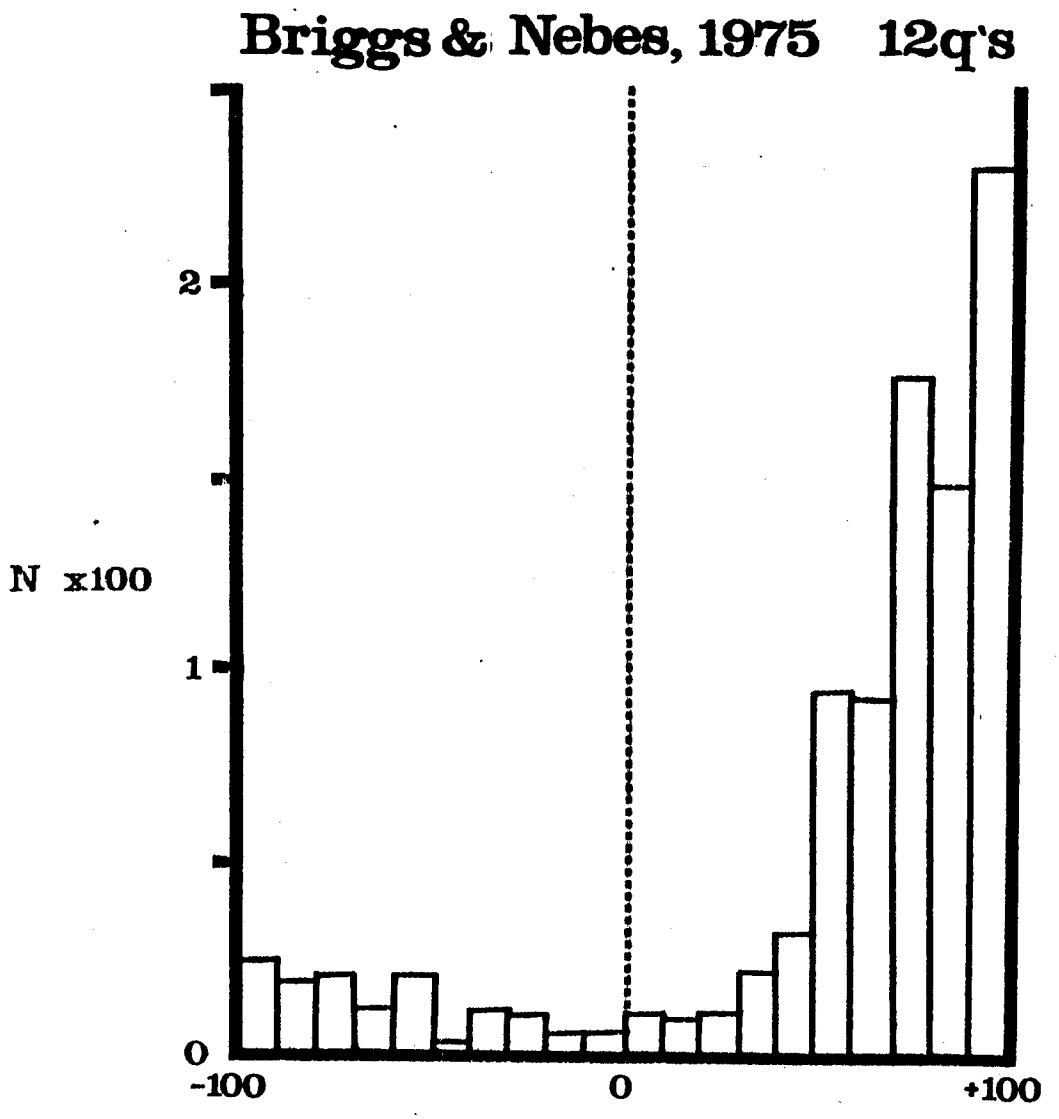

Briggs & Nebes, 1975 12q's
N x100
2
1
0
-100
0
+100

Figure 2.5 Shows the scores of subjects on the Crovitz and Zener Handedness scale (CZHS).

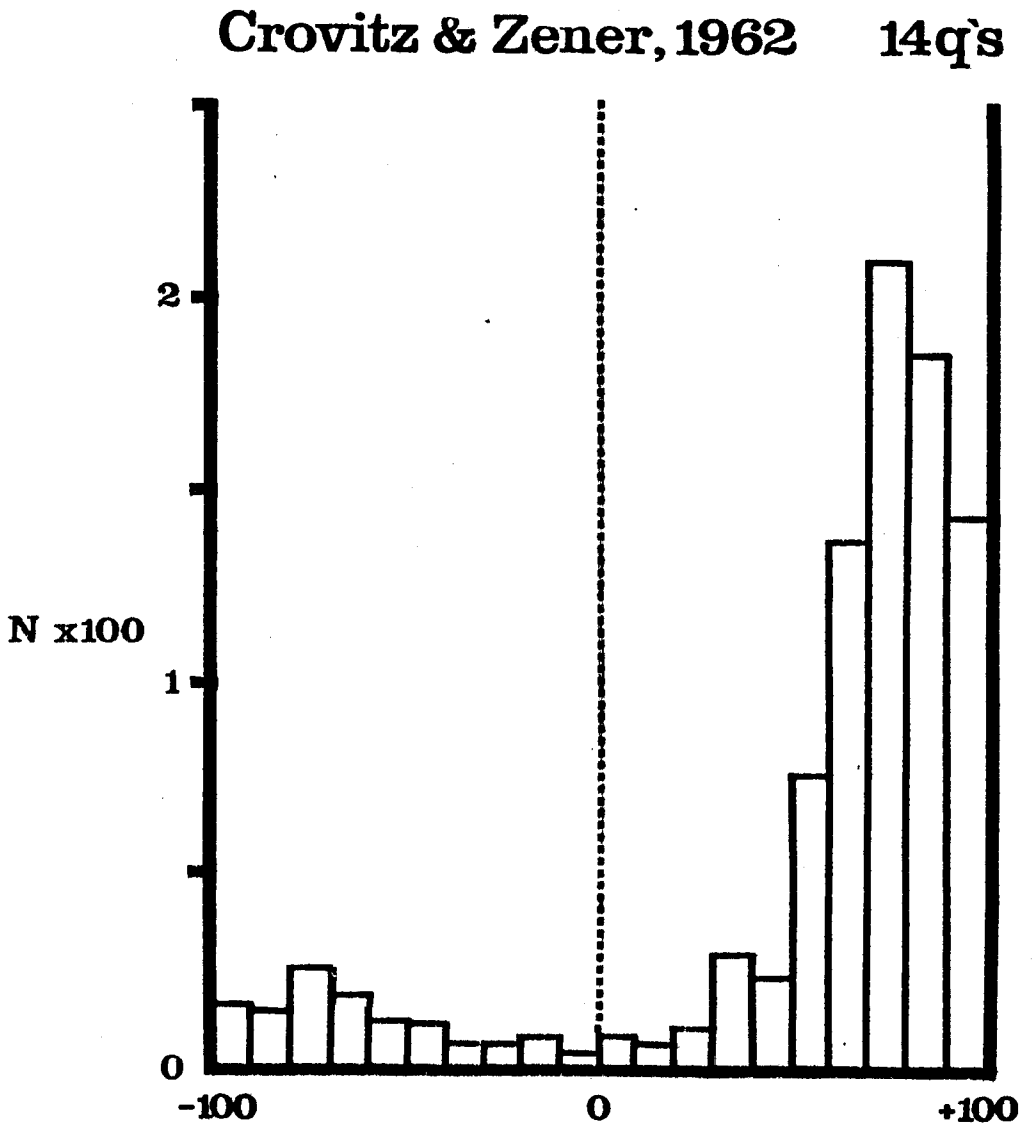

Crovitz & Zener, 1962 14q's
N x100
2
1
0
-100
0
+100

Figure 2.6 Shows the distribution of scores on the 28 item HS1. The dotted line in the left half of the distribution represents the reflection of the right half of the distribution around the mid-line, and in proportion to the total number of left-handers.

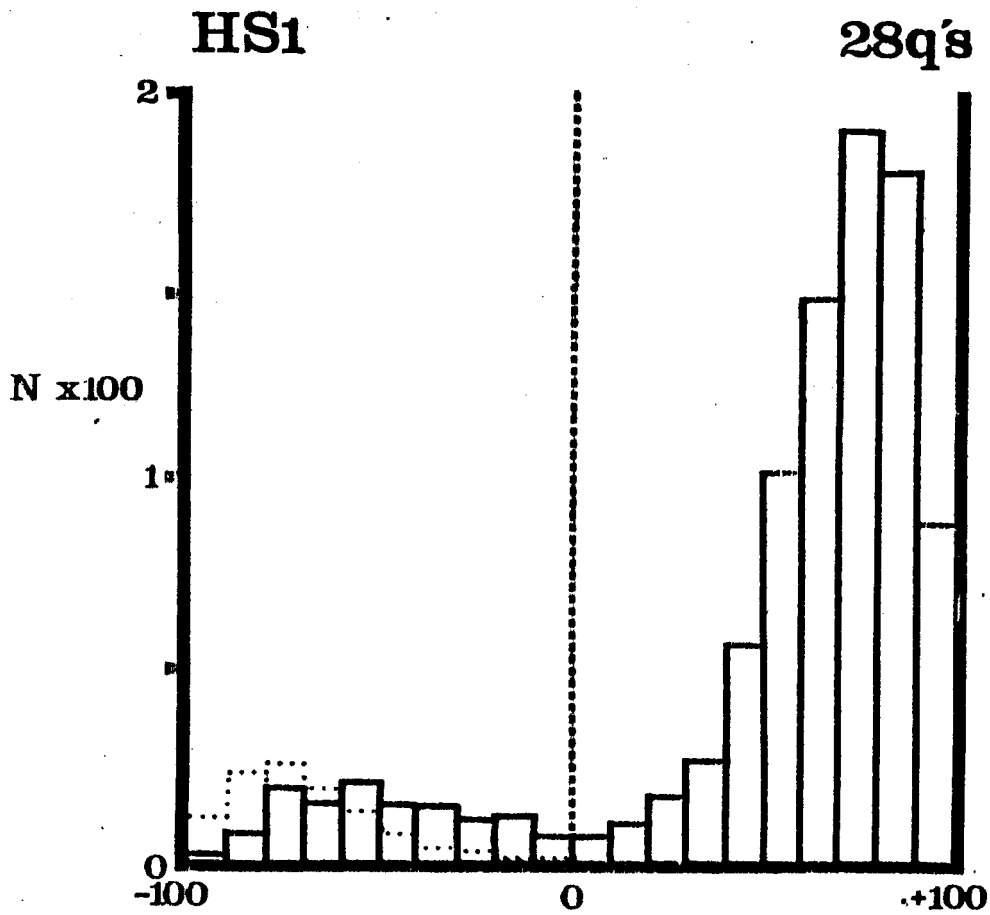

HS1
28q's
N x100
2
1
0
-100
0
+100

Figure 2.7 Shows, for each of the 28 individual questions, the mean scores of right and left-handers. Differences between right and left handers were tested by means of a t-test; points shown as open squares are not significantly different in right or left handers with $P > 0.05$. Solid squares show a significant difference; these have been arbitrarily divided into large differences (large squares), and small differences (small squares). Numbers alongside points represent question numbers. Question 1 has been omitted due to almost all subjects answering in extreme categories (see text).

Handedness Questions

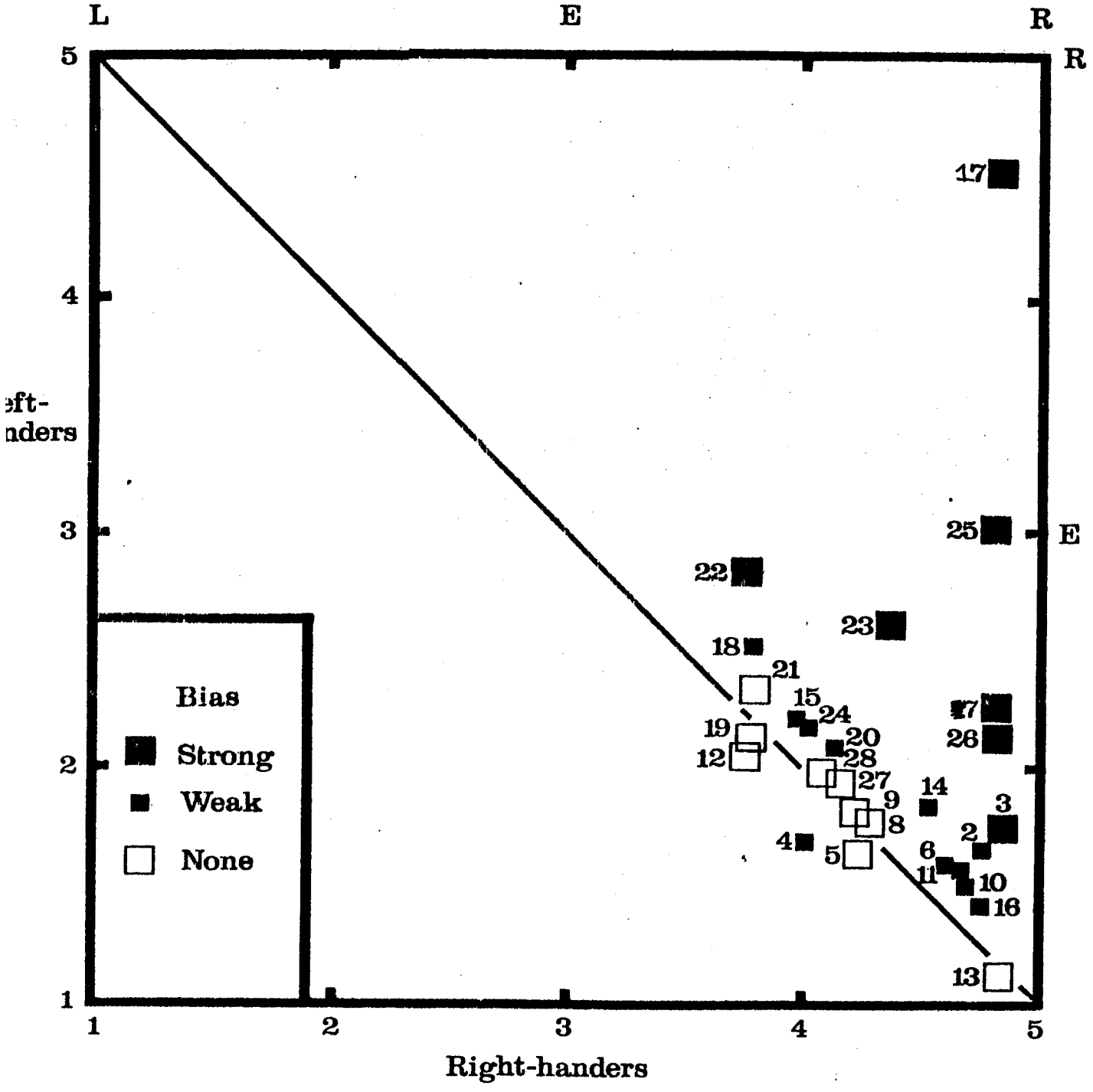


Figure 2.8 Shows the distribution of scores on a 21-item handedness scale (HS2), as described in the text, and in Appendix A2:1. Dotted line in left-hand half is as in Figure 2.6

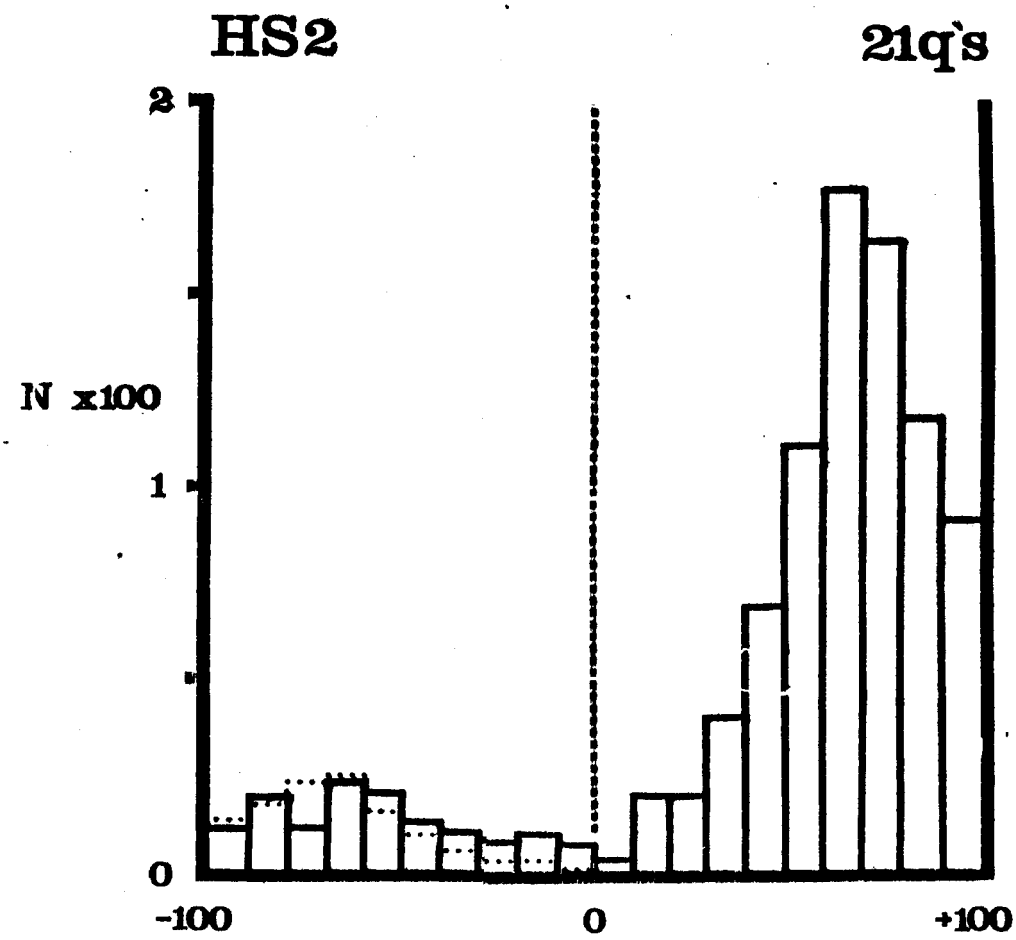

HS2
21q's
N x100
2
1
0
-100
0
+100

Figure 2.9 Shows the distribution of subjects divided into their classification of Right, Mixed and Left (Annett, 1967), as a function of their scores on the HS2. Note differences in scale of ordinates.

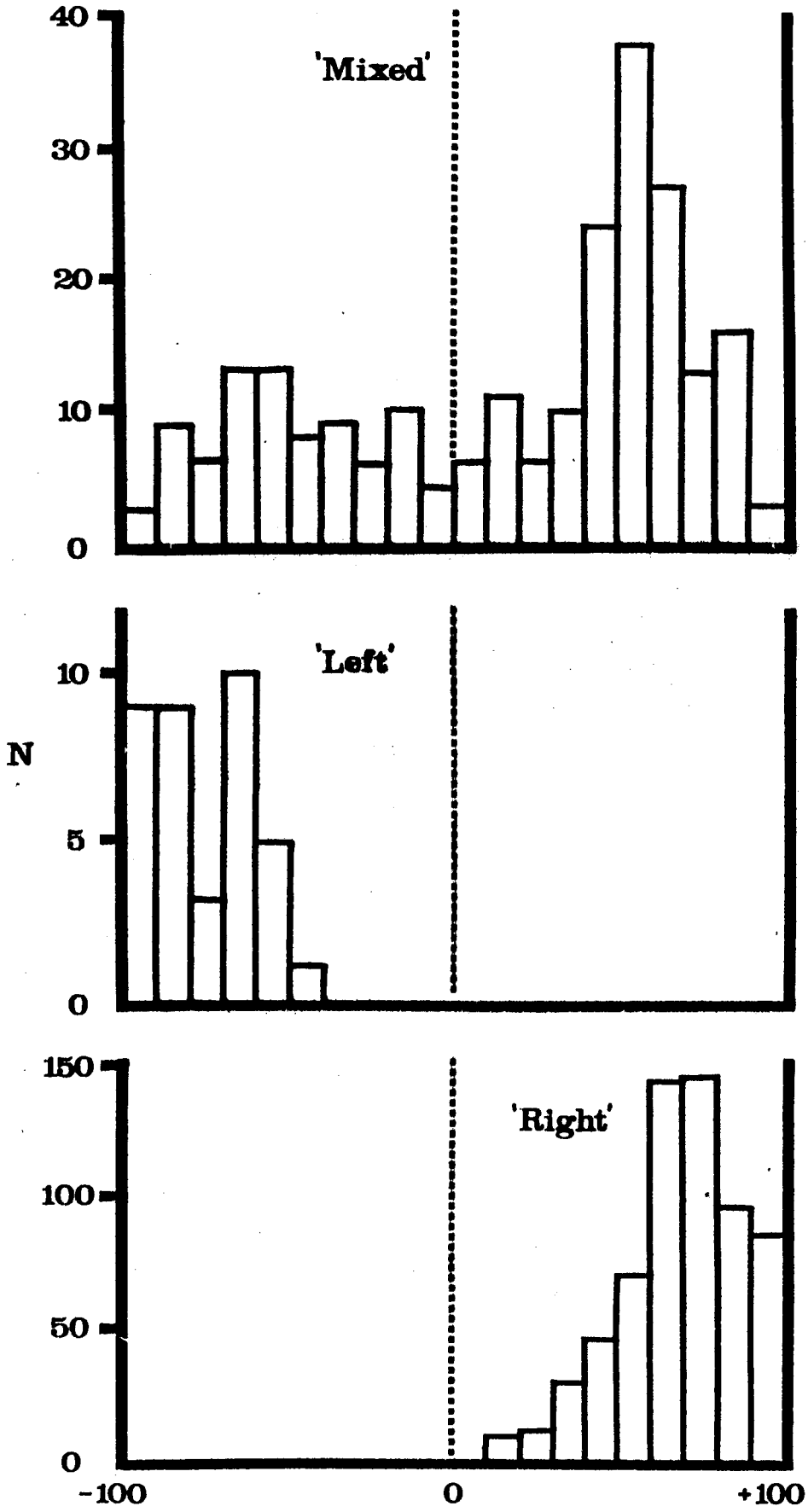

'Mixed'
'Left'
'Right'
N
40
30
20
10
0
10
5
0
150
100
50
0
-100
0
+100
HS2

Figure 2.10 Shows a plot of eigen-value against factor number for factor analysis of questions in HS2. Solid squares indicate factor analysis of left and right-handers combined; solid circles indicate factor analysis of just right-handers, and open circles indicate factor analysis of just left-handers. Note that each curve has been staggered along the abscissa to avoid confusing overlap.

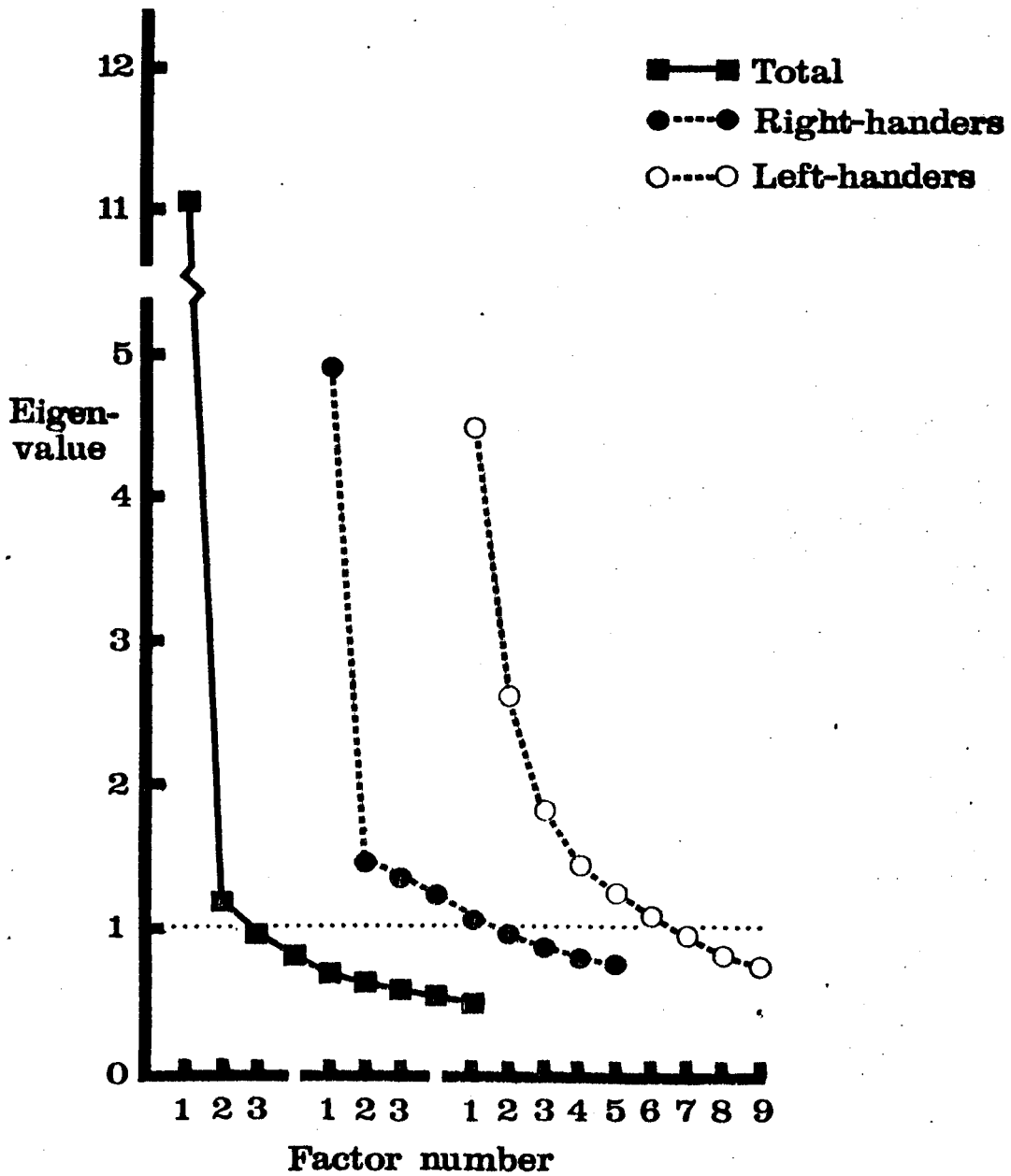

Total
Right-handers
Left-handers
Eigen-value
12
11
5
4
3
2
1
0
1 2 3
1 2 3
1 2 3 4 5 6 7 8 9
Factor number

Figure 2.11 Shows the mean (abscissa) and standard deviation (ordinate) of the responses of individual subjects on the HS3.

Figure 2.12 Shows, for different groupings within each handedness category, the percentage with a positive family history of left-handedness (see text for definition). Subjects are grouped into deciles on the HS2 scale. For the top seven right-handed categories each solid circle indicates one handedness decile. For the remaining points adjacent groups have had to be concatenated to produce adequate sample size; horizontal lines through each point indicate range of that particular point. Vertical bars through points indicate ± one standard error.

Dotted line represents expected distribution if there is no relation between degree of handedness and family history

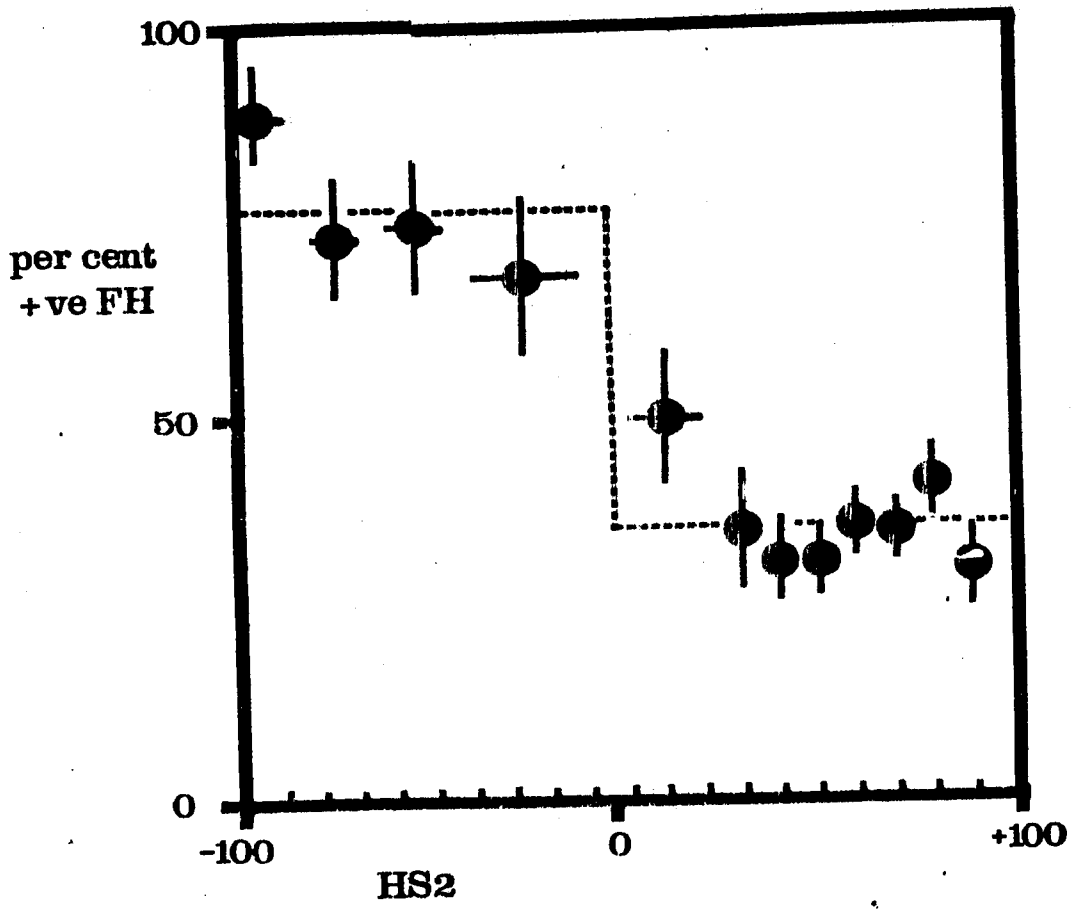

100
per cent
+ve FH
50
0
-100
0
+100
HS2

Figure 2.13 The degree of handedness (ordinate) of right-handed children of two right-handed parents in Survey 2 is shown as a function of the mid-parental handedness score (abscissa). Solid circles indicate mean, vertical bars representing ± one standard error. In view of skewness of overall distribution, dotted lines indicate medians. Figures along the top indicate sample size for each point.

R,R parents: R children

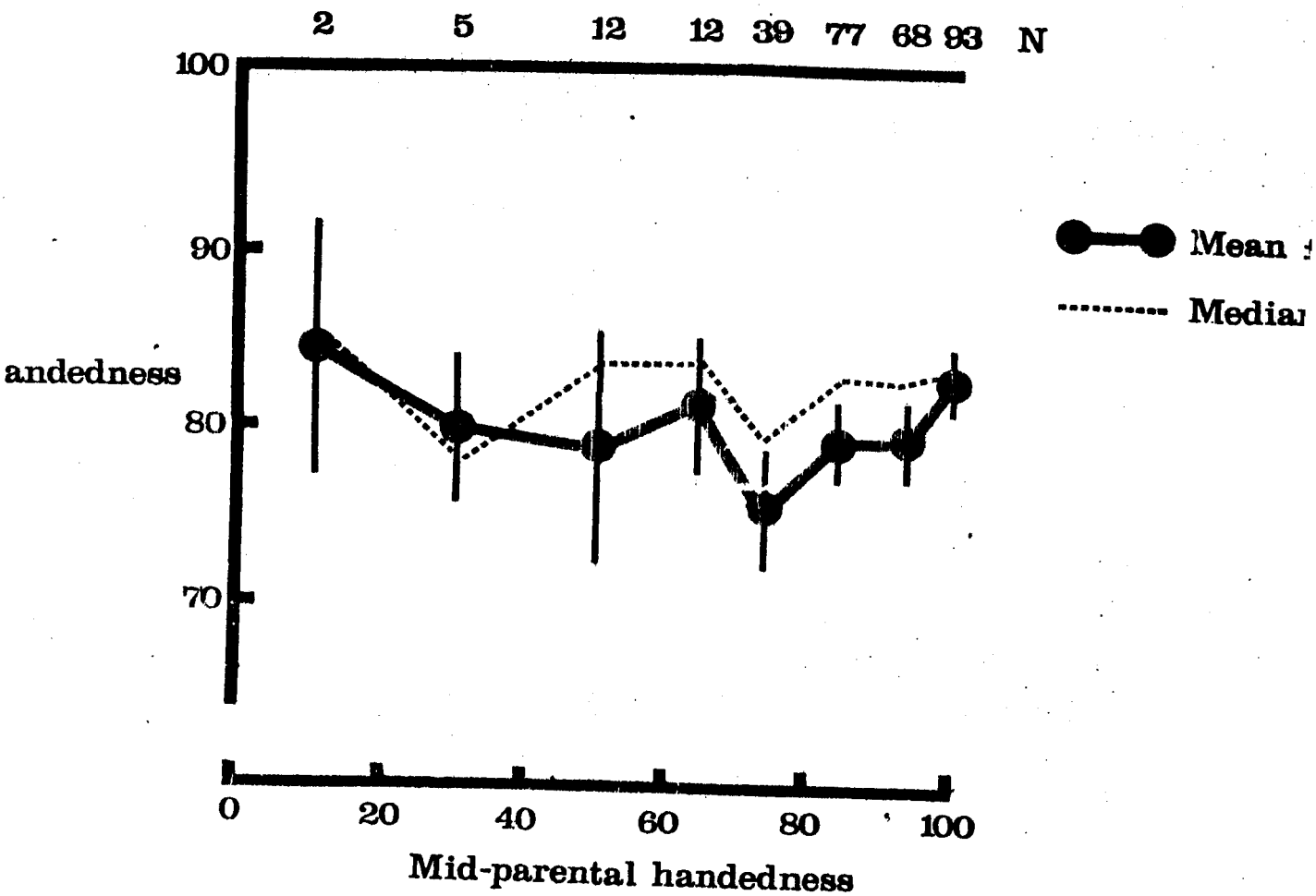


Figure 2.14 Shows the population distribution of an asymmetry score (100 x (R-L)/(R+L)) on a unimanual task, marking as many squares as possible on a piece of graph paper in one minute, as ascertained in 12777 11-year old children in the National Child Development Study. Overall Mean = 12.84; SD = 13.46; Skewness = -0.852; Kurtosis = 2.687.

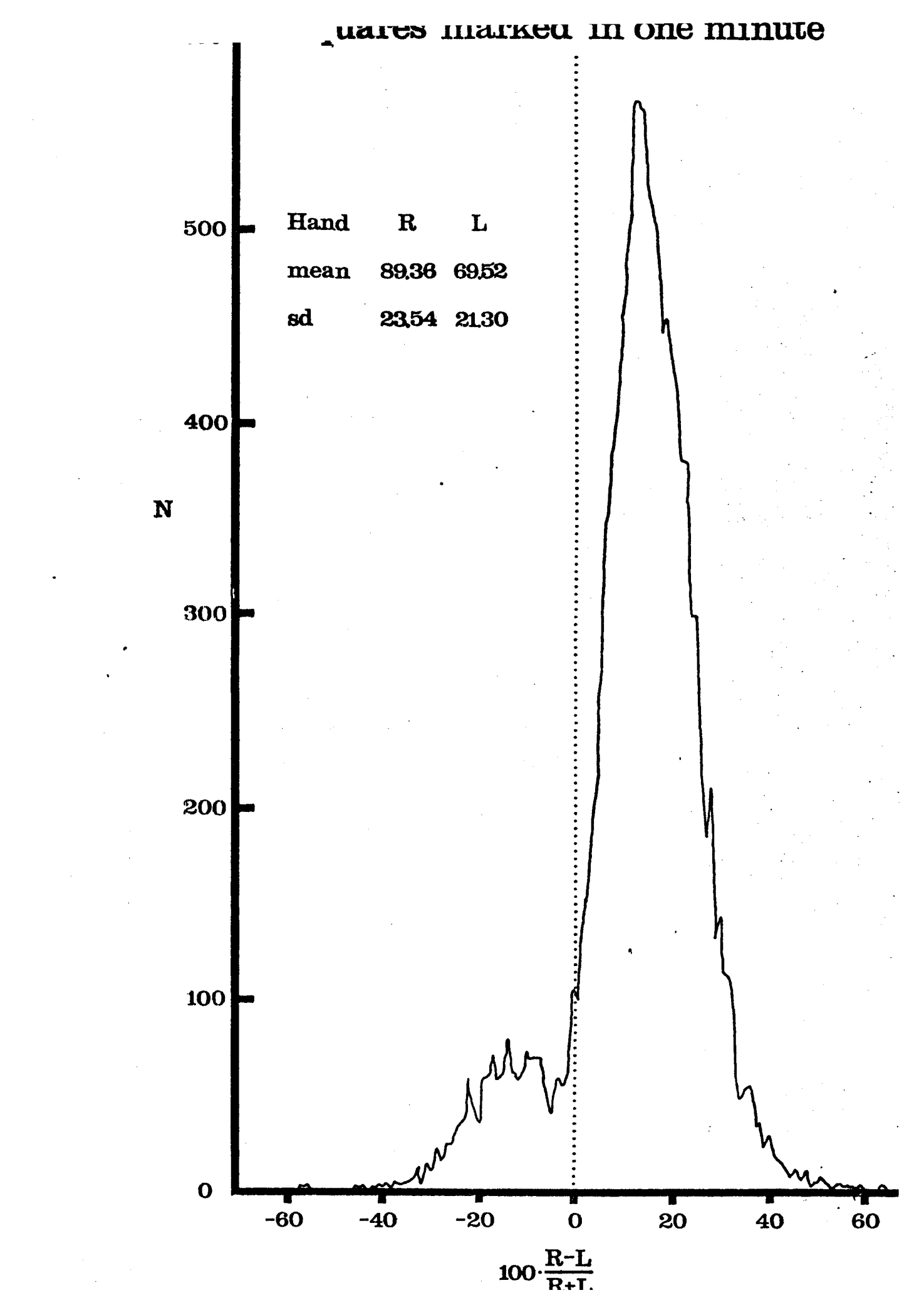

uares marked in one minute
Hand R L
mean 89.36 69.52
sd 23.54 21.30
500
400
300
200
100
0
N
-60
-40
-20
0
20
40
60
100·R-L/R+L

Figure 2.15 Shows the same data as Figure 2.14 but broken down by handedness (see Chapter 3.3 for details). For right-handers the mean score = 16.225, whilst for left-handers the mean score is -13.805; the respective SD's are 8.348 and 11.207 whilst skewnesses are 0.480 and -0.031. Kurtoses are 3.337 and 6.812, and sample sizes are 11208 and 1416 respectively.

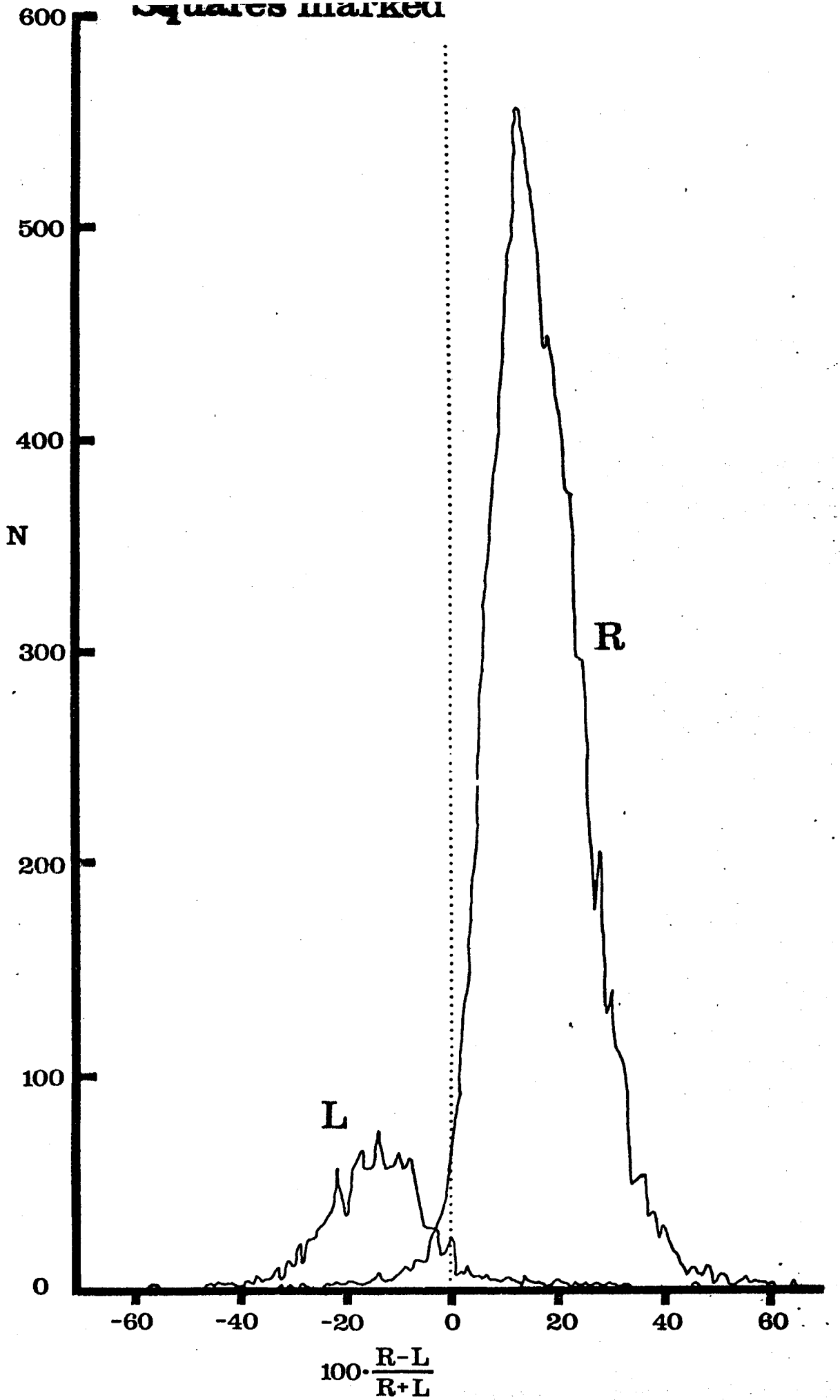
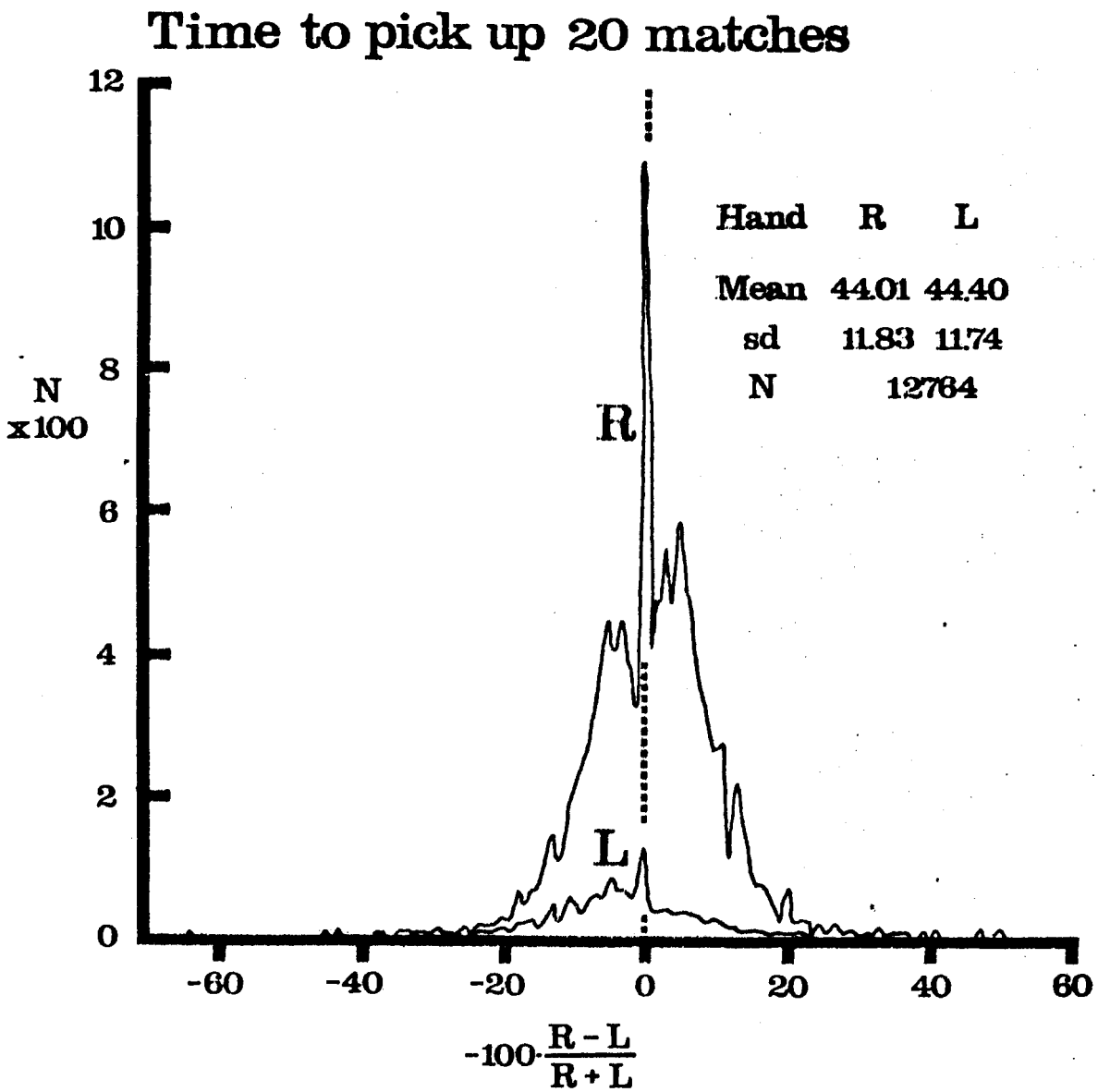

600
500
400
N
300
200
100
0
R
L
-60
-40
-20
0
20
40
60
100·R-L/R+L

Figure 2.16 Shows the distribution of an asymmetry score (-100 x (R-L)/(R+L)) for a task in which 11-year old children were timed at picking up 20 matches unimanually. For the entire population (N = 12747), the mean = 0.485, SD = 8.98, skewness = 0.262, and Kurtoses = 4.985. For right- and left-handers the respective distribution parameters are means = 0.982 and -3.408; SD's = 8.747 and 9.882; skewnesses = 0.331 and -0.351; Kurtoses = 4.319 and 10.467; and sample sizes = 11184 and 1412 respectively.


Time to pick up 20 matches
12
10
8
6
4
2
0
N
x100
R
L
Hand R L
Mean 44.01 44.40
sd 11.83 11.74
N 12764
-60
-40
-20
0
20
40
60
$-100\cdot\frac{R-L}{R+L}$

CHAPTER 3: HANDEDNESS AND BIRTH STRESS

"For he himself is subject to his birth"

Hamlet, I.3.18

Summary

No evidence was found, either in two retrospective studies, or one large prospective study, for a relation between left-handedness and birth complications.

3:1 Introduction

Bakan (1971) proposed that left-handedness might be a result of "neurological insult associated with pre-natal or delivery factors". At that stage his only substantive evidence was a relation between handedness and birth order, those birth ranks associated with a higher incidence of perinatal mortality (1, 4+) having a higher frequency of left-handedness than the lower-risk birth ranks (2,3). Bakan, Dibb and Reed (1973) attempted a slightly more direct approach to the problem, asking students whether there had been any complications during their own birth. Left-handers reported more birth stress, as also did both left- and right-handers with a sinistral family history as compared with the appropriate control group without a sinistral family history. The incidence of left-handedness was also raised in the children of older mothers (greater than 30 years), another high-risk group for perinatal morbidity and mortality. Bakan, Dibb and Reed proposed that familial tendencies in left-handedness might be a secondary result of a familial tendency to birth stress. Extrapolating further from their hypothesis, they suggested that "the frequency of left-handedness ... might serve as a general index of the prevalence of birth-related neurological insult". They also implied that left-handedness should be more common in lower socio-economic groups, although the opposite to this was later claimed by Bakan (1977).

Bakan (1975) without presenting any further data took his hypothesis even further. Birth stress, and by direct implication left-handedness, "may be related to a variety of genetic or environmental factors, such as maternal physique, parity, nutrition, drug ingestion, smoking, or even pain sensitivity which may influence the amount of anaesthetic used in delivery". Left-handedness was regarded as "an indication of neurological insult associated with pregnancy and birth. It is something to be reduced rather than tolerated ... In fact handedness deviation in a group indicates that the group is at risk for neuropathological conditions. Preventive interaction in such groups could play tremendous dividends, both economically and in the reduction of suffering". Clearly such a theory, if true, is of the greatest importance, both for neuropsychology and for obstetrics.

Bakan's theory has not had smooth running the whole way. Hubbard (1971) was unable to replicate Bakan's birth order effect: neither was Schwartz (1977). Bakan (1977) attempted to account for these discrepancies in terms of socio-economic differences between samples. He also presented another sample of data which showed the birth order effect at the 0.05 level. Further evidence cited by him in favour of his hypothesis was that Leviton and Kilty (1976) had found a 'dose-related effect', especially high incidences of left-handedness being found in birth ranks of 6 and higher. Despite this apparent

sophistication the authors omitted to give the sample size within each birth rank, and also gave no statistical tests; a reconstruction of the data suggests that there is no statistical significance, their sample being far too small to find effects of the size postulated (Chi-square = 9.58, 6 df, NS).

Hicks, Pellegrini and Evans (1978) also failed to find a birth order effect, and Hicks, Evans and Pellegrini (1978) pointed out that a compilation of all the studies in the literature still failed to show a significant birth-order effect of the type postulated by Bakan. My own analysis of surveys I and II came to the same conclusion (data not given here).

The only published replication of the study by Bakan, Dibb and Reed (1973) is that of Schwartz (1977) who failed to find any significant effect. My own data from surveys I and II are shown briefly in Table 1.

Details of Surveys I and II may be found in Chapter 2:2. In summary, Survey I consisted of a questionnaire returned by 936 undergraduates in which they were asked to report any known history of complications during birth, and also to complete a 28-item handedness questionnaire, handedness being defined as in Chapter 2. Survey II consisted of a larger questionnaire, distributed on the eve of the graduation ceremony, to all graduates. The

students, and also their parents, were asked to complete items relating to handedness, and the whole family was asked to report birth complications in the propositus and the siblings of the propositus. A total of 512 adequately completed questionnaires was received, and these replies contained information on a total of 1245 propositi and siblings. Information on birth complications in Survey II might be expected to be slightly more reliable (and also more frequent) than that in Survey I, since the mother in particular would have a more detailed recollection of the events of the birth. Nevertheless given the work of Chamberlain and Johnstone (1975) which demonstrated the fallibility of mothers' memory for events of the previous parturition (only a few years previous), the present data should not be relied upon to any great extent.

Table 3:1a shows that in neither survey is there any evidence for a relationship between handedness and birth complications. Table 3:1b shows that, contrary to the findings of Bakan, Dibb and Reed (1973), there is no relationship, either in right or left-handers, between a history of birth complications and a family history of sinistrality. In both surveys, family history of propositi and siblings is defined in terms of the handedness of all the <u>remaining</u> family members, a single report of left-handedness being sufficient to define a positive family history.

There has been little other evidence to support the birth stress hypothesis (although this has not prevented it from being widely quoted as established truth). Bakan (1977) has pointed out that in a small prospective study, Barnes (1975) found that the time to regular breathing after delivery correlated closely with subsequent handedness measured at the age of three. Barnes however puts a totally different interpretation upon this fact, suggesting "that left-handers are more likely to have a stressful birth, rather than the stressful birth causing the left-handedness". Whether or not the hypothesis of Barnes deserves serious consideration will depend upon the same evidence which would test Bakan's hypothesis, for unless an effect is demonstrated both hypotheses must surely fail.

Bakan appears to have missed one small piece of evidence which lends some support to his theory. Turkewitz, Moreau and Birch (1968) found that head turning in response to tactile stimulation was less asymmetric in a group of babies with low Apgar scores, than in babies with high Apgar scores.

Bakan (1978) has cited experimental evidence by Brann and Myers (1975) that experimental asphyxia in neonatal rhesus monkeys effects only the left hemisphere if the hemispheres are affected asymmetrically; whilst superficially impressive, the study in fact refers to a

total of only 3 monkeys, and is thus less than convincing in statistical terms.

As both Bakan (1977) and Hicks, Pellegrini and Evans (1978) recognise, the only way of resolving these questions is by a prospective study. In this Chapter I will report the results of such a study, about 12,000 children being followed from birth to at least the age of 11 years. The children comprise a nationally representative sample from all social classes. Ironically this data has been in existence for some years, and was available even before the start of the present controversy over birth-stress and left-handedness. Other aspects of handedness in the NCDS data have previously been considered by Calnan and Richardson (1976, a, b).

3:2 Method

The data for the present study were those collected by the National Child Development Study (NCDS), and now available to *bona fide* researchers via the Social Science Research Council's Archive of computer-readable data at the University of Essex. The NCDS study was originally the 1958 Perinatal Mortality Survey (PMS), which was sponsored by the National Birthday Trust Fund. It looked at a wide range of obstetric factors relating to *all* children born in England, Wales and Scotland during the period 3rd to 9th March 1958. Obstetric and other data were

collected by the mid-wife or doctor present at the birth, and are undoubtedly of a very high quality and accuracy. Extensive analyses of these data have already been published (Butler and Bonham, 1963; Butler and Alberman 1969).

In 1964 the NCDS was set up in order to trace as many as possible of the children studied during the 1958 PMS. This study (NCDS I) looked at the children at about the age of 7, and obtained interviews with parents and teachers, gave each child several ability tests, and also had each child examined by a doctor. Naturally, it was not possible to obtain adequate answers on all items for all children, and some children were not subjected to a whole section of the survey (e.g. the medical examination). Nevertheless the response rate was extremely high. Many results have already been reported from that study (National Child Development Study, 1966, 1972).

The 1958 cohort was studied again in 1969, when they were aged about 11, in a second study (NCDS II), which was essentially similar in content to that of NCDS I. A third study (NCDS III) was also carried out when the children were 16 years of age, but these data have not been used in the present study, being not yet available for public re-analysis.

For the present study, information was requested

on some 500 variables on each of a total of 18,285 children. This information was supplied on magnetic tapes which were analysed using the SPSS programme package (Nie et al, 1975), on the IBM 370/165 computer at the University of Cambridge.

In view of the potential importance of the NCDS data for research on handedness, since the comprehensive nature of its information allows a whole range of studies, it is worthwhile in the present paper devoting a little space to an analysis of the formal structure of the handedness information. This analysis can be regarded as being complementary in some sense to the study of the structure of handedness reported in Chapter 2, and provides further evidence that a simple classification of handedness into two categories, left and right, is adequate for most purposes.

3:3 The structure of handedness in the NCDS

The NCDS contains five variables explicitly concerned with hand preference. It also contains several items on differential skill of the two hands, and also questions on foot laterality and eye dominance; the first of these items has been considered further in Chapter 2.

Table 3:2 summarises the five items concerned with hand preference. Table 3:3 gives for completeness, the

number of children (out of a total of 11029 for whom valid information was available on all five hand preference items) in each of the possible 108 response categories. Note that 25 (23.1%) of each of the categories of Table 3:3 are actually empty, despite the large sample size. Table 3:4 shows an inter-correlation matrix for the five hand-preference items. All correlations are high, positive, and highly significant. Factor analysis of this 5 x 5 matrix gives eigen-values of 4.08, 0.36, 0.29, 0.17 and 0.07, the first factor alone accounting for 81.7% of the total variance. Clearly this first factor is the only significant factor. There is thus only a single dimension of handedness in the NCDS data. The question then arises as to the best way of dividing this continuum into categories, if at all. Factor scores were calculated for each subject on the single dimension derived from the factor analysis, and these factor scores were standardised so that the maximum and minimum possible scores were +100 and -100 (as in Chapter 2). Figure 3:1 shows the frequency distribution of individuals with particular factor scores. Note that the ordinate of this figure is logarithmic, and thus the 'dip' in the centre is far steeper and deeper than it appears. From Figure 3:1 it is clear that there are only two major categories present, right handers having positive scores and left-handers having negative scores. Using a criterion of zero on the standardised factor scores, the present population contains 11.25% left-handers. Whilst computationally correct, the

criterion described is somewhat inconvenient. Table 3:5 shows the relation of handedness, as classified above, to each of the five variables of which the scale was initially constructed. The relation with item 1275, the writing hand of the child at the age of 11, is extremely close, only one child being mis-classified if this variable is used instead of the above criterion derived from the factor analysis. In this and all future studies therefore, 'handedness' will refer to the response recorded in item 1275. It is noteworthy that this conclusion is consistent with the arguments put forward in Chapter 2 that the single best criterion of handedness is the writing hand.

3:4 Handedness and birth stress

The NCDS data contains a large amount of information on the pre-natal and peri-natal condition of each child, the information being collected at the time of birth, and therefore having a high degree of accuracy. In all the following tables, children have been considered only if they are singletons without congenital defects.

Table 3:6 shows the relation between left-handedness and the occurrence of each of 28 dichotomous items which might be predicted to increase, or to be associated with birth stress. Results are given for all births (i.e. singletons without congenital defects), male births alone,

and female births alone. In the NCDS data there is a significantly higher incidence of left-handedness in male children (12.81%) than in female children (9.64%), and therefore data combined by sex might sometimes give spuriously significant results, particularly if a complication were itself of different incidence in the two sexes. The combined male and female data are given, both for completeness, and also to assist interpretation of data from relatively rare conditions. Chi-squared values and p values in Table 3:6 are two-tailed values, Yates' correction being applied in all cases.

The great majority of the differences in Table 3:6 fail to reach significance, thus arguing against a relation between handedness and birth trauma. However, it might be argued that many of the variables shown in Table 3:6 actually show a small correlation with handedness and that this correlation is in the expected direction, and thus the combined impact of all of these minor correlations might be to produce a correlation overall which is significant. To assess this possibility the exact probability values for each 2 x 2 matrix were found, and these two-tailed probabilities converted to one-tailed probabilities (by taking into account the direction of the result found), and these one-tailed probabilities then combined, using the method of Kendall (1951). Even when this has been carried out for the results shown in Table 3:6 the data still show no significant relation between left-

handedness and birth stress, either for males (Chi-squared = 53.31, 56 df, p = 0.577), or for females (Chi-squared = 71.12, 56 df, p = 0.083) or for males and females combined (Chi-squared = 124.45, 112 df, p = 0.2005). Note that in the present data it is in the females in whom the greater effect is found, contrary to the findings of Bakan, who suggests that males are more likely to show such a link between handedness and birth stress.

Looking at the probability values in the first part of Table 3:6 (i.e. for males and females combined) it is apparent that for three items ('Caesarian Section', 'Neonatal convulsions or cyanosis', and 'Other abnormalities of presentation') that the p values reach a conventional significance level of 0.05. If one examines the probabilities for these items for males and females separately, then for the first two items, none of the probabilities within sex are significant, whilst for the third item, only the value for females is significant. Combining the male and female chi-squared results gives Chi-squared values of 5.22, 3.25, and 5.61 respectively, none of which are significant with 2 degrees of freedom. In summary, the three 'significant' results in Table 3:6 cannot be accepted as evidence for a real effect.

Table 3:7 shows similar results to Table 3:6 but is instead for those variables for which more than two possible response categories are available. Chi-squared

values for homogeneity are given for all variables. For those variables which are ordinal in their classification a Kolmogorov-Smirnov two-sample test was also carried out to check for differences in overall distribution between the left-handed and the right-handed children.

The significance values of Table 3:7 may also be combined by addition of Chi-squared values. This has been carried out for the first 14 items (i.e. 'social class' to 'smoking', inclusive) and gives values of Chi-squared = 57.03, df 61, for males and Chi-squared = 52.55, 61 df for females, neither value being statistically different from chance expectations, as neither is the combination of the male and female values (Chi-squared = 109.25, 122 df, NS).

Table 3:8 shows the breakdown of those variables which were cardinal and for which there were a sufficiently large number of possible responses to allow the use of statistics designed for continuous distributions. F-tests of significance have been carried out for all these items; in addition the Kolmogorov-Smirnov two-sample test is also reported in view of the possibility of small degrees of non-normality being confounded by the large sample size to produce spuriously significant or non-significant results.

Several composite indices of the data presented in

Tables 3:6, 3:7 and 3;8 have also been produced. Goldstein (1969) has used the variables age, parity, social class, height, smoking and pre-eclampsia to predict the perinatal mortality rate. All are independent predictors of differing strengths, and after their combined use (with appropriate weighting), a non-significant degree of variance is left unaccounted. Table 3:8 shows my own reconstruction of this variable (called the perinatal mortality predictor scale, PMPS), in left and right-handers. There is no significant difference between the two groups, when a non-parametric test is used (the distribution being strongly non-normal; skewness = 1.22, kurtosis =).

Three other indices have also been constructed, these being simple counts of adverse events occurring during a particular period. The ante-natal risk factor count (ARFC) was a simple count of the following variables:-

- maternal haemoglobin of less than 9 gms/100mls
- placenta praevia with bleeding
- abruptio placentæ with bleeding
- any vaginal bleeding before 28 weeks
- any other vaginal bleeding after 28 weeks
- maternal diabetes
- maternal heart disease
- maternal TB (active)
- hydramnios

Note that none of these items overlap with the items in the PMPS.

The Intra-partum risk factor count (IRFC) was a simple count of the number of events from the following list:-

- Caesarean section
- use of forceps during delivery
- breech presentation
- occipito-posterior presentation
- other abnormality of presentation (face, shoulders, etc.)
-
- umbilical cord prolapse
- meconium staining of amniotic fluid
- fetal distress as indicated by abnormality of fetal heart rate
- other signs of fetal distress

The neonatal risk factor count (NRFC) was a simple count of all adverse events recorded after delivery of the child, i.e.

- rhesus incompatability
- hyperbilirubinaemia
- neonatal convulsions or cyanosis
- neonatal hypothermia
- respiratory distress
- pyloric stenosis

- other miscellaneous neonatal conditions

A composite risk factor count (CRFC) was also constructed by using all the variables already described for the ARFC, IRFC and NRFC, and also adding to this list dichotomised versions of the variables used in the PMPS, as well as several others which might be thought pertinent to the question of birth stress and handedness. The extra variables were:-

- mother's weight greater than 10 stones 13 pounds
- mother's height less than 62 inches
- gestation period less than 36 weeks or greater than 42 weeks
- parity zero or greater than 2
- history of previous spontaneous abortions or ectopic pregnancies
- history of previous premature live births
- history of previous still-births or neonatal deaths
- history of previous toxaemia
- history of previous ante-partum haemmorhage
- history of previous Caesarean section
- first stage of labour longer than 24 hours
- second stage of labour longer than 2 hours
- membrane rupture longer than 48 hours
- mother's age greater than 29 years
- mother smoked after 4th month of pregnancy
- surgical induction of labour carried out
- oxytocin usage during labour

- moderate or severe toxaemia
  (as defined by Butler and Bonham, 1963).

The number of items on each of the scores ARFC, IRFC, NRFC and CRFC was 9, 9, 7 and 43 respectively. Clearly it is not possible to attain these maximum scores since some of the items are mutually exclusive. Results for the ARFC, IRFC and NRFC have already been presented in Table 3:7; none show significant relations with handedness. Table 3:9 shows the distribution of scores for males, females, and both sexes combined, on the composite risk factor score. For none of the groups is there a significant difference between right- and left-handers; this is also reflected in the absence of a difference in means of right- and left-handers.

3:5 Conclusions

In this Chapter I have reported results from two moderately large retrospective studies, and one very large prospective study of the relation between birth complications and subsequent left-handedness. None of the studies produced any substantial evidence for a relation between birth stress and left-handedness.

There seems little doubt that birth stress is unlikely to play any role in the development of left-handedness in the majority of the population. It may be

concluded safely that the incidence of left-handedness need be of no concern to obstetricians anxious to monitor the efficiency of their services.

Table 3:1a shows separately for Surveys I and II, the relation between handedness and a history of birth stress (or birth complications). In the table + indicates a history of birth complications, and - their absence.

Survey I

Birth complications	Handedness Right	Left	N	% Left
-	721	108	829	13.0
+	95	12	107	11.2

$X^2 = 0.14$, 1 df, $p = 0.708$

Survey II

Birth complications	Right	Left	N	% Left
-	803	129	932	13.8
+	262	51	313	16.3

$X^2 = 1.14$, 1 df, $p = 0.285$

TABLE 3:1.b Shows, separately for Surveys I and II and for right and left-handers, the relation between reported birth stress (or complications), and a family history (FH) of sinistrality.

RIGHT-HANDERS

		SURVEY I				SURVEY II			
		FH-	FH+	N	% +	FH-	FH+	N	% +
Birth stress	-	463	55	518	10.6	349	131	480	27.3
	+	252	37	289	12.8	454	131	585	22.4

x^2 = 0.67, 1 df, p = 0.411 x^2 = 3.41, 1 df, p = 0.061

LEFT-HANDERS

		SURVEY I				SURVEY II			
		FH-	FH+	N	% +	FH-	FH+	N	% +
Birth stress	-	25	2	27	7.4	30	17	47	36.2
	+	83	10	93	10.8	99	34	133	25.6

x^2 = 0.02, 1 df, p = 0.884 x^2 = 1.93, 1 df, p = 0.161

TABLE 3:2 Gives details of the five items in the NCDS data set which are concerned with hand preference

NCDS item number	Age at assessment	Survey	Person assessing child	Method of assessment	Item description in data manual	Possible responses
291	7	NCDS I	Mother (structured interview)	From memory	"Laterality (hand) - mother"s report"	-Right-handed -Left-handed -Mixed right and left
373	7	NCDS I	Doctor	Direct examination	"Task: Throw a crumpled paper ball Draw a cross	-Only right hand used -Only left hand used -Both right and left hand
1274	11	NCDS II	Mother (interview)	From memory	"Ask mother if the child is:-	-Left-handed -Right-handed -Mixed right and left
1275	11	NCDS II	Mother (interview)	From memory	"Which hand does your child write with?"	-Left hand -Right hand
1582	11	NCDS II	Doctor	Direct examination	"Ask child to throw ball to you. Did he/she use;-	-Right hand -Left hand

TABLE 3.3 Shows the numbers of children, out of a total of 11029 for whom adequate information was available on all five hand preference items, who gave each particular combination of responses.

		Right	Right	Mixed	Mixed	Left	Left	← item 373
		Right	Left	Right	Left	Right	Left	← item 1275
Item 1274 ↓	Item 291 ↓							item 1582 ↓
Right	Right	7849	1	852	0	31	0	Right
Right	Right	62	0	19	0	3	0	Left
Right	Mixed	338	1	63	1	0	0	Right
Right	Mixed	2	0	8	2	3	1	Left
Right	Left	31	0	12	0	2	0	Right
Right	Left	1	0	1	1	4	3	Left
Mixed	Right	223	3	27	4	2	1	Right
Mixed	Right	10	2	13	0	2	1	Left
Mixed	Mixed	131	6	31	16	1	2	Right
Mixed	Mixed	5	1	22	6	0	14	Left
Mixed	Left	0	4	5	27	1	8	Right
Mixed	Left	1	1	1	7	3	30	Left
Left	Right	13	7	3	7	0	0	Right
Left	Right	0	1	1	7	2	7	Left
Left	Mixed	3	7	2	25	0	9	Right
Left	Mixed	1	2	2	14	0	41	Left
Left	Left	1	21	0	136	0	68	Right
Left	Left	0	16	0	142	0	589	Left

Table 3:4 shows the inter-correlation matrix for the five hand preference items described in Table 3:2.

	1275	1274	291	1582	373
1275	-	0.917	0.861	0.744	0.766
1274	0.917		0.840	0.726	0.728
291	0.861	0.840	-	0.692	0.713
1582	0.744	0.726	0.692	-	0.701
373	0.766	0.728	0.713	0.701	-

Table 3:5 shows the relationship between the single variable derived from factor analysis ('Hand': see Chapter 3:3 for description) and the five simple variables from which it is derived.

		Hand	
		Right	Left
Item 291			
	Right	9113	40
	Mixed	612	145
	Left	63	1056
		Right	Left
Item 373			
	Right	8672	72
	Both	1062	385
	Left	54	774
		Right	Left
Item 1274			
	Right	9282	9
	Mixed	478	133
	Left	28	1099
		Right	Left
Item 1275			
	Right	9787	0
	Left	1	1241
		Right	Left
Item 1582			
	Right	9622	356
	Left	166	885

TABLE 3;6 Shows, for those variables which can be regarded as dichotomous, the proportion of those children with and without the condition, who are left-handed. Chi-squared values use Yate's correction for continuity, and probability values are two-tailed.

		Males Only						Females Only					
ondition	NCDS code number	condition absent N	%L	condition present N	%L	Chi^2	p (two tailed)	condition absent N	%L	condition present N	%L	Chi^2	p (two tailed)
hesus negative blood group	518	5050	12.8	941	13.6	0.352	0.552	4778	9.5	940	9.5	0.000	0.991
bruptio placentae	522	6815	12.7	8	12.5	0.263	0.607	6483	9.5	8	12.5	0.097	0.755
leeding per vaginam rior to 28 weeks	522	6627	12.7	196	12.8	0.007	0.929	6273	9.4	218	11.9	1.299	0.254
lacenta praevia	522	6805	12.7	18	27.8	2.458	0.117	6454	9.5	37	13.5	0.313	0.575
ther bleeding in pregnancy	522	6696	12.7	127	15.0	0.403	0.525	6354	9.4	137	10.9	0.200	0.654
aesarean Section	534	6637	12.6	186	16.1	1.713	0.190	6344	9.4	147	14.3	3.505	0.061
orceps	534	6494	12.7	329	12.8	0.002	0.958	6253	9.5	238	10.1	0.045	0.830
reech presentation	534	6741	12.7	82	14.6	0.129	0.718	6370	9.5	121	9.1	0.000	0.991
ciput-Posterior resentation	534	6678	12.7	145	11.7	0.054	0.815	6335	9.5	156	9.0	0.006	0.938
her abnormality of pres-itation(face,shoulders,etc.)	534	6803	12.7	20	15.0	0.001	0.978	6468	9.4	23	26.1	5.610	0.018
bilical cord prolapse	535	6488	12.8	8	12.5	0.251	0.615	6164	9.5	9	11.1	0.160	0.688
conium staining of liotic fluid	535	6388	12.8	108	13.9	0.035	0.850	6087	9.4	86	12.8	0.769	0.360
al heart distress	535	6306	12.8	190	12.6	0.001	0.976	6048	9.4	125	11.2	0.267	0.605
ier signs of fetal stress	535	6134	12.8	362	13.3	0.031	0.861	5870	9.4	303	11.2	0.947	0.330
esus incompatability	1831	6800	12.7	23	13.0	0.070	0.791	6451	9.4	40	15.0	0.857	0.354
onatal hyper-bilirubin-nia	1831	6814	12.7	9	22.2	0.127	0.721	6482	9.5	9	0.0	0.161	0.687
Neonatal convulsions or	1831	6792	12.7	41	19.5	1.160	0.281						

esentation	534	6678	12.7	145	11.7	0.054	0.815	6335	9.5	156	9.0	0.006	0.938
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conium staining of niotic fluid	535	6388	12.8	108	13.9	0.035	0.850	6087	9.4	86	12.8	0.769	0.360
tal heart distress	535	6306	12.8	190	12.6	0.001	0.976	6048	9.4	125	11.2	0.267	0.605
her signs of fetal stress	535	6134	12.8	362	13.3	0.031	0.861	5870	9.4	303	11.2	0.947	0.330
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onatal hyper-bilirubinmia	1831	6814	12.7	9	22.2	0.127	0.721	6482	9.5	9	0.0	0.161	0.687
Neonatal convulsions or cyanosis	1831	6782	12.7	41	19.5	1.160	0.281	6473	9.4	18	22.2	2.091	0.148
Neonatal hypothermia	1831	6816	12.7	7	14.3	0.195	0.658	6487	9.5	4	25.0	0.042	0.836
Respiratory Distress	1831	6813	12.7	10	0.0	0.536	0.464	6482	9.5	9	11.1	0.161	0.687
Pyloric stenosis	1831	6803	12.7	20	5.0	0.490	0.483	6480	9.5	11	0.0	0.312	0.567
Other miscellaneous neonatal problems	1831	6782	12.8	41	4.9	1.624	0.202	6454	9.5	37	13.5	0.313	0.577
Maternal diabetes	1837	6817	12.7	6	16.7	0.103	0.747	6484	9.5	7	14.3	0.044	0.833
Maternal heart disease	1837	6769	12.7	54	11.1	0.022	0.822	6440	9.5	51	3.9	1.253	0.262
Hydramnios	1837	6807	12.7	16	12.5	0.123	0.725	6475	9.5	16	12.5	0.000	0.989
Maternal tuberculosis	1837	6812	12.7	11	9.1	0.008	0.926	6470	9.5	21	14.3	0.145	0.703
History of previous toxaemia	509	6076	12.7	418	14.8	1.420	0.233	5802	9.5	368	9.5	0.003	0.951
History of previous ante-partum haemmorhage	509	6346	12.8	148	12.8	0.014	0.904	6038	9.4	132	11.4	0.363	0.546
History of previous Caesarean Section	509	6413	12.8	81	13.6	0.001	0.970	6103	9.5	67	6.0	0.597	0.439

Table 3:7 Shows, for those variables which have three or more categories, the proportion of each class which are left-handed. Chi-squared values are given for all items= Kolmogorov-Smirnov two-sample tests are reported for items which can be regarded as ordinal in structure.

Condition	NCDS code number	group	males N	males %L	males Chi^2 test
Social class	492	I	272	12.9	Chi^2 = 2.07
		II	824	11.5	4 df
		III	3737	13.0	p = 0.721
		IV	755	13.6	
		V	607	13.7	
Parity	504	0	2336	12.4	Chi^2 = 5.38
		1	2105	13.2	6 df
		3	1002	13.9	p = 0.496
		4	241	10.0	
		5	127	16.5	
		6+	175	12.6	
Previous abortions, or ectopic pregnancies	505	0	5740	12.9	Chi^2 = 3.06
		1	616	13.0	2 df
		2	103	7.8	p = 0.215
		3+	36	8.3	
Previous premature live births	506	0	5938	12.7	Chi^2 =0.09
		1	415	13.3	2 df
		2	76	11.8	pp = 0.951
		3+	24	16.6	
Previous still births	508	0	6147	12.9	Chi^2 = 1.40
		1	311	11.3	2 df
		2+	36	8.3	p = 0.496
Mother's minimum haemoglobin level (gms/100mls)	519	10.5+	3595	12.3	Chi^2 = 0.76
		9.0-10.4	495	13.3	2 df
		<9.0	113	10.6	p = 0.683
Length of first stage of labour (hours)	527	<3	600	14.8	Chi^2 = 7.93
		3-6	1458	12.7	6 df
		6-12	2128	12.6	p = 0.242
		12-24	1498	12.4	
		24-48	472	10.8	
		48-72	72	18.1	
		>72	22	22.7	
Length of second stage of labour (minutes)	528	<30	2977	13.9	Chi^2 = 12.93
		30-60	1720	11.6	8 df
		60-90	736	11.8	p = 0.114
		90-120	338	10.4	
		120-150	189	12.7	
		150-180	75	13.3	

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	— females —			
Chi^2 test	Kolmogorov-Smirnov test	N	%L	Chi^2 test	Kolmogorov-Smirnov test
Chi^2 = 2.07 4 df p = 0.721	K-S z = 0.437 NS	248 789 3544 744 536	9.7 8.0 9.7 8.9 9.1	Chi^2 = 2.61 4 df p = 0.623	K-S z = 0.439 NS
Chi^2 = 5.38 6 f p = 0.496	K-S z = 0.356 NS	2289 1886 973 480 238 153 154	8.8 9.8 8.5 7.9 10.9 7.8 10.4	Chi^2 = 4.08 6 df p = 0.644	K-S z = 0.610 NS
Chi^2 = 3.06 2 df p = 0.215	K-S z = 0.215 NS	5450 582 110 31	9.5 9.3 8.2 9.6	Chi^2 = 0.18 2 df p = 0.912	K-S z = 0.104 NS
Chi^2 =0.09 2 df pp = 0.951	-S z = 0.083 NS	5629 406 69 25	9.3 9.9 11.6 22.2	Chi^2 = 2.25 2 df p = 0.323	K-S z = 0.323 NS
Chi^2 = 1.40 2 df p = 0.496	K-S z = 0.242 NS	5842 300 27	9.5 8.3 22.2	Chi^2 = 5.57 2 df p = 0.061	K-S z = 0.150 NS
Chi^2 = 0.76 2 df p = 0.683	K-S z = 0.683 NS	3402 467 94	9.5 10.5 4.3	Chi^2 = 3.55 2 df p = 0.169	K-S z = 0.266 NS
Chi^2 = 7.93 6 df p = 0.242	K-S z = 0.474 NS	565 1325 2095 1439 433 48 17	8.3 10.3 8.4 10.1 10.6 10.4 17.6	Chi^2 = 7.7 6 df p = 0.255	K-S z = 0.733 NS
Chi^2 = 12.93 8 df p = 0.114	K-S z = 1.376 p = 0.045	2982 1601 669 266 159 70 31 15	9.7 9.2 8.2 9.4 10.7 7.1 16.1 6.7	Chi^2 = 4.51 8 df p = 0.989	K-S z = 0.445 NS

						K-S z = 1.13,				
						NS	484	9.9	8 df	NS
rupture (hours)		<3	3860	13.4	Chi^2 = 14.26		471	8.7	p = 0.903	
		3-6	603	10.6	8 df		428	8.9		
		6-12	565	11.3	p = 0.075		281	10.0		
		12-24	462	10.0			93	11.8		
		24-48	303	10.9			66	12.0		
		48-72	105	16.2			12	0.0		
		72-120	73	12.3			18	5.6		
		120-168	23	21.7						
		>168	21	23.8						
							231	8.2	Chi^2 = 5.09	
Inhalational anaesthesia	536	Not available	223	11.7	Chi^2 = 5.08		3357	8.9	8 df	
		Gas & Air	3548	12.9	8 df		1389	10.0	p = 0.747	
		Trilene	1461	12.0	p = 0.748		152	11.2		
		Gas, Air & Trilene	136	14.0			14	7.1		
		Gas & Oxygen	14	7.1			497	10.9		
		Not indicated	537	15.1			152	10.5		
		Contra-indicated	169	11.8			166	11.4		
		No time	160	15.0			205	10.2		
		Refused	227	11.9						
							5785	9.6	Chi^2 = 3.04	
Surginal induction	531	None	6054	12.7	Chi^2 = 0.820		262	8.0	4 df	
		Low	276	13.0	4 df		132	9.8	p = 0.550	
		High	150	14.7	p = 0.935		202	6.4		
		Yes, type NK	207	11.6			110	10.0		
		OBE, oestrogen or strip	136	13.2						
							6186	9.6	Chi^2 = 0.97	
Oxytocin induction	531	None	6483	12.7	Chi^2 = 0.30		291	7.9	2 df	
		Yes	307	13.4	2 df		14	7.1	p = 0.615	
		Only in labour	33	15.2	p = 0.858	K-S z = 0.759 NS	4711	9.5	Chi^2 = 7.84	K-S z = 0.282 NS
							1079	8.8	4 df	
							254	9.4	p = 0.0973	
Toxaemia	548	None	4833	13.1	Chi^2 = 3.02		222	14.4		
		Mild	1154	11.4	4 df		225	7.6		
		Moderate	280	12.1	p = 0.554					
		Severe	259	12.0		K-S z = 0.245 NS	4082	9.2	Chi^2 = 1.25	K-S z = 0.344
		Proteinuria (?cause)	297	12.1			955	10.4	3 df	NS
							711	9.1	p = 0.739	
							351	9.7		
Smoking after 4th month of pregnancy	639	None	4285	12.7	Chi^2 = 1.27					
		Medium	983	13.9	3 df	K-S z = 0.090	3509	9.4	Chi^2 = 1.48	K-S z = 0.16
		Heavy	754	12.3	p = 0.734		403	10.7	2 df	NS
		Variable	395	12.9			48	6.3	p = 0.477	
							3	0.0		
Number of antenatal problems (ARFC)		0	3785	12.4	Chi^2 = 1.19					
		1	385	11.7	2 df	K-S z = 0.296 NS	4823	9.4	Chi^2 = 1.69	K-S z = 0.24
		2	33	19.4	p = 0.550		659	9.1	5 df	NS
		3	2	0.0			560	10.5	p = 0.889	
							90	8.9		
Number of intra-partum problems (IRFC)		0	5023	12.8	Chi^2 = 1.17		35	11.4		
		1	654	11.9	5 df		6	0.0		
		2	630	13.8	p = 0.947					
		3	138	13.8		K-S z = 0.079 NS	6371	9.4	Chi^2 = 2.12	K-S z = 0.1
		4	44	13.6			112	13.4	2 df	NS
		5	7	14.3			8	12.5	p = 0.344	
Number of neo-natal		0	6680	12.7	Chi^2 = 0.316					

TABLE 3:8 Shows for those variables which are continuous in their distribution, the mean, standard deviation, and sample number for left- and right-handers. F-values are given for a one-way analysis of variance, and in view of possible non-normalities being confounded by the large sample size, a Kolmogorov-Smirnov two-sample test is also reported.

		Males (Mean / sd / N)		
Variable	number	right handers	left handers	F-test
Husband's age	494	30.53 6.49 5484	30.14 6.21 811	F(1,6293)=2.55 p = 0.110
Mother's age	553	27.47 5.70 5661	27.16 5.34 832	F(1,6491)=2.11 p = 0.146
Mother's height (inches)	510	63.39 2.50 5440	63.43 2.52 806	F(1,6244)=0.16 p = 0.687
Mother's weight (stones)	496	8.87 1.50 5529	8.84 1.46 811	F(1,6338)=0.291 p = 0.589
Gestation period (days)	497	280.9 11.81 5123	280.4 11.99 744	F(1,5865)=1.15 p = 0.639
Birth weight (ounces)	646	120.8 18.42 5467	119.5 22.45 794	F(1,6259)=3.57 p = 0.058
Perinatal mortality predictor scale (PMPS		-476.4 208.6 5385	-478.1 196.0 798	F(1,6181)=0.047 p = 0.828

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Kolmogorov Smirnov test	Females (Mean / sd / N) right handers	Females (Mean / sd / N) left handers	F-test	Kolmogorov Smirnov test
K-S z = 0.903 NS	30.63 6.32 5381	30.75 6.78 555	F(1,5934)=0.186 p = 0.665	K-S z = 0.578 NS
K-S z = 0.822 NS	27.50 5.67 5587	27.41 6.01 33	F(1,6168) =0.143 p = 0.705	K-S z = 0.894 NS
K-S z = 0.293 NS	63.42 2.50 5385	63.40 2.47 556	F(1,5939)=0.039 p = 0.843	K-S z = 0.363 NS
K-S z = 0.032 NS	8.85 1.48 5460	8.84 1.57 572	F(1,6030)=0.020 p = 0.88	K-S z = 0.606 NS
K-S z = 0.743 NS	281.6 11.92 4999	281.2 12.39 515	F(1,5512)=0.625 p = 0.793	K-S z = 0.649 NS
K-S z = 1.433 p = 0.033	115.9 17.45 5418	115.1 18.22 572	F(1,5988)=1.283 p = 0.699	K-S z = 0.699 NS
K-S z = 0.740 NS	-479.8 203.4 5308	-463.3 225.9 545	F(1,5851)=3.205 p = 0.073	K-S z = 0.977 NS

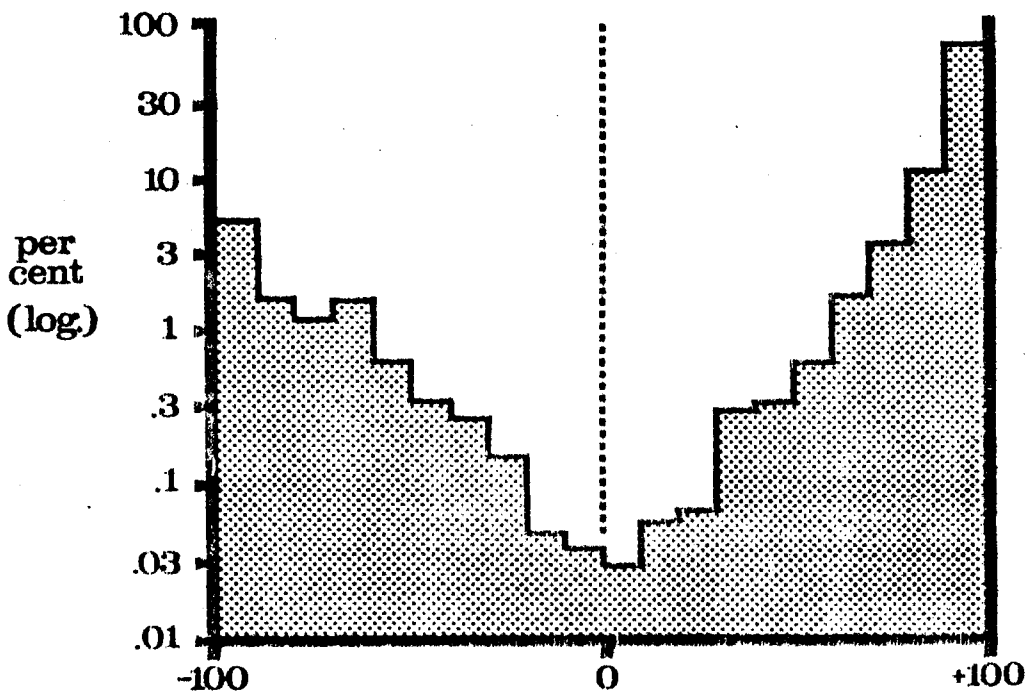
Table 3.9 Shows, for males and females ~~combined~~ the number of adverse events reported during pregnancy and the neonatal period by handedness. The results of Chi-squared test for homogeneity, and of a Kolmogorov-Smirnov two-sample test are reported, non-parametric tests being used in view of the clear non-normality of the data.

	males		females	
n(adverse events)	N	%L	N	%L
0	764	13.6	733	7.9
1	1272	13.1	1256	9.7
2	1235	11.5	1156	9.5
3	956	13.1	852	10.6
4	678	11.5	633	9.3
5	413	14.5	385	8.6
6	258	9.3	230	9.1
7	144	15.3	143	10.5
8	86	10.8	73	4.1
9	45	17.8	39	17.9
10	24	8.3	27	18.5
11	10	0.0	5	0.0
12	4	0.0	1	0.0
13	1	0.0	1	0.0
Mean (right)	2.622		2.567	
Mean (left)	2.624		2.640	
SD (right)	2.068		2.052	
SD (left)	2.112		2.073	
Chi^2	12.98		15.62	
df	13		13	
p	0.449		0.269	
K-S z	0.518 NS		0.448 NS	

Figure 3.1 Shows the distribution of the first factor of handedness (standardised to a range of +100 to -100) of 11028 children from the National Child Development Study. Note the logarithmic scale of the ordinate.

NCDS: Handedness

n=11029



CHAPTER 4: HANDEDNESS IN TWINS

Summary

The incidence of left-handedness is the same in MZ twins, DZ twins, and singletons. The proportions of R-R, R-L and L-L pairs in MZ twins are not in a binomial distribution. Twins have not been shown to be subject to any special factors modifying their handedness and are thus suitable for genetic analysis.

4:1 Introduction

Twins, it is usually claimed, are more likely to be left-handed than are singletons, and monozygotic (MZ) twins are more likely to be left-handed than dizygotic (DZ) twins (Nagylaki and Levy, 1973; Corballis and Beale 1976). It is also claimed that the distribution of concordant and discordant MZ handedness pairs approximates to that of a binomial distribution (Collins, 1970). In this chapter I would like to suggest that none of these assertions is supported by adequate evidence, the published evidence being unsatisfactory. I shall consider the items in the reverse order to that just given.

4:2 The distribution of pair-types in MZ and DZ twins

Twin data have represented the Achilles heel of genetic models of handedness, the lack of any difference in concordance between MZ and DZ twins forcing Corballis and Beale (1976) to suggest, and Collins (1970) to directly claim, that R-R, R-L and L-L pairs, in both MZ and DZ twins, were present in binomial proportions, and hence to infer that there could be no genetic control over handedness. Similar conclusions have been made for the genetic control of hand-clasping (Martin, 1975), and have been shown to be false, since some genetic models predict very low degrees of concordance (see Chapters 7, 8 and 9).

Collins (1970) added together the results of several large studies of twin handedness and concluded that the twin pairs were present in binomial proportions. Nagylaki and Levy (1973) demonstrated that Collins' results were invalid, but in so doing they made a further error, of adding together data which are not capable of addition. It is not statistically valid to combine data from different studies unless the incidence of left-handedness is similar in the individual studies. Table 4.1 shows data on twin pairs from 19 studies of MZ twins, and 18 studies of DZ twins. There are highly significant differences in the incidence of left-handedness between studies, both for MZ twins (X^2 = 73.26, 18 df, $p < 0.001$) and for DZ twins (X^2 = 53.32, 17 df, $p < 0.001$). The differing incidence of left handedness in the separate studies implies that the criterion for left-handedness is different between studies: hence the data within a study cannot be regarded as strictly independent, and hence <u>data</u> from different studies may not be summated, although <u>significance levels</u> may be (Lewis and Burke, 1949.)

Differences between studies are partly due to an interesting difference between the large and small studies (a large study being defined as one in which there are both more than 60 MZ pairs and more than 60 DZ pairs). In both MZ and DZ twins there is a tendency for there to be more left-handers reported in small studies than in large studies, although this effect is only significant

in MZ twins (MZ twins $p < 0.05$, Mann-Whitney U test). Nevertheless even when one considers only the large studies ($n = 10$) there are still differences in the proportions of left-handers in the different studies (MZ twins $X^2 = 35.0$, 9 df, $p < 0.001$; DZ twins $X^2 = 30.28$, 9 df, $p < 0.001$).

In order to test whether data are fitted by a binomial distribution it is therefore necessary to test each data set separately, and then to combine the X^2 goodness of fit values. Figure 4.1 shows data from the ten large MZ studies, showing the incidence of R-L and L-L pairs. In 9 of the 10 cases there are less R-L pairs than would be expected by a binomial distribution, and in the same cases there are more L-L pairs than would be expected by a chance distribution (exact binomial probability = 0.0107). Table 4.1 shows, in the columns marked X^2 Binom., for both MZ and DZ twins, the individual X^2 values for the goodness of fit of a binomial distribution. For MZ twins three of the studies are significantly different from a binomial distribution in their own right; together the 19 MZ studies have a X^2 value of 30.30, which with 18 df gives $p < 0.05$ for a binomial fit; for just the large studies $X^2 = 24.27$, with 10 df, for which P is less than 0.001. We may therefore accept Nagylaki and Levy's conclusion that MZ twin pairs do not occur in binomial proportions, even though their own evidence on this point was insufficient. Unlike MZ twins, the proportions

of DZ pairs do not differ significantly from the predictions of a binomial distribution, the X^2 value for all the studies being 15.63 (17 df), and for the large studies alone being 11.26 (10 df, NS). A genetic explanation of handedness should not be ruled out merely because the DZ pairs do not differ significantly from binomial distributions; the genetic explanation merely has the constraint that for DZ twins its predictions should be close to those of a binomial distribution.

It might be argued that the small difference between MZ and DZ twins is not good evidence for a genetic component in handedness. Far stronger evidence would be the incidence of the three types of handedness pair in MZ and DZ twins from parents of known handedness. There are no adequate data of this form in the literature, the only approximation being that of Rife (1950), which are shown in Table 4.2. There is a far higher proportion of L-L pairs amongst those MZ twins with a family history of left-handedness than in those without a history of left-handedness.

4:3 The difference in incidence of left-handedness between MZ and DZ twins

Nineteen studies in the literature have analysed the incidence of left-handedness in twins (including the study of Weitz (1924) who looked only at MZ twins). The

study of Shields (1962) is difficult to interpret, since the author gives incomplete information regarding the six "ambidexters", and thus I have here only considered the overall proportions of right and left-handers and have omitted the twin-pair from the analysis.

Overall 15.09% of 5140 MZ twins showed left-handedness as compared with 12.80% of the 4436 DZ twins. It is not however permissible to combine data from studies in this way, since the incidence of left-handedness is clearly different between studies (see above). It is therefore necessary to look at each study separately, and to combine results only after analysis. Figure 4.2 shows the incidence of left-handedness in MZ and DZ twins in 18 studies. In only 11 of the 18 studies is the incidence greater in MZ twins than in DZ twins. Furthermore the correlation between MZ and DZ incidences is only 0.297, which is not significantly different from zero. If one excludes the eight 'small' studies (defined as above), the correlation is still only 0.333, which is also not significantly different from zero. Even if the differences in incidence in MZ and DZ twins were zero we might still expect a correlation between the two incidences. Since the value is not significant we may infer that there are systematic differences in measurement within studies as well as between them.

Table 4.1 shows, for each study, the incidence of

left-handedness in MZ and DZ twins. In the column marked 'Difference' there is a '+' if the incidence is higher in MZ twins than in DZ twins, and '-' if the incidence is higher in DZ twins. Of the 18 studies, 7 found the MZ twins to have a lower incidence of left-handedness. The column marked 'X^2' gives the X^2 value for a homogeneity test for a difference between MZ and DZ pairs in either direction, the probabilities being given in the column marked 'Two-tailed'. Only 4 studies show significant differences, and one of these (Lauterbach, 1925) shows the difference in the opposite direction to that expected. Note also that all of the significant results were obtained before 1930 when we may assume that determination of zygosity was rudimentary, and when we know that both Newman (1928; 1940) and Dahlberg (1926) felt that symmetry reversal in twin pairs was actually pathognomic of monozygosity. The data of Table 4.1 may be combined to give an overall probability value for a difference between MZ and DZ twins. The column marked 'One-tailed' gives the probability of a significant difference in the direction of MZ twins having a higher incidence of left-handedness than DZ twins (calculated by taking the two-tailed probability, p_2, and calculating $p_1 = p_2/2$ if MZ is greater than DZ, or $p_1 = 1 - (p_2/2)$ if DZ is greater than MZ). These one-tailed probabilities may be combined using the method of Kendall (1951) (see also Jones and Fiske 1953 for further details). Overall the differences between MZ and DZ twins are significant ($X^2 = 70.28$, 36 df,

$p < 0.001$). However if one divides the data according to date of publication, then for studies produced up to and including 1930, the difference between MZ and DZ is highly significant ($X^2 = 34.86$, 12 df, $p < 0.001$), whilst for the periods 1931-1945, 1946-1960 and 1961-1976, the differences are not significant, either separately ($X^2 = 14.86$, 10 df, NS; $X^2 = 12.26$, 8 df, NS; and $X^2 = 8.30$, 6 df, NS respectively) or combined ($X^2 = 35.42$, 24 df, NS).

In summary the only evidence in favour of MZ twins having a higher incidence of left-handedness than DZ twins was obtained prior to 1930, when we may assume that classification of laterality was not entirely independent of zygosity determination, due to theoretical prior conceptions about the nature of mirror-imaging.

It should be noticed that the demonstration of differences between pre- and post-1930 studies does not invalidate earlier conclusions about the non-binomiality of MZ twins. Thus, in the post-1930 studies there is a significant departure from binomiality ($X^2 = 17.56$, 7 df, $p < 0.02$).

4:4 Handedness in twins and singletons

Nagylaki and Levy (1973) claim that the incidence of left-handedness in twins is higher than it is in

singletons. Their evidence for this consists of a comparison between published studies of twins and singletons. However, in none of these cases is evidence quoted of twins and singletons assessed by the same criteria, in the same study, by the same investigators. In view of the wide differences in reported incidences of left-handedness in MZ twins, and also in DZ twins (See earlier) it is clearly fallacious to compare different studies in such a manner.

There are almost no studies in the literature in which twin and singleton handedness have been assessed by the same method. A notable exception is Zazzo (1960) who comments (p. 124) "Pour ma part, je n'ai jamais trouvé, dans mes multiple recherches sur les jumeaux, cet excès de gaucheurs dont parlent tous les auteurs", and he supports this statement with statistical evidence (his Table 8). Wilson and Jones (1932) found no difference in usage of the left hand for <u>writing</u> in twins or singletons, but did find a small difference in ball throwing.

In summary, there is no adequate evidence for the claim of Nagylaki and Levy. It is perhaps worth suggesting that any further studies on this topic should be carried out either by questionnaire, or preferably, by personal examination, with the experimenter blind to the twin status of each child, who should be examined individually not in pairs.

4:5 Handedness in twins: Secular trends

Despite the problems of twin data, we might expect that the handedness of twins has been rather more accurately determined than that of singletons in large population surveys. Thus, the probability of a true left-handed twin being recorded as a right-hander would be low, although the probability of a right-hander being recorded as a left-hander would be concomitantly higher, due to a tendency towards over-inclusive definitions of left-handedness (e.g. Newman et al, 1937, sometimes relied entirely on asymmetry of finger-tapping as evidence of left-handedness). Figure 4.3 shows the incidence of left-handedness in the 18 studies (MZ and DZ studies combined). Only the large studies have standard error bars. Note the tendency for small studies to have higher incidences of left-handedness than large studies (Mann-Whitney U test, $p < 0.05$). There is a hint that the incidence of left-handedness is increasing, although this trend is not significant. The relatively low value reported by Loehlin and Nichol (1976) might be a result of their use of a postal questionnaire rather than personal examination, as used by most of the other studies.

4:6 Discussion

Evidence submitted here suggests that the incidence of left-handedness in both MZ and DZ twins, is the same as that in singletons. This removes the objection of Nagylaki

and Levy (1973) "that it is impossible to assess the heritability of a trait by using twin data if the frequency of the trait among twins differs from that among non-twins".

Nevertheless Nagylaki and Levy provide several other reasons why twins should have an increased incidence of left-handedness, and these must be considered, for if valid these reasons will put us in the awkward position of positing a true incidence of left-handedness which, if it were not for pathological factors, would be lower in twins than in singletons. Two main suggestions were made:-

" ... prenatal pathogenic factors are responsible for the high frequency of sinistrality in twins ..." There seems little doubt that twins, both MZ and DZ, suffer an increased intra-uterine mortality and morbidity, as well as increased perinatal death-rates (Anon, 1977, passim) and, as Nagylaki and Levy point out, this could be responsible for decreased IQ, and other mental sub-normalities. However these facts do not bear upon the main issue, since these associates have not been shown to relate causally to left-handedness, and until they have been, they need not concern us. The particular hypothesis of left-handedness being secondary to acute anoxia during delivery (Bakan, 1971) has received little further support (Hubbard, 1971; Schwartz, 1977; Hicks et al, 1978).

" ... some cases of handedness discordance in one-egg twins derive from asymmetry reversal". Asymmetry reversal, or mirror-imaging, has been proposed to account for the discordance of handedness, hair-whorling and dermatoglyphics. It is a much quoted concept for which there is no adequate published evidence. There would be little problem in demonstrating its existence if, say, all singletons were of phenotype R, and all MZ pairs were of type R-R or R-L, with none of type L-L, for then mirror-imaging would be indisputable. In the presence of a high singleton incidence of type L, and relatively high frequencies of L-L twin pairs, to demonstrate true mirror-imaging is a statistical problem of some complexity requiring rejection of a null hypothesis. This has never been done satisfactorily, Nagylaki and Levy (1973) quote Newman (1916) on the quadruplets of the nine-banded armadillo, as evidence of ectodermal mirror-imaging. However, Newman himself said that his data was so complex that he did not know how to analyse it, and he certainly gives no formal statistics, (Newman 1916). In the absence of a statement of chance probabilities his assertions of apparent mirror-imaging are difficult to assess.

In the case of hair-whorling, Morgan (1976) has demonstrated that the work of Bernstein (1925) is dubious, and therefore Rife's observations of a binomial distribution of hair-whorling in twins actually refutes the possibility of mirror-imaging, since binomiality is surely the null

hypothesis to be rejected in such a case. Collins (1977) has shown there to be no evidence for hair-whorling mirror-imaging in twins.

There is no adequate evidence for mirror-imaging in dermatoglyphics (Newman 1930; Rife and Cummins, 1943); neither is there evidence for mirror-imaging in tooth cusps, another ectodermal derivative (Staley and Green, 1974; Potter and Nance, 1976).

Nagylaki and Levy (1973) accept that there is no evidence for mirror-imaging of the viscera, or other non-ectodermal structures.

One may only concur with Bulmer (1970) who commented that mirror-imaging "does not occur more frequently than would be expected by chance".

We may conclude, in the absence of other evidence, that MZ and DZ twins are suitable for testing genetic models of handedness, not being subject to any special influences which may be demonstrated statistically. An important consequence of this conclusion is that any genetic model has to explain a significant but non-binomial degree of discordance in MZ twins. As Corballis and Beale (1976) have clearly shown, the model of Nagylaki and Levy (1972) cannot cope with such a possibility, and it must therefore be regarded as inadequate.

TABLE 4.1

Study	Size designation	N_{MZ}	$\%L_{MZ}$	N_{DZ}	$\%L_{DZ}$	Difference	X^2_{MZ-DZ}	Two-tailed	One-tailed	X_{MZ}	X_{DZ}
Siemens (1924)	Small	37	16.21	31	27.41	-	2.52	0.108	0.946	0.00	0.09
Weitz (1924)	Small	18	25.00							0.02	-
Lauterbach (1925)	Large	75	5.33	126	12.69	-	5.69	0.016	0.991	0.24	0.0(
Dahlberg (1926)	Large	69	14.49	128	7.03	+	5.72	0.015	0.992	6.14	0.2
Verschuer (1927)	Large	244	20.28	278	14.04	+	5.51	0.018	0.009	0.14	7.7
Newman (1928)	Small	50	31.00	50	17.00	+	5.37	0.019	0.009	0.62	0.3
Hirsch (1930)	Small	43	20.93	58	6.03	+	2.90	0.084	0.042	3.01	0.24
Wilson & Jones (1932)	Large	70	10.71	123	11.38	-	0.04	0.832	0.583	0.06	0.13
Stocks (1933)	Small	42	9.52	94	10.63	-	0.08	0.776	0.611	1.23	1.03
Newman et al (1937)	Small	50	19.00	50	11.00	+	2.51	0.108	0.054	1.21	0.76
Bouterwek (1938)	Small	122	18.85	35	17.14	+	0.105	0.744	0.372	0.04	1.50
Rife (1940)	Large	223	11.88	146	15.41	-	1.91	0.163	0.918	3.32	0.09
Thyss (1946)	Large	103	18.44	86	16.27	+	0.305	0.587	0.293	5.24	0.05
Rife (1950)	Large	343	12.82	211	11.61	+	0.356	0.557	0.278	0.03	0.32
Dechaume (1957)	Small	33	24.24	33	19.69	+	0.397	0.535	0.267	0.00	0.44
Zazzo (1960)	Large	259	13.32	335	10.89	+	1.63	0.198	0.099	5.62	1.24
Shields (1962)	Small	88	10.23	25	12.00	-	0.129	0.720	0.639	-	-
Carter-Saltzmann (1976) et al.	Large	187	17.11	176	19.31	-	0.59	0.552	0.723	3.30	0.04
Loehlin & Nichols (1976)	Large	514	14.10	333	11.11	+	3.21	0.069	0.034	0.08	1.37
Total		2570	15.09	2218	12.80						

TABLE 4.2

Data of Rife (1950) showing the effect of a family history of left-handedness upon the handedness pairs of MZ and DZ twins.

Twin type	Family History	R-R	R-L	L-L	n(pairs)
MZ	+	56.41	41.02	2.56	78
MZ	-	81.88	16.60	1.50	265
DZ	+	54.16	43.75	2.08	48
DZ	-	84.66	14.72	0.61	163

$\underline{X}^2$ MZ: $X^2 = 21.74$, 2 df, $p < 0.01$

DZ: $X^2 = 17.28$, 2 df, $p < 0.01$

Figure 4.1 Shows, for the ten large studies of monozygotic twins, the percentage of pairs that were L-L (open squares), and the percentage that were R-L (solid squares). All points are ± 1 standard error. The abscissa shows the overall proportion of left-handers in the sample. The solid lines indicate the expected values under a binomial hypothesis.

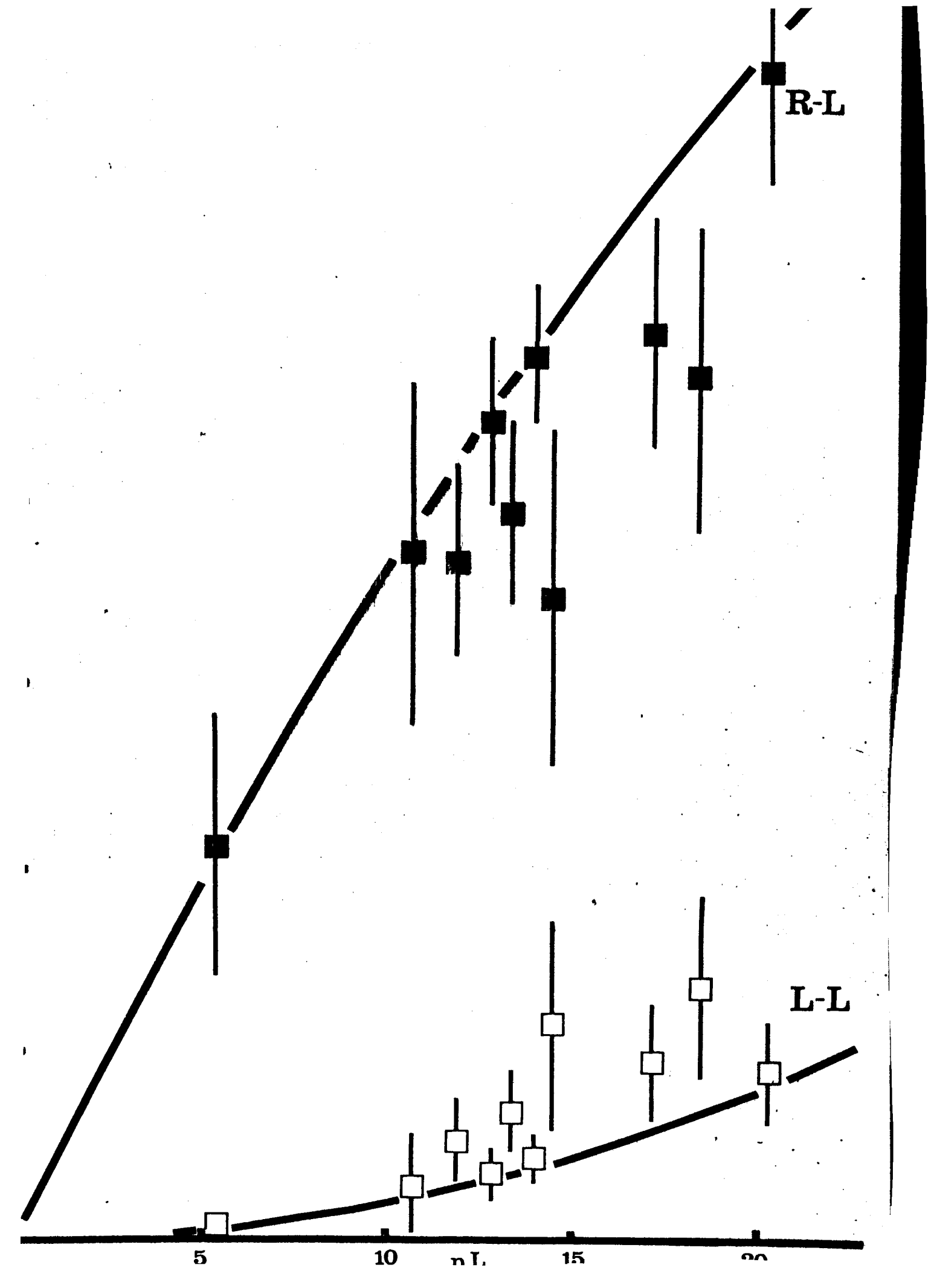

R-L
L-L
5
10
15

Figure 4.2 Shows, for 18 separate studies, the percentage of left-handers in dizygotic twins (abscissa), and in monozygotic twins (ordinate). Solid circles are the 10 'large' studies, and open circles are the eight 'small' studies. The dotted line at 45 degrees represents the point of equality for MZ and DZ twins.

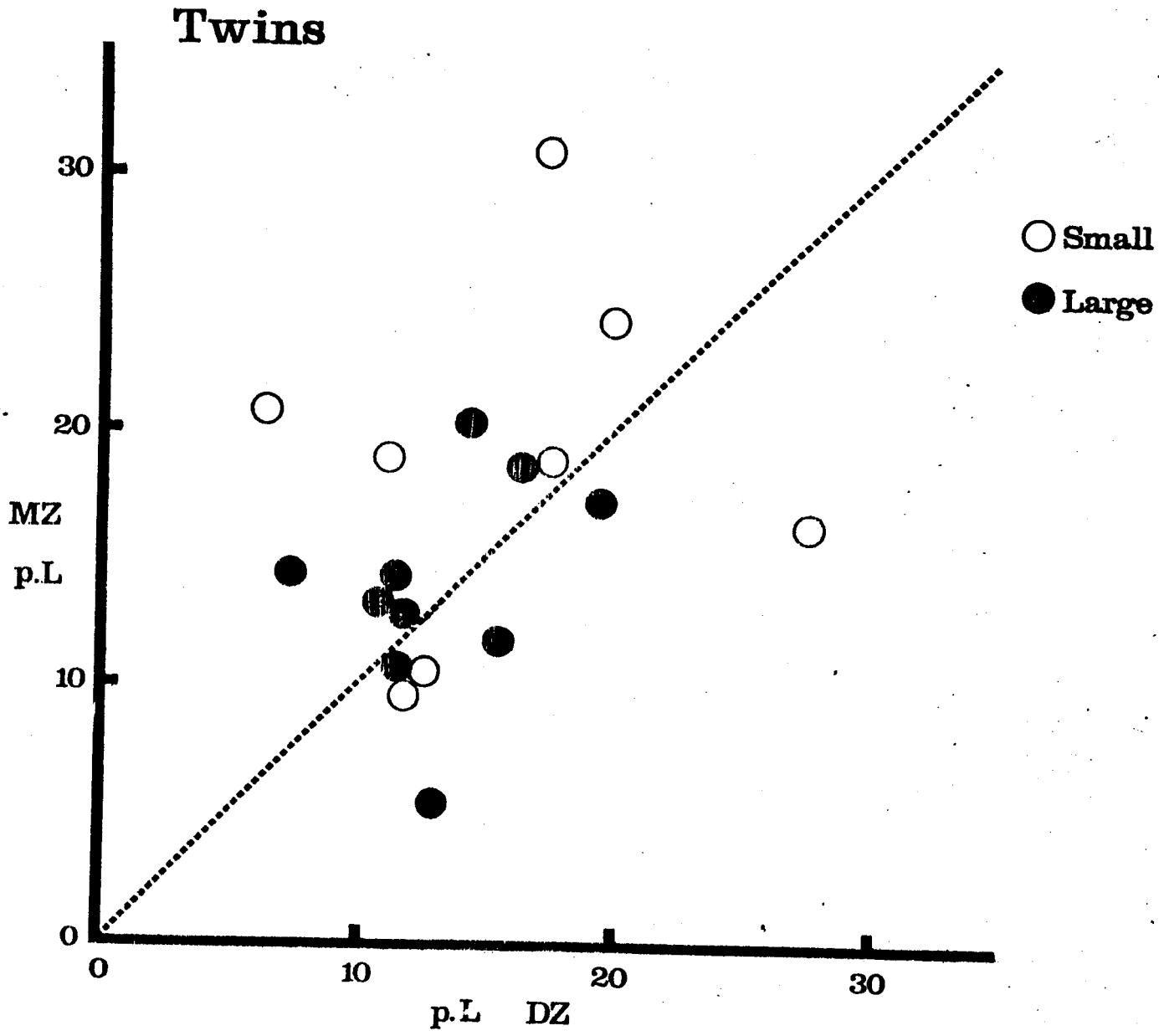
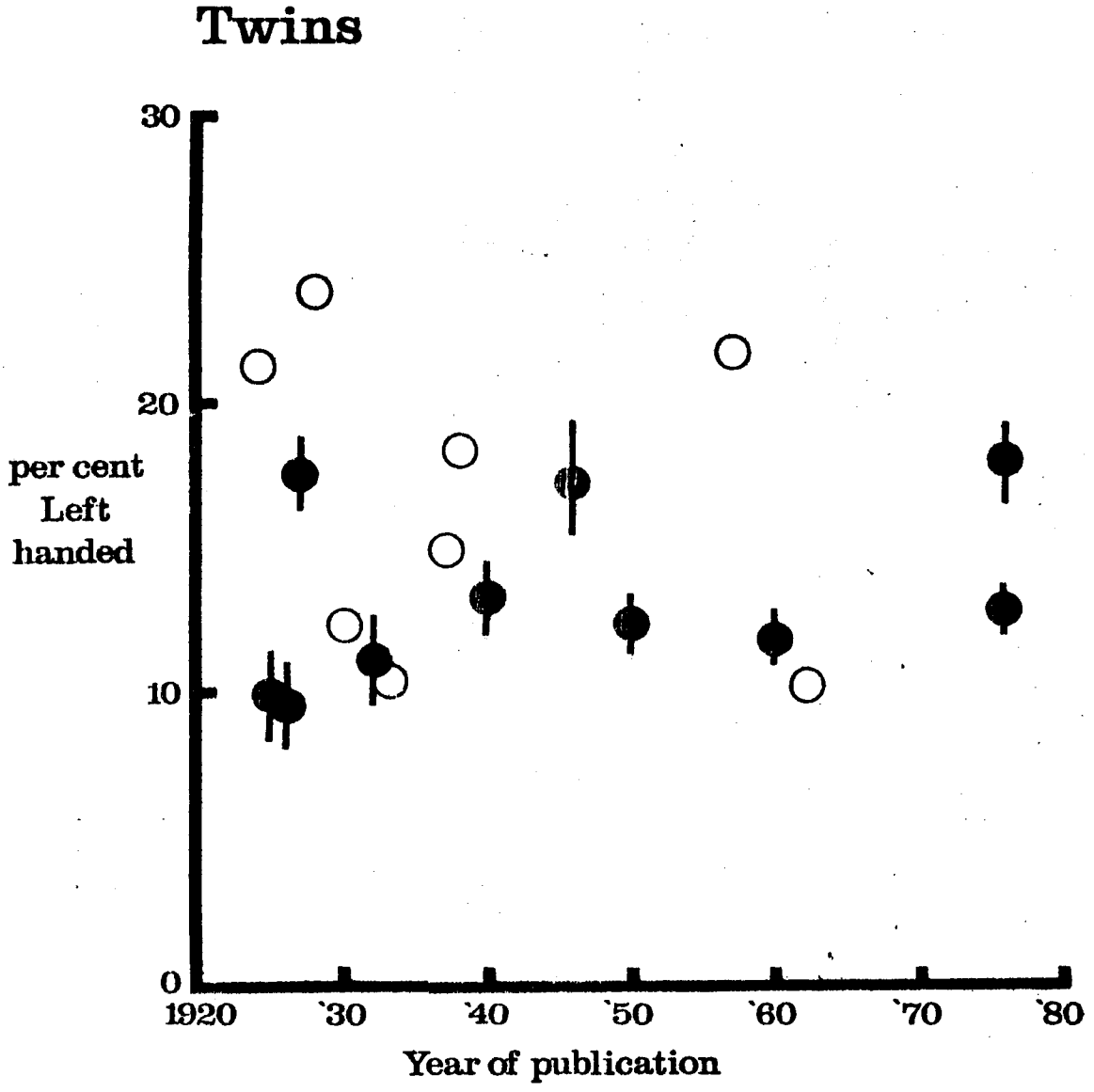

Twins
30
20
10
0
MZ
p.L
0
10
20
30
p.L DZ
Small
Large

Figure 4.3 Shows the overall incidence of left-handedness in eighteen different twin studies (MZ and DZ twins combined), plotted against the date of publication of each study. Solid circles represent the ten 'large' studies, and open circles the eight 'small' studies. Only the large studies have bars, which represent ± one standard error.


Twins
30
20
10
0
per cent
Left
handed
1920
'30
'40
'50
'60
'70
'80
Year of publication

CHAPTER 5: LEFT-HANDEDNESS AND EPILEPSY

"Should ... (the) ... routes for the passage of phlegm from the brain be blocked, the discharge enters the blood-vessels ... This causes loss of voice, choking, foaming at the mouth, clenching of the teeth and convulsive movements of the hands; the eyes are fixed, the patient becomes unconscious ...

The discharge of phlegm takes place more often on the right side of the body than on the left because the blood-vessels on that side are more numerous and of greater calibre than on the left".

Hippocrates, The Sacred Disease, 10; 13.

5:1 Introduction

Left-handedness is commonly claimed, usually in passing, to be more common amongst epileptics (e.g. Hicks and Barton, 1975; Bakan, 1978; Bradshaw, 1978 and Hicks and Kinsbourne, 1978). In this chapter I will present evidence from the large-scale NCDS study which does not support the popular view, at least as far as the less severe forms of epilepsy are concerned.

Hecaen and de Ajuriaguerra (1964) reviewed several studies which had examined the incidence of left-handedness in epileptics, usually without an adequate control group. Redlich (1908) claimed an increased sinistrality in epileptics, although he also suggested that there was no increased familial sinistrality. Stier (1911) claimed both increased sinistrality and an increased familial sinistrality in epileptics. Hordijk (1952) claimed an increased incidence of epilepsy in sinistral families. Bodin (1953) suggested an increased incidence of left-handedness in epileptics as compared with schizophrenics and oligophrenics; he however used no normal control group, and his results are only just statistically significant (Chi-squared = 7.87,3df, p = 0.048).

Several studies have examined the incidence of left-handedness in chronic severe epileptics undergoing neuro-

surgical operations (e.g. Penfield and Roberts (1959) and Milner et al (1964)). Whilst these studies appear to show increased sinistrality, there is neither an appropriate control group, nor any evidence to negate the possibility of a selection bias in favour of operating on left-handed patients (due for instance to a diminished risk of post-operative aphasia).

A recent study by Brittain (1978) compared a group of epileptic patients (n = 157), with a group of matched controls (n = 80); there was no evidence of increased sinistrality amongst the epileptics.

5:2 Method

In 1958 the Perinatal Mortality Study collected data from every child born in Britain during the first week of March. These children were later followed up at the ages of seven and eleven as a part of the National Child Development Survey (for further details see Butler and Bonham, 1963; Butler and Alberman, 1969; National Child Development Study, 1966, 1972). Handedness was assessed in a large proportion of the study population, and the detailed structure of these responses has been discussed earlier in Chapter 3:3, the main finding being that it is relatively simple to divide the children into two distinct categories, right- and left-handers. These same categories will be used in the present chapter.

At birth evidence was collected of the presence of neonatal convulsions (this was regretably coded with neonatal cyanosis, and thus the two are indistinguishable). At ages 7 and 11 detailed information was collected from the parents (interviewed by a trained interviewer) and also from doctors, as to the presence of epileptic symptoms during the child's life. An overall summary of the child's elileptic status was also constructed by the NCDS.

5:3 Results

Table 5.1 shows, for both sexes separately, and for the two sexes combined, the incidence of left-handedness as a function of various items which are or could be associated with epilepsy. Only singletons have been included in the present analysis. Almost none of the Chi-squared values are statistically significant. Table 5.2 shows the NCDS's own summary classification of the individuals' epileptic status at the age of 11. Two of the statistical tests approach significance. Nevertheless, scrutiny of the sub-groups of the table shows that, if anything, the incidence of left-handedness in the true epileptic groups (i.e. groups 5, 7, 8 and 10) is lower than in the other groups. In tables 5.1 and 5.2 there is no evidence to suggest that left-handedness is more common amongst epileptics. Table 5.3 shows data from a question in NCDS II, in which mothers were asked to

classify any attacks that their child had had (it is accepted that this classification may well be suspect). There are no differences in incidence of left-handedness between the groups. Of more interest is that the mothers were also asked to state the age at which the child first started having the attacks. Table 5.3 also shows the mean age of starting attacks, and also an analysis of variance of age and attack type, by handedness. If one were to propose that, say, birth stress caused both childhood epilepsy and handedness (e.g. Bakan, 1971), then one would predict that the age of onset of epilepsy might well be lower in left-handers than right-handers. There is no evidence of such a process in Table 5.3 and the trend in the results is actually in the opposite direction.

5:4 Conclusions

There is no evidence, in a large population sample, of an association between left-handedness and epilepsy. It is of course possible that there may be such an association in severe epileptics (of whom there are relatively few in the present sample), but to establish that would involve partialling out any effects due to mental sub-normality or cerebral palsy, both of which are probably associated with left-handedness.

TABLE 5.1 Shows the proportions of left-handers in those who gave particular answers on NCDS items connected with epilepsy. The analysis is for males, and females separately. Only singletons have been included.

Variable	NCDS No.	Survey	Person
Neonatal convulsion (or cyanosis)	1831	PMS	Doctor-midwife
Fit or convulsion in first year of life	274	I	Mother
Fit or convulsion after the first year of life	275	I	Mother
One fit only before five years of age	1817	I	NCDS Summary
More than one fit before five years of age	1817	I	NCDS Summary
Fits after five years of age	1817	I	NCDS Summary
Doubtful fits before seven years of age	1817	I	NCDS Summary
Petit mal or blank spells	276	I	Mother
Fits before age of seven	1502	II	Doctor
Doctors summary of abnormal conditions present= 'Epilepsy' (groups 2,3,4, & 5)	415	I	Doctor
Fits after the age of seven	1502	II	Doctor
Absent from school for more than one week during last year due to convulsions, fits, or turns	1321	II	Mother
Parent, brother or sister has had a fit or a convulsion	290	I	Mother

Males						Females					
Condition absent		Condition present				Condition absent		Condition present			
N	%L	N	%L	Chi2	p	N	%L	N	%L	Chi2	p
6782	12.7	41	19.5	1.160	0.281	6473	9.4	18	22.2	2.091	0.148
5903	12.9	125	12.0	0.027	0.868	5661	9.4	95	10.5	0.034	0.853
5870	12.9	171	11.7	0.115	0.733	5635	9.5	134	8.2	0.115	0.734
6717	12.7	107	13.1	0.141	0.708	6395	9.5	96	9.4	0.001	0.971
6724	12.7	100	11.0	0.266	0.612	6416	9.5	75	8.0	0.192	0.665
6784	12.7	40	7.5	0.982	0.321	6459	9.5	32	9.4	0.001	0.982
6787	12.7	37	19.3	0.413	0.527	6470	9.5	21	14.28	0.568	0.451
6009	12.8	36	11.1	0.003	0.952	5734	9.5	37	5.4	0.314	0.575
6072	12.6	201	11.9	0.765	0.381	5792	9.5	191	8.4	0.286	0.59
5849	12.8	37	8.1	0.723	0.395	5573	9.5	33	9.1	0.006	0.93
6235	12.6	38	10.5	0.146	0.704	5979	9.5	24	8.3	0.038	0.84
68	12.7	11	27.2	0.998	0.317	5597	9.4	17	23.52	2.432	0.11
5485	12.9	491	13.6	0.166	0.683	5271	9.5	439	8.0	0.993	0.31

Table 5.2 Shows the proportion of left-handers in each of the NCDS's summary of epilepsy categories (variable 1842), by the subject's sex.

	Males		Females	
Disease group	N	%L	N	%L
1. No disease	4	25.0	2	50.0
2. Indefinite	63	22.2	67	6.0
3. Faints	84	11.9	141	14.2
4. Hysterical	36	8.3	25	8.0
5. Consensus epilepsy	27	0.0	21	4.8
6. Suspect diagnosis	3	0.0	0	-
7. ?Febrile epilepsy	15	0.0	8	12.5
8. Definite febrile. ?epileptic	15	0.0	8	12.5
9. Not diagnosed	53	9.4	39	15.4
10. Febrile convulsion	113	11.5	92	9.8
11. Breath-holding	42	9.5	30	6.7
12. Non-epileptic blank spells	3	33.3	3	0.0
13. Epilepsy never suspected	6375	12.8	6061	9.4
	Chi-squared=22.63 12df, p=0.0310		Chi-squared=11.57 12df, p=0.3961	

TABLE 5.3 Shows, for the mothers description of their child's attacks at the age of 11, the proportion of left-handers in each group, the mean age of onset of attacks in right and left handers, and an analysis of variance of age on onset by attack type and handedness

	Males				Females			
Mother's description	N	%L	Mean AGE R	Mean AGE L	N	%L	Mean AGE R	Mean AGE L
Grand mal	58	8.6	4.50	5.25	26	11.5	1.61	2.50
Petit mal	81	13.6	3.36	2.09	88	2.3	1.64	1.00
Other, or mixed epilepsy	8	25.0	6.20	3.00	11	18.2	1.00	5.50
Fainting or blackouts	103	10.7	8.43	9.36	124	14.5	7.70	8.52
Other attacks or turns	98	11.2	4.68	5.40	104	11.5	4.45	4.41
No attacks at all	6386	12.8	-	-	6064	5.4	-	-
	Chi-squared = 2.66 5df, p=0.7521				Chi-squared = 10.64 5df, p=0.0591			

Analysis of variance
of age at first attack by
epilepsy type and handedness

Main effects	df	p	df	p
Convulsion type	4	< 0.001	4	< 0.001
Handedness	1	0.811	1	0.261
Interaction	4	0.797	4	0.567
Residual	313		323	

CHAPTER 6: PATHOLOGICAL LEFT-HANDEDNESS: DOES IT EXIST?

"the study of the thing caused must precede the study of the cause of the thing ....."

(Hughlings Jackson; quoted by Wilson, 1908, p. 211)

Summary

The concept of pathological left-handedness is reviewed, both from a historical and from an empirical point of view, and it is suggested that there is no adequate evidence to justify its continued use. That the concept is still much used may be the result of a desire to restore to the brain its lost asymmetry, by allowing Dax's law once more to be true.

6:1 Introduction

In this chapter I would like to suggest that pathological left-handedness does not exist, and that it is only one of a number of erroneous concepts which have been developed in an attempt to account for the relationship between handedness and speech dominance as perceived through the study of aphasic patients. I will suggest that the concepts have arisen, not in response to the careful observation of patients, and the interpretation of their history, symptoms, and signs, but rather as ad hoc concepts whose primary purpose has been to revive otherwise moribund theories. But in so doing the theories transgress the limits of science and the theories become unfalsifiable. To understand the concept of pathological left-handedness it will be necessary to consider the literature on aphasia and cerebral dominance, and to critically assess the theoretical interpretations and the explanatory concepts which have been generated.

6:2 Definition: and a challenge

Before commencing this review I feel obliged to state what I understand by the concept of pathological left-handedness. Pathological left-handedness could be agreed to occur in a patient previously of known right-handedness, who, as a result of some injury to the brain, or similar cause, changes from a preferential use of the right hand to

a preferential use of the left hand. The concept of hand dominance inevitably implies a relative preference between two otherwise similar organs or systems. One cannot have dominance if there is only one organ; and hence it is clearly trivial or absurd to claim that a man has pathological left-handedness if for some reason his right hand has been severed from his body. But it is similarly trivial to describe as pathological left-handedness a case in which a man has one half of his brain destroyed; the shift to left-hand usage must be preferential, not inevitable, and must occur in the absence of a hemiplegia, hemiparesis, lower-motor-neurone weakness, or other defect of the limb, or lack of control due to tremor, athetosis, ataxia, etc. The shift in hand preference in pathological left-handedness also implies that the previous preference had been ascertained; one cannot claim that a change has occurred unless one knows the nature of the earlier state. This might seem to impose unacceptable limits in the case of very early brain damage, for it would be very difficult, nay impossible, to assess the handedness of an unborn fetus. This problem may be readily and satisfactorily surmounted by the use of a control group; the incidence of left-handedness must be shown to be significantly higher in a pathological group than in an appropriate control group (both groups being assessed blind etc.). Whilst one might not be able to know with certainty the handedness which an individual would have shown if he had not received a particular brain insult, one may make estimates of the

various probabilities.

Defining pathological left-handedness as above I would suggest that there is not a single case in the literature which could be regarded as acceptable proof of the existence of the phenomenon.

6:3 Aphasia and left-handedness

To understand the origin of the concept of pathological left-handedness we must return to the period of the first realisation of the asymmetry of the brain. In 1861 Broca described a patient in whom a lesion of the left hemisphere had resulted in aphasia (for translation see Rottenberg and Hochberg, 1977), the lesion being confirmed by post-mortem. At this time the particular importance of the left hemisphere does not seem to have been realised*. The first mention of the predominance of the left side is in Broca (1863), where it is noted in passing.

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*And this was not particularly unusual. As Benton and Koynt (1960) have pointed out:

> "The association of aphasia with right hemiplegia was not remarked, despite the repeated incidental observation of aphasia and dextral paralysis. This relationship escaped the notice of even so keen and careful a student as Morgagni, who had had the opportunity of observing so many instances of this occurrence. The failure to make this correlation was not, of course, peculiarly distinctive of medical observation before 1800 ...."

See also Giannitrapani (1967), and Riese (1947).

Broca's best description of "la singulière prédilection ... pout l'hémisphère gauche", was published in 1865. For a good account of the early history of aphasia see Ombredane (1951). Whether Broca was truly anticipated by M. Dax, in an obscure paper of 1836, is still controversial. Dax, fils, in 1865 (and also anticipated by a memoir of 1858, and another of 1860 - see Anon (1863)), published a paper written by his father and presented, it is claimed, at a meeting of the Montpelier Medical Association. Certainly Broca himself could not find any published account in the Revue de Montpelier of 1836, and he could only conclude that the work was presented but not published. Dax fils produced ~~a copy of the~~ his father's manuscript, which Broca declared to be in a different style to that of the son (a hint perhaps of suspected forgery). This manuscript does not apparently exist today; and Critchley (1964) has suggested that it is conceivable that it never existed. Alternatively, he suggested that the elder Dax wrote the account, but never quite got round to presenting it at the meeting, leaving it 'in the bottom drawer of his desk'. Hecaen and Dubois (1969) appear to believe that the paper was actually presented in the meeting, although they quote little evidence for their position. There seems little doubt throughout the entire controversy that Broca conducted himself with scrupulous fairness, even presenting Dax to the Society of Anthropologists himself, on the 21 April, 1867.

From my own point of view the most important point

is the attitude of Dax, *père et fils*, to the question of aphasia after right-sided cerebral lesions. Dax *père* had never seen a case of aphasia after a right lesion (and indeed went so far, on the strength of his observations, to apply leeches only to the left-side of the head in cases of aphasia.) Dax *fils* put things a little more strongly:

> "Mais la lésion cérébrale d'où résulte primitivement cette altération de la parle est toujours localisée, pour moi et comme mon père, dans l'hémisphère gauche, et jamais dans l'hémisphère droit ..... Congestion et hémorrhagie, traumatisme, anemie, agissant sur l'hémisphère gauche, troublent la parole; les mêmes causes, agissant sur l'hémisphère droit, n'influencent pas la faculté de parler".
>
> (The cerebral lesion which early on results in this alteration of speech is always localised, for me as well as for my father, in the left hemisphere, and never in the right hemisphere ..... Congestion and haemmorhage, trauma or anaemia, act on the left hemisphere damaging speech; the same conditions acting on the right hemisphere do not influence speech).

A few patients had however been described in whom aphasia was associated with a *left* hemiplegia (i.e. right hemisphere damage). One reaction to such cases was to try and explain them away, and to simultaneously explain the overall predominance of left-sided lesions in aphasia, by suggesting that left-sided brain lesions were more common than right-sided brain lesions (e.g. due to emboli being more likely to enter the left carotid artery than the right artery; Bateman 1870). Such a possibility was rapidly removed by the simple statistical analysis of Ogle (1867); whilst 90% of aphasics had a right hemiplegia, only

55% of hemiplegias affected the right side (and hence were due to defects in the circulation of the left side of the brain)*.

Broca's response to the problem of aphasia with left hemiplegia was entirely theoretical, since he never actually saw a patient with the condition. In response to estimates by Charcot and Vulpian, that amongst aphasics only one in twenty had a left hemiplegia, he suggested that:-

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*Bateman (1865) put forward a further argument against those physiologists who claimed

> "that in an organ as symmetrical as the brain it is impossible to admit a difference of function between the two sides. One hemisphere exactly resembles the other in form, and must therefore exactly resemble it in its office" (Ogle, 1867).

Bateman said in reply:-

> "I must however remind (the physiologist) that this is not the only singular and inexplicable fact which physiology presents to us. Professor Trousseau alluding to this subject says he has never seen an intercostal neuralgia except on the left side; why is this? We know nothing about it except that it is a symptom depending exclusively on an affection of the left side of the cord" (Bateman, 1865).

Trousseau (e.g. 1877) was however one of the physiologists who rejected the possibility of an absolute functional asymmetry of the brain. His error in the case of 'intercostal neuralgia' was probably in regarding as neuralgia those cases in which "real neuralgia was correctly distinguished from pleurodynia, pleuritic shingles and hepatic colis" (Ireland, 1880). Angina pectoris is conspicuously absent from this list and in view of controversy as to the neural or cardiac origin of angina, we may speculate that Trousseau had failed to correlate the gross anatomical asymmetry of the heart with this gross asymmetry of disease symptomatology (Gibson, 1902). Despite Trousseau's reservations, the asymmetry of the brain was the received view within a decade of Broca's 1865 paper (see e.g. Brown-Sequard, 1874).

> "Just as there are left-handers in whom the inherent pre-dominance of the motor activity of the right hemisphere confers a natural and irreversible pre-dominance to the activity of the left hand, so in the same way it is conceivable that there may be a certain number of people in whom the inherent predominance of the convolutions of the right hemisphere will reverse the order of the phenomena which I have just described" (Broca, 1865; translated by Hecaen and Piercy, 1956).

In other words, left-handers will have aphasia after <u>right</u> sided lesions. Note that Broca is assuming that the system is symmetric (indeed is restoring its lost symmetry; hence perhaps the enduring attraction of the hypothesis). Also he feels the predominance of the brain to be primary, and that of the hand secondary, and this dominance of the brain is "irreversible".

Broca's corollary for left-handers was necessary, for aphasics with right-sided lesions were soon to be described; the simplicity of the challenge was perhaps too great, and by 1870 Bateman could refer to 28 cases of aphasia with <u>left</u> hemiplegia; however Bateman was aware of only two cases in which the handedness of the patient was known - and both were left-handers.

In 1868 Hughlings Jackson published a short case history describing a case of aphasia associated with a <u>left</u> hemiplegia (i.e. a right brain lesion), justifying the case's publication:-

> "We make a note of this case as it has been supposed by some that exceptional cases are not put on record" (Jackson, 1868a).

Strangely though, this case was not an exception to the rule, and a few weeks later Jackson sent off a further letter to the Lancet pointing out that he had omitted to state that the patient was left-handed (Jackson, 1868b).

Further cases were produced, which supported Broca's symmetric conception of aphasia and handedness. Wadham (1869) described an 18-year old youth with an aphasia and a left hemiplegia. This youth:-

> "When asked his name, wrote (it) correctly with his right hand, although his mother asserted that she had never seen him previously do so, and that he and four of his brothers were left-handed". (Wadham, 1869).

Wadham suggests that this case might be explained:-

> "in the supposition which has been made, that in left-handed individuals the organ of speech is exceptionally placed in the opposite hemisphere (to normal)".

We may find the same assumption in Pye-Smith (1870).

Exceptions to this revised rule, of the language centre being contra-lateral to the dominant hand, were however occurring. Ogle (1871) discussed such cases and introduced a new concept which was to have disastrous consequences, resulting eventually, and inexorably, in non-falsifiable hypotheses.

> "It may, perhaps, be urged that there are recorded cases in which the aphasia coincided with left hemiplegia, and yet the patient was not reported to be left-handed. But in answer to this I would say that none such has been recorded since special attention was directed to the probability of right- or left-handedness being concerned in the latter, and that without special enquiry it is very easy, as Dr. Jackson's case shows (vide supra) for the co-existence of left-handedness to escape notice".

Thus far Ogle's claim is reasonable. He continues however:

> "Even should such a case occur it would not be incompatible with the views now expressed. For, as I have already stated, there are probably persons with a natural left-handed tendency in whom the bias is so feeble that its external manifestations become completely masked by education. In such a person aphasia might occur with left hemiplegia, and such a case would thus appear erroneously to stand in contradiction with Dax's law". (Ogle, 1871)*.

In continuing thus, Ogle has completely transformed the problem. Handedness and side of lesion in aphasics can no longer be determined independently, but instead the determination of handedness is itself contingent upon the presence or absence of aphasia and the side of the hemiplegia. Dax's law has become impossible to disprove.

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*Critchley (1964) has suggested that the term 'Dax's law' was first used by Grasset, in 1873; the present usage clearly pre-dates that essay, In the present paper I shall use the term in the sense of all right-handers having left hemisphere speech, and all left-handers having right hemisphere speech.

Despite Ogle's stricture ("even should such a case occur"), cases of crossed aphasia (i.e. dominant hemisphere for speech ipsilateral to dominant hand; the term was apparently first used by Bramwell, 1899) continued to appear, and presented theoretical problems. In his Gulstonian Lecture of 1878, Ferrier could only point out that:-

> "In several at least of the cases of aphasia with disease of the right speech centre, the patients have been left-handed".

The double implication is that the majority of cases were not left-handed, and that according to Dax's law they really ought to have been.

Jackson (1880), despite his earlier interest in the specific side of the lesion, switched his theoretical interpretations, and neatly side-stepped the whole problem:-

> "It is admitted that there are cases of left hemiplegia with aphasia in persons who are not right-handed. Besides granting fully the significance of the fact that in the vastly greater number of cases loss of speech is caused by disease in the left half of the brain, the thing of infinitely greater significance is that damage in but one half can produce speechlessness"

Paget (1887) described an excellent case of crossed aphasia in a sinistral; but, in a theme to be expanded later, the difficulties were explained away:-

"The explanation of the anomaly may, I think, be found in the fact that, though left-handed for other actions, (the patient) wrote with his right hand, and was in the habit of writing much .... The habit of writing much seems to be the only assignable cause for this reversal" (Paget, 1887).

William James, in 1892, totally accepted Dax's Law:-

"The injury (of Broca's aphasia) in right-handed people is found on the left hemisphere, and in left-handed people on the right hemisphere. Most people, in fact are left-brained, that is, all their delicate and specialised movements are handed over to the charge of the left hemisphere. The ordinary right-handedness for such movements is only a consequence of that fact ...." James (1892).

In the last two years of the nineteenth century, two works appeared which introduced a series of ideas which would modify approaches to cerebral dominance. Both authors assumed at least partial veracity of Dax's Law:-*

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*At this point I cannot resist quoting from the writings of at least one man who believed in the strict truth of Dax's Law. Collins (1898), in an essay awarded the 'Alvarenga Prize of the College of Physicians of Philadelphia, 1879', writes thus:-

"Every one knows that the faculty of speech is maintained by the left hemisphere of the brain, and by the right hemisphere in those who are left-handed. And this for the same genetic reason that the organism is right-handed in the majority of people and left-handed in the few; when the organism is left-handed, then the right hemisphere contains the zone of language. It would seem that this is one of the facts of physiology which is indisputable; nevertheless, every now and then someone brings forward evidence purporting to deny it. One of the most recent purveyors of such evidence is Moltschanow, of Moscow, ... (he describes a case reported by Moltschanow). The ...

"Cases of "crossed" aphasia are, however, occasionally, though very rarely met with in which the aphasic symptoms are ... permanent. But so far as I know cases of this kind very rarely indeed occur in right-handed persons, indeed I know of no recorded case; as far as I know they almost always occur in left-handed persons". (Bramwell, 1899).

Note that by now it seems to have been conceded that in the case of left-handers, Dax's Law did not hold strictly; nevertheless the law was persisted with for right-handers. Indeed Bastian (1898) believed in it whole-heartedly for both types of handedness:-

"... the now ascertained fact that in the great majority of cases in which aphasia has occurred as a result of brain lesions in the right hemisphere (with or without the association of left hemiplegia) those so affected have been left-handed persons" (Bastian, 1898)

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writer says that this case speaks unequivocally against what he is pleased to call Broca's dictum concerning the location of the speech area. He says further that other authors have probably had similar cases, for in looking over the literature of aphasia he had noted that it is oftentimes impossible to determine from the reported cases whether the patient was right-handed or left. This point the writer need have no difficulty in determining in the future. If the patient whose history he reads has a left hemiplegia with aphasia, that patient is a right-handed man, and vice versa. It is almost incredible that physicians will attempt to convince themselves that evidence of this kind can have the slightest effect in overthrowing such an invariable rule as the one relating to the location of the speech area, just cited. An attempt to discredit a law so firmly established as this by the citation of testimony of a woman concerning the right-handedness or left-handedness of her husband (the source of the handedness information in Moltschanow's case) is like trying to trip up Atlas by putting a microbe to obstruct his path".

In support of his claim Bastian references a work by Seguin in which, after finding that the incidence of left-handedness and the incidence of aphasia after right-sided lesions were numerically similar, he assumed that left-handedness and right-sided aphasia must be related in a perfect one-to-one fashion (i.e. perfect Dax's Law).

From the works of Bramwell and Bastian we may trace the development of four separate sets of ideas.

5:3.1 The concept of hand usage modifying speech laterality

Bastian (1898) had required that Dax's Law be true, for he continued:-

> "It would thus seem that the predominant use of the right hand or of the left hand carried with it, as one of its associated effects, the leading activity in the production of speech by the left or the right hemisphere respectively; and that we must consequently push our question further back, and inquire as to the causes that have led to this predominant use of the right hand".

Notice that now, unlike Broca's conception of the problem, the brain does not have primacy; the use of one particular hand induces the development of the contralateral cerebral hemisphere. This hypothesis is implicit in Moxon (1866) ("the brain of educated individuals is manifestly more unsymmetrical than the brains of uneducated individuals"), and possibly also in the case-history described by Jackson (1880b), and almost certainly in Paget (1887).

The intellectual origin of the concept may probably be traced back to two cases reported in 1878 and 1880. Gowers (1978) described a case in which the left hand was congenitally absent and the contra-lateral parietal cortex was apparently atrophied and vestigial. His assistant at the post-mortem was the young Victor Horsley, still an undergraduate. Bastian and Horsley (1880) reported an almost identical case, commenting only that "It seems something more than can be accounted for by mere chance coincidence".

Wernicke (1906) felt that the lateralisation of speech function was secondary to hand usage, even after childhood:-

> "... the right hemisphere may be able to take over the function of speech originally laid down in the left hemisphere ... the left hemisphere may lose the speech functions it previously had developed if use of the left hand replaces that of the right. I do not know of a similar case (as described earlier in the paper) which so convincingly supports our hypothesis that localisation of the speech centre is (a) functional acquisition of each individual" (1905; 1977 trans., p. 268).

In childhood, "one need not develop left-handedness for localisation of speech to be transferred to the right side ..." (ibid).

The concept is stated most clearly (and perhaps most absurdly) by Coley, who gave instructions on 'The Prophylaxis of Aphasia':-

> "... no harm, and probably a great deal of good, might come from instructing the patient how to provide against the contingency of a lesion involving the left Broca's convolution ... My suggestion is that a graphic centre should be made to develop in the right side of the brain by practising writing with the left hand" (Coley, 1909).

The importance of utilising the left hand had earlier been stressed by the Ambidextral Culture Society, which had been formed by 1905 by one John Jackson "for the promotion of education reform and two-handed training" (see Barsley, 1966, for an account)*. By 1926, no less a neurologist than the esteemed Kinnier Wilson described the argument of Weber (1904) that it would only have been in the mid-19th Century that Broca could make his discovery, since:-

> "The dissemination of the accomplishment of writing among all classes, determined the lead of the left hemisphere over the right, and at the same time finally established the localisation of the speech centres in the cerebral cortex".

This argument is, he admits, over-stated, but "must play a role". He concludes that:-

> If these facts were better known the cultivation of ambidexterity would receive that impetus which it assuredly deserves (Wilson, 1926).

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*The utility of ambidexterity was not always accepted. Gould (1905) arguing from the same neurological premises as Coley, Jackson, etc., could only sympathise with the "pitiable victims!" of the Ambidextral Culture Society.

Weisenberg and McBride (1935) echoed the same suggestions:-

> "It would seem as if the right brain, while not directly concerned in language in the right-handed individual, nevertheless is in a state of receptivity for language acquisition, the degree varying in accord with the use of the left hand in writing". (my emphasis).

Needles (1942) analysed nine cases from the literature and concluded that in usage of the non-preferred hand:-

> "... the function of speech is not relinquished by one hemisphere and taken up by the other, as the shift from the use of one hand to the use of the other occurs, but continues rather to be participated in by both hemispheres..."

He continues, with disarming candour, "There is, of course, no way of confirming this postulate experimentally". This is, of course, untrue.

Outside of medicine such theories were of great influence, as may be seen in the case quoted by Burt (1950), and briefly reported in the Times Educational Supplement of May 6th, 1929:-

> "At the special schools in the Lingfield Colony for Epileptics a group of children who were stationary in learning were given a training in left-handedness in the hope that 'additional' centres in the brain might be opened up" (Burt, 1950; p. 336).

6:3.2 The concept of early brain damage modifying dominance

After discussing cases of crossed aphasia in sinistrals, Bramwell (1899) says:-

> "I am speaking of cases in which there is no reason to suppose that the normal development of the speech centres was interfered with in early life".

Here Bramwell is talking only about the transfer of speech laterality, not of pathological left-handedness as such. This concept however transmutes. Redlich (1908) is reputed to have been the first to posit 'pathological left-handedness' (Bingley, 1958), although the idea is usually attributed to Gordon (1921), who said:-

> "Something may have affected the dominant (in these cases the left) hemisphere. It may have been a lesion, causing among other things slight hemiplegia, or it may have been due to defective development of the left-hemisphere. It would then follow that such left-handedness differed from what is usually termed 'natural' left-handedness".

Gordon defines two types of pathological left-handedness. The first group consisted of, "Very simple cases and requiring no explanation", and he instances the case of a loss of the right arm. And a second group:-

> "Various forms of hemiplegia; in some cases the affected hand cannot be used at all, in others to a limited extent, and in still others the effect is apparently only transitory, or perhaps often unnoticed," (my emphasis).

The twin concepts of pathological shift of language laterality and pathological shift of handedness, either able to occur in the absence of further evidence, provided a theoretical carte blanche to the neurologist. No case was now beyond explanation, or incapable of being fitted to a particular theoretical pre-conception,be it Dax's Law, or some modification of it. Thus Kinnier Wilson (1921) wrote:-

> "It seems superfluous to point out that by universal consent we are justified in stating that in right-handed persons the lesions apt to be associated clinically with disorders of the speech function are situatedin the left cerebral hemisphere ... I do not ignore the exceptional cases ... several valid arguments have been advanced in such cases".

Indeed, but any cases could be so explained. Nevertheless, the explanatory armanentarium of the theoretician was to be further expanded with the addition of the concept of 'stock-brainedness'.

6:3.3 Stock-brainedness

Once more this concept may be traced back to Bramwell (1898):-

> "One would expect that in cases in which 'crossed' aphasia ... occurred in a right-handed person some of the near relations or ancestors of that person would probably have been left-handed".

This idea was expanded upon by Foster Kennedy (1916) in the

concept of 'stock brainedness'. He cited a crossed aphasic described by Bramwell, (1897):-

> "However I would like to point out that perhaps not sufficient weight is given to his isolation as a left-handed person in a stock entirely right-handed".

Once again Kinnier Wilson (1926) finds the concept useful in explaining awkward cases:-

> "In other words, ectopia of the speech centres is a possibility in some right-handed members of a sinistral stock, and in some left-handed members of dextral stock. This is not mere speculation, but a hypothesis which satisfactorily accounts for not a few recorded instances of 'crossed aphasia', and which receives support from the pathological side, as has been shown by Mendel [K, not G.]."

6:4.3 Latent left-handedness

From stock-brainedness developed a yet more powerful weapon: latent left-handedness. Pick, writing just before his death in 1924, grappled with the problem of crossed aphasia, and also with the possibility of bilateral language representation:-

> "The behaviour of left-handed individuals is opposite to that described for dextrals. However there are a few poorly explained cases which in spite of right-handedness, have right-sided lesions followed by aphasic defects. Lastly a few cases have been reported in which the pertinent foci are partly in one and partly in the other hemisphere. This phenomenon, probably related to ambidexterity, is explained by the conflict of a

> pre-disposition (e.g. to right-handedness) with individual (latent) left-brainedness". (Presumably here the pre-disposition is latent, and by latent is intended 'manifest').

Note that manifest and true 'brainedness' need not be related; in the event of awkward cases, then clearly not only handedness but also brainedness could have been determined erroneously.

Brain (1945) emphasised the distinction between natural and pathological left-handedness:

> "Natural or innate left-handedness is either inherited or, in the case of uniovular twins, produced by mirror-imaging".

Note that there are now only two causes of true left-handedness; all others are, by implication, pathological. Note also that there is actually no adequate evidence for the phenomenon of mirror-imaging, it also being simply a useful concept to explain away apparently awkward data - see Chapter 4:6. Brain continues, "Natural handedness is important for neurological diagnosis". He then cites apparent cases of:-

> " ... the speech centres [being] situated in the right hemisphere in a right-handed person ... The clinical importance of these cases is obvious. When the left cerebral hemisphere is damaged early in life, the right hemisphere usually takes over the speech function of the left" (and by implication, once more, true crossed aphasia does not occur in dextrals).

The differentiation of handedness into natural and pathological led to a search for clues to discriminate the two states. Brain (1945) in discussing the slant of handwriting in sinistrals, said:

> "The slope of the writing is naturally reversed, and this reversal is often present in the right-handed writing of a left-handed person, and may thus provide a clue to latent sinistrality (Brain 1945).

Weisenberg and McBride had initiated this search for the true handedness:-

> "Handedness is often difficult to determine ... Furthermore a knowledge of handedness alone is not sufficient; it is important to find out as much as possible about 'sidedness' and consequently to determine which is the preferred foot, and which is the preferred eye".

This hunt for clues to latent sinistrality is found in its most hypertrophied form in Luria's monograph on Traumatic Aphasia (published in Russian in 1947; translated 1970).

> "Thus it is possible to determine subtle signs of left-handedness [all emphases are in the original] in cases where gross observation gives no basis for such classification ... Among the morphological signs of latent left handedness are a large left hand, a well developed venous system on the back of the left hand*, a wide finger nail on the fifth finger of the left hand, and highly developed expressive musculature

*This was suggested by Dr. Minor of Woscow, to the first International Neurological Congress in Berne, August 1931 (Anon, 1932). No statistical evaluation is known to me.

on the **right** side of the face*.

"The number of functional signs of latent left handedness is considerably greater. [These include that] in more primitive functions ... under condition of high affect ... latent left handedness may manifest itself, and the individual may switch to his left hand. Aside from such observation, a detailed interrogration [sic] ... often reveals irregularities in the dominance of the right hand ..."

"Another technique for the discovery of latent left handedness involves special tests. Among these are such wellknown tests as clasping the hands** ... clapping movements ... tonus tests ... [and] sensory dominance ....

These types of test are not limited to functions involving the hand. The procedures include a determination of the dominant eye.

"These tests permit us to detect latent signs of hemispheric dominance ..."

"The detection of latent left-handedness is not the only approach to a more precise characterisation of cerebral dominance [he discusses the genetic literature] ... we shall [thus] expect that, along with the overt forms of left handedness, it should be possible to find latent forms of left-handedness which give no external signs of their presence. Such forms could be identified only by reference to the presence of overt left-handedness in other members of the family ..."

Using this concept of latent left-handedness, Luria is able to explain the occurrence of right-handed patients with lesions of the left hemisphere and yet no aphasia. Case 9

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*This suggestion might originate in the findings of "a friend of Dr. Wilbur, of Syracuse, New York" - see Ireland, 1880.

**The earliest suggestion that hand-clasping might indicate latent sinistrality seems to be Alfred Adler's comment that "the left hand willinstinctively do this so that the left thumb is over the right thumb" (Problems of Neurosis, p. 114, 1929); see Levine (1935) who calls this "an indication of latent handedness tendencies".

gave him the most problems:-

> "This patient had always been right-handed; in his family there were no cases of left-handedness. But he himself showed a number of slight signs of left-handedness (when he clasped his hands, the left thumb was on top; the nail of his left fifth finger was larger than that of the right; the left eye was dominant)".

This cataloguing of the stigmata of 'latent left-handedness' reminds one of nothing more than that notorious index, the Malleus Maleficarum, in which could be found those diabolic signs (the 'witchmarks') necessary for the unequivocal detection of witches.

Parenthetically, it is perhaps ironic to note that after cataloguing his signs of latent left-handedness, Luria puts in a footnote that "A new and apparently effective method of detecting hemispheric dominance was introduced by ... Wada". (Note that it is only "apparently" effective).

In many places Luria's text seems only comprehensible if one assumes some confusion and interplay between handedness and dominance, such that the author must have assumed the veracity of Dax's Law.

6:3 A Re-capitulation

From the works of Bastian (1898) and Bramwell (1899)

we may trace four concepts; hand usage modifying speech laterality; early brain damage modifying speech laterality and handedness; stock-brainedness; and latent left-handedness. These four concepts arose in response to a single problem, the presence of crossed aphasics. If we consider the two types of handedness, and the two sides of possible aphasia producing brain lesions, we may construct a table thus (ignoring the possibility of bilateral representation of language):-

		Side of aphasia-producing lesion	
		Left	Right
Handedness	Right	a%	b%
	Left	c%	d%

Let a, b, c and d be the precentage occurrence of the four possible combinations. Broca's initial (implicit) assumption was that a + c = 100%. This was modified by cases of aphasia after right-sided lesions, in supposed left-handers; Dax's law is thus that a + d = 100%. The concept that both b and c were equal to 0% held for several decades, admittedly under pressues. Thompson (1921) felt that b and c were either each zero or each very small. Even in 1936 Chesher felt that the normal form was for a and d together to equal 100%. But by 1899 Bramwell had begun to realise that c was not equal to 0%, but he still suspected that $c \ll d$. Conrad (1949) realised that for left-handers the true relation might actually be that

c > d, and this was supported by Zangwill (1960) and by Brain (1961). Espir and Rose (1970) however felt that c = d.

The zero nature of b held for much longer, being clearly held by Elliott, Hughes and Turner (1951), and still held by Zangwill (1960), although doubt was beginning to develop. Russell and Espir (1961) concluded that:-

> "... there is of course no known reason which would prevent some right-handers and left-handers from developing the dominant hemisphere on the right, without there being any gross abnormality in the left hemisphere".

Penfield and Roberts (1959) doubted the utility of familial left-handedness as an explanation of dextral crossed aphasia:-

> "we have found left-handedness in the family of right-handed patients who had dysphasia with involvement of the left-hemisphere, and we doubt that there is any significant difference (from right-handers with dysphasia after right-sided lesions)".

Milner et al (1964) presented empirical evidence for b not being zero and accepted that "the overwhelming predominance of left-sided speech representation in right-handed persons of right-handed ancestry rests unchallenged" (i.e. a $\gg$ b and b is not equal to zero). Clarke and Zangwill (1965) stated:-

> "It certainly cannot be said that aphasia resulting from right hemisphere lesions in right-handers is invariably linked with sinistrality; there is, indeed, some reason to believe that this association may have been exaggerated".

Weinstein (1978) claimed that b might be as high as 10% (although he also claimed that for left-handers, c was only 30%).

If we now return to our four explanatory concepts we find that, certainly at the time of their introduction, there was no adequate empirical evidence for them; they were post hoc postulates to maintain Dax's Law, or variants upon it*. It should be clear by now that the non-zero nature of b and c cannot support the existence of all four of the original speculations; further evidence is required or else some or perhaps even all of them must flounder.

Handedness cannot, and indeed must not, be defined

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*In the case of stock-brainedness this is readily admitted by one of its originators, Bramwell (1898) tells us, in a footnote:

> "In my lectures on Aphasia ... I have thrown out the suggestion that in some right-handed persons whose ancestors were left-handed the active or driving speech centre may possibly in rare instances be situated in the right hemisphere; but while this is, I think, theoretically probable, no case in which it actually occurred has, so far as I know, been recorded". (It is regrettable that in the above quotation 'thrown out' is ambiguous in its meaning).

in terms of language laterality, or vice-versa. To say, as has Girard (1952) that, 'Le gaucheur n'est pas celui qui écrit avec la main gauche, mais celui qui parle avec le cerveau droit" (The left hander is not one who writes with his left hand, but one who talks with his right brain), is to make a fundamental confusion between data and explanatory concepts. One may say, in a form of short-hand, that a person acts '<u>as if</u>' he were a left-hander, but one may not say that he <u>is</u> a left-hander. To expand the short-hand he may be 'a right-hander with a history of severe cerebral trauma to the left hemisphere', but he cannot be a 'latent left-hander'; to say otherwise is to render hypotheses untestable and necessarily, and analytically, true.

We may find this process of 'truth by definition' occurring in the large series of patients described by Hécaen and de Ajuriaguerra, and analysed in some detail by Annett (1975), who suggests, somewhat discretely, that:-

> "Among the dysphasics 33 (29 percent) were recorded as left-handed ... for non-dysphasics the incidence was 11 percent. Thus the criterion of sinistrality differed markedly between dysphasics and non-dysphasics ... it is possible that more careful enquiries were made about the laterality of the patient and his relatives for dysphasics than for non-dysphasics" (Annett, 1976).

To put it less discretely, Hécaen and de Ajuriaguerra were classifying a patient's handedness <u>after</u> they had known whether he was dysphasic and whether he had a right- or

left-sided lesion. This is the error exemplified by Girard (1952).

Note also the reference, in the last quote, to family handedness. Right-handers may be divided into those with or without a family history of left-handedness, and these two groups may differ in their responses to right or left brain lesions, but this does not entitle those with a positive family history to be called left-handers: they are still right-handers. To call them otherwise is to see the basic data of aphasia through the distorting spectacles of a prior theoretical conception.

The confusion of data and hypothesis was recognised long ago by Moutier, a pupil of that great inconoclast of aphasia, Pierre Marie.

> "Chaque fois en effet qu'un fair contradictoire de lésion du centre de Broca sans aphémie a été observé, il s'est toujours trouvé quelque partisan de la localisation classique pour déclarer que le sujet atteint était un gaucher méconnu, un ambidextre au besoin. Ou bien, on admet que l'aphémie a été fugitive, et que ce caractère transitoire des accidents est dû à une suppléance rapide de l'hémisphère gauche par l'hémisphère droit. Gaucherie, ambidextérité, suppléance, sont des théories que l'on ne saurait appliquer également a l'interprétation de tous les faits. La part de vérité pouvant exister ici est du reste des plus difficiles à déterminer; nous allons nous en rendre compte" (Moutier, 1908, p. 115).
>
> (In effect, each time that a contradictory example is observed, of a lesion of Broca's area without aphasia, there is always found a supporter of

> classical localisation who declares that the affected patient was an unrecognised left-hander, if need be an ambidextral. Or instead, one suggests that the aphasia was fugitive, and that this transitory nature must be the result of a rapid substitution of the left hemisphere by the right hemisphere. Left-handedness, ambidexterity, plasticity, are theories that one does not manage to apply equally to the interpretation of all the facts. The element of truth which exists here is moreover very difficult to distinguish ...").

One does not have to look very far for the effects of this confusion of data and theory. Ettlinger et al's influential review of crossed aphasia in dextrals concluded that:-

> "... it will be seen that some degree of ambidexterity was present in three of the cases, and a familial sinistral tendency in nine ... It would therefore seem justifiable to anticipate some sinistral tendency in a case such as ours" (Ettlinger et al, 1955).

The effect of received theory upon interpretation of cases may be seen in two further examples:-

> "Indeed there are only two authentic cases ... in which neither personal nor familial indications of left-handedness could be established. Our patient's elder brother was left-handed, left-footed and right-eyed, which proves that in our case there was also a tendency to familial left-handedness" (Botez and Westheim, 1959).

(Note that here there cannot be "a tendency to familial left-handedness" - either the patient has left-handed relatives or he does not).

The effect of an apparent intimidation upon the

interpretation of data can be found in Archibald and Wepman (1968). They found evidence of language in the right hemisphere of eight right-handers, but were forced to conclude:-

> "The eight right-brain damaged dextral patients with language difficulties reported in this paper appear anomalous in view of the relative dearth of such cases in the literature ... (They may be explained away if) ... their language behaviour may be attributable to general cognitive deficit with, perhaps, some central visual involvement" (Archibald and Wepman, 1968).

Observations of patients are inevitably made in the presence of prior theories as to the nature of such observations; patients reported in the literature suffer even more from this deficit. Figure 6.1 shows the cumulative number of reported cases of left-handed patients with crossed and non-crossed aphasia (data from Goodglass and Quadfasel, 1954). Note that we are now fairly sure that crossed aphasia in left-handers is actually more common than non-crossed aphasia; but it is only in the late 1940s when such a hypothesis began to be acceptable, that cases started being reported. Prior to that, reported cases supported the prevailing theory that left-handers talked with the right-brain.

6:6 Evidence

Having noted that the concepts of stock-brainedness,

alteration of dominance by brain damage, and modification of language representation by hand usage were all introduced in the absence of any adequate evidence (that is, apart from single clinical cases), it is necessary to review evidence now available for them.

6:6.1 The modification of language laterality by hand usage

Even recent work has assumed that this is a potent factor. Humphrey and Zangwill (1952) describing the case of a left-handed man with a right occipito-parietal injury, noted that although aphasic the patient did not suffer from agraphia. They comment:-

> "It is probable that the absence of any real agraphia in the present case is related to the patient's long-standing right-handedness in writing. It may be surmised that his preference for the right hand (whether inborn or as a function of training) in writing indicates the central mechanisms subserving this function had been organised in the left hemisphere".

Zangwill (1955) commented that:-

> "In 'congenital' sinistrals ... the hemisphere dominant for language is typically the right. In these cases, education of the right hand for skilled activity, more particularly writing, may lead to some measure of transfer of dominance to the left hemisphere. It is however most doubtful if this transfer is ever complete, except possibly in the case of writing itself. None the less it is to be expected that many 'congenital' sinistrals by virtue of early right-handed tranning, will come to acquire some measure of bilateral representation of speech" (Zangwill, 1955).

There is little published evidence on the question. Goodglass and Quadfasel (1954) analysed 50 cases of cerebrally damaged left-handers, 35 of whom wrote with the right hand, and 15 of whom wrote with the left-hand. The side of lesion is shown in Table 6.1. Whilst there is a small trend towards a higher incidence of left-sided language laterality in those writing with the right hand, the trend is far from significant (Chi-squared = 1.52, NS).

Gloning et al (1969) reported a study of 57 left-handers, all of whom had been forced to write with their right-hand at school, but of whom seventeen reverted to left-hand writing in adult life. All had suffered brain damage as adults, Although sample sizes were small, the particular writing hand had no effect upon speech comprehension or expression (although there was a tendency for patients who wrote with the hand ipsilateral to the lesion to be less impaired on reading, writing and calculation).

Nielsen (1946; see Clark,1957, p30) cites a definitive case in which there appears to be prima facie evidence of transfer of the speech centre secondary to hand usage. A 10 year old boy who was right-handed was forced to write with his left hand as a result of the amputation of his right arm. At the age of 31, 21 years after the forced transfer of writing, a localised cyst of the right angular gyrus procuced a reversible aphasia, thus confirming that speech was in the right hemisphere. However this patient

also had a sinistral family history (one uncle and two cousins on his mother's side) and an alternative explanation to that of Lovell, Waggoner and Kahn (1932) is that the loss of his hand had not caused transfer of his speech centre, but that it had always been in the right hemisphere (see Chapter 8:4 for details of the proposed genetic model). Certainly a single case cannot prove the possibility of transference of dominance. Chesher (1936) also described 3 patients who seemed to disconfirm the possibility of hand usage modifying language laterality.

6:5.2 Early brain damage modifying language dominance

There seems little real doubt that cerebral dominance for language can be modified by early insults to the brain.

Milner et al (1964) determined language dominance by the Wada sodium amytal technqiue. Of 44 left-handers with no evidence of early brain damage, 63.6% had language located in the right hemisphere, and 20.4% had language located only in the right hemisphere. Of those 27 cases of left-handers with evidence of early brain damage before the age of 2, only 22.2% had language exclusively in the left hemisphere, and 66.6% had language exclusively in the right hemisphere (the remaining cases had evidence of bilateral language representation). These differences are highly significant, whether one excludes those with bilateral language (Chi-squared = 13.17, 1 df, $p < 0.001$), or

includes them in the non-left hemisphere group (Chi-squared = 9.90, 1 df, $p < 0.01$).

The evidence of Penfield and Roberts (1959) is not so clear. They operated on patients with chronic epilepsy and noted the incidence of post-operative aphasia, according to handedness and side of operation. Their data were presented with patients divided according to the presence or absence of a history of early brain damage. Amongst left-handers without early brain damage, 72.2% of 18 had aphasia after a hemisphere operation, compared with 6.7% of 15 after a right hemisphere operation ($p < 0.001$). The corresponding figures for left-handers with early brain f damage are 12.2% of 49 and 14.1% of 7 (no significant difference) This might seem to indicate a degree of randomness in speech allocation amongst those with early brain damage, but such a conclusion is far from certain. A closer examination of the data presented in my Table 6.2 reveals several anomalies. Whilst early brain damage seems to produce a decreased incidence of aphasia after left hemisphere operations, in both right and left handers (Chi-squared = 5.78, 1 df, $p < 0.05$; Chi-squared = 20.47, 1 df, $p < 0.001$), there is no compensatory increase in aphasia in those with early brain damage after right-sided lesions. This is a surprising finding, Taken overall the incidence of post-operative aphasia is far lower in early brain-damage patients (12.6% of 136) than in those without brain damage (33.6% of 386) (Chi-squared = 21.25, 1 df, $p < 0.001$), a finding true for

both left-handers and right-handers (Chi-squared = 8.72, 1 df, $p < 0.01$; Chi-squared = 12.0, 1 df, $p < 0.001$ respectively). Early brain damage thus seems to protect against aphasia secondary to neurosurgical operation; this might imply some degree of bilateral speech representation, and as such might be consistent with the hypothesis that brain damage modifies language dominance, but the data are not totally convincing.

Early studies of the effects of early brain damage and of hemispherectory in infancy suggested an almost total plasticity, language being readily inducible in the opposite hemisphere. Recent reviews have however suggested that the situation is not quite so simple. Dennis and Whittaker (1977) reviewed the 19th and 20th Century literature and found that whilst 40.1% of 533 cases of left hemisphere damage resulted in dysphasia, this was true of only 18.4% of 422 cases of right hemisphere damage. The implication is that whilst early brain damage might modify language dominance, there is still evidence of an intrinsic asymmetry which sets upper limits on the effects of modifiability secondary to trauma or damage.

6:5.3 Stock-Brainedness

Despite Bramwell's candid admission of a lack of evidence for stock-brainedness, modern work has confirmed the partial validity of the concept, its modern form being

that left-handers without a history of left-handedness in the family will be more like right-handers than those with such a history; and the converse for right-handers, a positive history of left-handedness making them more like left-handers, as a group.

However evidence of the type presented by Ettlinger et al (1955) is not valid as evidence of stock-brainedness. They found that of 15 cases of crossed aphasia in dextrals, 9 (60%) had evidence of familial sinistrality. This neither explains away such cases (they are still, after all, right-handed), and nor does it prove that a positive family history is significantly more common in such cases than in non-crossed dextrals, for a control group is lacking; indeed Penfield and Roberts (1959) doubted whether the incidence would be greater than in a control population (and a glance at Figure 2 might also support this position).

Studies have usually compared persons with a family history of left-handedness (+FHLH) with those without such a history (-FHLH). Criteria vary; Hécaen and Sauguet (1971) considered a patient to have +FHLH if "there was knowledge of at least one parent, sibling, or direct descendant who was left-handed, or at least two other left-handed relatives, e.g. cousins, uncles, or grand-parents". Zurif and Bryden (1969) had a more stringent criterion; "if either a parent or sibling was left-handed; all other left-handers were considered to be non-familial". Clearly these

criteria will produce different results; neither criterion is perfect, since family size will vary (ten dextral siblings being rather more convincing evidence of -FHLH than one dextral sibling), and there is a possibility of reporting biasses, those with left-handed relations being more likely to report the fact. The problem of dividing a population into -FHLH and +FHLH groups is clearly shown in Figure 6.2. This shows data from survey 2, which I have described earlier in Chapter 2, in which a series of 511 students and their parents jointly reported the handedness of themselves and of their relatives, the questionnaire specifically asking for information on the handedness of the aunts, uncles, siblings, grand-parents, and great-grand-parents of the student. Figure 6.2 shows, separately for right and left-handers, the number of left-handed relatives reported as a function of the total number of relatives reported. It would be difficult to argue that there was any clear clustering of either right- or left-handers into two groups, those with and those without a sinistral family history.

Despite the problems of defining +FHLH, useful conclusions may probably be derived from reports in the literature. Two useful studies have examined clinical populations with dysphasia. Hécaen and Sauguet (1971) looked at 73 left-handers. Those with +FHLH showed no difference in verbal symptomatology between right and left-sided lesions, whilst for those with -FHLH there was a strong tendency for speech deficits to be nearly absent

after right-handed lesions, but to be similar to +FHLH for left-sided lesions. Luria (1970) considered right-handers with +FHLH or -FHLH(his groups A + Ba, and Bb + Bc respectively). Of those with -FHLH, 6.7% had no initial aphasia after injury of the left frontal area, whilst 30.3% of those with +FHLH had no aphasia (Chi-squared = 6.77, 1 df, $p < 0.01$). Similarly no residual aphasia was present in 22.1% of -FHLH cases as compared with 71.5% of +FHLH cases (Chi-squared = 28.21, 1 df, $p < 0.001$). Both of these sets of data are compatible with a modern concept of stock-brainedness.

Several studies have looked at cerebral dominance as measured by dichotic or visual half-field studies. Zurif and Bryden (1969) found that amongst left-handers +FHLH was associated with a greater degree of right cerebral dominance than was -FHLH. Satz, Achenbach and Fennell (1967) suggested a similar result, crossed dominance being more common with +FHLH; however they gave insufficient data for an adequate stastistical analysis.

Briggs and Nebes (1976) found no significant difference between +FHLH and - FHLH on a dichotic task; Lake and Bryden (1976) found a similar result for right-handers, and a non-significant trend in the wrong direction for left-handers (i.e. -FHLH in left-handers resulted in more right cerebral dominance). Higenbottam (1973), using a dichotic task, found no evidence of differences between -FHLH and

+FHLH in left-handers. Using unilateral ECT as a method of assessment of language laterality, Warrington and Pratt (1973) found a non-significant effect of +FHLH in the opposite direction to that expected (i.e. in the same direction as Lake and Bryden, 1976).

In summary therefore the clinical evidence for a role of stock-brainedness is fairly good, in both right and left-handers (despite these studies often being marred in other ways). The earlier experimental literature also seemed to support the concept, but more recent work has been unable to positively support it, although no significant results in the opposite direction have been found.

6:5.4 Latent left-handedness

Brain (1945) proposed that the slant of the hand-writing might provide a clue to latent sinistrality (by which it is assumed is meant a sub-group of right-handers who will tend to behave more like left-handers than ordinary right-handers). No evidence on this point is known to me. Brain also proposed that early brain damage might modify language dominance, there thus being a sub-group of pathological right-handers, who otherwise behave as if left-handed. Evidence on this point will be deferred until later, the question being identical to that of the existence of pathological left-handers (see section 6, below).

Weisenberg and McBride (1935) cite foot dominance and eye dominance as clues to latent sinistrality. The relation of either form of dominance to handedness is far from clear; there is certainly no adequate study in the literature showing that right-handers with unusual eye or foot dominance are more akin to left-handers than to right-handers.

Luria's list of signs of latent left-handedness is more extensive. Well developed veins on the back of the left hand, a wide finger nail on the left little finger, and right-sided predominance of facial expression are not known to have eny empirical evidence in their support. Hand-clasping (and arm-folding) do not correlate at all with handedness (see Chapter 9:5.1 and 9:5.2), and have never been shown to be related to speech dominance, or any other characteristic of right- or left-handers.

There is no statistical evidence that "... irregularities of the dominance of the right hand ..." relate significantly to any other aspect of handedness or of speech dominance. Clapping movements, tonus tests, and sensory dominance have not been shown to be related to handedness, or to language laterality.

In conclusion there is no reasonable evidence for the concept of latent sinistrality, except in the particular case of 'stock-brainedness', and there its usage must be

particularly restricted (see above).

6:6 Pathological left-handedness

Pathological left-handedness has become a major explanatory concept, Zangwill (1955) commented:-

> "If my view is correct, at least half of the normal left-handed population are in reality 'shifted dextrals', i.e. persons without family history of sinistrality who have acquired their left-handedness in the course of early development ... Such an individual tends to show left cerebral dominance for language in accordance with his inherited bias ... In 'congenital' sinistrals on the other hand the hemisphere dominant for language is typically the right".

(Note that here the concept of pathological left-handedness once more allows Dax's Law to be true).

Bingley (1958) divided the factors determining handedness simply into hereditary and environmental, and assumed that pathological factors could modify handedness in a significant proportion of cases, particularly amongst epileptics.

The degree of theoretical confusion surrounding pathological left-handedness is nowhere better shown than by Geschwind in his summary of handedness and aphasia in the influential Beeson and McDermott's Textbook of Medicine (1975). Here we are told that:-

> "[A non-pathological left-hander] ... becomes aphasic as a result of [a] lesion in either hemisphere, although left-hemisphere lesions generally produce more lasting disability ... Pathologic left-handers, who have suffered early childhood injury to the left hemisphere, are usually right-brained for speech".
>
> (Geschwind, 1975)

Note the complete contrast with the views of Zangwill.

Corballis and Beale (1976) made a tentative estimate that 40% of left-handedness is pathological in origin (p. 139), once more deriving their evidence from an assumption that Dax's Law is true.

Hicks, Pellegrini and Evans (1978), after finding no support for one version of the evidence for pathological left-handedness, conclude, " ... we do not doubt the existence of pathological left-handers ...".

Satz (1972) proposed an 'explanatory model' for pathological left-handedness, but had to accept that the syndrome's occurrence was "untested and rarely contested, (and) has been tacitly accepted by neurologists and psychologists for decades ...".

Statistical evidence for the concept of pathological left-handedness usually comes from one of four sources:-

i. the role of acute anoxia of delivery in inducing left-handedness.

ii. the increased incidence of left-handedness in twins, particular mono-zygotic twins.

iii. left-handedness in epileptics.

iv. the increased incidence of left-handedness in the mentally sub-normal, the mentally retarded, and the mentally ill.

There is also implied evidence that reports of clinical cases support the concept of pathological left-handedness; earlier, in Section 2, I have argued that, to my knowledge, there is not a single satisfactory case in the literature. Certainly, no such cases have been cited by the proponents of pathological left-handedness.

It is now worth considering the other evidence for pathological left-handedness.

6:6.1 <u>The role of acute anoxia of delivery in inducing left-handedness</u>.

I have investigated this question extensively in Chapter 3 and need only briefly report the conclusions of that study. In two moderately large retrospective studies, and one very large prospective study (N = 12,000), using data derived from the 1958 Perinatal Mortality Survey, and the National Child Development Study (NCDS), I was unable to find any evidence for a relation between handedness and birth stress.

6:6.2 The increased incidence of left-handedness in twins, particularly those of monozygotic origin.

It is usually claimed, a) that MZ twins have a higher incidence of left-handedness than do DZ twins, and that b) twins in general have a higher incidence of left-handedness than do singletons. Since neither of these items could conceivably have a genetic basis, it is argued that pathological factors, plus mirror-imaging in the MZ twins are responsible for the increased sinistrality. In Chapter 4 I have critically reviewed the evidence for these statements, and have concluded that neither is supported by the evidence.

6:6.3 Left-handedness in epileptics

Milner, Branch and Rasmussen (1964) found a higher incidence of left-handedness in chronic epileptics who had an early as opposed to a late cerebral lesion. Penfield and Roberts (1959) (data presented in section 6:5.2 above) found a higher incidence of left-handedness in chronic epileptics whose lesion had occurred before the age of 2, than in those whose lesion occurred later in life. Neither of these studies contributes anything to the present study's purposes. The definition quoted in Section 6:2 specifically says that hand preference alone must be altered by a lesion. In the studies described above we have no idea as to the frequency of right hemiplegia,

etc., and thus many of these 'pathological' left-handers might well fit into Gordon's first category, that is left-handers of necessity.

Amongst less severe epileptics the evidence for an increased incidence of left-handedness is poor. In Chapter 5 I have reported evidence from the NCDS data in which there was no relation between handedness and a history of epilepsy (defined in a large number of ways). It is possible that other studies have found an increased incidence of left-handedness in epileptics; but these studies are possibly all invalidated by the a priori expectations of the researchers causing a criterion shift in the definition of left-handedness in the epileptics. Certainly future studies in this case, as with those concerning twins (see Chapter 4:4), should be carried out with the experimenter blind as to the epileptic status of the child being examined.

6:6.4 The increased incidence of left-handedness in the mentally sub-normal, retarded and ill.

It has been pointed out by several investigators (see Satz, 1972 for a review), that left-handedness is more frequent amongst the mentally sub-normal, and those who are disabled learners. Stutterers also appear to have an increased incidence of left-handedness.

Table 6:3 shows data from my own analysis of the NCDS data (see Chapter 3:3 for further details of this dataset); it is clear that mental retardation does indeed correlate with left-handedness. Calnan and Richardson (1976b) using the same NCDS data-base, found that left-handedness related significantly to poor speech, and to several attainment tests.

Several studies (Lishman and McMeekan, 1976; Gur, 1977; Colby and Parkinson, 1977; Boklage, 1977) have found an increased incidence of left-handedness in psychotic patients.

None of the facts so far presented in this section are disputed; what I do find contentious is the logic which allows these data in isolation to be interpreted as evidence for pathological left-handedness, as do, for instance, Satz (1972) and Bakan (1978). In none of the cases is it known, for instance, whether the familial incidence of left-handedness is the same as an appropriate control group; it may well be that left-handers are more prone to certain conditions, rather than the conditions causing their left-handedness.

Thus far all the evidence criticised has been very indirect. To my knowledge there is no direct evidence of a link between cerebral trauma and left-handedness. Table 6:4 shows my own analysis of NCDS data in which mothers

of children were asked if their child had ever been knocked unconscious, or suffered a fractured skull, Whilst not perfect these two variables might be expected to correlate with cerebral trauma of the closed head injury type. Despite a large sample size, there is no evidence of a relation between handedness and cerebral trauma. Although very inadequate, these data are, as far as I am aware, unique, and provide yet further evidence for the invalidity of the concept of pathological left-handedness.

6:7 Conclusions

> " ... if I wish to prove anything, experiment and reason for me take the place of authorities ..."
>
> Paracelsus

Despite the claims of many authorities, a critical review of pathological left-handedness reveals that there is no adequate evidence to substantiate the concept, and indeed the idea probably finds its origins in repeated attempts to prove Dax's Law. The present author would accept that pathological left-handedness might possibly occur very rarely, although as yet there is no convincing evidence for such a phenomenon. Certainly pathological left-handedness cannot, as many authors would claim, be very frequent.

Table 6.1 Shows, using data from Goodglass and Quadfasel, (1954), the assessed language laterality of 50 left-handers according to their usual hand of writing

	Assessed language laterality			
Writing hand	Left	Right	Total	% Left
Right	20	15	35	57.1%
Left	5	10	15	33.3%
Total	25	25	50	50.0%

Table 6.2 Data from Penfield and Roberts (1959), showing the incidence of post-operative aphasia after right or left-sided operations, in right and left-handed chronic epileptics. Patients are divided into those with (+) and those without (-) evidence of early brain damage.

		Left-sided lesions			Right-sided lesions		
hand-edness	early brain damage	N	aphasics N	aphasics %	N	aphasics N	aphasics %
Right	-	157	115	73.2%	196	1	0.5%
	+	22	10	45.4%	58	0	0%
Left	-	18	13	72.2%	15	1	6.7%
	+	49	6	12.1%	7	1	14.0%

Table 6.3 Doctor's assessment of child's degree of mental retardation at the age of 7.

	N	% L
None	11538	11.2%
Present, but no handicap	68	13.2%
Handicap slight (ESN)	101	18.8%
Handicap moderate (ESN)	62	21.0%
Handicap severe (severely subnormal, not at school)	16	50.0%

$$X_4^2 = 35.57,\ p < 0.001$$

Table 6.4 Mother's history of whether the child has ever had any head trauma; information collected at the NCDS second sweep when the child was 11 years old

Question	Response	N	% L	Chi2	Sig
Has the child ever had a fracture of the skull?	No	12010	11.1%	2.69	NS
	Yes	1646	12.5%		
Has the child ever been knocked unconscious in an accident?	No	13096	11.3%	0.004	NS
	Yes	559	11.4%		

Figure 6.1 Shows the cumulative number of cases reported of left-handers with either right or left-sided lesions which produced aphasia, by date of publication. Data from Goodglass and Quadfasel (1954).

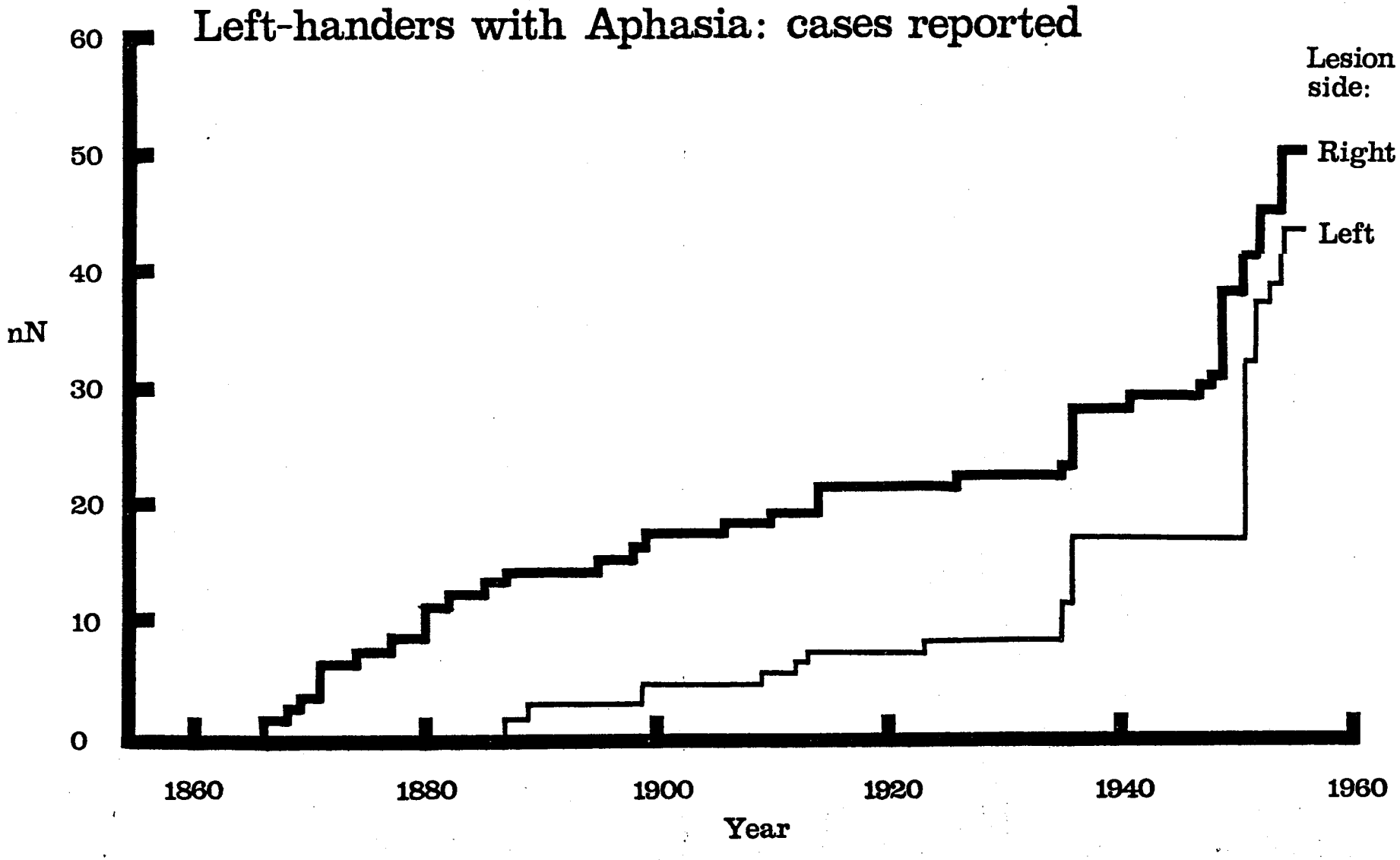
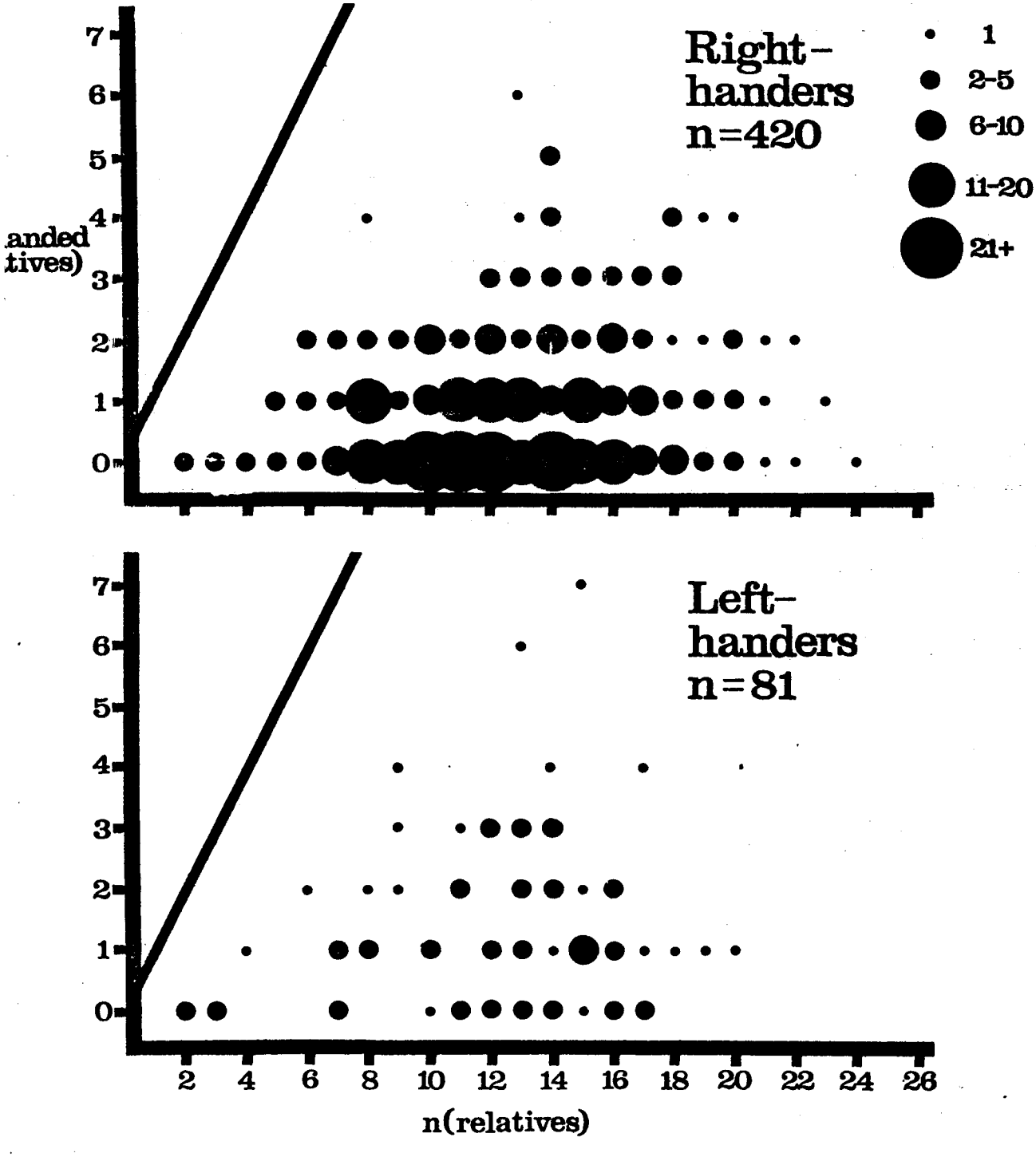

Left-handers with Aphasia: cases reported
60
50
40
nN
30
20
10
0
1860
1880
1900
1920
1940
1960
Year
Lesion side:
Right
Left

Figure 6.2 Shows, separately for right and left-handers, the number of left-handed relatives reported as a function of the total number of relatives reported. The data are described in more detail in Chapter 2.

Family history of handedness



CHAPTER 7: A GENETIC MODEL OF HANDEDNESS

"the determinant for the distribution of
right and left in the animal kingdom is
what we generally call chance"

Ludwig (1932) cited in Fritsch (1968, p 103)

7:1 The requirements of a model of handedness

Any model of handedness has to meet certain minimal requirements.

i) It must account for the data in the literature showing a familial trend in handedness. Left-handers represent about 10% of the children of two right-handed parents; 20-25% of the children of one right and one left-handed parent; and about 40% of the children of two left-handed parents. (see Table 7.1 for further details).

ii) It must explain the relatively high proportion of monozygotic twin pairs which are discordant for handedness (that is, in which one is right-handed and the other is left-handed).

iii) It should account economically for the phenomenon of cerebral speech dominance, which is partially correlated with handedness; thus using dichotic listening tests, unilateral ECT or the sodium amytal test, about 10% of right-handers and about 35% of left-handers show right hemisphere speech dominance. It should also account for the discrepancy between the incidence of crossed speech dominance in right-handers as assessed by dichotic tests (about 10%) and that assessed from clinical studies of aphasia (about 1-3% at maximum).

iv) The model should be compatible with the known inheritance of other biological asymmetries e.g. <u>situs</u> (the laterality of the viscera), and possibly also hand-clasping and arm-folding (see Chapter 9 for an account).

v) The model must be biologically convincing. Morgan (1976) has pointed out that a genetic model may be fitted to any data as long as sufficient allellic pairs of varying penetrance at different loci are postulated.

In this and the following chapter I wish to present a two-allele, single-locus model, with complete penetrance in the homozygotes, and 'additivity' of the heterozygote; this model I will suggest, can satisfy all of the above requirements.

Before starting to fit a model to the handedness data it is necessary to clarify several points.

It is not possible to fit a genetic model unless the phenotypes are clearly known. Earlier, in Chapter 2, I have discussed the phenotypic description of handedness at some length and have concluded that there are, at least for genetic purposes, exactly two types of handedness, right and left, and that the best criterion for determining to which of the two categories an individual belongs, is the hand used for writing. In adopting such a position I am in complete disagreement with the position of Annett (e.g.

1976, "Hand preferences vary not discretely but continuously"); this inevitably conditions the nature of our respective genetic models, since, of necessity, hypothesised genotypes are closely dependent upon supposed phenotypes.

I am probably in disagreement with a large number of authors in that I feel that pathological left-handedness (and of course, by symmetry, pathological right-handedness), are either very rare, or possibly even non-existent. Certainly I do not feel that left-handedness can be regarded as secondary to birth trauma or birth stress (as does for instance Bakan, 1978), and in Chapter 3 I have given extensive empirical evidence for this position. But neither do I feel that pathological left-handedness occurs in any other form; my evidence for this position has been presented in the previous chapter.

I take such an apparently iconoclastic position on these issues since I feel that thus the solution to the problem of the origin of left-handedness is rendered less obscure.

7:2 The biological background

In discussing the biology of asymmetry we must clearly distinguish fluctuating asymmetry from directional asymmetry.

7:2.1 Fluctuating asymmetry

Biological and physical systems inevitably contain noise; Molecules buffer against one another in quantal jumps according to the stochastic laws of thermodynamics. The result is that any system which starts off being symmetric will ultimately show some degree of asymmetry simply as the result of chance accumulation of noise. (Note that this is independent of any fundamental sub-atomic asymmetry). These asymmetries will show a symmetric distribution about a mean of zero.

During early embryonic development small numbers of cells are involved and thus the relative role of chance variations becomes much larger. As a result of what Waddington (1957) has called 'canalisation' such asymmetries are liable to become fixed, and may result in the relatively large asymmetries of adulthood (larger, that is, than might be expected on a purely chance basis). The net result of such chance or fluctuating asymmetries is that 50% of a population will tend to have one side 'dominant' (of whatever organ, tissue, or function is being considered), and the rest of the individuals will have the opposite side 'dominant'; this is equivalent therefore to a chemist's racemic mixture of stereo-isomers. Fluctuating asymmetry may be demonstrated experimentally, and is usually investigated in the teeth or dermatoglyphics, both systems becoming fixed early in embryonic

life. Events increasing the level of 'biological noise' during early fetal development, such as heat (Siegel et al, 1977), audiogenic stress (Siegel and Smookler, 1973), and behavioural stress (Siegel and Doyle, 1975), result in greater degrees of fluctuating asymmetry (measured as increased variance, the mean remaining zero).

An important point about fluctuating asymmetry is that none of its variance can ever be genetically controlled (hence the name: the asymmetry fluctuates randomly from generation to generation).

7:2.2 Directional asymmetry

Fluctuating asymmetry is undoubtedly (and inevitably) a common event during ontogeny. Nevertheless on its own it cannot account for any of the asymmetries which are of interest to the psychologist or biologist, that is, asymmetries in which the mean of the distribution of (L-R) is not zero (or in the case of unilateral conditions, n(L) is not equal to n(R)); such conditions are said to show directional asymmetry.

Directional asymmetry is biologically very different from fluctuating asymmetry. That it is of greater importance is suggested by the fact that in its extreme forms we do not even question its occurrence. Why the heart is on the left is neither a simple not a trivial

question. If the heart were on the left in 50% of cases then the answer might be simple - that fluctuating asymmetry followed by canalisation inevitably produced it that way. To use a physical metaphor, to maintain an asymmetry we must search for a stabilising force, for otherwise the system would inevitably return to symmetry; or rather, a directional asymmetry will return to a fluctuating asymmetry.

Whilst directional asymmetry can (and one might argue, must) have some form of genetic control, there is an important limitation upon such control. Morgan (1976) has argued that it is, in principle, unlikely that a gene carries direct information about chirality; that is, paired alleles have not been shown, in any convincing case, to produce enantiomorphic phenotypes in their carriers. Thus, if one homozygote of an allelic pair produces one particular directional asymmetry, then the other homozygote cannot produce the mirror-image directional asymmetry. (An apparent exception to this rule is discussed in detail in Chapter 9). The present model relies heavily upon Morgan's principle for its origins, although not, of course, for its justification as a satisfactory fit to the data.

The need for a new model of the genetic control of handedness is two-fold. First I will argue that no previous model adequately fits the available data. Second, I will suggest that all previous models (with the single exception

of Annett, 1978) have not satisfied Morgan's principle, since they have alleles which are symmetric in their effects. As a result I will argue that any adequate model of the inheritance of asymmetry must itself be inherently asymmetric. This principle may be satisfied in the case of handedness by allowing one homozygote from an allelic pair to produce a directional asymmetry, and the other homogygote to 'produce' a fluctuating asymmetry; that is, the latter genotype has no control over the asymmetry and hence it is, in effect, a null gene.

The inter-relation of directional and fluctuating asymmetry may be seen in a series of morphological examples.

In most vertebrates the heart and stomach are on the left and the liver is on the right. This situation (situs solitus), (SS), is occasionally mirror-reversed to give situs inversus, (SI). Spemann and Falkenberg (1919) found that tying a fine thread around the mid-line of a developing newt embryo produced conjoined twins. The left-hand member of the pair almost always showed SS, whilst the right-hand member showed SI in about half of the cases. A similar situation exists if the embryo is completely split in two (Ruud and Spemann, 1923). Analogous findings have been reported in conjoined trout (Lynn, 1946) and conjoined human twins (Newman, 1940). In other animals SI has been shown to be produced teratogenically (Shehenfelt, 1974), by cold (Newman, 1925)m or by irradiation

(Wilson et al, 1953), but in no case does the incidence rise above 50%

The iv mutation in the mouse, when homozygous, produces SI in exactly 50% of cases (Layton, 1976). Nevertheless the iv gene shows complete penetrance. When two iv homozygotes are mated then the progeny show 50% of SI, irrespective of the particular phenotype of the parents. A similar situation exists in another mouse mutation (Tihen et al, 1948) producing SI, and also in the platyfish Xiphorus maculatus (Baker-Cohen, 1961). In man, Kartagener's syndrome, of chronic sinusitis, bronchiectasis and situs inversus, is inherited as an autosomal recessive, with the limitation that only 50% of homozygotes show the SI component of the syndrome (Afzelius, 1976). Amongst human monozygotic twins showing situs inversus, 46% of 13 reported pairs are concordant, the rest discordant (Lowe and McKeown, 1953); the expected values are 33.3% concordant and 66.6% discordant.

Directional and fluctuating asymmetry can also be seen in the arrangement of the optic chiasma in the flatfish, the Heterosomata (see Chapter 9 for further discussion).

A behavioural example of fluctuating asymmetry is that of Collins (1970), who looked at the pawedness of mice which had been inbred for 28 generations (and hence we may assume were almost certainly homozygous at all loci).

Of these animals 50% were left-pawed and 50% were right-pawed, with no evidence of any inheritance over a further three generations (Collins, 1970).

7:3 The data to be explained

7:3.1 Familial data

The literature contains seven studies of the incidence of left-handedness in the progeny of R x R, R x L and L x L matings (Ramaley, 1913; Chamberlain, 1928; Rife, 1940; Hubbard, 1971; Annett, 1973; Ferranato, 1974; and Annett, 1978). In using these data sets certain modifications have been made. The data of Chamberlain (1928) have been used as quoted by Annett (1973), not withstanding the trivial inconsistencies demonstrated by Levy, 1976. In using the Chamberlain data I have considered only those individuals obtained by random sampling since the incidence of left-handedness is higher amongst those families obtained by special appeal (Chi-squared = 3.43, 1 df, $p < 0.10$), and thus one suspects a response bias. Similar considerations for the data of Annett (1973) mean that only the randomly obtained data have been included. For the data of Hubbard (1971) it has been assumed that all of those progeny with one left-handed parent have only one left-handed parent; on the basis of random assortative mating one would have expected only four individuals to have come from L x L matings. Whilst I would also have liked to use the data of

Falek (1959), this was not possible due to the sample not being complete, and there being no indication of the overall incidence of left-handedness in the propositus generation.

As well as the above published studies, I have also been fortunate in being able to use the results of two unpublished studies. Dr. C.G.N. Mascie-Taylor, of the Department of Physical Anthropology, University of Cambridge, allowed me to use data from a survey carried out by him in 1977 in a Cambridge suburb; other aspects of this study have been reported elsewhere (Mascie-Taylor, 1979a, b; Mascie-Taylor and Gibson 1978, 1979). The data were obtained from a comprehensive study of a population, there being almost no failures to respond, handedness being directly assessed by the researcher, and sampling was truly random. Second, Drs. Chaurasia and Goswami, of the School of Biological Sciences, Bhopal University, India, have allowed me to quote data they obtained from a questionnaire distributed to a large number of their students.

I have also used 5 distinct sets of data obtained from two large-scale surveys of my own. The data of survey 1 were obtained by questionnaire given to undergraduates at the University of Cambridge in May 1977; the students were asked to provide information on their own handedness and on the handedness of their siblings,

parents and grand-parents (it was stressed to them that it was preferable to not reply to a question rather than to guess at an answer). Left-handedness was classified on the basis of writing hand, with the single exception that those right-handed writers who originally wrote with their left-hand, but had been forced to write with their right hand,were classified as left-handed. Two sets of family data were obtained from this study; first, the students' and students' siblings handedness as a function of parental handedness (ICM-1 propositus generation); and second, the parents' handedness as a function of the grand-parental handedness (ICM-1 parental generation). The response rate to the questionnaire was difficult to assess accurately but was of the order of 50%. The second study, survey 2, was carried out in June 1977, and the questionnaire was given to all Cambridge graduates who were collecting their degrees in person. The questionnaires were distributed on the eve of the graduation ceremony. Information was thus obtained not only from the students, but also from other members of the students' family, so that fairly accurate information could be obtained on the handedness of parents, parents' siblings, the grand-parents, and the great-grand-parents, as well as on the students' siblings. Once more it was emphasised that questions should be answered only if the answer was fairly certain, guessing being discouraged. Handedness was classified as in the previous study. From this study three separate sets of family data could be obtained; students and students's siblings as a

function of parental handedness (ICM-2 propositus generation); parental and parental siblings' handedness as a function of grand-parental handedness (ICM-2 parental generation); and grand-parental handedness as a function of great-grand-parental handedness (ICM-2 grand-parental generation). The response rate of this study was of the order of 20%. Other aspects of these questionnaires have been discussed in some detail in Chapter 2.

The data from all these studies are collected together in table 7.1 The data from L x L matings are relatively restricted, and, except in the case of Chamberlain (1928), I have lumped together data from separate studies with broadly similar overall incidences of left-handedness in the progeny.

Figure 7.1 shows the data of Table 7.1 plotted graphically, with the proportions of left-handed progeny of the three mating types shown as a function of the overall incidence of left-handedness amongst the progeny. Note that when this is done it is clear that there is a fairly clear relationship between the data from all of the studies.

That there are differences in incidence of left-handedness in the progeny of various studies is clear from Figure 7.1. These differences are far too large to be simply a function of sampling errors. The differences are

far too large to be simply a function of sampling errors. The differences are compounded in their complexity, by differences in incidence of left-handedness in the parental generations of different studies, even when the effect of progeny variation is partialled out.

These differences in incidence represent a major problem for any model of left-handedness. One can hardly make the usual assumption of classical genetics, that all differences in incidence are due to differences in allele frequencies, since it is clear that by using different definitions of left-handedness, the same study may be used to produce different progeny incidences of left-handedness. Clearly in such a case it would be absurd to argue that the allele frequencies had also changed. There are two approaches to the problem. We may follow Nagylaki and Levy (1972) and Levy (1976) who argue that data are invalid for genetic analysis unless the parental incidence of sinistrality is equal to the progeny incidence of sinistrality. That process of exclusion left them with only a single set of data, that of Rife (1940). They still however, make the assumption that Rife's criterion is the correct one; if one cared to dispute Rife's definition of handedness one would be left with absolutely no data at all with which to test a model. This position seems somewhat unsatisfactory. The alternative position, adopted by Annett (1978), seems far more realistic, In the first instance one assumes that all studies have the same allelic proportions, and hence

have the same proportions of the true phenotypes (i.e. in her case, RS+ and RS-). Differences in apparent proportions of sinistrality are then due to differences in criterion or threshold of left-handedness. As she points out (Annett, 1978), this has a close similarity to the methodology of signal detection theory where, despite differences in response bias (Beta), one is trying to find a true sensitivity (d'). In the following analysis I shall follow Annett in attempting to make corrections for differences in incidence of left-handedness, and thus will attempt to fit all of the available data, warts and all. My actual calculations for these corrections will however be somewhat different to those of Annett.

Appendix A7:3 discusses the possibility of assortative mating for handedness, and concludes that it is of little or no consequence.

7:3.2 Twin data

There are eighteen different sets of twin data in the literature; these are summarised in Table 7.2.

Several claims have been made about handedness in twins:-

i. that monozygotic (MZ) twins show binomial proportions of R-R, R-L and L-L pairs (and hence that handedness cannot be under genetic control).

ii. that MZ twins have a higher incidence of sinistrality than do dizygotic (DZ) twins.

iii. that twins in general have a higher incidence of sinistrality than do singletons.

In Chapter 4 I have critically reviewed evidence for these statements and find none of them adequately supported by the data. This is important, since on the basis of (ii) and (iii) in particular, Nagylaki and Levy (1973) have argued that twins are not suitable for fitting to genetic models, since twins show increased pathological left-handedness, and also, in MZ twins, "ectodermal mirror-imaging". Since there is no evidence for ii and iii, genetic models should be able to cope with data from twins as well as that from singletons. In particular, as Corballis and Beale (1976) have pointed out, the model of Levy and Nagylaki cannot cope with the MZ twin data at all.

As with data from the singletons, so in the case of twins it is necessary to account for differences in incidence of sinistrality between studies. Once more I shall assume, along with Annett, that the differences are due to variation in threshold or criterion of left-handedness, and that in all studies the underlying allele frequencies are the same in each case. Unlike Annett I shall assume that the phenotypes in twins are also the same as in singletons.

7:4 Previous models of handedness

It is instructive to compare previous models of handedness; for convenience the essential genetics of each are summarised in Table 7.3.

Ramaley (1913), not long after the re-discovery of Mendel's laws, proposed that there were two alleles which we may label L and R. L was recessive and there was complete penetrance. Thus all LL individuals were left-handed, whilst all RR and RL individuals were right-handed. As Ramalay himself recognised this model fails since it predicts that all of the children of two left-handers should themselves be left-handed; even a single exception, such as he reported, and as were later reported by Jordan (1914), disproves the hypothesis. Rife (1950) proposed a modified form of the Ramaley model. Not only were LL genotypes left-handed, but so also were a proportion of the heterozygotes. This model thus allows that neither right nor left-handers breed perfectly true, since the left-handers' genepool now contains R alleles, and vice-versa. Trankell (1955) produced a model which was also a variant of Ramaley's model, In Trankell's model it is the LL genotypes rather than the heterozygotes which show partial penetrance, and hence allow L x L matings not to breed true. Whilst both are improvements upon Ramaley's model, neither that of Trankell nor that of Rife will adequately fit all of the requirements. although as will be seen below, the Trankell

model can be made to fit quite closely.

Annett (1964) proposed yet another variant upon the Ramaley theme. Annett complicated the problem by arguing not that penetrances in the Ramaley model were wrong, but that the phenotypic classification was itself in error. Thus she proposed that the heterozygotes produced a separate and distinct phenotype known as 'mixed' handedness. This model clearly fails since both R x R and L x L (as opposed to M x M etc.) matings should breed true, and yet the data of Annett (1972) clearly shows that this is not the case. The logic of increasing the number of phenotypes is also somewhat worrying since it inevitably means that all previous data sets in the literature are inadequate for testing the model.

Levy and Nagylaki (1972) proposed a yet more complex model. They argued that it was necessary to postulate four alleles, two at each of two loci, the two loci segregating independently. There are thus nine genotypes. They also postulated yet another increase in the number of phenotypes, arguing that as well as individuals whose writing hand was the right or the left, each group could be sub-divided into two further groups according to whether cerebral control of the hand was ipsilateral or contralateral. The possibility of ipsilateral hand control, whilst intriguing, seems not to be supported by either anatomical or clinical evidence. That not all pyramidal fibres decussate in the medulla is

well accepted; and it seems probable that in a few cases no decussation takes place at all at the medullary pyramid. But this does not demonstrate that control of the hand is ipsilateral, since the fibres might well decussate at other levels. The critical observation, which receives no adequate support from the literature, is whether individuals occur in whom after a cerebral lesion there is ipsilateral loss of motor control

The most serious criticism however of the Levy and Nagylaki model is that it simply fails to fit the data adequately. Hudson (1975) has pointed out the model's failure to account for the data of Annett (1972) or Chamberlain (1928). More seriously still, as Corballis and Beale (1976) have pointed out, the model can in no way account for the twin data, since it makes no allowance for monozygotic discordance of handedness.

The most recent model of handedness is that of Annett (1978). This model was first postulated in 1972, but only in 1978 were the actual genetic mechanisms stated. The model is completely different to any of the others described. Its most important aspects are that it is compatible with Morgan's principle, and that it can account for different manifest incidences of left-handedness in different studies. It also makes predictions about clinical data on aphasia and speech dominance (Annett, 1975, 1976), although these predictions are rather less powerful when it is realised that

in the later paper these data are themselves used to define the parameters of the model.

Annett's model proposes that there are two phenotypes, RS+ and RS-. RS- individuals are normally distributed with mean 0.0 and variance of 1. RS+ individuals have a normal distribution, but are shifted to the right and have a mean of z, with a variance of 1. A threshold, x, differentiates right-handers from left-handers. Annett (1978) shows that the model is capable of dealing with all the family data, and indeed my calculations show that this is so for all of the 14 studies of Table 7.1 (see Figures 7.2 and 7.13). The great defect of the model is that in order to account for the twin data, Annett has to postulate that the RS+ distribution has a different mean to that in the singleton model. In other words, the model proposes that the inheritance of handedness is different in singletons and in twins, This is a serious objection, and certainly from my calculations there is no possible single pair of values of z and the proportion of RS- individuals, which will adequately fit all of the data of table 7.1 and of table 7.2,no values being acceptable at better than the one in a million probability level. Annett's error, I would argue, is not in the form of the model - that is correct, and I would argue that in some sense the model I wish to propose is a special case of the Annett model - but in her description of the phenotypes. By allowing handedness to be a continuous rather than a discrete variable, her model becomes

distorted, and hence fails. And of course the repeated fitting of yet more family data alone, as in Annett (1979), will never convince one of the overall adequacy of the model.

In summary, most of the previous models of handedness have failed as they do not satisfy Morgan's principle. The sole exception is the model of Annett (1978), which I would suggest fails due to its insistence upon an unrealistic phenotypic conception of handedness.

7:5 The Model

As stated earlier, the present model is explicitly based upon the information described in 'The Biological Background' (section 7:2 above). It is proposed that there are two alleles, D and C. Allele D (for Dextral), produces 100% right-handers when it is homozygous. Allele C (for Chance), 'produces', in its homozygous form, pure fluctuating asymmetry (i.e. there is no control over the phenotypes of the progeny). Thus exactly 50% of the CC genotype are right-handed, and 50% are left-handed. Note that in this case, 50% is not an arbitrary figure (45% or 23%, for example, could not be fitted to the same argument), but is a precise statement of a particular genetic manifestation. Also it must be emphasised that although only 50% of CC individuals are left-handed, this does not mean that the CC genotype is only partially penetrant. It shows 100% penetrance

for its real phenotyoes, which is fluctuating asymmetry, and hence, as a secondary consequence, produces only 50% of left-handers.

Thus far the model can be defined a priori from a knowledge of the biology of asymmetry. The next step is less predictable. The problem is that the proportion of left-handers to be expected from the heterozygote, DC, is undefined. If C is recessive (i.e. the heterozygote manifests as the commoner of the two homozygotes) then one would expect 100% right-handers from the DC genotype. Conversely if C is dominant (i.e. the heterozygote acts as the less common of the two homozygotes) then one would expect 50% of left-handers from the heterozygote. But of course there could also be an infinity of intermediate positions in which the heterozygote produces a proportion, x, of left-handers, where x is between 0 and 0.5. In particular I wish to call the position exactly intermediate between the two extremes, 'additive'. The additive DC genotype thus produces 25% left-handers and 75% right-handers. Whatever the particular value of $p(L|DC)$, the use of values other than 0 or 100 may be seen as examples of the 'random phenotype concept' (Birnbaum, 1972).

The models to be discussed are thus a restricted set of those to be called model I in Chapter 9.

Genotype	p(left-hander)	p(right-hander)
DD	0.0	1.0
DC	x/2	1.0 - x/2
CC	0.5	0.5

The parameter x controls the degree of dominance of the heterozygote. Thus if x = 1, then C is recessive, if x = 0 then C is dominant, and if x = 0.5, then C is 'additive'.

Thus far it is a relatively simple matter to choose a particular value of x (thereby defining the genetics entirely), and to predict the proportions of left-handers which would result from particular parental pairs, or in particular twin-types (for details of the calculations see the Appendix). The major problem is to fit the models when the apparent incidence of left-handedness is different in different studies, and one has no idea which, if indeed any, of the values is actually correct. To solve this problem it is necessary to define two incidences of handedness. Let $p(L_m)$ be the manifest proportion of left-handers in a particular population; thus $p(L_m)$ can vary between populations, or even within a population if the criterion for left-handedness should be changed.

By contrast, let $p(L_t)$ be the true incidence of left-handedness, which I would propose, at least in the first instance, is constant in all the different populations being studied. Clearly, whilst $p(L_m)$ can be directly estimated

from the data, the value of $p(L_t)$ may only be hypothesised. In reality, the process of calculation is to hypothesise particular values of x and $p(L_t)$, and then to find the fit of that particular model. The calculation then proceeds combinatorially, for all possible values of x and $p(L_t)$ in the desired range, finding all of those which fit adequately.

Given a particular pair of values of x and $p(L_t)$ one obtains predictions which are the same for all datasets. Clearly these must be modified for each particular dataset, from a knowledge of $p(L_m)$. To do this requires assumptions about the processes involved in altering the value of $p(L_t)$ to that of $p(L_m)$. There are two major ways in which $p(L_m)$ may not equal $p(L_t)$.

i. A different criterion of left-handedness may be used ('criterion shift').

ii. There may be a response bias, whereby left-handers tend not to respond to questionnaires (as perhaps may have been true at the turn of the century) or they tend to over-respond to questionnaires (as may well be true now). And of course separate corrections must be made for both the parental and propositus generations, $p(L_t)$ for parents, being assumed to be equal to $p(L_t)$ for the progeny.

Scrutiny of the origins of the data presented in Table 7.1 suggests that in all parental cases, and almost all of the progeny cases, criterion shifts cannot be eliminated. But similarly in most surveys, response biasses cannot be eliminated either. Exceptions are my own surveys, in which I am forced to argue that the correct criterion of left-handedness has been used, and the study of Mascie-Taylor, who I would also argue has used the correct criterion, and in which study there was probably no response bias since there was almost one hundred percent ascertainment; it is noteworthy that in that study the parental and progeny incidences are equal, and the incidence of sinistrality is very close to that which I will later hypothesise. In Appendix 7:2 I give details of the calculations using both methods, and suggest that in most cases they give sufficiently similar results to mean that they are, for most practicable purposes, indiscriminable. I have therefore, for simplicity, assumed that all differences in incidence of $p(L_m)$ are due to criterion shifts alone.

Clearly in applying the corrections for a criterion shift one is assuming that two separate processes are occurring simultaneously:-

a. some true right-handers are manifesting as left-handers, and
b. some true left-handers are manifesting as right-handers.

Without further data it is not possible to discriminate between these two processes. In order to be able to carry out the calculations I have therefore assumed that if the manifest incidence of left-handedness is less than the true incidence, then this is entirely due to true left-handers manifesting as right-handers (i.e. there are no true right-handers manifesting as left-handers); and vice-versa for $p(L_t) < p(L_m)$.

Making these assumptions it is possible to fit the data of Tables 7.1 and 7.2 to a whole series of models, and to find which are compatible with the data. The details of all calculations are given in Appendix 7.2

7:6 Fitting the model

Fitting the model of handedness requires estimation of two parameters, x, the degree of dominance (i.e. $p(L|DC)$), and $p(L_t)$, the true indidence of left-handedness. This fitting has been carried out by predicting the expected results for particular values of $p(L_t)$, and x, and then testing the goodness of fit by means of a Chi-squared test. This is carried out for all possible values of x in the range 0(0.05)1.0, and $p(L_t)$ in the range 0.05(0.005)0.25; values of $p(L_t)$ have been used outside of this range, but they do not give interesting results (there being no fit at all), and they will not be considered here. Details of the method of goodness of fit testing are given in Appendix 7:1.

Before fitting a new model it is necessary to confirm that none of the old models is capable of fitting the data when the new corrections are made for differences in the incidence of handedness. Figure 7.3 shows the goodness of fit of the Rife model, that is a model in which $p(L|RR) = 0.0$, $p(L|LL)=1.0$, and $p(L|RL) = x$, where x is in the range of 0.0 to 1.0. A special case of this model, when $p(L|RL) = 0.0$, is the Ramaley model. Figure 7.3 shows that particular values of x, and the true incidence of left-handedness will adequately fit either the twin data, or the family data, but that there is no overlap at all of the distributions, and indeed all Chi-squared values for the combined data are massively significant, the lowest being, for $p(L_t) = 0.25$, and $p(L|RL) = 0.4$, equal to 194.5, with 66 degrees of freedom (liberal estimate of df, see appendix 7:1).

Figure 7.4 shows the rather different result for the Trankell model, in which $p(L|RR) = 0.0$, $p(L|RL) = 0.0$, and $p(L|LL)$ can vary in the range from 0.0 to 1.0. As with the Rife model, when $p(L|LL) = 1.0$, this is also the Ramaley model. Once more there are adequate fits for both the twin data and the family data considered on their own; this time there are also adequate fits for the combined data, the best fit being with $p(L_t) = 0.075$ and $p(L|LL) = 0.30$ (Figure 7.5). Despite, therefore, the original reservations about the theoretical inappropriateness of the Trankell model, it is capable of fitting the available data adequately; this point will be returned to

later in a more general discussion of my own model, and of the models in general.

We may now fit the data to the new model.

Figures 7.6a, 7.6b and 7.6c show the goodness of fit of various forms of the model to the data from R x R, R x L and L x L matings. It can be seen that the R x R data discriminates little between models, the R x L data discriminates well, and that the L x L data also discriminates moderately well (although [redacted] inevitably hampered by small sample sizes). The goodness of fit of the combined family data is shown in Figure 7.6d. A whole range of models will fit this data, from dominant through to recessive, with different values of $p(L_t)$ for particular models.

Figures 7.6e and 7.6f show the goodness of fit of the data from MZ and DZ twins. Whilst DZ twins appear to discriminate very little between models, they clearly exclude the a priori probable models, i.e. the recessive and dominant. The MZ twins show a far greater selectivity. Combining the results of the family studies, and the MZ and DZ twins, we obtain the distribution of Figure 7.7. It can be seen that there is now a relatively small area which can account for all of the data. The minimum Chi-squared value of 70.6 is found at values of $p(L_t)$ of 0.080. and degree of dominance 0.25 (that is the hetero-

zygote produces 12.5% left-handers). However there are several other pairs of values which produce almost equally good fits. In particular I would like to emphasise those models in which the degree of dominance is 0.5 (that is, that the heterozygote produces 25% left-handers, exactly mid-way between the two homozygotes). I will call this model additive, after the usage adopted in polygenic models, although the analogy is not exact, since in polygenic systems it is the <u>individual</u> phenotype which is mid-way between extremes, whereas in the present case it is the <u>population</u> phenotype which is mid-way between the homozygotes. One advantage of the additive model is that as will be shown in Chapter 9, models to be fitted to hand-clasping and arm-folding data also have to have similar 'additive' genes, and that at least one such pair is necessary.

Considering the additive models, the miniumm Chi-squared value is 79.4, at a $p(L_t)$ of 9.0%: however, at $p(L_t)$ of 9.5% the Chi-squared value is only 79.5. For reasons which will becomeapparent later, I have chosen to concentrate upon this latter value.

Tables 7.1 and 7.2 show, as well as the raw data for families and twins, the predictions of these two particular versions of the model (i.e. $p(L_t)$ and degree of dominance of 8.0% and 9.5%, and 0.25 and 0.5 respectively). Scrutiny of this table shows several features. First, the two models

often show only minimal differences in their predictions, thus making discrimination very difficult. Second, the overall Chi-squared values are heavily dependent upon just a very few datasets. In particular the data of Hubbard (1971), and my own study (ICM-2-grandparents) account for much of the Chi-squared in the family data. The Hubbard study was difficult to fit as R x L and L x L data had been lumped together; my own grand-parental study had a high a priori probability of being in error since it was assessing the handedness of the propositus' grand-parents as a function of the propositus' great-grand-parents. Amongst the twin studies, the data of Loehlin and Nichols (1976) seems to fit badly for both MZ and DZ twins. This study was the only twin study to be conducted by means of a postal questionnaire, and thus might be less reliable than the others.

When these aberrant values are excluded, one obtains Chi-squared values of 51.21, with 60 df for the 'a' model, and 46.58, with 60 df for the 'b' model, both values being highly acceptable in statistical terms. These values are also acceptable if one uses a more conservative estimate of the number of degrees of freedom, i.e. 48.

The fit of this slightly reduced data set (i.e. with only 12 sets of family data, and 17 MZ and 16 DZ twin data sets), isshown in Figures 7.8 and 7.9 for my own model, Figure 7.10 for the Rife model, Figures 7.11 and 7.12 for the

Trankell model, and Figure 7.13 for the Annett model. Clearly all of these models might be expected to show some improved fitting to the more homogeneous data sets.

My own model now shows a better fit over a slightly wider range. The Rife model also shows a slightly better fit, but there is still not even a hint of a combined twin and family fit. The Trankell model shows a somewhat improved fit for both family and twin data. The Annett model, like the Rife model, shows better family and twin fits, but still there is no combined fit even at the one in a million level.

In summary, by making assumptions about the nature of the differences in incidence of handedness in different studies, and by using a genetic model in which fluctuating asymmetry plays a large part, it is possible to fit all of the existing data using a single model, and a single prediction of the incidence of $p(L_t)$.

7:7 Discussion

It is still however necessary to consider the fact that the Trankell model seems to fit the data. We may consider both my own model and also the Trankell model (and indeed many other models as well), as mathematical (although not biological) variants of a general model in which $p(L|DD)$ may vary between 0.0 and 1.0, and $p(L|DC)$ and $p(L|CC)$ may also

vary between 0.0 and 1.0. As well as this, of course the value of $p(L_t)$ may also vary to fit the model; the result is a vast array of possibilities. If we allow there to be 21 discrete value of each of the conditional probabilities, and there to be 41 discrete values of $p(L_t)$, then a total of 379701 models can be formed; of course a large number of these models are not possible on logical or mathematical grounds. Nevertheless it should be clear that there is a high probability that models with apparently very different theoretical bases, may well have remarkably similar predictions for the range of situations we are considering here,(although this of course is not to say that they must <u>always</u> be the same). It is also necessary to remember ~~that~~, as Morgan has pointed out, that if one has enough free parameters in a model then of course it may be made to fit almost any data; in this respect my own model, and that of Tranke l, are both superior to the 'general' model in that they both specify clearly in advance two of the four parameters. I would however, like to suggest that there are other reasons for taking more notice of these two models, and indeed in particular of my own one model. At the very beginning of this paper I stressed that there were two further requirements of a model; it must be biologically convincing; and it must give some account of the data from studies of cerebral speech dominance. I would like to argue that the Trankell model is not biologically convincing, since there are good <u>a priori</u> reasons for favouring a model which contains

fluctuating asymmetry.

That my own model is precise in its predictions may be shown by a set of tables. Table 7.4 shows the expected proportions of the three genotypes in individuals of particular handedness and of particular parental types, for a model in which $p(L_t) = 9.5\%$, the inheritance is additive, and it is assumed that manifest incidence is equal to true incidence. The figures are given to 8 significant places to assist any further calculations from the table. Table 7.5 shows the expected proportions of MZ twin pairs by parental handedness, and Table 7.6 shows the probability of MZ twins being of a particular genotype, given their own twin pair-type, and the parental handedness. These data will be used further in the next chapter.

If the present model of handedness is correct then it allows a re-interpretation of the several sets of data in the literature associating left-handedness with mental deficiency (see chapter 6:6.4). These data sets have often been interpreted as evidence that left-handers in general are of lower intelligence, as if the handedness were somehow the <u>cause</u> of the mental deficiency. An alternative view is that a prior severe brain insult, e.g. due to trauma, metabolic or chromosomal abnormality, may have caused the low intelligence, and also have caused a massive amount of 'biological noise' which has over-ridden any pre-existing directional asymmetry, to produce

fluctuating asymmetry, and hence left-handedness in up to 50% of cases. If true then one might also predict that any other developmental abnormality of the brain might also produce a local increase in biological noise, and hence fluctuating asymmetry, and 'phenocopy left-handers'. One possible example of this is in congenital partial or total agenesis of the corpus collosum. The condition is associated with normal intelligence, and is often detected only at routine post-mortem, or as an incidental finding on ventriculography or computerised tomography. Nevertheless a review of the few cases in the literature for whom the handedness is given, shows that of the 19 cases, 9 (47.5%) were described as left-handed (Bossy, 1972; Dennis, 1976; Ettlinger et al, 1972; Ferris and Dorsen, 1975; Field et al, 19 Gardner et al / 1975; Sadowsky and Reeves, 1975). Although this is a small sample, it is tempting to conclude that the incidence of left-handedness in callosal agenesis is indeed raised, as might be predicted from the theory.

As yet no account has been given of why the heterozygote in my own model should be exactly intermediate between the two homozygotes in its expression. Three possible mechanisms can be suggested:-

i. If the D allele produces a buffering substance, and the C allele a disrupting substance, then genotype DD will produce only buffer, and genotype CC will produce only disruptor, thereby resulting in either directional or

fluctuating asymmetry respectively. The heterozygote, DC, would produce both substances, and the particular effect of this would be dependent upon the dose-response curves of the two substances, and might possibly result in an effect midway between the two homozygotes.

ii. The additivity of the alleles might be produced by the phenomenon of allelic restriction (Melnick and Shields, 1976), which is equivalent to autosomal lyonisation. This could only be so if the gene produced its effect at an early enough stage in embryogenesis (i.e. that its action was only expressed in a single cell). This could produce a heterozygote expression midway between the two homozygotes.

iii. The manifestiation of the genes in the two sexes might be different. The classic example of this is the gene for <u>hornless</u> in sheep, which acts as a dominant in males and a recessive in females (Wood, 1905). The manifest incidence of left-handers is often higher in males than females, and there is a possibility also for a tendency for children of left-handed mothers to show a greater incidence of left-handedness than the children of left-handed fathers (Annett, 1973). (Whether such effects are differences in true handedness, or only in manifest handedness is not at all clear at present). The effect of a male-dominant/female-recessive gene is formally equivalent to an additive model when the two sexes are combined.

APPENDIX A7:1: TESTING THE STATISTICAL SIGNIFICANCE OF FIT

Whilst it is a relatively simple task to calculate Chi-squared values for the goodness of fit of particular models, it is necessary to test the significance of such fits. To do this requires knowledge of the necessary number of degrees of freedom. For the twin data this seems fairly simple, one degree of freedom being used to test the fit of the theory to a 3 x 1 matrix (i.e. n(R-R), n(R-L) and n(L-L)). One degree of freedom is used since there are a total of three cells in the matrix, thus setting an upper bound of three degrees of freedom. But one of these degrees of freedom is consumed since it is necessary to know the total sample size (i.e. N), and a further degree of freedom is used since one needs to know the overall incidence of left-handedness (i.e. $p(L_m)$). This leaves a single degree of freedom; and here my calculations agree with those of Annett (1978), who also uses one degree of freedom for fitting twin data.

Fitting the 3 x 2 matrix of family data is not so simple. Classically one would argue that a 3 x 2 table has (3-1) x (2-1) = 2 degrees of freedom; and indeed it would do so if one were testing simple homogeneity of the table with respect to its edge totals. Certainly this argument gives us a conservative lower bound for the degrees of freedom of two. However the table is not strictly a 3 x 2

but is instead 3 tables each of size 2 x 1. It would be quite possible to carry out a survey in which firstly one randomly ascertained the manifest incidences of left-handedness in the separate generations, and then looked for, say, just children of one right and one left-handed parent. One could then predict the expected percentage of left-handed progeny and see how this compared with the observed value, using a Chi-squared with one degree of freedom to test the result. This indeed is what, in effect, I have done in the R x R, etc, sections of Figures 7.2 - 7.13. Since there are three separate tables it seems illogical to have only two degrees of freedom for the combined table. Consider the hypothetical study above in which I ascertained the progeny of only R x L matings. From this I obtain a value of Chi-squared, say X (R-L), with one df. I later look at the same population again and examine just R x R matings, and then obtain a Chi-squared value of X(R-R), also with one degree of freedom. The combined result thus has two degrees of freedom. If now I finally ascertain the L x L matings, I obtain a third value of Chi-squared, X(L-L). If I were now to have to test the combined result of all three studies with only two degrees of freedom, I would be in an awkward position. Let the probability of obtaining the result from the first two combined studies (with a Chi-squared value of X(1,2)), be p(1,2). Then by adding the third study, I produce X(1,2,3), where X(1,2,3) = X(1,2) + X(L-L). And X(1,2,3) has a probability p(1,2,3). But since X(L-L) can only be equal to or greater

than zero, and in all probability, X(L-L) will be greater than zero, it is necessarily true that $p(1,2,3) > p(1,2)$. Thus in obtaining further data I am merely disadvantaging the theory, since by using X(1,2,3) there is necessarily a greater probability of rejecting the theory than by using X(1,2). This seems an absurdity, an absurdity which is readily removed by testing X(1,2,3) with three degrees of freedom.

I would therefore argue, on logical rather than statistical grounds, that one must use three degrees of freedom for fitting the sort of data I have described. In so stating I am disagreeing implicitly with Annett (1978), who uses two degrees of freedom, but does not justify her actions in so doing. Ultimately the best way of resolving this dispute is probably by some form of Monte Carlo simulation, and finding whether two or three degrees of freedom fits the observed values better. In the graphs shown earlier in this paper I have given in general the results for a 'conservative' test (i.e. with 2 dfs), but have also indicated the 5% limits for a more liberal test with three degrees of freedom.

A further statistical problem concerns low expected values in the Chi-squared calculations. Most textbooks stat that expected values must be greater than 5; a few state that 3 is an acceptable lower limit. Most calculations are however looking for significant heterogeneity, where as in t

present study I am primarily concerned with the goodness of fit of predictions to data. Since low expected values can only produce spuriously high values of Chi-squared (and thus produce falsely significant results), I have used a minimum acceptable expected value of 1.5; in so doing I may have only rejected some models erroneously, I am unlikely to have accepted any by mistake. The utility of such an approach is that it allows one to fit data from relatively small studies without having to combine otherwise disparate studies.

APPENDIX A7:2 THE CALCULATIONS

Consider a system with two alleles at a single locus, and hence three possible genotypes, G(1), G(2) and G(3). There are two possible phenotypes from these three genotypes, P(1) and P(2). Each genotype produces a particular proportion of phenotype P(1) (the proportions beimg symbolised by x(1), x(2) and x(3) respectively), and hence proportions 1-x(1), 1-x(2) and 1-x(3) of phenotype P(2) respectively. These values may be represented in tabular form:-

Genotype	Genotype frequency	Proportions of each phenotype from each genotype (i.e. $p(P_A \mid G_B)$	
		P_1	P_2
G_1	f_1	x_1	$(1-x_1)$
G_2	f_2	x_2	$(1-x_2)$
G_3	f_3	x_3	$(1-x_3)$

Let the frequencies of the genotypes be f_1, f_2 and f_3, where f_1, f_2 and f_3 are linked by the Hardy-Weinberg equilibrium. If we assume that G_2 is the heterozygote, then the frequency of one of the alleles, A_1 is clearly:-

$$A_1 = \sqrt{f_1}$$

and hence the frequency of the other allele, A_2, is:

$$A_2 = 1 - \sqrt{f_1}$$

Hence the three genotype frequencies can be represented:-

$$f_1 = A_1^2$$

$$f_2 = 2.A_1.A_2$$

$$f_3 = A_2^2$$

Let L be the incidence of P_1 in the population. Then it is clearly the case that:-

$$L = p(P_1) = \sum_{n=1,3} r_n.f_n \qquad \ldots 1$$

For a particular model, defined by x_1, x_2 and x_3, then since L is known (from the data to be fitted or predicted), and since f_2 and f_3 are expressible in terms of f_1, then f_1 can be determined.

The conditional probabilities $p(G|P)$

In order to calculate the expected offspring from particular parental pairs, it is necessary to know, for an individual, the probability that he is a particular genotype, given that he is a particular phenotype.

In general it may be shown that:-

$$p(G_n|P_m) = \frac{p(G_n).p(P_m|G_n)}{p(P_m)} \qquad \ldots 2$$

where $p(P_m|G_n)$ is the probability of phenotype m given that the genotype is type n, and $p(G_n)$ is equivalent to f_n, the frequency of genotype G_n.

Calculation of family tables, that is $p(P|Q \times R)$

In order to calculate the probability of an individual being a particular phenotype, given the particular phenotypes of his parents, it is necessary to have a function which generates the Mendelian breeding ratios. Let $M(G_a|G_b \times G_c)$ give the probability that for a mating between genotypes G_b and G_c that the progeny will be of genotype G_a. Thus if G_1 and G_3 are the homozygotes, and G_2 the heterozygotes, then from simple Mendelian principles,

$$M(G_2|G_1 \times G_3) = 0.5$$
$$M(G_1|G_2 \times G_3) = 0.25$$
$$M(G_3|G_1 \times G_3) = 0.25$$

Consider two parents of known genotype. In general:-

$$p(P_m|G_a \times G_b) = \sum_{x=1,3} p(P_m|G_x).M(G_x|G_a \times G_b) \qquad \ldots 3$$

But the problem is more typically to calculate the probability of the progeny's phenotype given the parents phenotypes not genotypes. For such a situation one may extend the

above equation 3, so that we may calculate:-

$$p(P_m|P_c \times P_d) = \sum_{y=1,3} \sum_{z=1,3} p(G_y|P_c).p(G_z|P_d).p(P_m|G_y \times G_z) \quad \ldots 4$$

Monozygotic twins

The problem is to calculate $p(P_a \ \& \ P_b)$, that is the proportion of twin-pairs in whom twin 1 is of phenotype a, and twin 2 is of phenotype b. Note that using this notation then to find the proportion of discordant twin-pairs we must remember that:-

$$p(\text{discordant}) = p(P_1 \ \& \ P_2) + p(P_2 \ \& \ P_1) \quad \ldots 5$$

Consider a pair of twins of genotype G_m. Then for these twins:-

$$p(P_a \ \& \ P_b|G_m) = p(P_a|G_m).p(P_b|G_m) \quad \ldots 6$$

(assuming that chance processes occur independently in the two twins).

For the progeny of parents of genotypes G_j and G_k, then:-

$$p(P_a \& P_b|G_j \times G_k) = \sum_{m=1,3} M(G_m|G_j \times G_k).p(P_a|G_m).p(P_b|G_m) \quad \ldots 7$$

Hence for the progeny of parents of known phenotypes:-

$$p(P_a\&P_b \mid P_j x P_k) = \sum_{m=1,3} \sum_{n=1,3} p(P_a\&P_b \mid G_m x G_n).p(G_m \mid P_a).p(G_n \mid P_b) \quad \ldots 8$$

And thus for the total population of MZ twins:-

$$p(P_a\&P_b) = \sum_{j=1,3} \sum_{k=1,3} p(P_a\&P_b \mid P_j x P_k).p(P_j).p(P_k) \quad \ldots 9$$

This equation (9) can however be much simplified. In the total population the genotype frequencies of MZ twins must be the same as in the remaining singleton population. For the calculation of the overall proportions of concordant and discordant pairs irrespective of parental phenotypes, the parental calculations are strictly irrelevant, and:-

$$p(P_a \,\&\, P_b) = \sum_{n=1,3} p(P_a \mid G_n).p(P_b \mid G_n).p(G_n) \quad \ldots 10$$

Dizygotic twins

Using the same notation as for monozygotic twins, but remembering that dizygotic twins do not have the same genotype, but do have the same parents, we may calculate the proportions of concordant and discordant pairs. In making the calculations we must remember that due to the DZ twins having shared parents, $p(G_a \,\&\, G_b)$ is not

the same as $p(G_a).p(G_b)$ (an easy error to make).

Consider parents of genotypes G_m and G_n. Then:-

$$p(P_a\&P_b \mid G_m x G_n) = \sum_{x=1,3} \sum_{y=1,3} m(G_x \mid G_m x G_n).M(G_y \mid G_m x G_n).p(P_a \mid G_x).p(P_b \mid G_y) \quad \ldots 11$$

And thus for parents of known phenotypes:-

$$p(P_a\&P_b \mid P_m x P_n) = \sum_{x=1,3} \sum_{y=1,3} p(P_a\&P_b \mid G_x x G_y).p(G_x \mid P_m).p(G_y \mid p_n) \quad \ldots 12$$

Consequently for the whole population, irrespective of parental phenotype:-

$$p(P_a\&P_b) = \sum_{m=1,3} \sum_{n=1,3} p(P_a\&P_b \mid P_m x P_n).p(P_m).p(P_n) \quad \ldots 13$$

An example

Lest the above equations seem impossibly complex (which they only are as a result of being completely general), a very simple worked example should help to clarify the arguments.

Consider a genetic model in which there are two alleles,

D and C, which produce phenotypes as follows:-

Genotype	Genotype frequency	Phenotypes Left	Right
DD	d^2	0.0	1.0
DC	2dc	0.25	0.75
CC	c^2	0.5	0.5

The above table is thus analogous to that presented earlier in the first part of this appendix.

Let L represent the overall incidence of left-handedness. Then from equation 1:-

$$L = (0).d^2 + (0.25).2dc + (0.5).c^2$$

Since d = 1 - c, then:-

$$L = (0.5).c.(1-c) + (0.5).c^2$$

$$= 0.5 - 0.5c^2 + 0.5c^2 = 0.5c$$

$$\therefore \quad c = 2L$$

The numerical advantage of this particular model is that L is a linear function of c; however for all other models L is a quadratic function of C, which complicates the equations.

We may now re-write the genotype frequencies:-

$$p(CC) = 4L^2$$
$$p(DD) = (1-2L)^2$$
$$p(DC) = 4L(1-2L)$$

From equation 2 we may now calculate the conditional probabilities of a particular genotype given that the person is of a particular phenotype. Thus:-

$$p(DC|L) = \frac{4L(1-2L).(0.25)}{L}$$

$$= 1 - 2L$$

And thus for the rest of the table:-

Genotype	p(Genotype | Left-handed)	p(Genotype | Right-handed)
DD	0	$\frac{(1 - 2L)^2}{(1 - L)}$
DC	$1 - 2L$	$\frac{3L(1 - 2L)}{(1 - L)}$
CC	$2L$	$\frac{2L^2}{(1 - L)}$

In order to calculate the family breeding tables we need to know the Mendelian genetics of the three genotypes; that is, for all combinations of a, b and c, we must know

the value of $p(G_a | G_b \times G_c)$. Simple genetical theory gives us:-

		G(c)		
		DD	DC	CC
G(b)	DD	p(DD)=1.0 p(DC)=0.0 p(CC)=0.0	p(DD)=0.5 p(DC)=0.5 p(CC)=0.0	p(DD)=0.0 p(DC)=1.0 p(CC)=0.0
	DC	p(DD)=0.5 p(DC)=0.5 p(CC)=0.0	p(DD)=0.25 p(DC)=0.5 p(CC)=0.25	p(DD)=0.0 p(DC)=0.5 p(CC)=0.5
	CC	p(DD)=0.0 p(DC)=1.0 p(CC)=0.0	p(DD)=0.0 p(DC)=0.5 p(CC)=0.5	p(DD)=0.0 p(DC)=0.0 p(CC)=1.0

If we wish to calculate $p(L | L \times L)$, then firstly using equation 3:-

$$p(L|DD \times DD)=0.0$$
$$p(L|DD \times DC)=p(L|DC \times DD)=0.125$$
$$p(L|DD \times CC)=p(L|CC \times DD)=0.25$$
$$p(L|DC \times DC)=0.25$$
$$p(L|DC \times CC)=p(L|CC \times DC)=0.375$$
$$p(L|CC \times CC)=0.5$$

These values may then be entered into equation 4, giving:-

$$p(L|L \times L) = \frac{1-2L}{8} + \frac{2L}{4} + \frac{(1-2L)^2}{4} + \frac{4L^2}{2}$$

$$+ 2(1-2L).2L.(0.375)$$

$$= \frac{1+2L}{4} = \frac{1+c}{4}$$

And similarly:-

$$p(L|R \times R) = \frac{3(3-c)}{4(2-c)}$$

$$p(L|R \times L) = \frac{1 + c(2-c)}{4(2-c)}$$

(c is used instead of L as the equations are simpler).

Notice how, despite the complexities of equations 3 and 4, for particular cases there are solutions which are relatively simple. Nevertheless the general equations are rather more complex sinze a). Thev are applicable to any two-allele genetic model, and b), they are particularly suitable for use in a computer, and most persons wishing to carry out such calculations would probably use a computer.

In the particular model being discussed the equati ons for monozygotic twins also reduce to fairly simple results. From equation 10 it can be shown that:-

$$p(L\&L) = c^2.(0.5)^2 + 2dc.(0.25)^2 + d^2.(0.0)^2$$

$$= \frac{c^2}{2} + \frac{2dc}{16}$$

$$= \frac{c^2}{2} + \frac{dc}{8}$$

Similarly, it can be shown that:-

$$p(R\&R) = d^2 = \frac{9dc}{8} + \frac{c^2}{2}$$

$$p(R\&L \text{ or } L\&R) = \frac{c^2}{2} + \frac{3dc}{4}$$

If the reader has progressed thus far in replicating the above equations then he will surely understand the (relatively simple) principles involved, and thus there will be no need to produce an exact solution for the numerically more complex case of the dizygotic twins.

Corrections for actual incidence of left-handedness not equalling true incidence

The measured incidence of left-handedness in most of the studies being used as a data-base is different, both within studies between parents and children, and also between studies within a particular generation. These differences might possibly represent real differences in allele frequency, and that would be the classic genetic approach to such discrepancies (and indeed is the approach which I have used elsewhere in fitting the hand-clasping data and arm-folding data, where there is good reason to believe that response biasses and criterion shifts are of little significance). However, in the case of handedness there is reason to be doubtful and I will assume that the true incidence of left-handedness is in fact constant, and that differences in apparent or manifest incidence are due instead to alterations in the criterion of left-handedness, or to differences in response rates of right and left-handers to questionnaires, etc. Annett (1978,1979) has shown how by varying the criterion of left-handedness widely differing incidences of 'left-handedness' can be obtained.

I would of course dispute that many of these scales are truly measuring left-handedness, and would argue that the criterion put forward elsewhere, of writing hand alone, is probably the best criterion. Nevertheless there are still such shifts in other data and instead of simply discarding unacceptable data (the approach of Levy and Nagylaki, 1972), I will instead attempt to correct for these biasses.

The main types of error, which I will call criterion shifting and response bias, do not, regrettably, have identical corrections. And for criterion shifting it is not strictly possible, on logical and arithmetic grounds, to say that there is even a single correction.

Despite all such problems I will nevertheless attempt to fit the data bearing in mind that some small discrepancies may still be present and be essentially irremovable.

A further complication to be borne in mind is that the corrections need not be the same type, nor in the same direction, nor of the same magnitude, in parents and children.

Notation

Let the true handedness, H, be notated as H_t and the manifest handedness as H_m, where H can be either R or L

specifically, or 'H', meaning either phenotype.

Corrections for simple criterion shift when $p(L_m) < p(L_t)$

This is very simple. For any individual who is truly left-handed there is a finite probability, q, that he will manifest as a right-hander, hence:-

$$p(L_m) = q.p(L_t) \qquad \text{where } q = \frac{p(L_m)}{p(L_t)} \qquad \ldots 14$$

More importantly, since the correction is indepeddent of propositus genotype, or of parental phenotype or genotype, then for any given population subset:-

$$p(L_m \mid \text{subset}) = q.p(L_t \mid \text{subset})$$

and particularly:-

$$p(L_m \mid H_a x H_b) = q.p(L_t \mid H_a x H_b)$$ where H_a and H_b are both true handedness ... 15

For monozygotic twins a similar correction may be made:-

$$p(L_m \,\&\, L_m) = q^2.p(L_t \,\&\, L_t)$$

$$p(R_m \,\&\, R_m) = (1-q)^2.p(L_t \,\&\, L_t) + 2q.(1-q).p(L_t \,\&\, L_t) + p(R_t \,\&\, R_t)$$

$$p(R_m \,\&\, L_m) = 1. - p(L_m \,\&\, L_m) - p(R_m \,\&\, R_m)$$

Corrections for criterion shifting when $p(L(m)) > p(L(t))$

This is basically similar to the previous case, although it is not immediately obvious that it is valid to use the same equation with $q > 1.0$. The situation is that there is now a finite probability, r, that a true right-hander will manifest as a left-hander, all true left-handers being presumed to manifest as left-handers. Hence:-

$$p(R_m) = r.p(R_t) \quad \text{where} \quad r = \frac{p(R_m)}{p(R_t)} \qquad \ldots 16$$

Using the same argument as before:-

$$p(R_m \mid H_a \times H_b) = r.p(R_t \mid H_a \times H_b)$$

And thus:-

$$p(L_m \mid H_a \times H_b) = 1 - r.(1 - p(L_m \mid H_a \times H_b)) \qquad \ldots$$

If equation 17 is identical to equation 15 (i.e. if it is valid to use $q > 1$ in equation 15), then we would expect that:-

$$p(L_m \mid H_A \times H_B) = 1 - r(1 - p(L_t \mid H_A \times H_B)) = q.p(L_t \mid H_A \times$$

..

Expanding the second part of equation 18, and substituting $T = p(L(t)\,|\,H(A)xH(B))$ and $M=p(L(m)\,|\,H(A)xH(B))$, then:-

$$1 - r(1-T) = q.T \qquad \ldots 18a$$

From equation 14 we know that $M = qT$, and from equation 16 that $r=p(R(m))/p(R(t))$, and these may be substituted into equation 18a, to produce:-

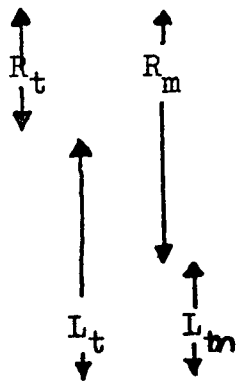
$$1 - \frac{(1-qT)}{(1-T)}.(1-T) = q.T$$

which is an identity. Hence equation 14 is valid both for $q < 1$ and $q > 1$.

Corrections for differences in parental left-handedness incidence

Thus far, corrections have only been made for $p(L_m)$ not being equal to $p(L_t)$ in the progeny. Parents however may also have incidences of left-handedness which are different to those of the true incidence, and corrections must also be made for this as well. The corrections are best seen by setting out a 3 x 3 matrix.

Consider firstly the case in which $p(L_{m-par}) > p(L_t)$:-



	R_t & R_m	L_t & R_m	L_t & L_m	Frequency
R_t & R_m	R_t x R_t	R_t x L_t	R_t x L_t	$p(R_t)$
L_t & R_m	R_t x L_t	L_t x L_t	L_t x L_t	$(1-s).p(L_t$
L_t & L_m	R_t x L_t	L_t x L_t	L_t x L_t	$s.p(L_t)$

From this diagram, in which true left-handers are divided into two types, manifest left-handers (L_t & L_m), and manifest right handers (L_t & R_m), it is simple to write down correction equations: Let $s=p(L_m)/p(L_t)$. Then:-

$$p(L_t|R_m x R_m)=\left[p(R_t)^2.p(L_t|R_t x R_t)+2.p(R_t)(1-s).p(L_t).p(L_t|R_t x L_t)\right.$$

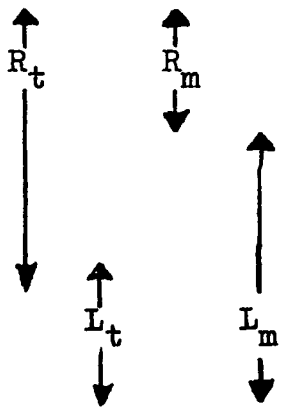
$$\left.+(1-s)^2.p(L_t)^2.p(L_t|L_t x L_t)\right]/(1-s.p(L_t)^2) \qquad \ldots 19$$

$$p(L_t|R_m x L_m)=\frac{\left[p(R_t).s.p(L_t).p(L_t|R_t x L_t)+s.p(L_t)^2.(1-s).p(L_t|L_T x L_t)\right]}{(1-s.p(L_t)).s.p(L_t)} \qquad \ldots 20$$

$$p(L_t|L_m \text{ x } L_m) = p(L_t|L_t \text{ x } L_t) \qquad \ldots 21$$

Alternatively consider the case in which $p(L_{m-par}) > p(L_t)$ It is now assumed that this is entirely due to true right-

handers manifesting as left-handers. Let $u = p(R_m)/p(R_t)$. Constructing a similar table to the previous one, thus:-



	R_t & R_m	R_t & L_m	L_t & L_m	Frequenc
R_t & R_m	$R_t \times R_t$	$R_t \times R_t$	$R_t \times R_t$	$u.p(R_t)$
R_t & L_m	$R_t \times R_t$	$R_t \times R_t$	$R_t \times L_t$	$(1-u).p(R$
L_t & L_m	$R_t \times L_t$	$R_t \times L_t$	$L_t \times L_t$	$p(L_t)$

And once more reading off directly from the table, we may see that:-

$$p(L_t \mid R_m \times R_m) = p(L_t \mid R_t \times R_t) \qquad \ldots 22$$

$$p(L_t \mid R_m \times L_m) = p(L_t \mid R_t \times L_t) + \frac{(1-u).p(R_t).p(L_t \mid R_t \times R_t)}{p(L_t)} \qquad \ldots 23$$

$$p(L_t \mid L_m \times L_m) = \frac{((1-u).p(R_t).p(L_t \mid R_t \times R_t) + 2.p(R_t).(1-u).p(L_t).p(L_t \mid R_t \times L_t) + p(L_t).p(L_t \mid L_t \times L_t)}{(1 - u.p(R_t))} \qquad \ldots 24$$

Scrutiny of equations 22 to 24 will reveal, unlike the case of the progeny, described earlier, that the correction for $p(L_m)/p(L_t)$ being greater than 1 cannot be the same as that when the value is less than 1. Consider equations 19 and 22. Equation 19 is a function of s; equation 22 is independent of s. But if in equation 19, s were greater than 1, $p(L_t \mid R(m) \times R(m))$ must still be a function of s. Hence equations 19 and 22 cannot be alternative views of the same equations, and hence two separate sets of corrections must therefore be used for the situations in which $s > 1$ and $s < 1$.

Of course if the exact proportions were known for the two processes represented by the values s and u, then an exact correction could be made for the occurrence of both processes. But in general such information will not be available.

Since both parents and children will probably have $p(L_m)$ differrnt from $p(L_t)$, the procedure used in the calculations was firstly to correct for the parents, using equations 19 to 24 and then to correct for the children (using equation 15).

Corrections for response bias amongst propositus

Consider a population of right and left-handed propositi. Left-handers do not respond as often as right-

handers, only a proportion, k, of the true left-handers responding, whilst all of the right-handers respond. (The argument is the same if only x of the right-handers respond, and only kx of the left-handers respond; the corrections are needed whenever k is not equal to 1).

We may represent the situation thus:-

	L_t		R_t
	non-responders	responders	responders
prop'n	$(1-k).p(L_t)$	$k.p(L_t)$	$p(R_t)$

The ratio of $p(L_m)$ to $p(L_t)$ may be represented by j, where:- $j = p(L_m)/P(L_t)$. It may readily be shown that:-

$$j = \frac{k}{k.p(L_t) + p(R_t)} \qquad \ldots 25$$

Consider progeny of a particular parental phenotype, H x I. Then:-

$$p(L_m|H \times I) = \frac{k.p(L_t|H \times I)}{k.p(L_t|H \times I) + p(R_t|H \times I)} \qquad \ldots 26$$

It is tempting to hope that this equation, 26, might be identical to equation 15, so that expressing equation 26 in terms of j would result in an equation of the form:-

$$p(L_m \mid H \times I) = z.p(L_t \mid H \times I) \qquad \ldots 27$$

Consideration however shows that this cannot be so. Let there exist an equation of the following form for R x R matings (derived fromequation 27).

$$p(L_m \mid R \times R) = z.p(L_t \mid R \times R)$$

But also, from equation 26:-

$$p(L_m \mid R \times R) = \frac{k.p(L_t \mid R \times R)}{k.p(L_t \mid R \times R) + p(R_t \mid R \times R)}$$

And hence:-

$$z.p(L_t \mid R \times R) = \frac{k.p(L_t \mid R \times R)}{k.p(L_t \mid R \times R) + p(R_t \mid R \times R)}$$

$$z = \frac{k}{k.p(L_t \mid R \times R) + p(R_t \mid R \times R)}$$

Thus z is a function of $p(L_t \mid RxR)$. If therefore z were valid for finding $p(L_m \mid RxL)$ and $p(L_m \mid LxL)$, these would also involve functions of $p(L_t \mid RxR)$; but this clearly contradicts the form of equation 27, and hence we may presume that no such equation can possibly exist.

Hence we may infer that corrections for criterion shift and corrections for response bias are <u>not</u> the same. How

different they are in practice is a question best examined by actual examples.

Consider a population in which $p(L_t) = 0.10$, and $p(L_m)$, due to a response bias, is 0.08. Then $j = 0.8$, and thus $k = 0.7826$. Let $p(L_t \mid R \times R)$ be 0.08. Then using the correction as in equation 26, $p(L_m \mid R \times R)$, = 0.0637, whereas using a correction as in equation 15, $p(L_m \mid R \times R) = 0.0640$.

Similarly for a value of $p(L_t \mid L \times L)$ of 0.35, the respective values of $p(L_m \mid L \times L)$ are 0.2964 and 0.2800.

Thus in general the predictions only really differ when $p(L_T \mid H \times I)$ is quite a lot greater than $p(L_t)$, but this only occurs, of course, for L x L matings, which are themselves relatively rare. The implication is that for most purposes the correction of equation 15 will be adequate.

As with the discussion earlier for a criterion shift, so it is not immediately clear that equation 26 is valid both for $p(L_m) > p(L_t)$, and for $p(L_m) < p(L_t)$. We may represent the situation in which there is a relative proportion of right-handers who do not respond, as

Proportion	L_t	R_t	
	responders $p(L_t)$	responders $f.p(R_t)$	non-responders $(1-f).p(R_t)$

We may then find:-

$$e = \frac{p(L_m)}{p(L_t)} = \frac{1}{p(L_t) + f.p(R_t)} \qquad \ldots 28$$

and hence:-

$$p(L_m \mid H \times I) = \frac{p(L_t \mid H \times I)}{p(L_t \mid H \times I) + f.p(R_t \mid H \times I)} \qquad \ldots 29$$

We may thus combine equations 26 and 29, since both equal $p(L_m \mid H \times I)$.

$$\therefore \frac{p(L_t \mid H \times I}{p(L_t \mid H \times I) + f.p(P_t \mid H \times I)} = \frac{k.p(L_t \mid H \times I)}{k.p(L_t \mid H \times I) + p(R_t \mid H \times I)}$$

$\ldots 30$

Inserting values for f and k from equations 25 and 28 respectively, it may be shown that the equation eventually reduces to the form, j = e. Hence equation 26 is valid whether $p(L_m)$ is greater than or less than $p(L_t)$.

To show the magnitude of such corrections, consider the situation in which $p(L_t) = 0.10$, and $p(L_m) = 0.20$. Then $j = 2.0$ and thus $k = 2.250$. If $p(L_t \mid R \times R) = 0.08$, then $p(L_m \mid R \times R) = 0.1636$ by the response bias method, or

0.1800 by the criterion shift method. Onee again the differences are relatively small, and I would like to propose that they can probably be ignored.

Corrections for response bias amongst parents

These are not strictly relevant since in most studies, a) the propositi respond, not the parents, b) it is unlikely that students with left-handed parents show a different response rate to those without, and c) the parental incidence of sinistrality in most studies is fairly low, and suggests that criterion shifts are probably of greater import.

In general the effects of differing parental incidences (of whatever sort) are relatively small in comparison with the effects due to propositus biasses, and thus differences in mode of parental correction are unlikely to reduce large effects. They will not be considered in further detail here,

APPENDIX A7:3: ASSORTATIVE MATING FOR HANDEDNESS

Assortative mating is the situation in which like preferentially mates with like (positive assortative mating), or like preferentially mates with non-like (negative assortative mating). Chance expectations for non-assortative mating may be obtained from population phenotype frequencies and from the binomial or multinomial distribution. If assortative mating is present then account must be taken of that fact in the calculation of expected frequences in progeny, etc. (see Cavalli-Sforza and Bodmer, 1971, p58, pp537-550 for a review).

Table 7.76 shows the proportions of R-R, R-L and L-L matings in various studies from the literature. These data sets are shown graphically in Figure 7.14. Table 7.7 also shows the Chi-squared value for the goodness of fit of a binomial distribution (note that in some of these cases the expected values are less than one, and thus care should be taken in interpreting these values). Of the thirteen data sets, six shows less R-L pairings than would be expected, and seven show more than would be expected. Four of the sets showing positive assortative mating are significantly different from chance expectations with $p < 0.05$, whilst only one of the sets showing negative assortative matings is significant at the 0.04 level. Of the significant cases of positive assortative mating, one is the Hicks and Kinsbourne (1976) study, which, as will be shown

in Chapter 10, shows several strange features, and should perhaps not be treated too seriously. Of the remaining studies, there seems little doubt that overall the data is heterogeneous, differing from a binomial distribution; but the fact that these differences are not in a consistent direction suggests that some factors other than assortative mating may be the cause. A response bias may easily produce a surplus of 'unusual' or 'interesting' families, such as those with two left-handed parents.

In summary, there is a significant heterogeneity amongst the parental mating pairs but this difference from predicted proportions is relatively small, and may well not be the result of true assortative mating. For the purposes of model fitting, it may safely be ignored until it is demonstrated more convincingly.

Table 7.1

Shows the data from fourteen studies of the incidence of left-handedness in families of different parental types. Column 1 gives the name of the study; columns 2 and 3 indicate the overall incidence of left-handedness in the propositi and in the parents; columns 4 and 5 and 6 show the proportion of left-handers, and the actual numbers of right and left-handers in the progeny of R x R matings, the top figure indicating the actual values, and the figures in brakkets indicating the predictions of the model, the first figure in the brakkets being for a model in which $p(L_t) = 0.08$ and $p(L|DC) = 0.125$, and the second figure being for a model in which $p(L_t) = 0.095$, and $p(L|DC) = 0.25$; column 7 gives the value of the Chi-squared goodness of fit statistic for the two models, for just the R x R data; columns 8 - 11 give similar information for R x L matings, and columns 12 - 15 for L x L matings; the final column gives the total Chi-squared values for the goodness of fit.

RxR

Study	p(L) propositi	p(L) parents	p(L)	n(R)	n(L)	Chi^2
ICM-2 grandparents	3.22	3.31	2.15 (2.98,2.99)	455 (451.1,451.1)	10 (13.9,13.9)	(1.118
Chamberlain (1928)	4.77	3.56	4.26 (4.39,4.40)	6917 (6907.5,6907.0)	308 (317.5,318.0)	(0.298
ICM-1 parents	6.66	6.10	2.91 (5.74,5.76)	167 (162.1,162.1)	5 (9.9,9.9)	(2.548
Mascie-Taylor (1977)	8.30	9.30	6.82 (6.49,6.81)	232 (232.9,232.0)	17 (16.1,17.0)	(0.048
Annett (1978) OU	8.50	5.47	7.27 (7.45,7.48)	1656 (1652.9,1652.4)	130 (133.1,133.6)	(0.077
ICM-2 parents	8.53	4.55	7.68 (7.66,7.68)	1924 (1924.3,1923.9)	160 (159.7,160.1)	(0.000
Rife (1940)	8.76	5.24	7.57 (7.73,7.75)	1842 (1839.0,1838.5)	151 (154.0,154.5)	(0.064
Ferronato (1974)	9.75	9.86	6.66 (7.57,8.00)	154 (152.5,151.8)	11 (12.5,13.2)	(0.192
Annett (1972)	10.63	4.40	9.73 (9.67,9.69)	6206 (6209.9,6208.6)	669 (665.1,666.4)	(0.025
Chaurasia & Goswami	14.07	10.40	11.96 (10.92,11.55)	1060 (1072.5,1064.9)	144 (131.5,139.1)	(1.33
ICM-2 propositi	14.39	9.82	11.35 (11.17,11.81)	796 (797.9,791.9)	102 (110.3,106.1)	(0.03
Hubbard (1971)	14.58	6.30	13.94 (12.49,12.55)	722 (734.2,733.7)	117 (104.8,105.3)	(1.62
Ramaley (1913)	15.56	8.03	12.03 (12.66,12.77)	841 (834.9,833.9)	115 (121.1,122.1)	(0.
ICM-1 propositi	15.69	9.66	13.51 (12.18,12.88)	755 (766.7,760.6)	118 (106.3,112.4)	(1.
Combined LxL data I	8.73	6.36				
Combined LxL data II	14.84	8.84				

)	Chi^2	RxL p(L)	RxL n(R)	LxL n(L)	LxL Chi^2	LxL p(L)	LxL n(R)	LxL n(L)	LxL Chi^2
		18.75	26	6					
3.9,13.9)	(1.118,1.128)	(6.53,6.44)	(29.9,29.9)	(2.1,2,1)	(7.819,8.050)	(10.08,12.05)	0	0	
3		11.42	411	53		28.0	18	7	
17.5,318.0)	(0.298,0.330	(9.67,9.52)	(419.1,419.8)	(44.9,44.2)	(1.639,1.956)	(14.94,17.85)	(21.3,20.5)	(3.7,4.5)	(3.357,1.755)
		25.0	15	5			0	3	
.9,9.9)	(2.548,2.583)	(13.30,12.97)	(18.3,17.4)	(2.7,2.6)	(2.376,2.562)	(20.86,24.93)			
		14.58	41	7			3	1	
6.1,17.0)	(0.048,0.000)	(16.24,16.97)	(40.2,39.9)	(7.8,8.1)	(0.097,0.194)	(25.99.26.93)			
0		19.04	170	40			4	0	
33.1,133.6)	(0.077,0.102)	(17.03,16.66)	(174.2,175.0)	(35.8,35.0)	(0.602,0.863)	(26.62,31.81)			
0		18.23	148	33			7	1	
59.7,160.1)	(0.000,0.001)	(17.19,16.86)	(149.9,150.5)	(31.1,30.5)	(0.139,0.241)	(26.71,31.92)			
1		19.54	140	34			5	6	
54.0,154.5)	(0.064,0.086)	(17.58,17.21)	(143.4,144.1)	(30.6,29.9)	(0.461,0.665)	(27.43,32.79)			
		22.50	31	9			0	0	
2.5,13.2)	(0.192,0.400)	(19.34,20.50)	(32.3,31.8)	(7.7,8.2)	(0.255,0.098)	(29.68,29.99)			
)		20.97	471	125			5	1	
5.1,666.4)	(0.025,0.011)	(21.48,21.11)	(468.0,470.2)	(128.0,125.8)	(0.091,0.007)	(33.29,39.78)			
		27.38	122	46			3	4	
1.5,139.1)	(1.334,0.199)	(28.53,30.35)	(120.1,117.0)	(47.9,51.0)	(0.108,0.701)	(41.19,41.32)			
		25.75	173	60			6	2	
.3,106.1)	(0.032,0.177)	(28.50,30.19)	(166.6,162.7)	(66.4,70.3)	(0.864,2.181)	(43.94,44.43)			
		19.01	90	23			0	0	
.8,105.3)	(1.626,1.494)	(29.07,28.34)	(85.8,86.7)	(35.2,34.3)	(5.945,5.193)	(45.66,54.57)			
5		32.33	113	54			1	7	
21.1,122.1)	(0.347,0.474)	(30.70,,29.79)	(115.7,117.3)	(51.3,49.7)	(0.211,0.519)	(48.73,58.00)			
8		25.65	142	49					
06.3,112.4)	(1.460,0.316)	(30.86,32.65)	(132.1,128.6)	(58.9,62.4)	(2.427,4.256)	(48.52,			
						33.3			
						(27.34,			
						43.4			
						(46.50			

RxL

	Chi^2	p(L)	n(R)	n(L)	Chi^2	p(I
,13.9)	(1.118,1.128)	18.75 (6.53,6.44)	26 (29.9,29.9)	6 (2.1,2.1)	(7.819,8.050)	(1(
5,318.0)	(0.298,0.330	11.42 (9.67,9.52)	411 (419.1,419.8)	53 (44.9,44.2)	(1.639,1.956)	28 (1·
9.9)	(2.548,2.583)	25.0 (13.30,12.97)	15 (18.3,17.4)	5 (2.7,2.6)	(2.376,2.562)	(2
,17.0)	(0.048,0.000)	14.58 (16.24,16.97)	41 (40.2,39.9)	7 (7.8,8.1)	(0.097,0.194)	(2
,133.6)	(0.077,0.102)	19.04 (17.03,16.66)	170 (174.2,175.0)	40 (35.8,35.0)	(0.602,0.863)	(
,160.1)	(0.000,0.001)	18.23 (17.19,16.86)	148 (149.9,150.5)	33 (31.1,30.5)	(0.139,0.241)	
,154.5)	(0.064,0.086)	19.54 (17.58,17.21)	140 (143.4,144.1)	34 (30.6,29.9)	(0.461,0.665)	
13.2)	(0.192,0.400)	22.50 (19.34,20.50)	31 (32.3,31.8)	9 (7.7,8.2)	(0.255,0.098)	
,666.4)	(0.025,0.011)	20.97 (21.48,21.11)	471 (468.0,470.2)	125 (128.0,125.8)	(0.091,0.007)	
,139.1)	(1.334,0.199)	27.38 (28.53,30.35)	122 (120.1,117.0)	46 (47.9,51.0)	(0.108,0.701)	
3,106.1)	(0.032,0.177)	25.75 (28.50,30.19)	173 (166.6,162.7)	60 (66.4,70.3)	(0.864,2.181)	
8,105.3)	(1.626,1.494)	19.01 (29.07,28.34)	90 (85.8,86.7)	23 (35.2,34.3)	(5.945,5.193)	
1.1,122.1)	(0.347,0.474)	32.33 (30.70,,29.79)	113 (115.7,117.3)	54 (51.3,49.7)	(0.211,0.519)	
6.3,112.4)	(1.460,0.316)	25.65 (30.86,32.65)	142 (132.1,128.6)	49 (58.9,62.4)	(2.427,4.256)	

		LxL		
Chi^2	p(L)	n(R)	n(L)	Chi^2
(7.819,8.050)	(10.08,12.05)	0	0	
(1.639,1.956)	28.0 (14.94,17.85)	18 (21.3,20.5)	7 (3.7,4.5)	(3.357,1.755)
(2.376,2.562)	(20.86,24.93)	0	3	
:0.097,0.194)	(25.99.26.93)	3	1	
0.602,0.863)	(26.62,31.81)	4	0	
0.139,0.241)	(26.71,31.92)	7	1	
0.461,0.665)	(27.43,32.79)	5	6	
0.255,0.098)	(29.68,29.99)	0	0	
0.091,0.007)	(33.29,39.78)	5	1	
0.108,0.701)	(41.19,41.32)	3	4	
0.864,2.181)	(43.94,44.43)	6	2	
5.945,5.193)	(45.66,54.57)	0	0	
(0.211,0.519)	(48.73,58.00)	1	7	
(2.427,4.256)	(48.52,49.19)	0	0	
	33.3 (27.34,32.67)	24 (26.2,24.2)	12 (9.8,11.8)	(0.651,0.007)
	43.4 (46.50,50.50)	10 (12.3,11.4)	13 (10.7,11.6)	(0.928,0.334)

n data, the observed and expected numbers of each type (
cted values (E) are for two different models, 'a' in which p(L)=0.095 and p(L DC)=0.25,
DC)=0.125.

		Monozygotic twins			
y	p(L)	N(R-R) E(a,b)	N(R-L) E(a,b)	n(L-L) E(a,b)	Chi2 (a,b)
ens (1924)	16.21	26 (26.5,26.4)	10 (8.8, 9.0)	1 (1.5,1.4)	0.045,0.027
z (1924)	25.00	10 (10.3,10.3)	7 (6.2,6.3)	1 (1.3,1.3)	0.029,0.030
erbach (1925)	5.33	67 (67.6,67.7)	8 (6.6,6.5)	0 (0.6,0.7)	0.067,0.083
berg (1926)	14.49	53 (51.6,51.3)	12 (14.7,15.2)	4 (2.6,2.3)	1.250,1.815
chuer (1927)	20.28	156 (158.6,157.9)	77 (71.6,73.1)	11 (13.6,12.9)	0.973,0.517
an (1928)	31.00	25 (24.3,24.2)	19 (20.2,20.5)	6 (5.3,5.2)	0.171,0.240
ch (1930)	20.93	25 (27.5,27.4)	18 (12.9,13.2)	0 (2.5,2.4)	4.696,4.313
on & Jones (1932)	10.71	56 (57.1,56.8)	13 (10.8,11.3)	1 (2.1,1.8)	1.070,0.066
ks (1933)	9.52	35 (35.2,35.0)	6 (5.6,5.9)	1 (1.2,1.0)	0.006,0.000
an, Freeman and inger (1937)	19.00	34 (33.5,33.4)	13 (13.8,14.2)	3 (2.6,2.4)	0.127,0.246
erwek (1938)	18.85	80 (82.2,81.8)	38 (33.6,34.3)	4 (6.2,5.8)	1.443,1.010
(1940)	11.88	176 (177.2,176.4)	41 (38.6,40.3)	6 (7.2,6.4)	0.366,0.037
ss (1946)	18.44	72 (70.1,69.8)	24 (27.8,28.4)	7 (5.1,4.8)	1.258,1.789
e (1950)	12.82	261 (266.7,265.5)	76 (64.5,66.9)	6 (11.8,10.5)	5.013,3.227
haume (1957)	24.24	19 (19.4,19.3)	12 (11.2,11.4)	2 (2.4,2.3)	0.122,0.071
zo (1960)	13.32	199 (199.2,198.2)	51 (50.7,52.6)	9 (9.2,8.2)	0.005,0.121
ter-Saltzmann et al 76)	17.11	132 (131.5,130.8)	46 (47.0,48.3)	9 (8.5,7.9)	0.054,0.276
hlin & Nichols (1976)	14.10	380 (338.1,386.3)	123 (106.7,110.4)	11 (19.2,17.3)	6.107,3.83

rs of each type of twin-pair (R-R, R-L and L-L). The
nd p(L DC)=0.25, and 'b' in which p(L)=0.08, and

		Dizygotic twins			
Chi2 (a,b)	p(L)	N(R-R) E(a,b)	N(R-L) E(a,b)	N(L-L) E(a,b)	Chi2 (a,b)
0.045,0.027	27.41	16 (16.5,16.4)	13 (11.9,12.0)	2 (2.5,2.4)	0 0.216,0.177
0.029,0.030	-	-	-		
0.067,0.083	12.69	96 (97.1,96.8)	28 (25.6,26.2)	2 (3.1,2.8)	0.648,0.382
1.250,1.815	7.03	111 (111.3,111.3)	16 (15.4,15.3)	1 (1.3,1.3)	0.006,0.008
0.973,0.517	14.04	136 (133.1,132.6)	34 (39.9,40.7)	8 (5.1,4.6)	2.649,3.600
0.171,0.240	17.00	35 (34.8,34.7)	13 (13.3,15.2)	2 (1.8,1.7)	0.002,0.006
4.696,4.313	6.03	51 (51.4,51.5)	7 (6.1,6.1)	0 (0.4,0.5)	0.032,0.035
1.070,0.066	11.38	97 (97.7,97.4)	24 (22.5,23.1)	2 (2.7m2.4)	0.293,0.110
0.006,0.000	10.63	76 (75.9,75.7)	12 (16.2,16.6)	2 (1.9,1.7)	0.000,0.005
0.127,0.246	11.00	39 (40.1,39.9)	11 (8.8,9.1)	0 (1.1,0.9)	0.143,0.119
1.443,1.010	17.14	23 (24.3,24.2)	12 (9.4,9.5)	0 (1.3,1.2)	0.231,0.205
0.366,0.037	15.41	104 (105.7,105.4)	39 (35.6,36.3)	3 (4.7,4.4)	0.961,0.656
1.258,1.789	16.27	60 (60.9,60.8)	24 (22.0,22.4)	2 (3.0,2.8)	0.519,0.354
.5) 5.013,3.227	11.61	164 (166.7,166.3)	45 (39.4,40.5)	2 (4.8,4.3)	2.448,1.751
) 0.122,0.071	19.69	21 (21.5,21.5)	11 (9.9,10.0)	1 (1.6,1.5)	0.037,0.028
) 0.005,0.121	10.89	264 (269.1,268.3)	69 (58.8,60.4)	2 (7.1,6. 3)	5.530,4.198
) 0.054,0.276	19.31	115 (115.9,115.6)	54 (52.2,52.8)	7 (7.9,7.6)	0.176,0.068
'.3) 6.107,3.830	11.11	261 (266.2,265.4)	70 (59.6,61.2)	2 (7.2,6.4)	5.673,4.348

Table 7.3

This shows a simple summary of the critical features of the main genetic models which have been proposed, including the present one.

Study	Phenotypes				
	Right			Left	
Ramaley (1913)	RR	RL		LL	
Trankell (1955)	RR	RL	(1-x) LL	x LL	
Rife (1950)		RR	(1-x) L	xRL	LL
			'Mixed'		
Annett (1964)		RR	RL	LL	
Levy & Nagylaki (1972)	CC:LL	CC:Ll	CC:ll	Cc:ll	
	Cc:LL	Cc:Ll	cc:LL	cc:ll	
	cc:ll				
	(L,l = Left/right language dominance)				
	(C,c = Contra/ipsi-lateral hand control)				
Annett (1972)	RS+	(RS-)	RS-	(RS+)	
McManus	DD	DC	DC	CC	

Table 7.4

Proportion of individuals of each genotype, by handedness and parental handedness, for a p(L-true) of 9.5%, and additive inheritance.

Parental type	p(DD)	p(DC)	p(CC)
		Handedness not known	
R x R	.72677923	.25146843	.02175223
R x L	.34526797	.56697715	.08775397
L x L	.16402560	.48195500	.35402500
NK	.65610000	.30780000	.03610000
		Right-handers	
R x R	.78464120	.20361667	.01174201
R x L	.42396487	.52215615	.05387786
L x L	.23348750	.51453737	.25197509
NK	.72497200	.25508300	.01994500
		Left-handers	
R x R	0.0	.8525137O	.14748630
R x L	0.0	.76362093	.23637900
L x L	0.0	.40500000	.59500000
NK	0.0	.81000000	.19000000

Table 7.5

Shows, the predicted proportions of the various types of twin pair, for monozygotic twins, by parental handedness.

Pair type / parental type	R - R	R - L	L - L
R x R	87.37	10.52	2.11
R x L	68.61	25.65	5.74
L x L	52.37	35.77	11.86
NK	83.82	13.34	2.82

Table 7.6

Shows the expected genotype probabilities for an individual from a monozygotic twin pair, according to the ty of the twin pair, and the parental handedness type.

Genotype	DD	DC	CC
		R-R pairs	
Pate-ntal type			
R x R	.8317	.1618	.0062
R x L	.5032	.4648	.0319
L x L	.3131	.5174	.1690
N^K	.7826	.2064	.0107
		R-L pairs	
R x R	0.0	.8966	.1034
R x L	0.0	.8289	.1711
L x L	0.0	.5051	.4948
NK	0.0	.8647	.1352
		L-L pairs	
R x R	0.0	.7429	.2570
R x L	0.0	.6176	.3823
L x L	0.0	.2539	.7460
NK	0.0	.6806	.3194

Table 7.7

Shows the observed values of the numbers of R-R, R-L and L-L matings in the parents of various study populations. The table also shows the total number of parental pairs (n), the overall parental incidence of left-handedness (p(L)), and the Chj-squared value for the goodness of fit of a binomial distribution.

Study	R-R	R-L	L-L	n	p(L)	Chi2
II: grandparents	465	32	0	497	3.21%	0.55
Chamberlain (1928)	2031	137	9	2177	3.55%	15.18
Annett (1973)	1978	171	2	2151	4.06%	0.74
II: parents	766	67	4	837	4.48%	3.50
Rife (1940)	620	62	5	687	5.24%	5.76
I: parents	172	20	3	195	6.66%	6.03
Mascie-Taylor (1978)	163	29	1	193	8.03%	0.07
Ramaley (1915)	258	45	2	305	8.03%	0.01
Hicks & Kinsbourne (1976) - biological parents	923	150	28	1101	9.35%	42.58
I: propositi	337	81	0	418	9.68%	4.81
II: propositi	351	79	3	433	9.81%	0.40
Ferronato et al (1974)	61	15	0	76	9.86%	0.91
Hicks & Kinsbourne (1976) - step-parents	54	39	15	108	31.94%	3.10

Figure 7.1 Shows the family data of Table 1 plotted, separately for the progeny of R x R, R x L and L x L matings, as a function of the overall incidence of left-handedness in the particular study. Data points are plotted plus or minus one standard error. R x R progeny are plotted as small filled circles, R x L progeny as open circles, and L x L progeny as filled squares. In view of the small numbers of L x L matings, several groups of studies have been combined, as indicated by the two large inclusion brackets. The abscissa represents the population incidence of left-handedness, and the ordinate represents the percentage of left-handedness in the particular progeny type being shown.

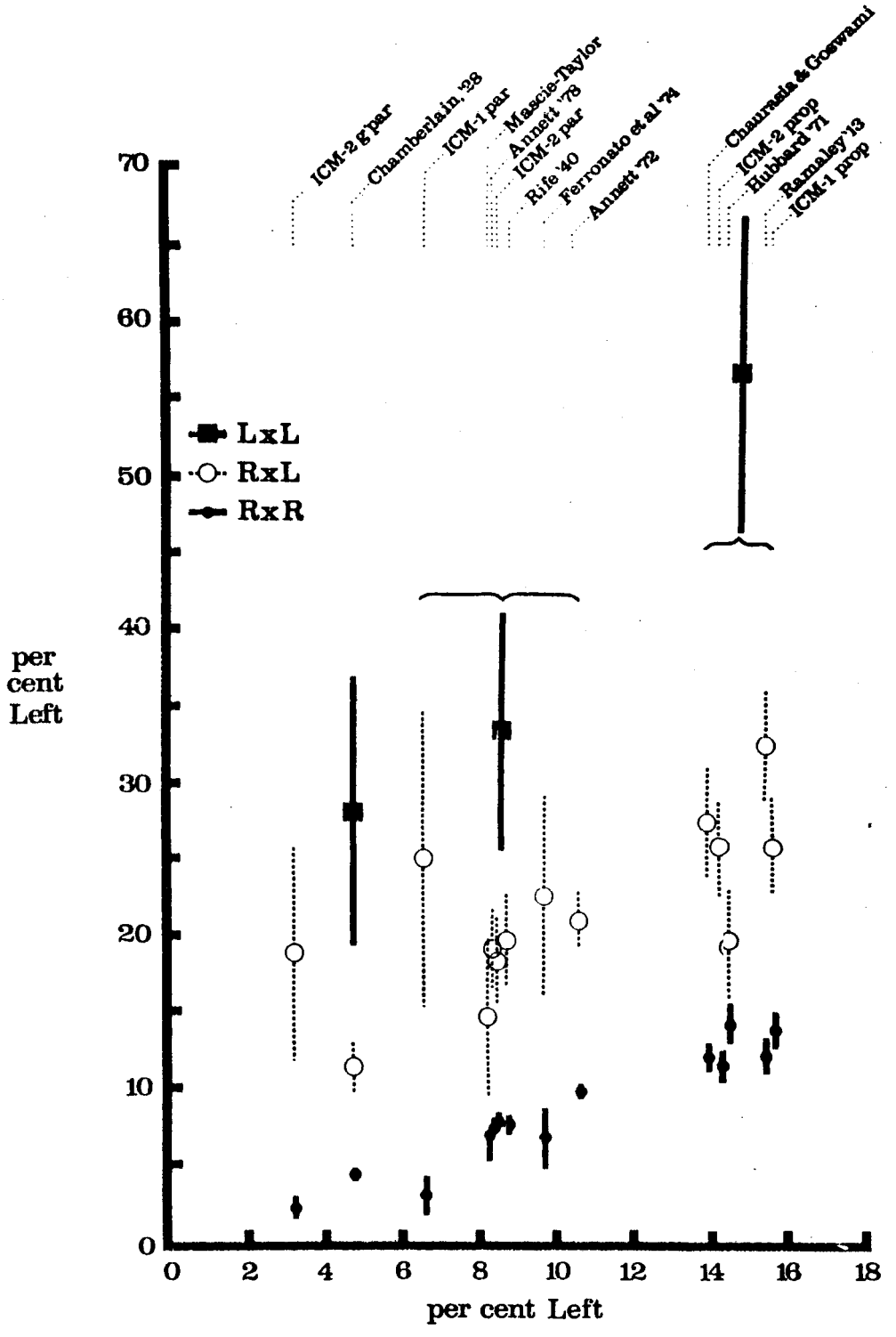

ICM-2 g'par
Chamberlain, '28
ICM-1 par
Mascie-Taylor
Annett '78
ICM-2 par
Rife '40
Ferronato et al '74
Annett '72
Chaurasia & Goswami
ICM-2 prop
Hubbard '71
Ramaley '13
ICM-1 prop
LxL
RxL
RxR
per cent Left
per cent Left
0
10
20
30
40
50
60
70
0
2
4
6
8
10
12
14
16
18

Figure 7.2 shows the goodness of fit of the Annett (1972) model to all the 14 data sets of Table 7.1, and all of the 18 MZ and 17 DZ twin data sets of Table 7.2. Figures 7.2 to 7.13 are all basically similar and have the same conventions. Figures 7.2, 7.3, 7.6, 7.8, 7.10, 7.11 and 7.13 each show six small contour maps. The upper left map (a) is for the goodness of fit of the progeny of R x R matings, the upper right map (b) is for the goodness of fit of the progeny of R x L matings and the middle left map (c) is for the goodness of fit of the progeny of L x L matings. The combined values of these three values are shown in the middle right map (d). The goodness of fit of MZ twins is shown in the lower left map (e) and the goodness of fit of the DZ twin data is shown in the lower right map (f). The combination of the MZ, DZ and Family data (i.e, maps d, e and f) is shown in Figures 7.5, 7.7, 7.9 and 7.12; it is not applicable in all cases.

The abscissa of each box represents a particular value of the degree of inheritance; in figures 7.3-12 it is the value of $p(L|DC)$, and in figures 7.2 and 7.13 it is the size of the 'right shift' (in z units). The ordinate in figures 7.3-12 is the hypothesised true incidence of left-handeness, and in figures 7.2 and 7.13 the ordinate is the proportion of individuals who are of phenotype RS-.

Within the boxes are plotted contour maps of the probability density of the goodness of fit of the particular models. In general, green contours are acceptable with $p > 0.05$ and red contours are rejected with $p < 0.05$. Green

contours are plotted at levels of 0.05, 0.10, 0.2o, 0.30, 0.40, 0.50 (thickened line), 0.60, 0.70, 0.80, and o.90, and 0.95. Red contours are plotted at levels of 0.045, 0.01, 0.005, 0.001 (thickened line), 0.0001, 0.00001, 0.000001 and 0.0000001, as well as at values of 0.955, 0.99, 0.999, 0.9999 and 0.99999 (i.e. the fit is too good).

In boxes a,b,c,e and f the red and green contours may simply be read off to show the goodness of fit of a particular model. For the combined family data (box d), and for the overall combined data (figures 7.5, 7.7, 7.9 and 7.12), the probability levels depend upon the particular number of degrees of freedom used. As explained in appendix 7.1, this is somewhat controversial. For the family and combined data I have therefore plotted two distinct sets of probability contours. The red and green contours represent the probability values stated earlier for the more <u>conservative</u> test of goodness of fit. In these boxes there is also a heavy black line, which is always outside the green area, and this represents the goodness of fit at the 0.05 level, on a more <u>liberal</u> test.

If for the combined data there is no fit both at the 0.0 level on the more liberal test, and at the 0.0000001 level on the more conservative test, then no graph is given.

For figures 7.3 to 7.7 the degrees of freedom for boxes a-f, and the combined data, are 14, 14, 3, 17 (31), 18, 17 and 52 (66) respectively, liberal tests, where relevant, being shown in brackets. For figures 7.8 to 7.12 the respective degrees of freedom are 12, 12, 3,15 (27), 17, 16 and 48 (60). For figure 7.2 the degrees of freedom are 14, 14, 9, 23 (37), 18, 17, 60 (74), and for figure 7.13 are 12, 12, 9 21(33), 17, 16 and 56 (68). The difference between the Annett and other models is that in the case of the Annett model, data sets have not been combined when the L x L values are relatively small; in consequence the estimate of the Chi-squared and the df may be slightly in error.

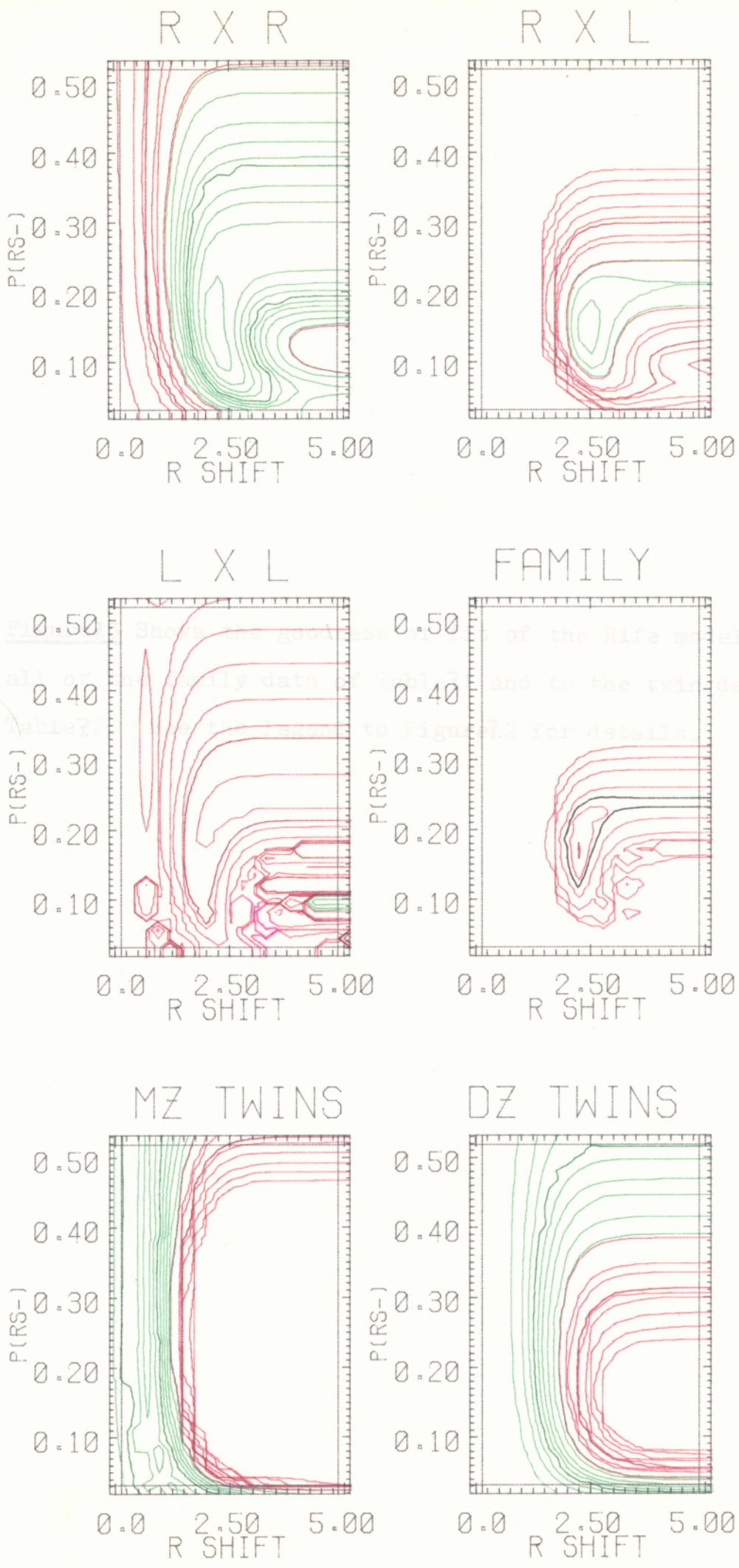
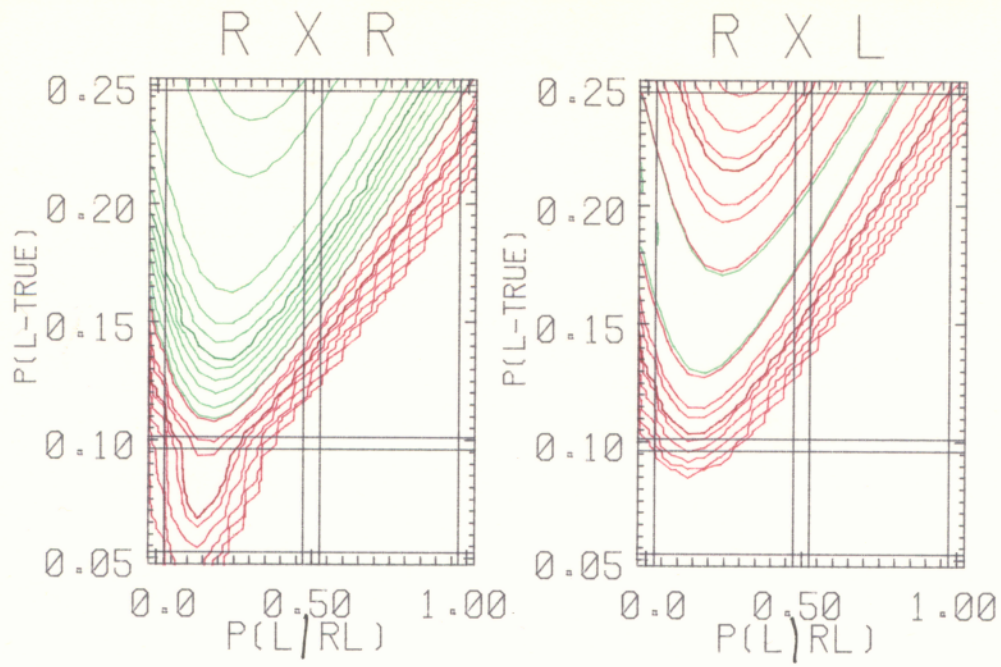
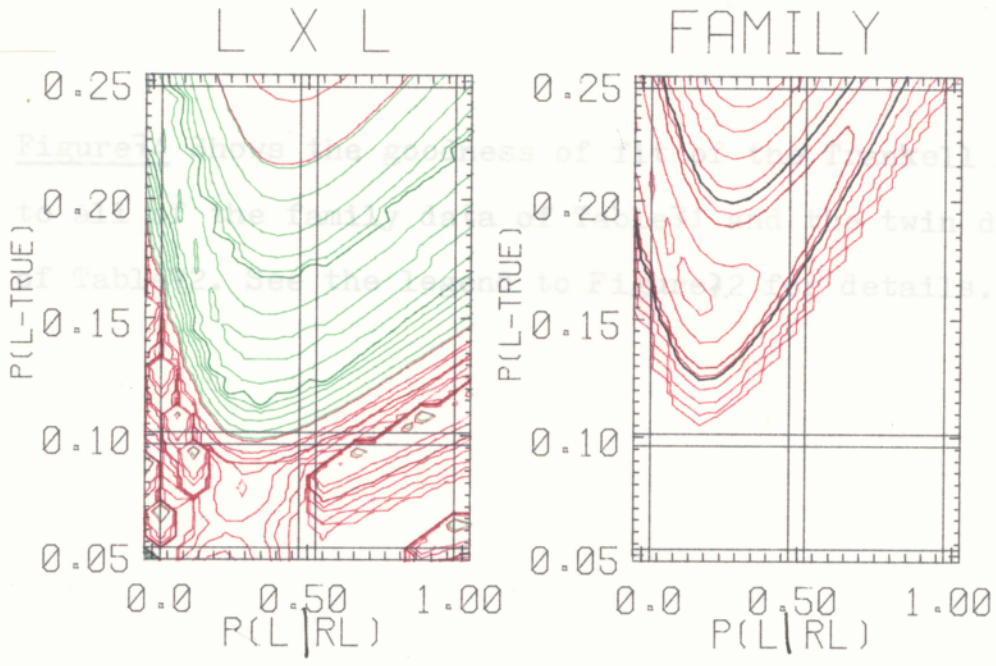

R X R
R X L
L X L
FAMILY
MZ TWINS
DZ TWINS
P(RS-)
0.50
0.40
0.30
0.20
0.10
0.0
2.50
5.00
R SHIFT

Figure 7.3 Shows the goodness of fit of the Rife model to all of the family data of Table 7.1 and to the twin data of Table 7.2. See the legend to Figure 7.2 for details.


R X R
R X L
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)


L X L
FAMILY
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)

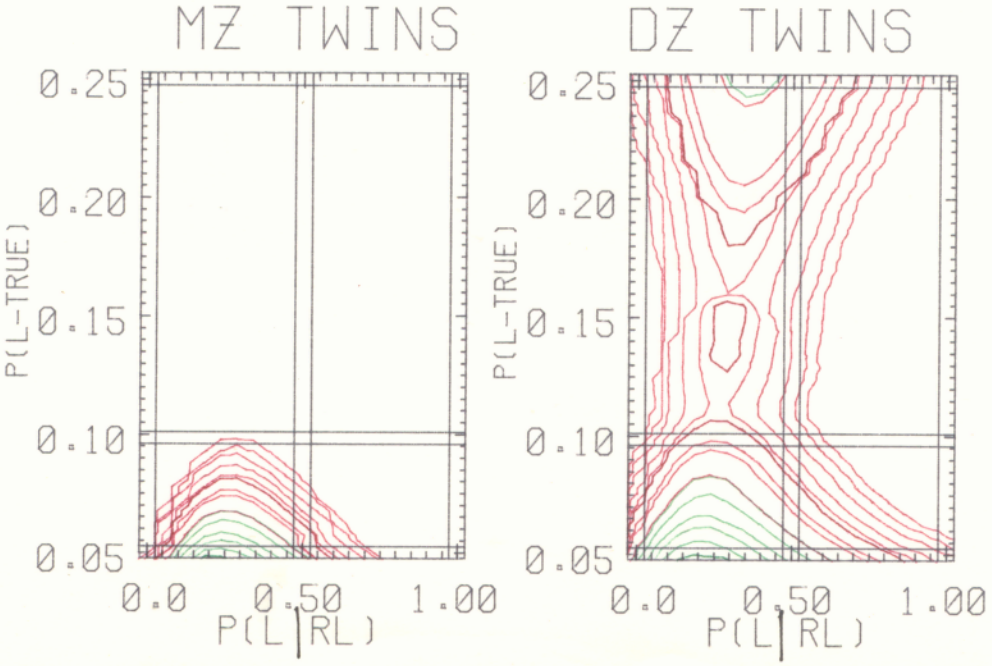

MZ TWINS
DZ TWINS
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)

Figure 7.4 Shows the goodness of fit of the Trankell model to all of the family data of Table 7.1 and the twin data of Table 7.2. See the legend to Figure 7.2 for details.

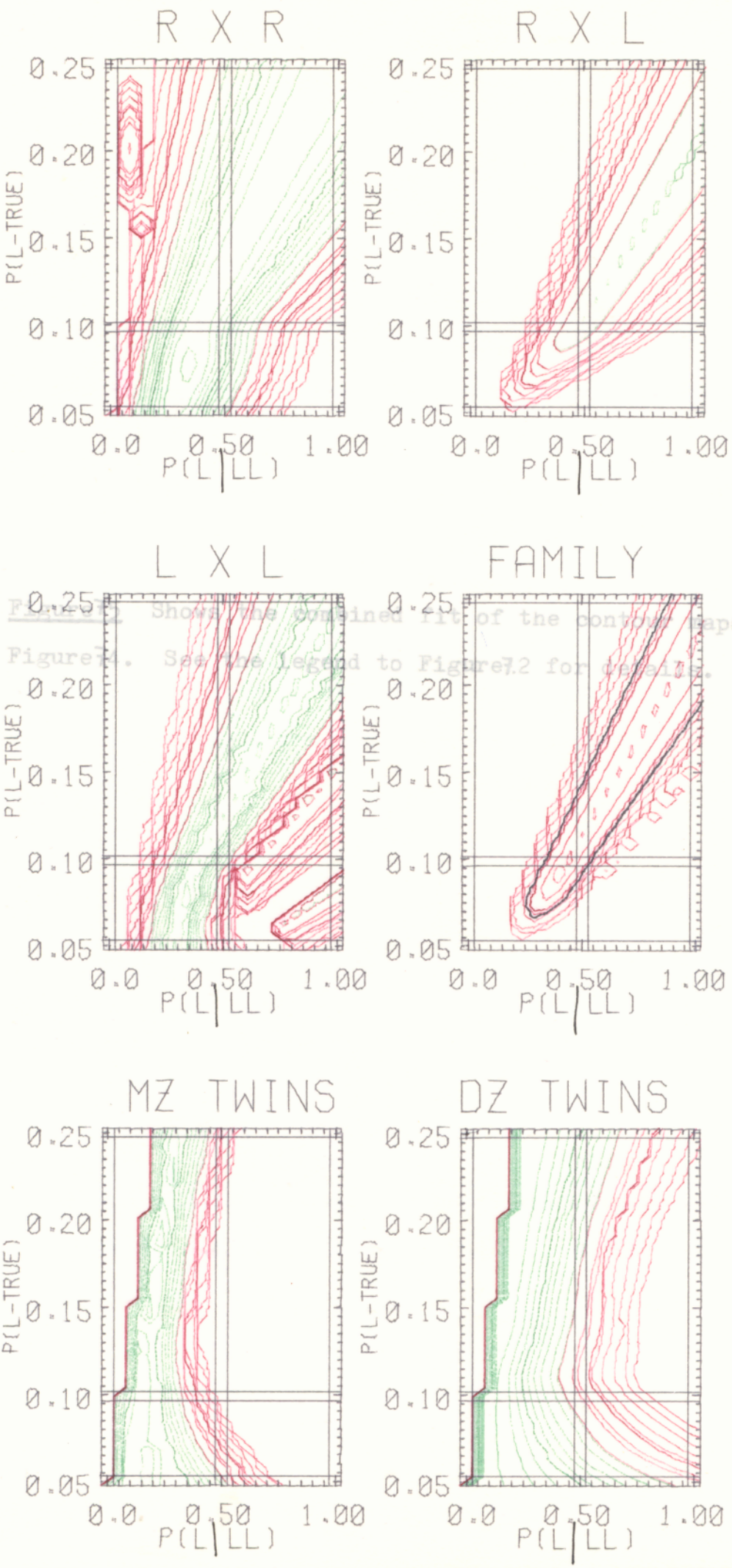


Figure 7.3 Shows the combined fit of the contour maps of Figure 7.1. See the legend to Figure 7.2 for details.

Figure 7.5 Shows the combined fit of the contour maps of Figure 7.4. See the legend to Figure 7.2 for details.

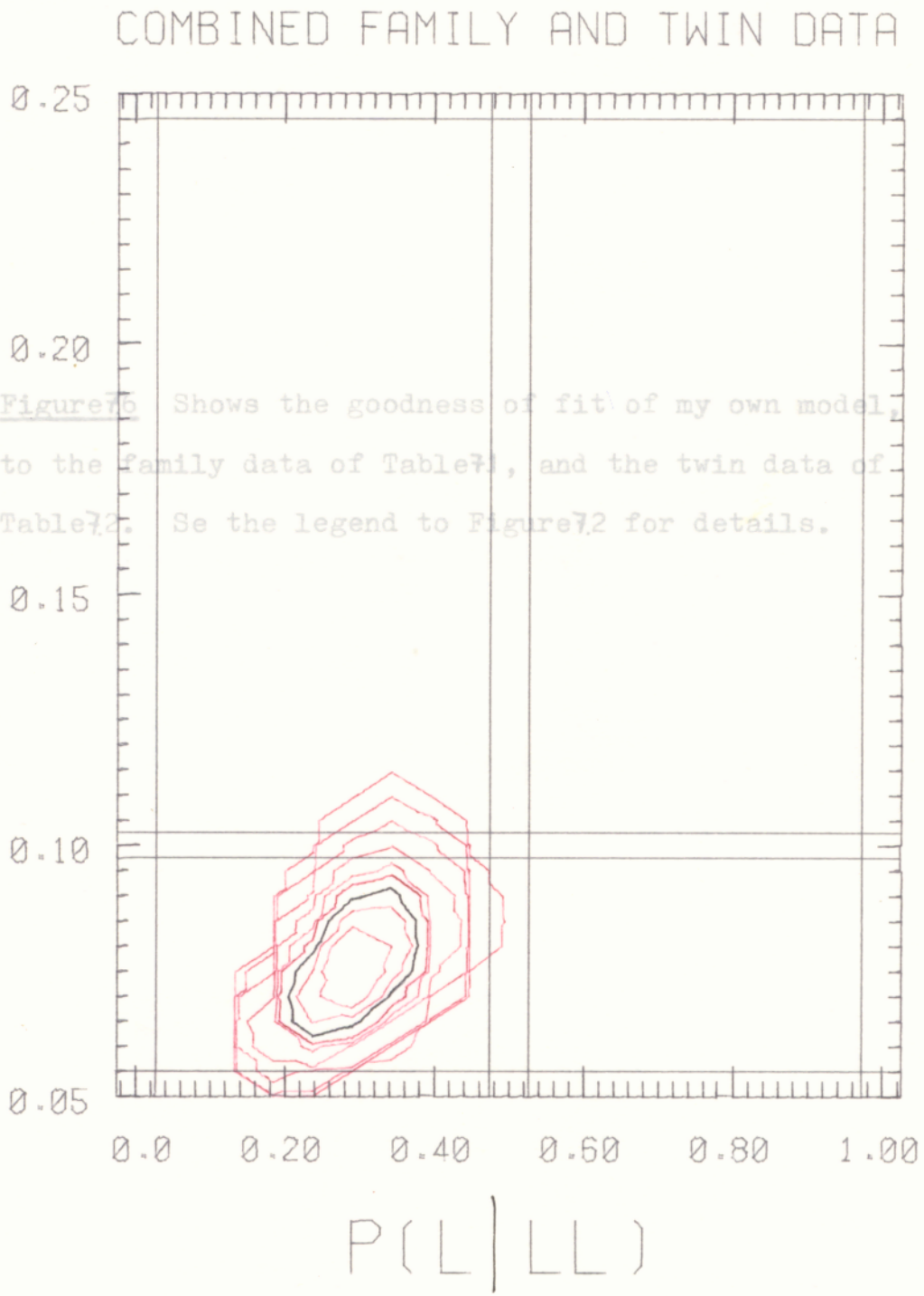
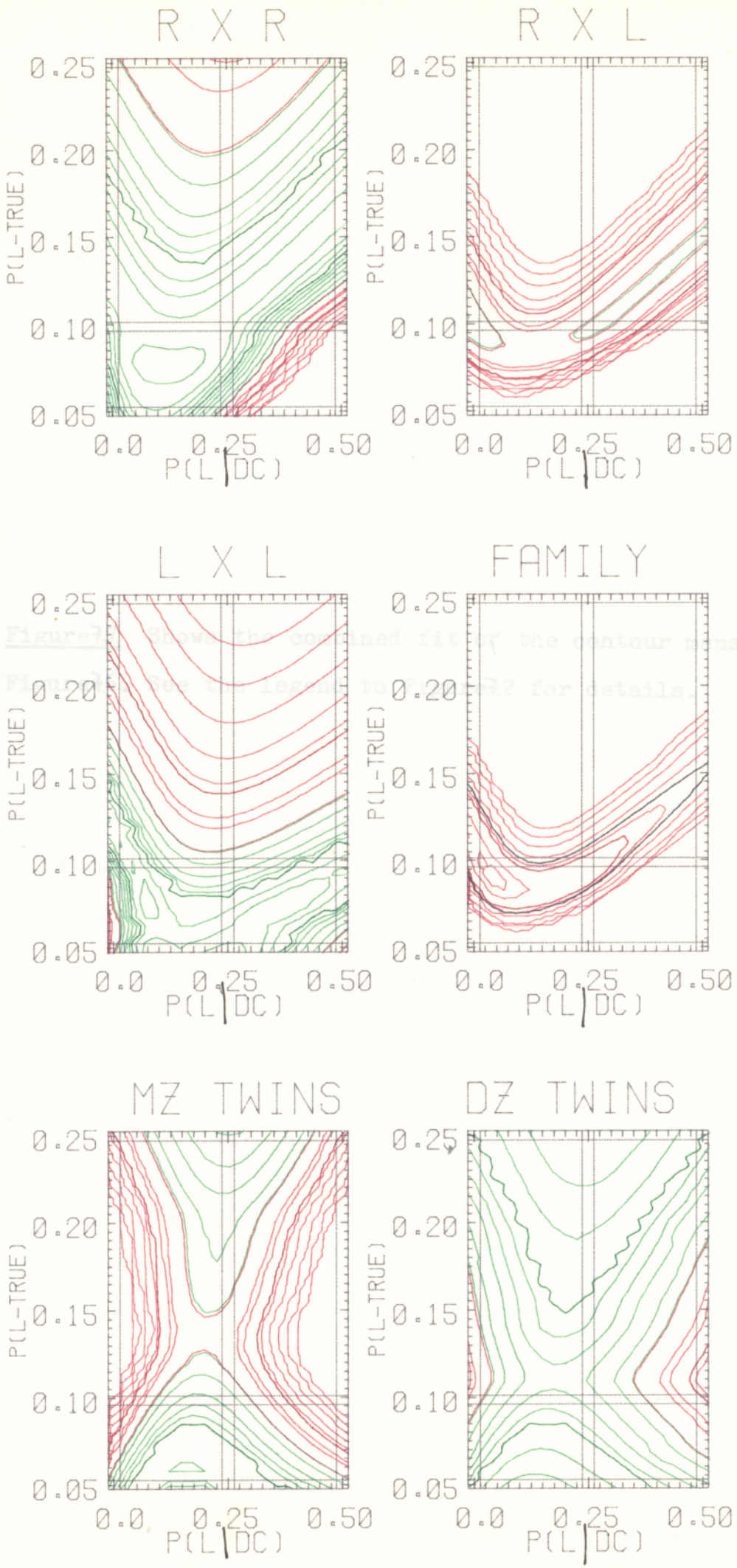


Figure76 Shows the goodness of fit of my own model, to the family data of Table71, and the twin data of Table72. Se the legend to Figure72 for details.

Figure7.6 Shows the goodness of fit of my own model, to the family data of Table7.1, and the twin data of Table7.2. Se the legend to Figure7.2 for details.


R X R
R X L
L X L
FAMILY
MZ TWINS
DZ TWINS
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

ICM-14

Figure 7.7 Shows the combined fit of the contour maps of Figure 7.6. See the legend to Figure 7.2 for details.

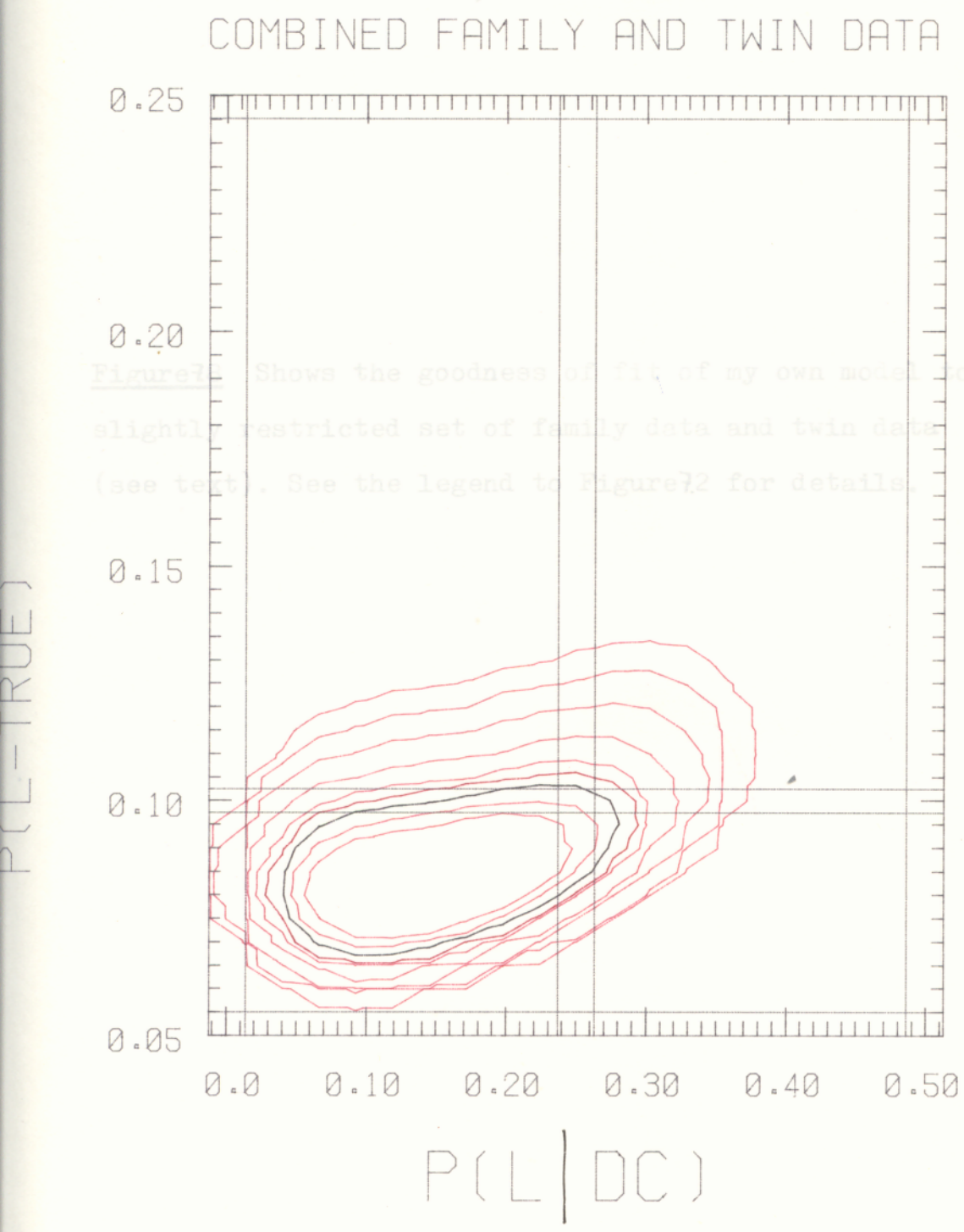

COMBINED FAMILY AND TWIN DATA
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.10
0.20
0.30
0.40
0.50
P(L|DC)

Figure 18 Shows the goodness of fit of my own model to a slightly restricted set of family data and twin data (see text). See the legend to Figure 12 for details.

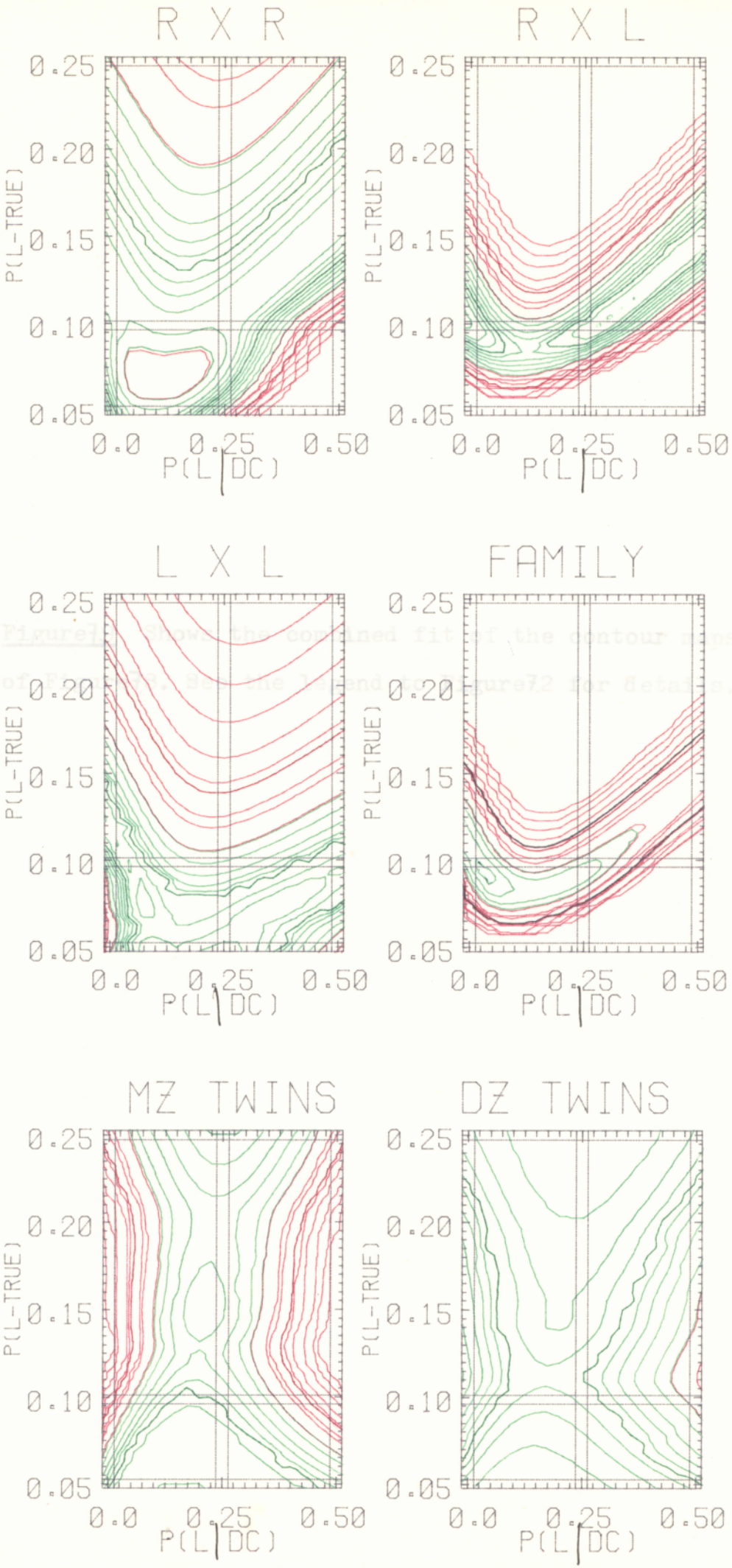

R X R
R X L
L X L
FAMILY
MZ TWINS
DZ TWINS
P(L-TRUE)
P(L|DC)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50

Figure 7.9 Shows the combined fit of the contour maps of Figure 7.8. See the legend to Figure 7.2 for details.

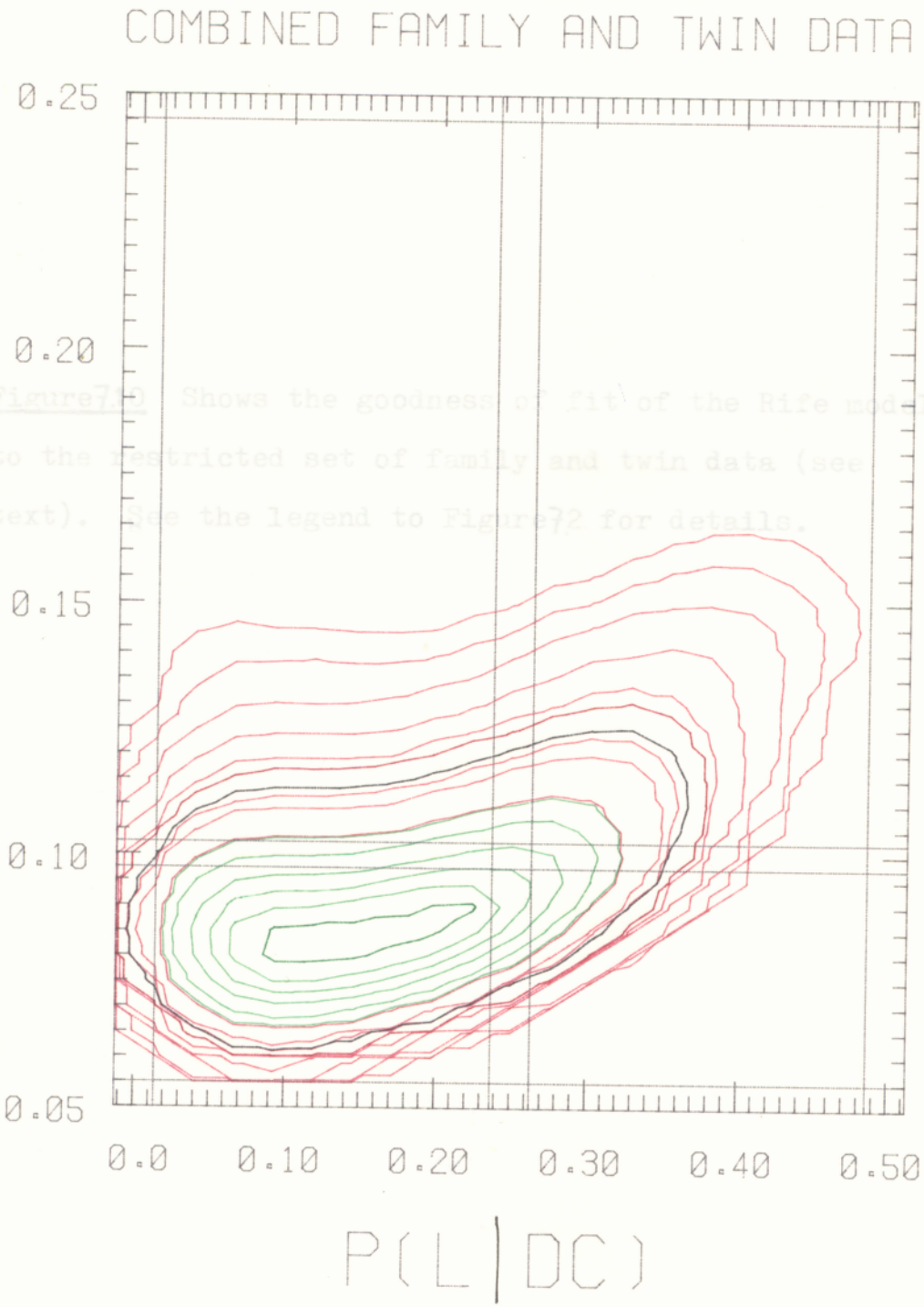
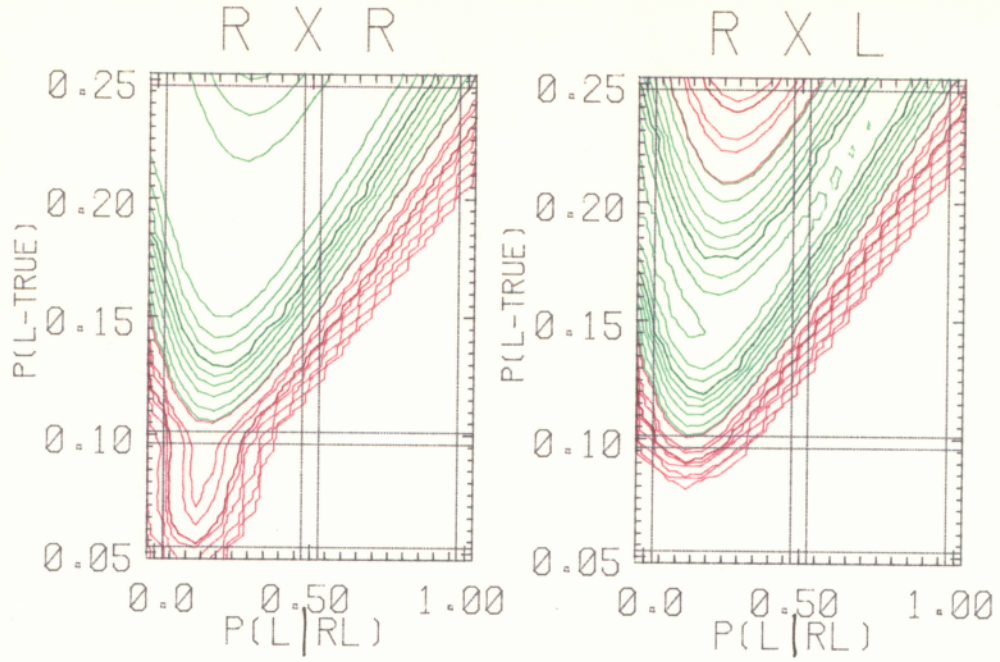
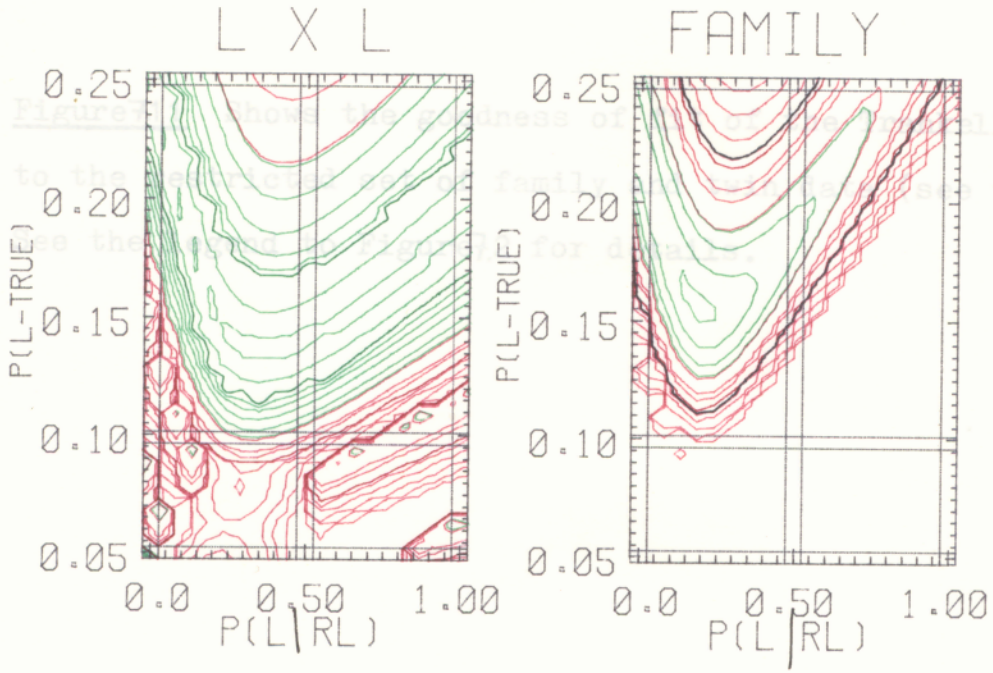

COMBINED FAMILY AND TWIN DATA
0.25
0.20
0.15
0.10
0.05
0.0
0.10
0.20
0.30
0.40
0.50
P(L|DC)

Figure7.10 Shows the goodness of fit of the Rife model to the restricted set of family and twin data (see text). See the legend to Figure7.2 for details.


R X R
R X L
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)


L X L
FAMILY
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)

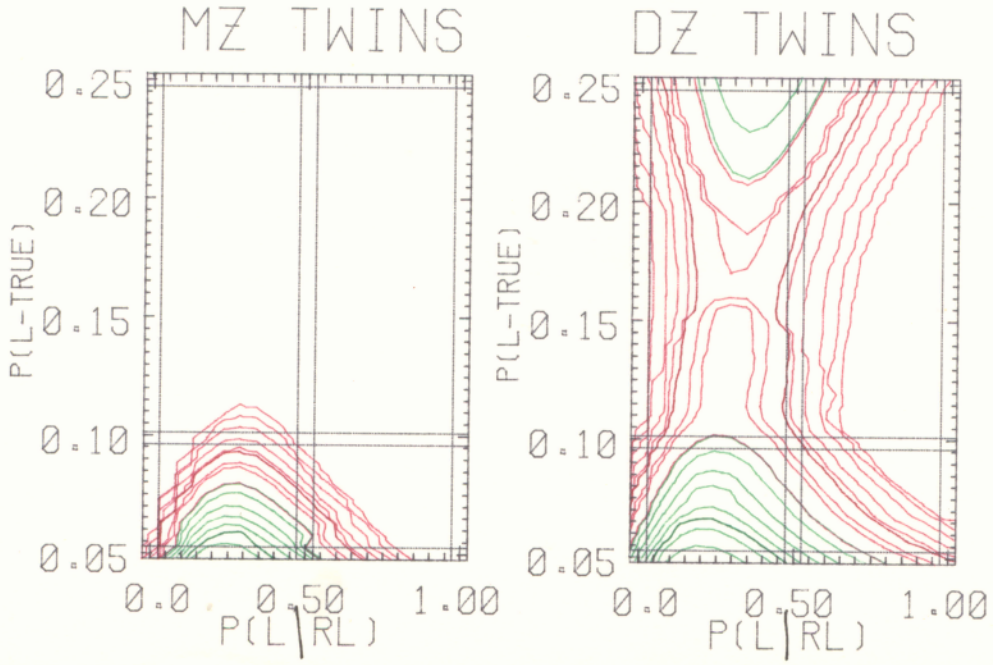

MZ TWINS
DZ TWINS
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|RL)

Figure7.11 Shows the goodness of fit of the Trankell model to the restricted set of family and twin data (see text). See the legend to Figure7.2 for details.

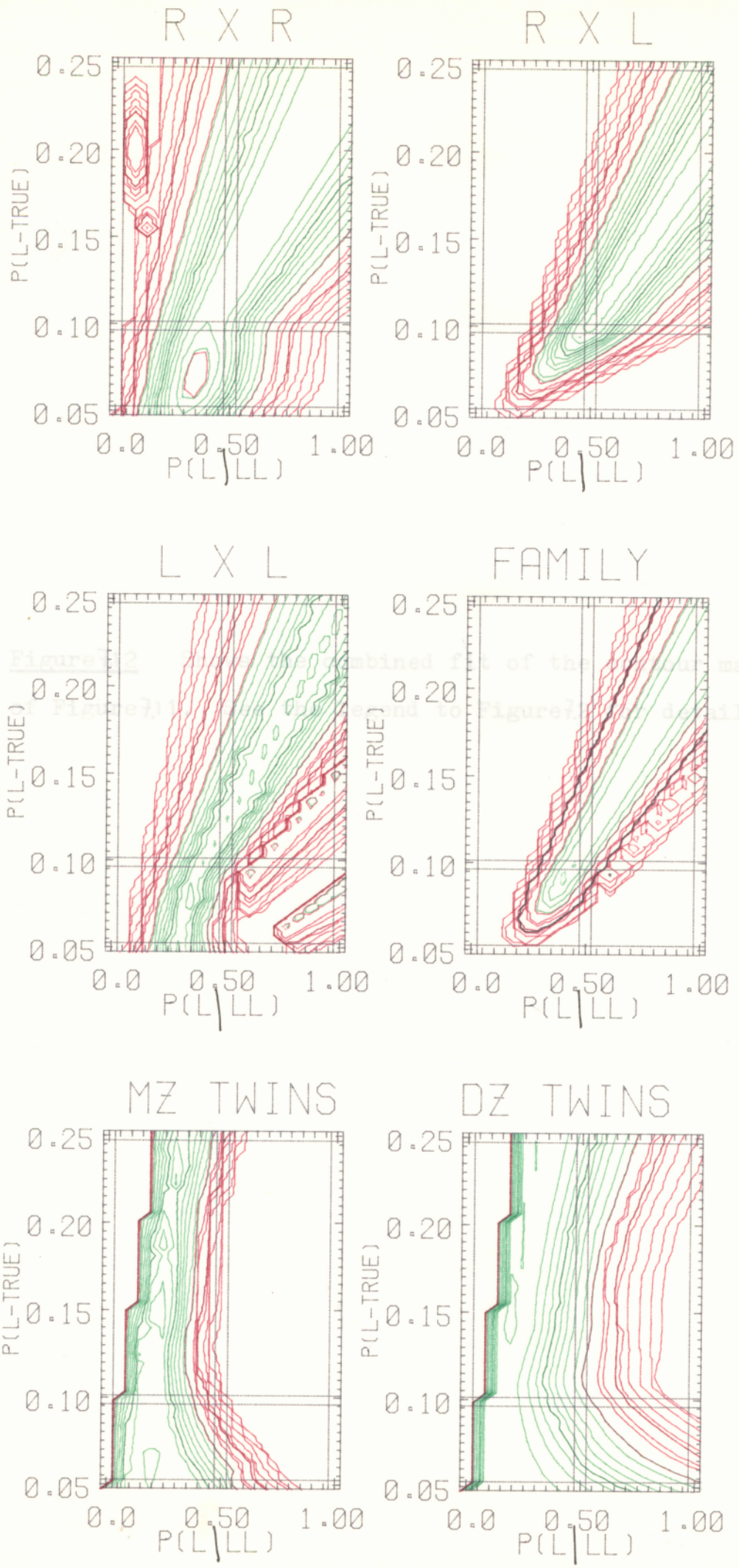

R X R
R X L
L X L
FAMILY
MZ TWINS
DZ TWINS
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|LL)

Figure 7.12 Shows the combined fit of the contour maps of Figure 7.11. See the legend to Figure 7.2 for details.

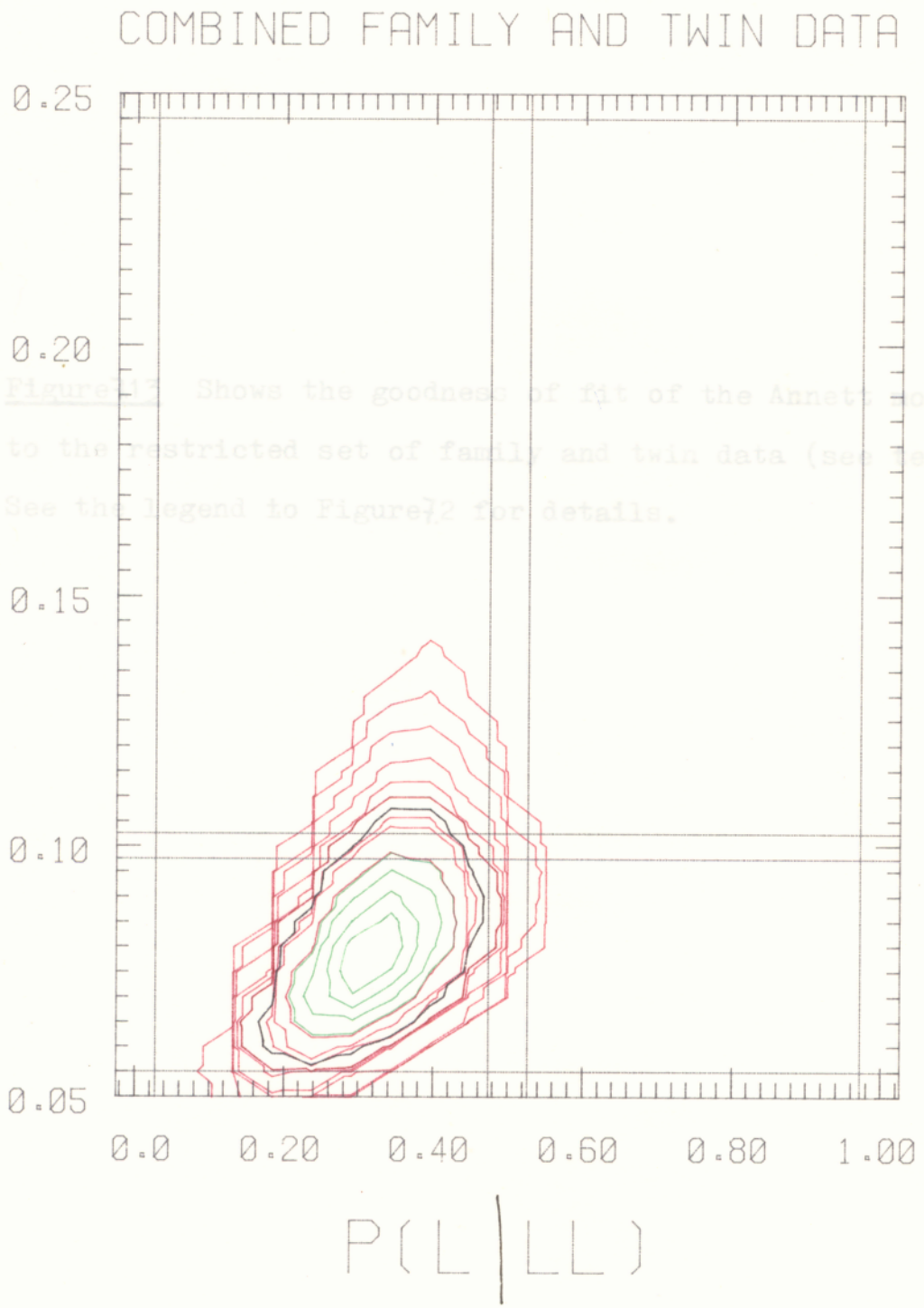
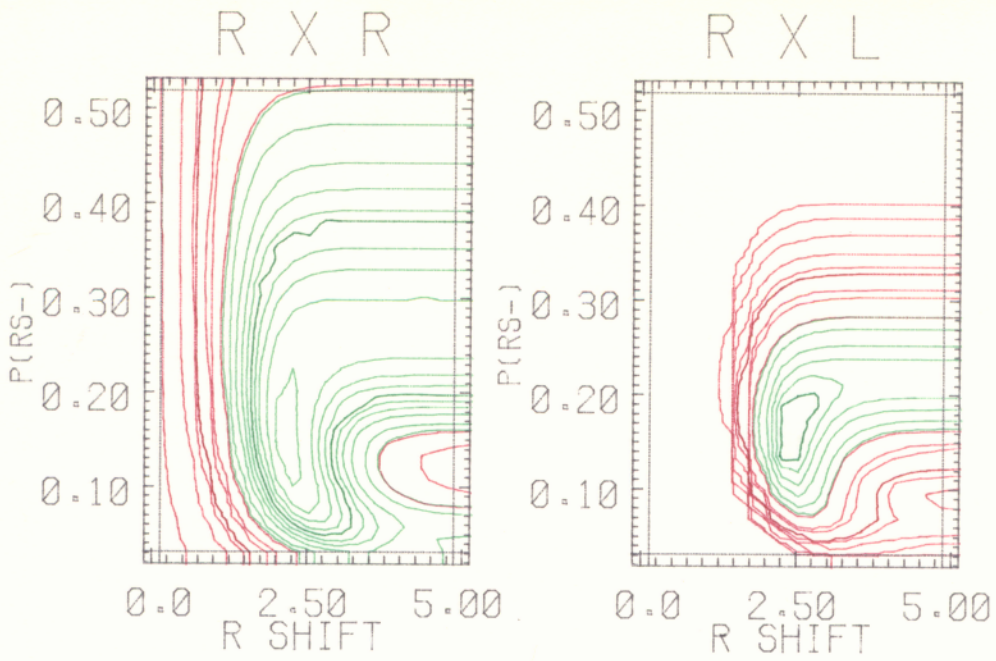
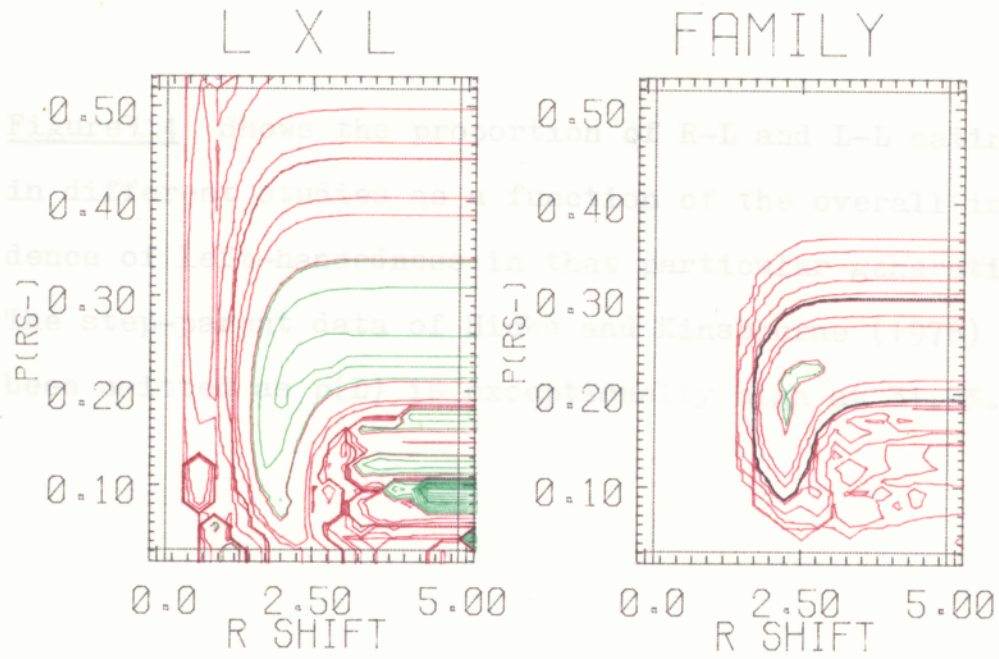

COMBINED FAMILY AND TWIN DATA
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.20
0.40
0.60
0.80
1.00
P(L|LL)

Figure 7.13 Shows the goodness of fit of the Annett model to the restricted set of family and twin data (see text). See the legend to Figure 7.2 for details.


R X R
R X L
0.50
0.40
0.30
0.20
0.10
P(RS-)
0.0
2.50
5.00
R SHIFT


L X L
FAMILY
0.50
0.40
0.30
0.20
0.10
P(RS-)
0.0
2.50
5.00
R SHIFT

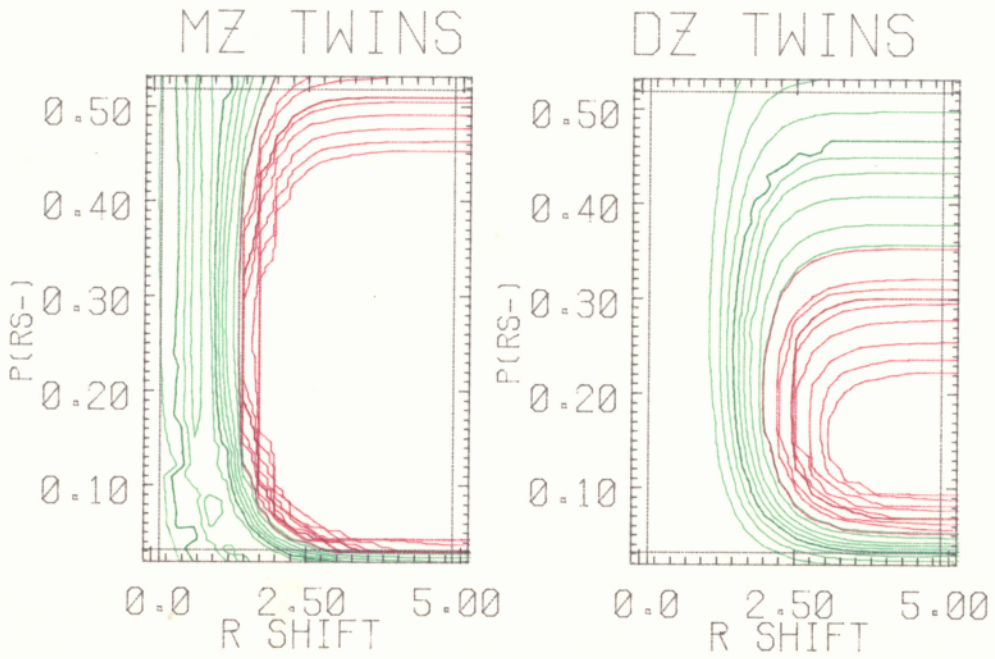
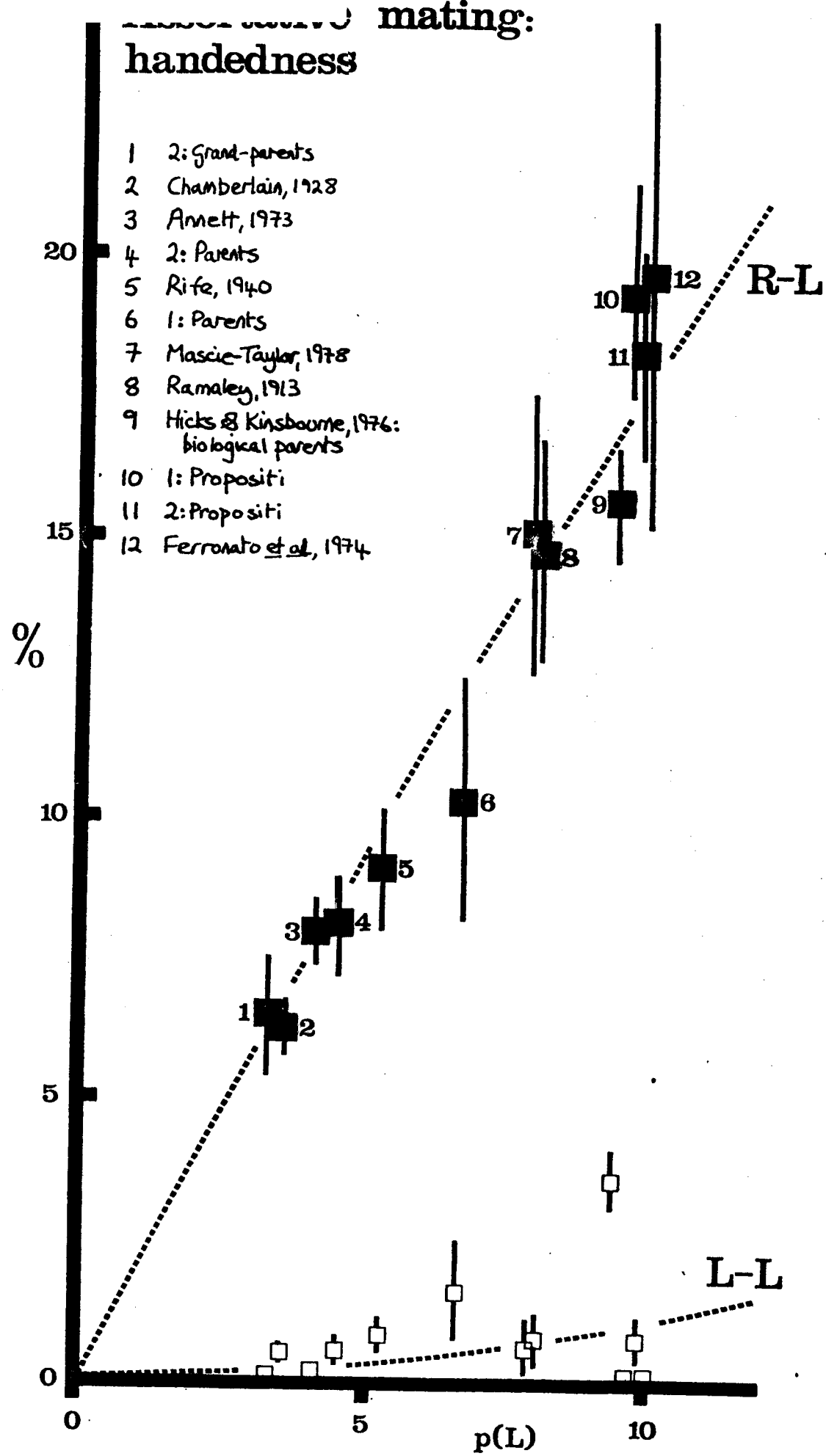

MZ TWINS
DZ TWINS
0.50
0.40
0.30
0.20
0.10
P(RS-)
0.0
2.50
5.00
R SHIFT

Figure 7.14 Shows the proportion of R-L and L-L matings in different studies as a function of the overall incidence of left-handedness in that particular generation. The step-parent data of Hicks and Kinsbourne (1976) have been omitted as p(L) is exceptionally high at 31.9%.


mating:
handedness
1 2: Grand-parents
2 Chamberlain, 1928
3 Arnett, 1973
4 2: Parents
5 Rife, 1940
6 1: Parents
7 Mascie-Taylor, 1978
8 Ramaley, 1913
9 Hicks & Kinsbourne, 1976: biological parents
10 1: Propositi
11 2: Propositi
12 Ferronato et al, 1974
R-L
L-L
%
20
15
10
5
0
0
5
10
p(L)

CHAPTER 8: A GENETIC MODEL OF SPEECH DOMINANCE

"Altogether unexpectedly my father had a shock of palsy; it lamed his right side and deprived him of the proper use of speech! We had to guess at everything that he required; for he never could pronounce the word that he intended."

Goethe, Wilhelm Meister's Apprenticeship;
translated by Carlyle

8:1 The requirements of a model of speech dominance

As with handedness, so with speech dominance there is a set of observations which any acceptable model must be able to explain:-

i. Cerebral speech dominance. This can be assessed in several different ways in normal subjects. The most common method, using dichotic listening, finds that about 15% of right-handers and 35% of left-handers show a left hemisphere effect (i.e. a right ear advantage). Results from unilateral ECT studies, from intra-carotid sodium amytal, and from the position of the hand during writing, produce basically similar results.

ii. Loss of speech after a unilateral cerebral lesion is more common if the lesion is on the left side of the head (about 40%) than if the lesion is on the right side of the head (3 or 4%).

iii. In persons who have suffered loss of speech after a unilateral brain lesion, about 2-3% of right-handers and 20-30% of left-handers, have a right-sided lesion. The incidence of crossed speech is thus estimated very differently from clinical cases, than from dichotic or ECT studies.

iv. Left-handers seem both more prone to aphasia after a

cerebral lesion, and also more likely to recover from such aphasia, so that overall the prognosis is better in left-handers than right-handers (Gloning et al, 1969; Subirana, 1958; Goodglass and Geschwind, 1976; Hécaen and Albert, 1978; Hicks and Kinsbourne, 1978).

v. A familial history of sinistrality seems to increase the probability of recovery from aphasia (Zangwill, 1960; Hicks and Kinsbourne, 1978).

In the present chapter I shall describe a genetic model of speech dominance which is explicitly derived from the earlier genetic model of handedness. That model, in its general form, accounted for most of the observed data; the data however did not allow a complete discrimination between possible models, although in principle the models are distinguishable. For the present chapter I will think primarily in terms of three major alternatives:-

1. $p(L) = 0.095$, $p(L|DC) = 0.25$ and $p(L|CC) = 0.05$. This is the 'additive' model, which is my own preference from the three.

2. $p(L) = 0.08$, $p(L|DC) = 0.125$, and $p(L|CC) = 0.05$. This is the other main version of my own model which takes fluctuating asymmetry as a conceptual starting point.

3. $p(L) = 0.075$, $p(L|RL)=0.0$ and $p(L|LL)=0.30$. This is

the Trankell model with corrections for differing incidences of left-handedness.

In the present chapter I will describe the extension of the handedness model to the problems of speech dominance and aphasia. The three separate versions of the genetic model will all be shown to be equally compatible with the available data; nevertheless, as with the handedness data, the failure to distinguish between models is primarily a defect of sample size, or of the collection of inappropriate or incomplete data, since the models are in principle distinguishable. Unless otherwise stated, I shall assume that the first model, the additive model, is actually the correct one.

8:2 Cerebral speech dominance in normal subjects

The literature contains, to my knowledge, ten separate studies of dichotic tests in relation to handedness. These studies are very variable. Some, but not all, use three separate categories for classification of dominance (Right, Left and Equal); in such cases I have lumped together right and equal groups (the method of data fitting would also work if I had concatenated left and equal groups, although the arithmetic would be slightly different). I have also added together results from different studies. I have done this, despite severe reservations, because a) some studies are too small to permit useful analysis on their

own, and b) most studies are not random population samples, but choose differing numbers of right or left-handers, in some cases choosing only right or left-handers. The data are summarised in Table 8.1. Most studies have been careful to select subjects of fairly strong handedness, and therefore I have taken handedness assessments at face value; from most studies it is anyway impossible to make a direct estimate of the manifest incidence of left-handedness. Overall of 373 right-handers, 19.03% were right hemisphere dominant (or showed no ear advantage at all); of 328 left-handers, 38.10% were right hemisphere dominant (right vs left handers , Chi-squared = 31.52, 1 df, $p < 0.001$).

In interpreting dichotic tests it is important to remember that the test-retest correlation is relatively low; thus Pizzamiglio *et al* (1974), found in adults that only 70% of individuals showed the same dichotic category (right, left or equal) on two separate occasions; Blumstein *et al* (1975) found a similar result, as did Bakker *et al* (1978) in children. Account must be taken of this when fitting genetic models, since the incidence of apparent right speech dominance will be artefactually raised (by a mechanism similar to that of Satz, 1972).

Table 8.2 summarises data from five studies of speech dominance assessed by unilateral ECT. As with the dichotic studies, right hemisphere dominance and 'no hemisphere

dominance' groups have been lumped together, and handedness judgments have been taken at face value. In total, of 127 right-handers, 8.66% had right-hemisphere dominance, whilst of 48 left-handers, 29.16% had right-hemisphere dominance (Chi-squared = 11.96, 1 df, $p < 0.001$).

Table 8.3 summarises data from two studies of speech dominance assessed by intra-carotid sodium amytal. Unfortunately I have ~~only used~~ used only the data of Milner et al (1964) for the calculations to be reported later in this chapter, since I did not discover the paper by Milner (1975) until too late. 9.09% of 48 right-handers, and 42.85% of 44 left-handers had non-left hemisphere speech (Chi-squared = 8.77, 1 df, $p<0.01$).

As well as these conventional ways of assessing dominance, I have also used data from a more unconventional and less well understood source - that of the position of the hand during writing. Levy and Reid (1976; 1978) have argued that a 'hooked' or 'inverted' writing position, in either right or left-handers, indicates crossed dominance for speech and hand control. Table 8.4 summarises data from two studies of my own and also one of Lawson (1978) of the incidence of an inverted writing position in right and left-handers, 12.9% of 968 right-handers, and 32.4% of 222 left-handers claim, on questionnaires, to have inverted writing positions, figures which closely resemble those from other sources of dominance. For this study I

have therefore trested handwriting position as an 'honorary marker of cerebral speech dominance', in much the same way as geneticists regard PTC tasting as an honorary blood group. However it should be noted that the data of Table 8.4 are in strict contradiction of Levy and Reid's hypothesis (as also is the result of Peters and Pedersen, 1978), and that the best interpretation of an inverted writing position is that it might indicate right-speech dominance (or at least atypical dominance of <u>something</u>) in both right <u>and</u> left-handers. The theories to be presented here do not depend critically upon the hand-writing position data, and thus should the present speculations prove false or unsubstantiable, then no change in the theories need occur; the data are merely included in view of their potential interest.

Figure 8.1 summarises graphically the data from Tables 8.1 to 8.4. The abscissa is the overall percentage of the condition in the population (and thus varies according to the method used; it has been calculated by making the assumption that 9.5% of the population are actually left-handed, and then weighting the data of Tables 8.1 to 8.4 appropriately). As with the studies of handedness, I therefore wish to propose that all these studies are actually measuring the same thing, and that differences between the studies are merely due to measurement error, differences in criterion and so on.

In order to fit the genetic model of handedness to the above data, a single assumption has had to be made: that if the effect of a particular genotype is to produce a probability, p, that an individual will be left-handed, then the same genotype produces right hemisphere speech dominance with the same probability, p, and that the two processes are strictly independent. Thus individuals with the DD genotype will all be right-handed and left-speech dominant, whilst those individuals who are of the CC genotype will be right-handed with right-speech dominance in a quarter of cases, right-handed with left-speech dominance in a quarter of cases, etc.

As with the case of handedness, one may fit variants of the genetic model to the data, fitting by a combinatorial method, all possible values of x (the degree of dominance), and $p(L_t)$, the true incidence of left-handedness. Figure 8.2 shows the goodness of fit of all four types of data considered together (i.e. combining significance values between each data type, but combining data itself within each data type). Table 8.5 gives the actual and expected values for the additive model.

It can be seen from Figure 8.2 that there is an adequate fit for models which are approximately additive in their degree of dominance, and that the hypothetical value of the incidence of sinistrality has little effect upon the goodness of fit. The fitted data lines in figure

8.1 are those which would be expected with an additive model with $p(L_t) = 9.5\%$.

Thus the model can cope adequately with these data on handedness and cerebral speech dominance. Note that although I am here fitting a genetic model to data, there are no actual data which a geneticist would find adequate. This is simply because there are no sufficiently large-scale, or well-defined, family studies of handedness and dichotic speech dominance; the single exception of Bryden (1975) was not large enough to fit.

It is to be expected that from the present type of model that there should be an increased incidence of right speech dominance on dichotic tests if there is a family history of left-handedness. Table 8.6 shows the expected proportions of right speech dominance in various family combinations. There are no adequate data in the literature to test these predictions exactly. Nevertheless Zurif and Bryden (1968) have found increased proportions of right speech dominance on dichotic tests in individuals with sinistral family histories, although the result is less than generally clear (see McKeever and Van Deventer, 1977 for a review).

A similar conclusion applies to twins. Table 8.7 shows the exact proportions of monozygotic twins who would to expected to have right speech dominance. It is worth

noting in this table that the probability of right speech dominance in each twin from an R-L pair is the same, irrespective of the handedness of that particular twin. The only data which might be suitable for testing these predictions are those of Springer and Searlemann (1978), who found that 13.2% of 106 R-R MZ twins, and 21.1% of 38 R-L MZ twins were right speech dominant. Expected proportions are 5.7% and 28.3%. However since the manifest incidence of left-handedness in MZ twins in that study is 16.66%, and the weighted manifest incidence of left-ear advantage is 18.45%. corrections must be made, since my model would predict that the true values in each case are 9.5%; after such corrections the predicted values are 15.03% and 25.78%, which give a Chi-squared goodness of fit value of 0.72, which is acceptable.

8:3 Clinical data on speech dominance and handedness

As stated earlier, there is a glaring discrepancy between the incidence of right speech dominance in right-handers as assessed by dichotic tests, and that as assessed by data from aphasic patients. Thus, one might expect that since nearly 20% of right-handers show evidence on a dichotic test of right speech dominance, that a similar proportion of right-handed individuals should show evidence of dysphasia after a right-sided lesion. The true figure is however closer to 2%, a difference of one order of magnitude.

To cope with this problem I wish to propose a more extensive model of cerebral speech dominance. This model will be compatible with the model proposed in the previous section, but will also cope with the dichotic-clinical discrepancy.

8:4 A model of speech dominance

In the model for handedness a single phenotype, hand dominance, was suggested to vary randomly in the presence of the CC genotype. In the earlier model of dichotic speech dominance, two phenotypes were supposed to vary randomly and independently with the CC genotype, i.e. handedness and dichotic asymmetry. In the present model I wish to extend this concept further and to propose that in the CC genotype, three, or even more, asymmetric phenotypes may vary randomly and independently.

Consider the case of speech dominance. As well as the handedness phenotype, let us now consider two speech 'dominances'. Let Speech-A (SA) and Speech-B (SB) both vary randomly and independently in genotype CC; of course in the DD genotype all individuals will have SA, SB and hand control in the left hemisphere. It must also be emphasised from the start that this is a purely formal model: there is no suggestion or implication that these two speech centres are related to Broca's or Wernicke's areas - indeed from the point of view of the present approach both of those areas

may each be considered as themselves possibly having several independent random asymmetric phenotypes. Eight phenotypes are thus proposed, from handedness (H), SA and SB all being in the left hemisphere, to H, SA and SB all being in the right hemisphere, Naturally some of these phenotypes will be far more common than others, with LLL (i.e. H, SA and SB in that order) being most common, and RRR being the rarest.

In order to make this model compatible with the previous dichotic model wer merely have to say that SA has more effect on dichotic tasks than SB. If SA and SB are both in the left hemisphere we would expect a strong right-ear advantage (REA), whilst if SA and SB are both in the right hemisphere we would expect a strong left-ear advantage (LEA). But if SA is in the left hemisphere and SB is in the right hemisphere we would expect only a modest REA, and if SA is in the right hemisphere and SB is in the left hemisphere we would expect only a modest LEA. Equating weak and strong ear advantages the present model of dichotic listening becomes the same as the previous one of Chapter 8.2.

The SA-SB model has several important consequences for dichotic listening tests. There has been much controversy over the optimal way in which to describe an individual's dichotic asymmetry score (e.g Marshall *et al*, 1975; Birkett, 1977); thus some authors use the simple score

(L-R), whilst others use (L-R)/(L+R), or other variants upon the basic data; Colbourn (1978) has even suggested it is possible that it is not valid to measure degree of dominance at all. Either way I would suggest that the present controversy is partially misguided in the absence of some understanding (or even simple description) of the underlying structure of the scores (in the same manner as the underlying structure of handedness is of critical importance for the description of handedness). Nowhere in the literature can I find a simple graph of the distribution of dichotic asymmetry scores. There are however, anecodotal reports to the effect that REA's seem more robust than LEA's; and that those individuals with REAs seem to divide into two gross categories, those with strong ear advantages and those with weak ear advantages (see Berlin and Cullen, 1977; Hicks and Kinsbourne, 1978).

Drawing upon a combination of such anecdotes and the genetic model I am proposing, I would therefore suggest that dichotic asymmetries have a structure of the form shown in Figure 8.3. There is a quadrimodal distribution heavily skewed to a right-ear advantage. The height of peaks B and C will be identical. In individuals with a right-ear advantage, the absolute size of the ear effect ought to be greater than in those with a lefteear advantage (since D > C and B > A). If the noise distributions (due to measurement errors) around A, B, C and D are large then (a) there will be a poor test-retest correlation for

those in categories B and C, and (b) the overall distribution will superficially resemble a skewed (or perhaps even a non-skewed) unimodal normal distribution. Such a model will explain several of the problems associated with dichotic listening tests; it perhaps also shows the potential folly of merely calculating the mean scores of individuals when underlying distributions have not been shown to be normal. Further predictions from the model, including the heights of A, B, C and D will become apparent later in the discussion.

The predictions of the earlier dichotic model for the effects of cerebral lesions were quite clear. If one has a speech centre in one hemisphere and that centre is damaged then aphasia will result. The proportion of aphasics with right cerebral lesions will hence be the same as the proportion of individuals with right cerebral speech, which will be the same as the incidence of left-handers. The SA and SB model is rather more complex. If both SA and SB are in the same hemisphere then there is no problem, for a lesion will incapacitate both simultaneously, and aphasia will result; throughout the argument it is assumed that SA and SB have the same cerebral location - clearly more complex models could be devised in which this is not the case. If SA and SB are in <u>different</u> hemispheres then two distinct models become possible. One may say that <u>both</u> speech centres are necessary for speech, alternatively that there is sufficient redundancy

to mean that either SA or SB will suffice for normal speech. I will in fact use both variants of the model by suggesting that in the event of an acute lesion then aphasia will result if either SA or SB is damaged; but that for a permanent aphasia to occur both SA and SB must be damaged. Thus aphasia will be only temporary if either SA or SB is damaged, or permanent, if they are both damaged. Table 8.8 shows detailed predictions of the proportions of individuals who will be of each of the various speech phenotypes, and for each group the probability that an acute or a permanent aphasia will result from a left or a right lesion.

This differentiation between acute and permanent aphasia produces several interesting predictions of the model.

Table 8.9 shows the proportion of individuals who will be expected to have a permanent aphasia after a unilateral lesion involving the relevant area, by handedness and familial handedness. Note that, overall, left-handers are less likely to suffer from permanent aphasia, and that in both right-and left-handers a positive sinistral family history also reduces the probability of a permanent aphasia. The present calculations assume an equal incidence of right and left-sided lesions, a true left-handedness incidence of 9.5%, and that all lesions are potentially aphasiogenic - the latter point will be returned to later.

Table 8.10 shows the probability of an acute aphasia, by handedness and familial handedness, and lesion side, for similar conditions to those of Table 8.9. Note that now left-handers are more likely to become aphasic than right-handers, and that a positive family history of left-handedness also increases the overall probability of acute aphasia. Thus left-handers are more likely to suffer from aphasia, and also more likely to recover from it, the overall result being a relatively lower incidence of permanent aphasia in sinistrals. Table 8.11 shows the proabbility of recovery from aphasia given that an individual has become acutely aphasic as a result of a unilateral lesion.

Table 8.12 gives the proportion of aphasics (both acute and permanent) who will be expected to have right-sided lesions. Note that for right-handed permanent aphasics from RXR families only 1.7% will have right-sided lesions. This figure would drop even further if one were also to exclude individuals with either sinistral grand-parents, aunts, uncles, siblings, children, etc. The figure is probably acceptable as an estimate of the incidence of aphasia after right-sided lesions in right-handers without left-handed relatives. Note also that the incidence of acute aphasia in right-handers from R x R families is far higher, at just over 9%; surveys of acute aphasia do indeed show a far higher incidence of right-sided lesions in right-handers. This discrepancy from conventional accounts may readily be explained when it is remembered that most patients

who are seen by neuropsychologists (and written up by them in the journals) are inevitably permanent aphasics, and thus the impressionis of a relatively low incidence of right-sided lesions.

One may speculate on the mechanism of this difference between acute and permanent aphaisa. In an ordinary individual with SA and SB in different hemispheres, there is presumably a high degree of interaction between the two centres. If one centre is suddenly destroyed then we may assume that this will, acutely, disrupt the functioning of the intact hemisphere, and that aphasia will result. However, given time such parients may be expected to recover the function of the intact centre. The mechanism of the interaction may perhaps be less psychological (i.e. functioning is impaired) and more physiological, the whole process perhaps being akin to spinal shock. Immediately after spinal section there is a complete loss of spinal reflexes below the lesion as a result of a loss of descending tone. After a variable period however, this shock disappears, and indeed the reflexes become hyperactive. Of more interest in this analogy is that if a second section is made below the first, then no spinal shock is seen. From this we may speculate on the role of the repeated insult to one or other of the speech centres*. The most relevant example of this would be found in chronic

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*My views here are remarkably similar to those of Riese (1970) who argues that one has to consider the role of diaschisis, or cerebral shock, in interpreting evidence on cerebral localisation and dominance. As he puts it:-

epileptics, who make up much of the population of persons whose speech is examined by amytal, split-brain techniques etc. (although other reversible lesions would also be relevant, such as aphasia as a result of transient ischaemic attacks, etc.). I would like to propose that in such cases (and I will justify this position later empirically) the continual insults render SA and SB autonomous, so that after an irreversible <u>acute</u> lesion the individual would only become aphasic if he would anyway have become <u>permanently</u> aphasic as a result of such a lesion.

The present model also has implications for work involving possible language functions in the right-hemisphere. Amongst right-handers, 28% may be expected to have <u>some</u> language function in the right hemisphere. Consequently the interpretation of individal cases of apparent language in the right-hemisphere, as after hemispherectomy, callosal section, or whatever, becomes very complex. The model would also predict that a proportion of individuals, particularly chronic epileptics, or those suffering repeated ischaemia. will apparently have

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"Speech defects of right-handed individuals may result from lesions of the <u>right</u> hemisphere throwing out of function the left hemisphere as the result of cerebral shock, or diaschisis; but as a rule, these effects of cerebral shock are transient. Thus the question of cerebral localisation has to be answered in a different way, according to the two major stages of brain injuries and brain disease, those of initial or transient, and those of residual or lasting symptoms".

bilateral language centres on a sodium amytal test; the incidence of such persons should be far higher amongst chronic epileptics than in normal individuals.

A further implication of the model is of some therapeutic and medical consequence. Earlier, in Table 8.10, I have given exact predictions for the probability of recovery from aphasia, I have not, in that table, made any allowance for the effects of treatment (merely assuming that the underlying pathology is irreversible, and ignoring potential effects due to the regression of intra-cerebral oedema, or whatever); in particular the model predicts that speech therapy will, and indeed, can, have no consequence on the overall rate of recovery from aphasia. It may affect the speed of recovery, but should not affect the overall incidence of recovery. This conclusion is compatible with several reviews of the effect of speech therppy (Sarno et al, 1970; Darley, 1975; Levita, 1978) which find it to have no effect upon the incidence of recovery. The single controlled trial of speech therapy in aphasia, of which I am aware, (Basso et al, 1975), found that speech therapy improved recovery if therapy was started within two months of a lesion, and that it had no effect six months after a lesion had occurred. This study however cannot differentiate between an increase in the speed of recovery of those who would anyway have recovered, and an actual increase in the overall incidence of recovery of speech.

Once one has opened the Pandora's box of multiple, independent random phenotypes contingent upon the same genotype, then many, many possibilities arise for theoretical explanation and speculation. As an example consider the usual description of the functions of the right hemisphere as dealing with 'holistic' or 'spatio-temporal' analysis, as opposed to the verbal, sequential or analytic analysis of the archetypal left-hemisphere. If we allow the possibility that the two speech centres and a 'visuo-spatial' centre, are each determined independently, then it is rapidly apparent that there may well be individuals with, say, SA on the left, SB on the right and also visuo-spatial analysis on the left; and so on. Naturally, the more bizarre arrangements (i.e. all phenotypes except SA, SB and hand control on the left, and visuo-spatial on the right) will be more common in left-handers. We now only have to speculate that it is functionally or operationally inconvenient to have, say, speech in opposite hemispheres, or speech and visuo-spatial analysis in the same hemisphere, to provide theoretical explanations of the data which finds an excess of left-handers in groups such as stutterers, or dyslexics, or that such individuals tend to have less asymmetry on dichotic or tachistoscopic tests. Or perhaps we may also explain simultaneously the increased incidence of left-handers and a thicker than usual corpus callosum in schizophrenics (Lishman and McMeekan, 1976; Gur, 1977; Rosenthal and Bigelow, 1972). And similarly some of the more bizarre neuro-psychological conditions

might fall within the scope of the model once we propose that say, a lateralised reading centre and a lateralised writing centre are both independent random phenotypes. For cases of aphasia and alexia without agraphia, or whatever, would now be possible, albeit rare, with a single lesion. The possibility that all possible lateral combinations of speech, handedness and visuo-spatial analysis might occur, has already been proposed by Kreindler et al (1966).

8:5 Fitting the SA-SB model of speech dominance*

The ideal data for fitting the SA-SB model would be ~~from~~ to persons of known handedness and familial handedness with definite cerebral lesions on one side only, and for whom aphasia has been tested immediately after injury, and also some while after injury, when recovery may have been expected to have taken place. Naturally no such evidence exists at present, and it is therefore necessary to examine a reduced subset of the relevant variables.

Table 8.13 lists thirteen different sets of data. Conrad (1950), Hécaen and de Ajuriaguerra (1964) and Newcombe and Ratliffe (1973-temp), examined the incidence

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*Since writing this chapter I have become aware of the paper by Satz (1979) in which he attempts to fit 'models' to data from left-handed aphasics. His methods of correction for varying incidences of aphasia are similar to mine; his predictions of the proportions of right, left and bilateral speech seem to be completely without theoretical foundation, except that they fit his not too exacting data. He assumes also that bilateral speech never occurs in right-handers, but gives no explanation of this statement.

of acute aphasia after cerebral lesions, Hecaen and Ajuriaguerra in patients with strokes, and the other two studies in patients with war wounds. Newcombe and Ratliff (1973-perm) looked at the long-term follow-up of aphasia in their head injury patients. Naumann (1955) examined patients with either gliomata or meningiomata. Bingley (1958) examined patients with temporal lobe gliomata, coding as aphasic those who developed aphasia either before operation or after operation. Penfield and Roberts (1954) looked at acute aphasia after surgery in chronic epileptics. Dennis and Whittaker (1977) reviewed a large amount of data from studies examining long-term aphasia in children with severe cerebral injury or congenital spasticity, as a function of the side of the lesion; they looked separately at 19th and 20th Century data. Hecaen and Piercy (1956) examined the incidence of paroxysmal ictal aphasia in epileptic patients with unilateral foci, as a function of the side of the focus,

Figure 8.4 summarises the data of Table 8.13. The abscissa is the overall incidence of aphasia in the study. It should be clear from this figure that the studies can be broadly grouped into three categories:-

i. The acute apahsias. The studies of Conrad, Hécaen and de Ajuriaguerra, and of Newcombe and Ratcliff (temp).

ii. The chronic aphasias. The studies of Bingley, Penfield

and Roberts, and Naumann, seem to cluster above group i for left-sided lesions, andbelow group i for right-sided lesions.

iii. The studies of Dennis and Whittaker, and Hecaen and Piercy are clearly distinct from either group i or ii. These studies are probably not suitable for the present form of analysis since there is little certainty that the lesions are unilateral, in both cases it being probable that bilateral damage has occurred (and hence the greater similarity of 'right and 'left' sided lesions), The reason for the inclusion of these studies will become apparent later.

Before fitting the model to these data-sets it is worthwhile to remember quite what is being fitted. The model for handedness inheritance has two paramters, $p(L_t)$ and $p(L|DC)$. Both of these may be varied over a wide range as in the figures of Chapter 7. Once these parameters are fixed then the proportions of the phenotypes of SA and SB are also fixed. In referring to a model as 'additive' one is describing the value of $p(L|DC)$, and nothing else. 'The additive model' refers to a model mentioned earlier in Chapter 8.1, in which $p(L_t)$ is also defined as 0,095 and which is one of the acceptable fits for the handedness data. In fitting the data for aphasia there is a further free parameter since, although the proportions of SA and SB are already determined, there are two possibilities as to the outcome of a lesion in an individual with SA and SB in different hemispheres. He may become aphasic, which I have referred to as the 'acute' or 'temporary' model,

or he may not be aphasic, the 'permanent' model. For a particular data-set it is not always clear which variant of the model should be fitted, and in the following discussion I have always fitted both variants of the model to all data-sets. This is necessary since (a) if for all data-sets then both variants fit, then the differentiation between the forms is clearly trivial, and (b) for some studies, particularly those involving chronic-epileptics, it is not at all clear which is the appropriate form of the model (see the earlier discussion).

Figure 8.5 shows the goodness of fit of various forms of my own model to those data sets whivh might be expected to have a temporary aphasia (i.e. group i above). In figure 8.5 the goodness of fit is shown in each case for both the 'temporary' (or 'acute') model, and the 'permanent' model. It is clear that the additive model will cope with all of the data if the aphasia is acute, but none of the data if the model is permanent.

Figures 8.6 and 8.7 show similar goodness of fit functions for the data of group ii. These data sets would be expected to have aphasias which are permanent in type. For Bingley (1958), Penfield and Roberts (1959), and Naumann (1955), there is an adequate fit for the permanent model, whilst only for the meningiomata data is there also a fit for the acute model. The long-term data of Newcombe and Ratliff are more problematic; they do not fit the

permanent model, but do fit the acute model. This point will be returned to later.

In fitting these data sets several assumptions have had to be made. Firstly the genetic model predicts the various proportions of the speech dominance phenotypes and then the expected incidences of acute and permanent aphasia after right- and left-handed lesions. However corrections must be made for the incidence of right-sided lesions not equalling the incidence of left-sided lesions (see Table 8.13 for the actual proportions). More controversial is the correction for the overall incidence of aphasia. Let the overall incidence of aphasia in the study be A_m. There is also an expected incidence of aphasia from the genetic model (i.e. equivalent to tables 8.9 and 8.11) which we may call A_t. A_m may not equal A_t for many reasons. Consider a study of aphasia after head injury; if one examined only patients who had had to have neurosurgical intervention then the incidence of aphasia would be high (and indeed may even be overly high, for aphasia may itself have been a criterion for operation). Alternatively if one had studied all patients with head injuries, however minor, but who had had, say, loss of consciousness and been brought into a casualty department, then the overall incidence of aphasia would be far lower. In the latter example we may postulate a simple dilution effect, whereby if an any sub-group the expected proportion of aphasics was p, and A_m/A_t equalled 0.001, then the corrected value for the sub-

group would be 0.001p. The severity of lesion is not the only factor which would affect A_m; the location of a cerebral lesion will also affect A_m. Thus if a large proportion of lesions is occipital, or cerebellar, we would expect a lower incidence of aphasia than if all lesions were fronto-temporal. A further complication for the model fitting is that, as with handedness and dichotic studies, different criteria may be adopted for aphasia. Some studies may have, perhaps inadvertently, included more expressive then receptive aphasics (due to the greater ease of detection); or studies might have had a very sensitive threshold for dysphasia so that the slightest hesitation, stammer, or mis-pronunciation might count as aphasia for the purposes of the study. All such factors will mean that A_m will vary between studies. I have used the single correction described above to account for all such effects, partily because of its simplicity, and partly due to the lack of any adequate, more detailed information on which to base more complex corrections. The single correction cannot hope to correct perfectly for all of the deviations of A_m from A_t. Consider too strict a criterion of aphasia; it should be true that all cases reported as aphasia really will be aphasia. If the criterion is too slack then an increasing proportion of non-aphasics will be included in the aphasic group. But these non-aphasics will have, on average, 50% of right-sided lesions, and thus the ratio of right to left-sided lesions will be distorted. No such distortion will

take place for too strict a criterion (this might, in principle, provide an empirical approach to the question of what is 'really' the correct definition of an aphasic).

It might be objected that I have used so many corrections in fitting the models to the data, that it would be possible to fit almost any data set which I cared to try. There are two arguemnts against this.

i. In Figures 8.5, 8.6 and 8.7 there are clearly models whcih do <u>not</u> fit, and hence we may assume that we have at least one remaining degree of freedom in our goodness of fit testing.

ii In Figure 8.4 I have included several data sets, those of Dennis and Whittaker, and Hecaen and Piercy, which were never intended to test the present model (since they clearly do not exclude possible bilateral lesions), and thus these models should <u>not</u> fit the present models; Figures 8.8 and 8.9 show that this is generally the case (the single exception being the rather small group of paroxysmal ictal receptive aphasics from the Hecaen and Piercy study).

Table 8.13 gives details of the actual and expected values for the data of Figures 8.5 and 8.9 as well as Chi-squared values for the additive version of the model. In fitting these data sets I have, as with the dichotic data sets, used two degrees of freedom. This is perhaps a liberal

estimate of the value, but I will use the same argument in its justification as I have earlier used in the appendix to the handedness paper.

Thus far the model fitting has taken no note of the handedness of individuals. There are rather fewer data sets with adequate information on handedness. Table 8.14 summarises six such data sets. In fitting such data there is no difference to the process described earlier, except that corrections have also to be made for the manifest incidence of left-handedness not equalling the true incidence, and for this a similar correction has been used as for the handedness models, and for the dichotic-type data shown earlier in Figure 8.2. Figure 8.10 shows the goodness of fit of the data which might be expected to be 'acute' in type. Conrad (1949) and Newcombe and Ratliff (1973) fit the acute model well, and strongly reject the permanent model. The data of Hécaen and de Ajuriaguerra fail to fit either model. This was expected, but is superficially surprising in view of the adequate fit of the acute model in Figure 8.5. The reason, as Annett (1975) has convincingly shown, is that the Hecaen and de Ajuriaguerra study did not classify handedness and speech dominance independently, but rather, due to a pre-existing theoretical bias, right-handers with aphasia and right-sided lesions were classified as 'left-handers'; in consequence no model could hope to fit such data. In contrast the earlier data of Figure 8.5, in which handed-

ness effects have been removed, are not subject to such pre-conceptions, and hence the model can fit the data adequately. If in the Hecaen and de Ajuriaguerra data one concatenates the results of right- and left-handers for right-sided lesions (but not for left-sided lesion , then the overall fit of the acute additive model is adequate (Chi-squared = 1.99), thus confirming Annett's interpretation of that data set.

Figure 8.12 shows the fit of the three data sets which would be expected to be 'permanent' in type. The Penfield and Roberts study clearly accepts the permanent model but rejects the acute model. The Bingley model discriminates little, but there is a marginally better fit for the permanent model. The Newcombe and Ratliff permanent data, whcih did not fit at all well in the earlier analysis (Figure 8.6) now fits the acute model, and almost fits the permanent model, although the fit is still far from adequate. At this point I can only plead, in mitigation, that several less than perfect corrections have had to be applied, and that the Chi-squared value is coming almost entirely from the two rather small sets of aphasics with right sided lesions (Table 8.14). In view of the adequate fitting of the other data sets it would seem churlish to reject the present, rather powerful, model because of a single disparate data set. A statistical significance test alone should not be the sole criterion of a useful

model*.

In making the calculations of Figures 8.10 and 8.11 I have used a liberal estimate of four degrees of freedom; I will use the same argument as given in Appendix 7.1 to justify this position.

Thus far little mention has been made in this Chapter of the Trankell model. The Trankell model also makes predictions for an SA:SB model, as long as one assumes that in the LL genotype, in which left-handers occur with a probability of 0.3, that SA and SB are also randomly and independently determined. Table 15 shows the detailed predictions of the three models described earlier, for the proportions of the various phenotypes. Whilst the models are in principle discriminable, in practice this will be fairly difficult. Figures 8.12 to 8.19 show goodness of fit testing of the Trankell model for the same data as that already tested for my own model in Figures 8.2 and 8.5-8.11. In general all of the conclusions made for the fit of my own model are also applicable to the fit of the Trankell model.

8:6 Speech dominance, spatial ability, and handedness

As stated earlier one may propose that not only speech

*Since writing this I have heard that Newcombe, at a meeting of the International Neuropsychological Society in Dubrovnik, in June 1979, reported that she wished to revise the data for her "permanent" aphasics. In particular she suggested that instead of there being 9 right handed right-lesioned aphasics, there was actually either one or none. These differences were put down to criterion problems. I do not know how this affects the other items of data in the set, and I have therefore not

dominance but also dominance for visuo-spatial ability could be an independent random asymmetry in the DC and CC genotypes. In such a case of course the DD genotype would be expected to produce all right-sided dominance for visuo-spatial ability. Levy (1976) has described data by McGlone and Davidson (1973) which can be used to test the predictions of this hypothesis. They tested a set of right- and left-handers for both dichotic speech dominance and tachistoscopic dominance. Table 8.16 shows their results, as taken from Table 1 of Levy (1976): I have omitted the 'equal' groups, as these were merely estimated; such an omission produces only minor changes in the calculations. We can fit this data to the predicted results of Table 15 (simply substituting visuo-spatial dominance for SB, and reversing left and right in that case). In the McGlone and Davidson data there is no indication of the overall incidence of left-handedness, and I have therefore assumed it to be 9.5%. Corrections must be made for the dichotic test, since taking a weighted average of the two handednesses shows a 30.6% manifest incidence of right-speech dominance. For visuo-spatial ability there was a weighted estimate of 13.3% manifest left dominance. I would predict that the true values of each should be 9.5% and I have corrected appropriately. Table 8.16 shows both the raw data and the predicted values; the Chi-squared goodness of fit statistic is 5.14, the vast majority of which is contributed by the single cell in which the observed value is 5 and the expected value is 10.9. Overall the fit of the model seems

to be adequate.

8:7 Discussion

In this chapter I have presented a model of the structure and genetics of cerebral speech dominance. The model has clearly defined predictions, and also has some important consequences, if true, for the therapy of aphasia. However, in stressing that the model is purely formal I have also over-simplified one or two points. In particular, I have presented the simplest possible version of the SA:SB model. Variants of the model are possible if one scrutinises the assumptions carefully; with the collection of further and more precise data, it is possible that these variants may prove superior to the conceptually simple version I have given so far. Taking these assumptions one at a time:-

i. It is assumed that if SA and SB are both in the same hemisphere, and that that hemisphere suffers a lesion then <u>either</u> both SA and SB will be damaged, resulting in aphasia, <u>or</u> that neither SA nor SB will be damaged, resulting in no aphasia. This assumes that SA and SB are topographically identical, which is perhaps an unreasonable assumption. If it is possible to damage SA and not SB, or vice-versa, then the predictions of the model are different, depending upon the degree of overlap (or correlation) of the two 'areas' (speaking semi-metaphorically).

ii. It is assumed that all lesions are neither progressive, nor reversible; that is, their effects are immediate and without change. Of course diseases or injuries do not work this way; in particular, any acute lesion causes cerebral oedema which is frequently reversible, often resulting in a decrease in the actual functional lesion size. Some lesions are progressive; neoplasms would be the obvious example, but cerebral thrombosis can produce oedema, which may produce further thrombosis in already compromised arteries. Account could perhaps be taken of these factors in a model, e.g. by assessing, say, reversal of hemiplegia as an index of decreased lesion extent.

iii. It is assumed that the lateral expressions of H, SA and SB in the DC and CC genotypes are statistically independent. A set of models could be constructed in which the events were not completely independent, but instead show some, but not perfect, correlation with one another; these correlations need not even be identical, so that that between SA and SB may be greater than that between H and SA.

iv. It is assumed, for all three genotypes, that the expression is identical for H, SA and SB. Thus if in the DC genotype there is a probability of left-handedness of 0.25, there is also assumed to be a probability of 0.25 of right SA, and 0.25 of right SB. Of course there is no necessary reason why these probabilities should be

identical, and it is thus possible that IC might produce 0.25 left-handers, 0.35 right SA, and 0.45 right SB.

To summarise, the model presented here is conceptually the simplest of a whole range of models; it copes with most of the relevant data moderately well, although the data are far from exacting in their test of the model, there being no adequate evidence which a geneticist would accept. Should the particular model be found to be lacking, there are many variants upon it which should be tested, before rejecting the <u>form</u> of the model, since the model is powerful on its predictions.

Table 8.1

Cerebral speech dominance as assessed by dichotic listening tests.

Study	right-handers			left-handers		
	Left	Right	N	Left	Right	N
Satz et al (1967)	60	9	69	37	15	52
Curry (1967)	19	5	24	16	7	23
Zurif & Bryden (1969)	18	2	20	12	7	19
Dee (1971)	40	9	49	38	32	70
McGlone & Davidson (1973)	31	4	35	26	18	44
Blumstein et al (1975)	27	11	38	-	-	0
Lake & Bryden (1976)	52	20	72	44	28	72
Davis & Wada (1967)	12	2	14	4	4	8
Lishman & McMeekan (1977)	19	3	22	13	7	20
Springer & Searleman (1978)	24	6	30	12	7	20
Total	302	71	373	203	125	328
Percent right or equal	19.03%.			38.10%.		

Table 8.2

Cerebral speech dominance as assessed by unilateral electro-convulsive therapy (ECT).

	right-handers			left-handers		
Study	Left	Right	N	Left	Right	N
Fleminger et al (1970)	26	6	32	-	-	0
Pratt & Warrington (1972)	51	1	52	-	-	0
Warrington & Pratt (1973)	-	-	0	26	9	35
Annett et al (1974)	17	2	19	2	2	4
Geffen et al (1978)	22	2	24	6	3	9
Total	116	11	127	34	14	48
Percent right or equal		8.66%			29.16%	

Table 8.3

Cerebral speech dominance assessed by intra-carotid sodium amytal.

	right-handers			left-handers		
Study	Left	Right	N	Left	Right	N
Milner et al (1964)	43	5	48	28	16	44
Milner (1975)	87	8	95	64	53	117
Percent right or equal		9.09%			42.85%	

Table 8.4

Hand-writing position as a function of handedness

	right-handers			left-handers		
Study	Normal	Inv-erted	N	Normal	Inv-erted	N
Studies Ib and II	731	117	848	62	40	102
Lawson (1978)	112	8	120	88	32	120
Total	843	125	968	150	72	222
Percent inverted		12.91%			32.43%	

Table 8.5 Shows the actual and expected proportions of right and left-handers with speech in the right or left hemisphere, for different types of data.

	right-handers			left-handers			
Speech dominance	L	R	Chi2	L	R	Chi2	Chi2-total
Dichotic studies	302 (300.9)	71 (72.05)	0.02	203 (211.8)	125 (116.2)	1.04	1.06
ECT studies	116 (116.1)	11 (10.84)	0.03	34 (33.45)	14 (14.5)	0.0	0.03
Amytal data	43 (42.75)	5 (5.24)	0.50	28 (30.16)	16 (13.83)	0.01	0.51
Hand position data	968 (975.8)	125 (117.1)	0.59	222 (201.8)	72 (92.1)	6.41	7.00

Table 8.6

Shows the expected percentages of individuals who are right speech dominant on a dichotic listening test, by handedness and parental handedness. As elsewhere these predictions are based on a model in which $p(L_t) = 0.095$, $p(L|DC) = 0.25$ and handedness and dominance are assessed correctly.

Handedness	NK	Right	Left
Parental handedness			
R x R	7.37	5.67	26.68
R x L	18.56	15.74	30.90
L x L	29.75	25.46	39.87
NK	9.50	7.37	29.75

Table 8.7

Shows the expected proportions of monozygotic twins who will be right speech dominant on a 'dichotic' type listening test, by twin pair type, and by parental handedness. Assumptions as in Table 8.6

Twin pair type	R - R	R - L	L - L
Parental handedness			
R x R	4.35	27.58	31.42
R x L	13.21	29.27	34.55
L x L	21.38	37.36	43.64
NK	5.70	28.37	32.98

Table 8.8 Probability of individuals being of particular speech dominance types, and of suffering from acute or permanent aphasia, by lesion side, handedness, and parental handedness. AA = Acute aphasia PA = Permanent aphasia LL = both speech centres on left LR = RL = one speech centre on each side; RR = both speech centres on the right RSL = Right-sided lesion LSL = Left-sided lesions.

Parental type	p(L)	p(LR) =p(RL)	p(RR)	p(AA|RSL)	p(PA|RSL)	p(AA|LSL)	p(PA|LSL)
		Handedness not known					
RxR	.873668	.052588	.021155	.126332	.021155	.978845	.873668
RxL	.686131	.128247	.057375	.313869	.057375	.942625	.686131
LxL	.523632	.178873	.118628	.476368	.118628	.881372	.523632
NK	.838262	.066739	.028262	.161737	.028263	.971737	.838262
		right-handers					
Parental type							
RxR	.902111	.041114	.015662	.097889	.015662	.984338	.902111
RxL	.731147	.111374	.046104	.268853	.046104	.953896	.731147
LxL	.585909	.159470	.095152	.414091	.095152	.904848	.585909
NK	.873442	.052814	.020929	.126558	.020929	.979071	.873442
		left-handers					
Parental type							
RxR	.516411	.196718	.090154	.483589	.090154	.909846	.516411
RxL	.488632	.202274	.106821	.511368	.106821	.893179	.488632
LxL	.376563	.224688	.174063	.623438	.174063	.825938	.376563
NK	.503125	.199375	.098125	.496875	.098125	.908175	.503125

Table 8.9 Proportion of individuals who will have a permanent aphasia, given that they have had a lesion, by handedness and by parental handedness.

Handedness	NK			Right			Left		
Lesion side	R	NK	L	R	NK	L	R	NK	L
Parental handedness									
RxR	2.11	44.73	87.36	1.56	45.88	90.21	9.01	30.32	51.64
RxL	5.73	37.17	68.61	4.61	38.86	73.11	10.68	29.77	48.86
LxL	11.86	32.11	52.36	9.51	34.05	58.59	17.40	27.52	37.65
NK	2.82	43.32	83.82	2.09	44.71	87.34	9.81	30.06	50.31

Table 8.10 Shows the probability of an acute aphasia given that a lesion has occurred, by the side of the lesion, the handedness of the patient, and the handedness of the patient's parents. The underlined percentages indicate the combined values for right and left lesions, irrespective of side, assuming an equal incidence of each.

Handedness		NK			Right			Left	
Lesion side	R	NK	L	R	NK	L	R	NK	L
Parental handedness									
RxR	12.63	55.25	97.88	9.78	54.10	98.43	48.35	69.66	90.98
RxL	31.38	62.82	94.26	26.88	61.13	95.38	51.13	70.22	89.31
LxL	47.63	67.88	88.13	41.40	65.94	90.48	62.34	72.46	82.59
NK	16.17	56.67	97.17	12.65	55.27	97.90	49.68	69.93	90.18

Table 8.11 Proportion of patients with acute aphasia who may be expected to recover their speech, by handedness and by parents' handedness.

Handedness		NK			Right			Left	
Lesion side	R	NK	L	R	NK	L	R	NK	L
Parental handedness									
RxR	83.26	47.00	10.75	84.00	46.18	8.36	81.36	62.30	43.25
RxL	81.72	54.47	27.22	82.85	53.10	23.36	79.11	62.20	45.29
LxL	75.10	57.84	40.59	77.02	56.13	35.25	72.08	63.24	54.41
NK	82.53	48.13	13.74	83.47	47.13	10.80	80.25	62.23	44.21

Table 8.12

Expected proportions of aphasics who will have right-sided lesions, by handedness, and parental handedness, and acute or permanent aphasia.

Handedness	NK		Right		Left	
Aphasia type	Acute	Perm	Acute	Perm	Acute	Perm
Parental handedness						
R x R	11.42	2.36	9.04	1.70	34.70	14.86
R x L	24.97	7.71	21.98	5.93	36.40	17.93
L x L	35.08	18.46	31.39	13.97	43.01	31.61
NK	14.26	3.26	11.44	2.33	35.52	16.31

Table 8.13

Shows the actual and the expected proportions of individuals with aphasia after right and left'sided lesions, in thirteen different studies.
LSL = Left-sided lesion. RSL = Right-sided lesion. A = Aphasia

Lesion side		right			left	
Lesion side:	Designated Aphasia type	A+	A-	Chi2	A+	A-
Conrad (1940)	Acute	18 (29.1)	249 (237.8)	4.8	185 (182.2)	172 (174.4)
Hécaen & de Ajuriaguerra(1964)	Acute	11 (16.6)	141 (135.4)	2.1	103 (102.1)	97 (97.8)
Newcombe & Ratliff (1973)	Acute	27 (40.4)	322 (308.5)	5.1	229 (226.9)	189 (191.1)
Newcombe & Ratliff (1973)	Permanent	14 (3.3)	335 (345.7)	35.0	166 (165.0)	252 (252.9)
Naumann (1955) - Gliomata	Permanent	6 (3.48)	275 (277.5)	1.9	141 (133.7)	118 (125.2)
Naumann (1955) - Meningiomata	Permanent	4 (1.31)	174 (176.6)	5.5	58 (56.6)	126 (127.3)
Penfield & Roberts (1959)	Permanent	2 (3.4)	209 (207.6)	0.6	128 (117.8)	47 (57.2)
Bingley (1958)	Permanent	4 (1.6)	105 (107.3)	3.3	70 (67.4)	35 (37.6)
Dennis & Whittaker (1977) - 19th C	Permanent	43 (23.9)	207 (226.1)	16.8	127 (152.9)	214 (188.1)
Dennis & Whittaker (1977) 20th C.	Permanent	35 (20.5)	137 (151.5)	11.6	81 (107.2)	142 (84.8)
Hécaen & Piercy (1956) -Total	Permanent	45 (20.4)	146 (170.6)	33.3	81 (111.3)	142 (111.6)
" - expressive	Permanent	13 (6.7)	32 (38.3)	7.0	48 (56.4)	33 (26.4)
" - receptive aphasia	Permanent	2 (1.8)	43 (43.2)	0.0	15 (15.7)	66 (65.3)

sia

	Chi2	Chi2 total	N	p(LSL)	p(A)	p(A|RSL)	p(A|LSL)
2 4)	0.1	4.9	624	57.2%	32.5%	6.7%	51.8%
7)	0.0	2.1	352	56.8%	32.4%	7.2%	51.5%
) l)	0.0	5.1	767	54.5%	33.4%	7.7%	54.8%
)	0.0	35.0	767	54.5%	23.5%	4.0%	39.7%
)	0.8	2.7	540	47.9%	27.22%	2.13%	54.44%
)	0.0	5.6	362	50.8%	17.1%	2.2%	31.5%
	2.7	3.3	386	45.3%	33.6%	0.9%	73.1%
	0.3	3.5	214	49.0%	34.5%	3.6%	66.6%
)	8.0	24.7	591	57.6%	28.7%	17.2%	37.2%
	8.6	20.2	414	53.8%	30.4%	23.5%	36.3%
)	16.5	49.9	414	53.8%	30.4%	23.5%	36.3%
	4.1	11.1	126	64.2%	48.4%	40.6%	59.2%
	0.0	0.1	126	64.2%	13.4%	4.4%	18.5%

RxR

Study	p(L) propositi	p(L) parents	p(L)	n(R)	n(L)	Chi^2
ICM-2 grandparents	3.22	3.31	2.15 (2.98,2.99)	455 (451.1,451.1)	10 (13.9,13.9)	(1.118,1.
Chamberlain (1928)	4.77	3.56	4.26 (4.39,4.40)	6917 (6907.5,6907.0)	308 (317.5,318.0)	(0.298,0.
ICM-1 parents	6.66	6.10	2.91 (5.74,5.76)	167 (162.1,162.1)	5 (9.9,9.9)	(2.548,2.
Mascie-Taylor (1977)	8.30	9.30	6.82 (6.49,6.81)	232 (232.9,232.0)	17 (16.1,17.0)	(0.048,0.
Annett (1978) OU	8.50	5.47	7.27 (7.45,7.48)	1656 (1652.9,1652.4)	130 (133.1,133.6)	(0.077,0.
ICM-2 parents	8.53	4.55	7.68 (7.66,7.68)	1924 (1924.3,1923.9)	160 (159.7,160.1)	(0.000,0.
Rife (1940)	8.76	5.24	7.57 (7.73,7.75)	1842 (1839.0,1838.5)	151 (154.0,154.5)	(0.064,0.
Ferronato (1974)	9.75	9.86	6.66 (7.57,8.00)	154 (152.5,151.8)	11 (12.5,13.2)	(0.192,0.
Annett (1972)	10.63	4.40	9.73 (9.67,9.69)	6206 (6209.9,6208.6)	669 (665.1,666.4)	(0.025,0.
Chaurasia & Goswami	14.07	10.40	11.96 (10.92,11.55)	1060 (1072.5,1064.9)	144 (131.5,139.1)	(1.334,0.
ICM-2 propositi	14.39	9.82	11.35 (11.17,11.81)	796 (797.9,791.9)	102 (110.3,106.1)	(0.032,0.
Hubbard (1971)	14.58	6.30	13.94 (12.49,12.55)	722 (734.2,733.7)	117 (104.8,105.3)	(1.626,1.
Ramaley (1913)	15.56	8.03	12.03 (12.66,12.77)	841 (834.9,833.9)	115 (121.1,122.1)	(0.347,0
ICM-1 propositi	15.69	9.66	13.51 (12.18,12.88)	755 (766.7,760.6)	118 (106.3,112.4)	(1.460,0
Combined LxL data I	8.73	6.36				
Combined LxL data II	14.84	8.84				

RxL

	Chi^2	p(L)	n(R)	n(L)	Chi^2
		18.75	26		
.9)	(1.118,1.128)	(6.53,6.44)	(29.9,29.9)	6 (2.1,2,1)	(7.819,8.
		11.42	411		
318.0)	(0.298,0.330	(9.67,9.52)	(419.1,419.8)	53 (44.9,44.2)	(1.639,1.
		25.0	15		
')	(2.548,2.583)	(13.30,12.97)	(18.3,17.4)	5 (2.7,2.6)	(2.376,2.
		14.58	41		
'.0)	(0.048,0.000)	(16.24,16.97)	(40.2,39.9)	7 (7.8,8.1)	(0.097,0.
		19.04	170		
33.6)	(0.077,0.102)	(17.03,16.66)	(174.2,175.0)	40 (35.8,35.0)	(0.602,0.
		18.23	148		
60.1)	(0.000,0.001)	(17.19,16.86)	(149.9,150.5)	33 (31.1,30.5)	(0.139,0.
		19.54	140		
54.5)	(0.064,0.086)	(17.58,17.21)	(143.4,144.1)	34 (30.6,29.9)	(0.461,0
		22.50	31		
.2)	(0.192,0.400)	(19.34,20.50)	(32.3,31.8)	9 (7.7,8.2)	(0.255,0
		20.97	471		
66.4)	(0.025,0.011)	(21.48,21.11)	(468.0,470.2)	125 (128.0,125.8)	(0.091,0
		27.38	122		
39.1)	(1.334,0.199)	(28.53,30.35)	(120.1,117.0)	46 (47.9,51.0)	(0.108,0
		25.75	173		
06.1)	(0.032,0.177)	(28.50,30.19)	(166.6,162.7)	60 (66.4,70.3)	(0.864,2
		19.01	90		
05.3)	(1.626,1.494)	(29.07,28.34)	(85.8,86.7)	23 (35.2,34.3)	(5.945,5
		32.33	113		
.122.1)	(0.347,0.474)	(30.70,,29.79)	(115.7,117.3)	54 (51.3,49.7)	(0.21
		25.65	142		
112.4)	(1.460,0.316)	(30.86,32.65)	(132.1,128.6)	49 (58.9,62.4)	(2.427

		LxL			
)	Chi^2	p(L)	n(R)	n(L)	Chi^2
1,2.1)	(7.819,8.050)	(10.08,12.05)	0	0	
9,44.2)	(1.639,1.956)	28.0 (14.94,17.85)	18 (21.3,20.5)	7 (3.7,4.5)	(3.357,1.755)
',2.6)	(2.376,2.562)	(20.86,24.93)	0	3	
3,8.1)	(0.097,0.194)	(25.99.26.93)	3	1	
8,35.0)	(0.602,0.863)	(26.62,31.81)	4	0	
1,30.5)	(0.139,0.241)	(26.71,31.92)	7	1	
6,29.9)	(0.461,0.665)	(27.43,32.79)	5	6	
',8.2)	(0.255,0.098)	(29.68,29.99)	0	0	
3.0,125.8)	(0.091,0.007)	(33.29,39.78)	5	1	
.9,51.0)	(0.108,0.701)	(41.19,41.32)	3	4	
.4,70.3)	(0.864,2.181)	(43.94,44.43)	6	2	
.2,34.3)	(5.945,5.193)	(45.66,54.57)	0	0	
54 (51.3,49.7)	(0.211,0.519)	(48.73,58.00)	1	7	
49 (58.9,62.4)	(2.427,4.256)	(48.52,49.19)	0	0	
		33.3 (27.34,32.67)	24 (26.2,24.2)	12 (9.8,11.8)	(0.651,0.
		43.4 (46.50,50.50)	10 (12.3,11.4)	13 (10.7,11.6)	(0.928,0.:

[illegible] shows, for each set of twin data, the observed and expected numbers of each type pected values (E) are for two different models, 'a' in which p(L)=0.095 and p(L DC)=0.25 L DC)=0.125.

		Monozygotic twins			
idy	p(L)	N(R-R) E(a,b)	N(R-L) E(a,b)	n(L-L) E(a,b)	Chi2 (a,b)
emens (1924)	16.21	26 (26.5,26.4)	10 (8.8, 9.0)	1 (1.5,1.4)	0.045,0.027
.tz (1924)	25.00	10 (10.3,10.3)	7 (6.2,6.3)	1 (1.3,1.3)	0.029,0.030
iterbach (1925)	5.33	67 (67.6,67.7)	8 (6.6,6.5)	0 (0.6,0.7)	0.067,0.083
ilberg (1926)	14.49	53 (51.6,51.3)	12 (14.7,15.2)	4 (2.6,2.3)	1.250,1.815
schuer (1927)	20.28	156 (158.6,157.9)	77 (71.6,73.1)	11 (13.6,12.9)	0.973,0.517
man (1928)	31.00	25 (24.3,24.2)	19 (20.2,20.5)	6 (5.3,5.2)	0.171,0.240
sch (1930)	20.93	25 (27.5,27.4)	18 (12.9,13.2)	0 (2.5,2.4)	4.696,4.313
.son & Jones (1932)	10.71	56 (57.1,56.8)	13 (10.8,11.3)	1 (2.1,1.8)	1.070,0.066
cks (1933)	9.52	35 (35.2,35.0)	6 (5.6,5.9)	1 (1.2,1.0)	0.006,0.000
man, Freeman and .zinger (1937)	19.00	34 (33.5,33.4)	13 (13.8,14.2)	3 (2.6,2.4)	0.127,0.246
iterwek (1938)	18.85	80 (82.2,81.8)	38 (33.6,34.3)	4 (6.2,5.8)	1.443,1.010
e (1940)	11.88	176 (177.2,176.4)	41 (38.6,40.3)	6 (7.2,6.4)	0.366,0.037
yss (1946)	18.44	72 (70.1,69.8)	24 (27.8,28.4)	7 (5.1,4.8)	1.258,1.789
fe (1950)	12.82	261 (266.7,265.5)	76 (64.5,66.9)	6 (11.8,10.5)	5.013,3.227
chaume (1957)	24.24	19 (19.4,19.3)	12 (11.2,11.4)	2 (2.4,2.3)	0.122,0.071
zzo (1960)	13.32	199 (199.2,198.2)	51 (50.7,52.6)	9 (9.2,8.2)	0.005,0.121
rter-Saltzmann et al 976)	17.11	132 (131.5,130.8)	46 (47.0,48.3)	9 (8.5,7.9)	0.054,0.276
ehlin & Nichols (1976)	14.10	380 (338.1,386.3)	123 (106.7,110.4)	11 (19.2,17.3)	6.107,3.830

of each type of twin-pair (R-R, R-L and L-L). The p(L DC)=0.25, and 'b' in which p(L)=0.08, and

Chi2 (a,b)	p(L)	Dizygotic twins N(R-R) E(a,b)	N(R-L) E(a,b)	N(L-L) E(a,b)	Chi2 (a,b)
.045,0.027	27.41	16 (16.5,16.4)	13 (11.9,12.0)	2 (2.5,2.4)	0 0.216,0.177
).029,0.030	-	-	-		
).067,0.083	12.69	96 (97.1,96.8)	28 (25.6,26.2)	2 (3.1,2.8)	0.648,0.382
1.250,1.815	7.03	111 (111.3,111.3)	16 (15.4,15.3)	1 (1.3,1.3)	0.006,0.008
0.973,0.517	14.04	136 (133.1,132.6)	34 (39.9,40.7)	8 (5.1,4.6)	2.649,3.600
0.171,0.240	17.00	35 (34.8,34.7)	13 (13.3,15.2)	2 (1.8,1.7)	0.002,0.006
4.696,4.313	6.03	51 (51.4,51.5)	7 (6.1,6.1)	0 (0.4,0.5)	0.032,0.035
1.070,0.066	11.38	97 (97.7,97.4)	24 (22.5,23.1)	2 (2.7m2.4)	0.293,0.110
0.006,0.000	10.63	76 (75.9,75.7)	12 (16.2,16.6)	2 (1.9,1.7)	0.000,0.005
0.127,0.246	11.00	39 (40.1,39.9)	11 (8.8,9.1)	0 (1.1,0.9)	0.143,0.119
1.443,1.010	17.14	23 (24.3,24.2)	12 (9.4,9.5)	0 (1.3,1.2)	0.231,0.205
0.366,0.037	15.41	104 (105.7,105.4)	39 (35.6,36.3)	3 (4.7,4.4)	0.961,0.656
1.258,1.789	16.27	60 (60.9,60.8)	24 (22.0,22.4)	2 (3.0,2.8)	0.519,0.354
) 5.013,3.227	11.61	164 (166.7,166.3)	45 (39.4,40.5)	2 (4.8,4.3)	2.448,1.751
0.122,0.071	19.69	21 (21.5,21.5)	11 (9.9,10.0)	1 (1.6,1.5)	0.037,0.028
0.005,0.121	10.89	264 (269.1,268.3)	69 (58.8,60.4)	2 (7.1,6. 3)	5.530,4.198
0.054,0.276	19.31	115 (115.9,115.6)	54 (52.2,52.8)	7 (7.9,7.6)	0.176,0.068
) 6.107,3.830	11.11	261 (266.2,265.4)	70 (59.6,61.2)	2 (7.2,6.4)	5.673,4.348

Table 8.14 Shows the actual and the expected proportions of aphasia in various groups by handedness and lesion side, in six different sets of data.

Handedness / Lesion side		Right						Left						total
		L			R			L			R			
		A+	A-	Chi2	A+	A-	Chi2	A+	A-	Chi2	A+	A-	Chi2	Chi2 total
	Designated Aphasia type													
Conrad (1949)	Acute	175 (171.6)	163 (166.3)	0.2	11 (18.2)	238 (230.7)	3.1	10 (8.9)	9 (10.1)	0.2	7 (4.6)	11 (13.4)	1.6	5.1
Hécaen & de Ajuriaguerra (1964)	Acute	81 (83.1)	82 (79.9)	0.2	0 (8.6)	130 (121.4)	9.2	22 (18.0)	5 (18.0)	1.7	11 (3.8)	11 (18.2)	16.0	27.1
Newcombe & Ratliff (1973)	Acute	218 (209.9)	170 (178.1)	0.7	19 (23.1)	297 (292.9)	0.8	11 (14.9)	9 (15.1)	2.1	8 (9.11)	25 (23.9)	0.1	3.7
Newcombe & Ratliff (1973)	Permanent	158 (168.3)	230 (219.6)	1.1	9 (3.4)	307 (312.5)	8.9	8 (7.5)	22 (22.5)	0.0	5 (1.6)	28 (31.4)	7.5	17.5
Penfield & Roberts (1959)	Permanent	115 (116.2)	42 (40.7)	0.0	1 (3.6)	195 (192.4)	1.9	13 (7.7)	5 (10.3)	6.3	1 (1.2)	14 (13.8)	0.1	8.4
Bingley (1958)	Permanent	68 (70.7)	33 (30.3)	0.3	1 (1.8)	98 (97.1)	0.4	2 (1.6)	2 (2.4)	0.2	3 (0.8)	7 (9.2)	6.6	7.5

Table 8.15 Shows the expected percentage proportions of the four speech dominance phenotypes, as a function of handedness, for the three proposed genetic models. Figures in brackets are percentages as a function of that particular handedness group, whilst figures not in brackets are overall percentages.

		SA:SB Phenotypes			
	Handedness	LL	LR	RL	RR
Genetic model					
1). Additive	Right	79.05	4.78	4.78	1.89
p(L)=0.095		(87.34)	(5.28)	(5.28)	(2.09)
p(L|DC)=0.25	Left	4.78	1.89	1.89	0.93
		(50.31)	(19.93)	(19.93)	(9.81)
2). McManus variant	Right	81.77	4.44	4.44	1.33
p(L)=0.080		(88.8)	(4.83)	(4.83)	(1.44)
p(L|DC)=0.125	Left	4.44	1.33	1.33	0.88
		(55.60)	(16.65)	(16.65)	(11.08)
3). Trankell	Right	83.57	3.67	3.67	1.57
p(L)=0.075		(90.35)	(3.97)	(3.97)	(1.70)
p(L|LL)=0.30	Left	3.67	1.57	1.57	0.67
		(49.00)	(21.00)	(21.00)	(9.00)

Table 8.16

Shows the observed and expected values of the number of individuals with right or left speech dominance, and right or left dominance for a visuo-spatial task, by handedness, (data from McGlone and Favidson, 1973, as quoted by Levy, 1976). Expected values are shown in brackets.

Speech dominance	Left	Left	Right	Right
Visuo-spatial dominance	Right	Left	Right	Left
Right-handers	19 (19.26)	2 (2.05)	8 (7.36)	1 (0.75)
Left-handers	15 (13.30)	8 (6.06)	5 (1o.90)	8 (5.66)

Figure 3.1 Shows the incidence of right speech dominance, as assessed by four different methods in right- and left-handers, as a function of the population incidence of right speech dominance (calculated by weighting the handedness groups, assuming that 9.5% of the population are truly left-handed). Points are shown ± one standard error.

Speech dominance & handedness

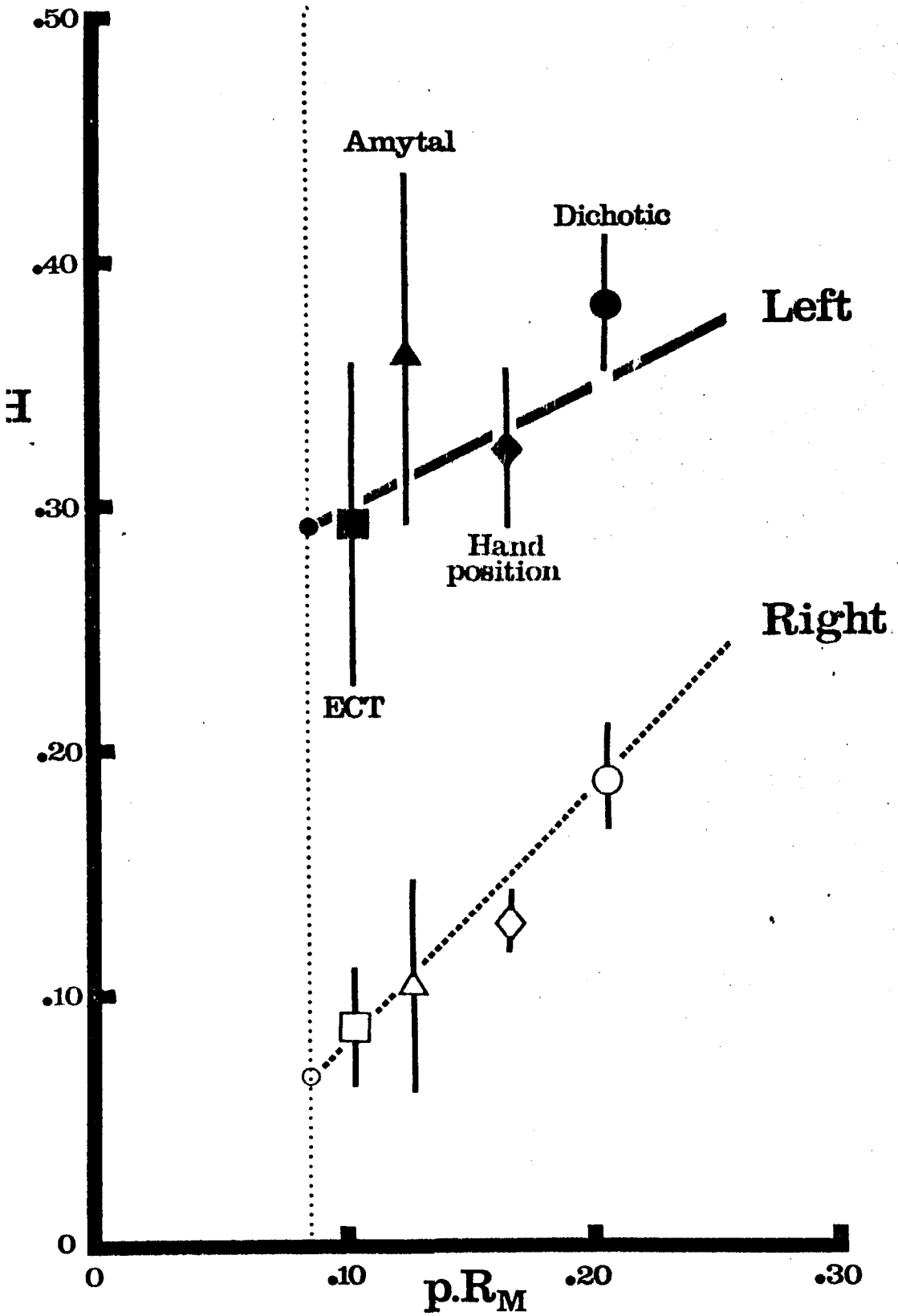
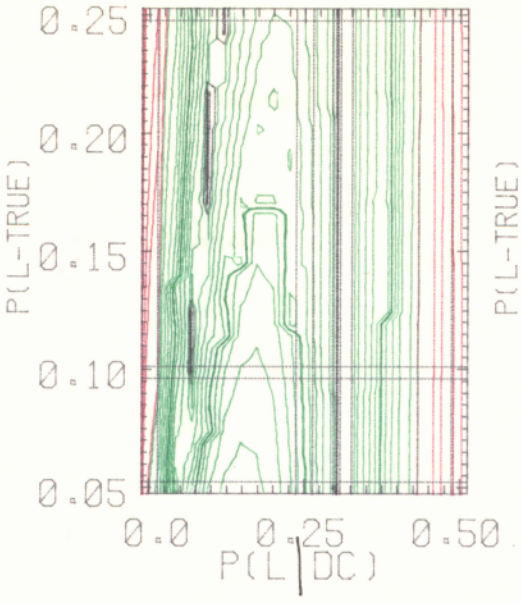
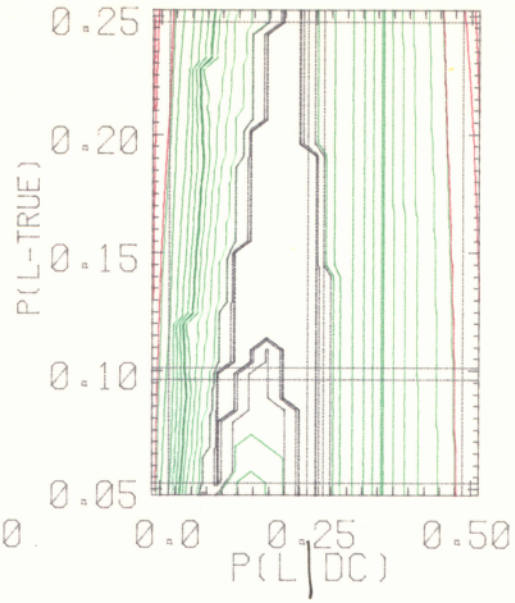


Figure 8.2 Shows the goodness of fit of my own model to the data of Figure 8.1 The contour maps are for the overall fit of the model, and are essentially similar to those of the previous paper (see caption to Figure 7.2), except that a 'liberal' number of degrees of freedom has been used throughout, and black conoour lines have been used to indicate goodness of fit probabilities of > 0.05. The abscissa shows the hypothesised proportion of left-handers produced by heterozygotes, and the ordinate the hypothesised true incidence of left-handedness. The goodness of fit has been calculated with two degrees of freedom.

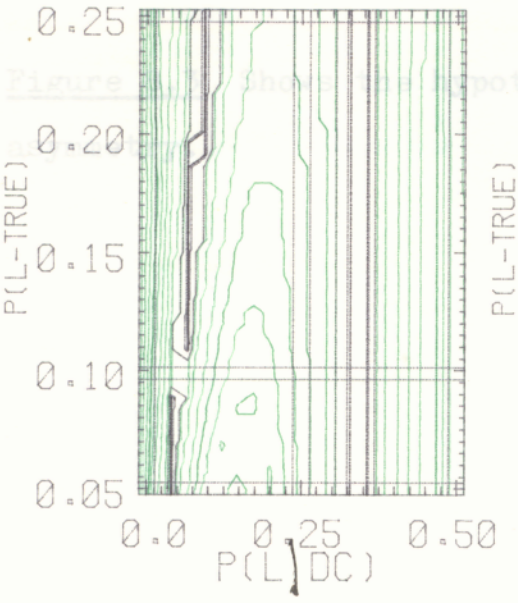
DICHOTIC LISTENING



UNILATERAL ECT



INTRA-CAROTID AMYTAL



HAND-WRITING POSITION

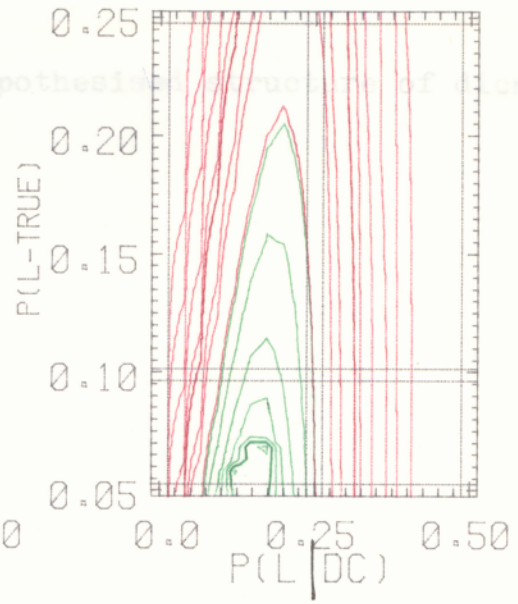


Figure 8.3 Shows the hypothesised structure of dichotic asymmetry.

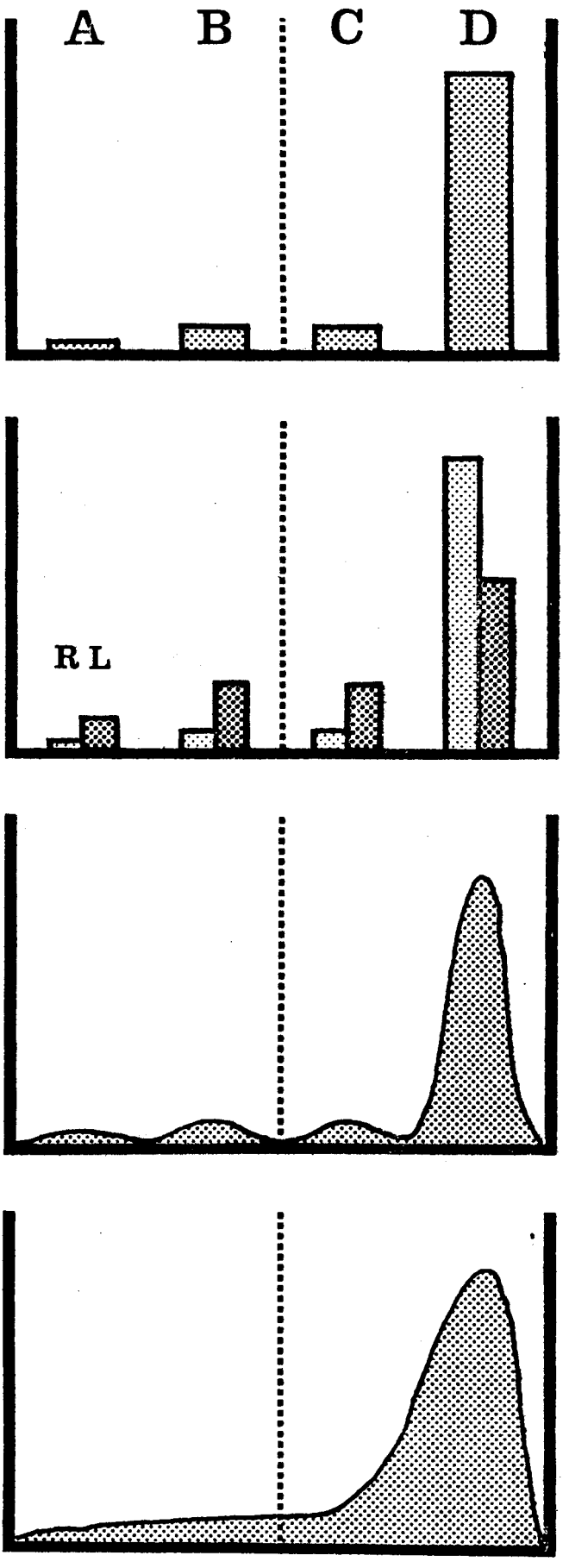

A
B
C
D
R L
LEA
REA

Figure 8.4 Shows, for a number of data-sets, the incidence of aphasia after right- and left-sided lesions, as a function of the overall incidence of aphasia in the study. Points are plotted ± one standard error.

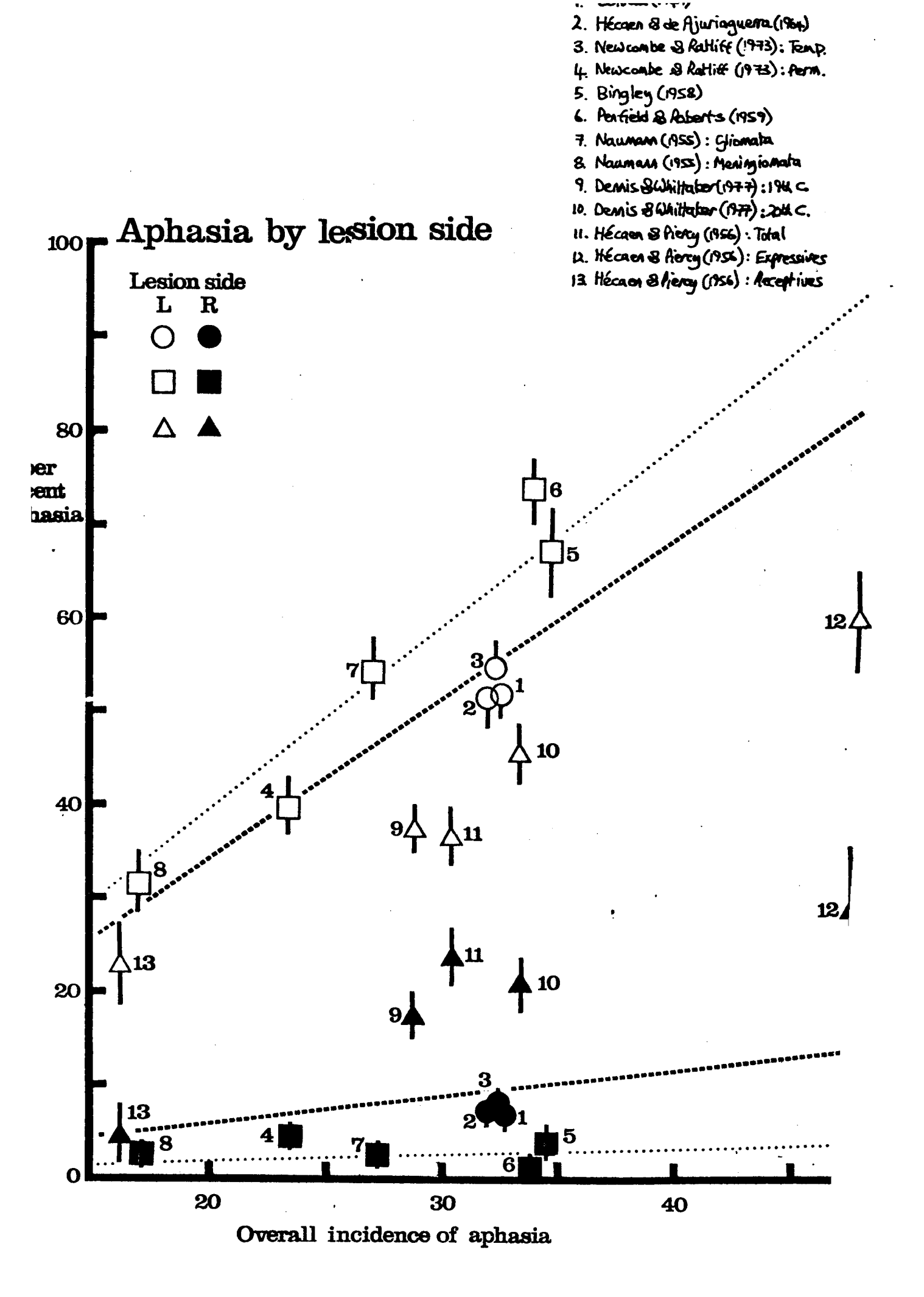
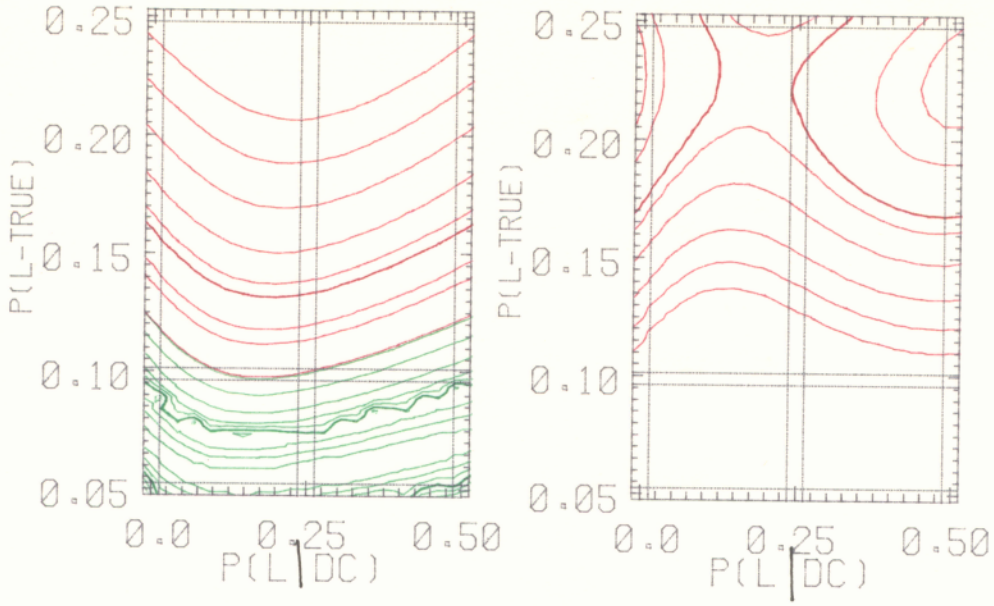
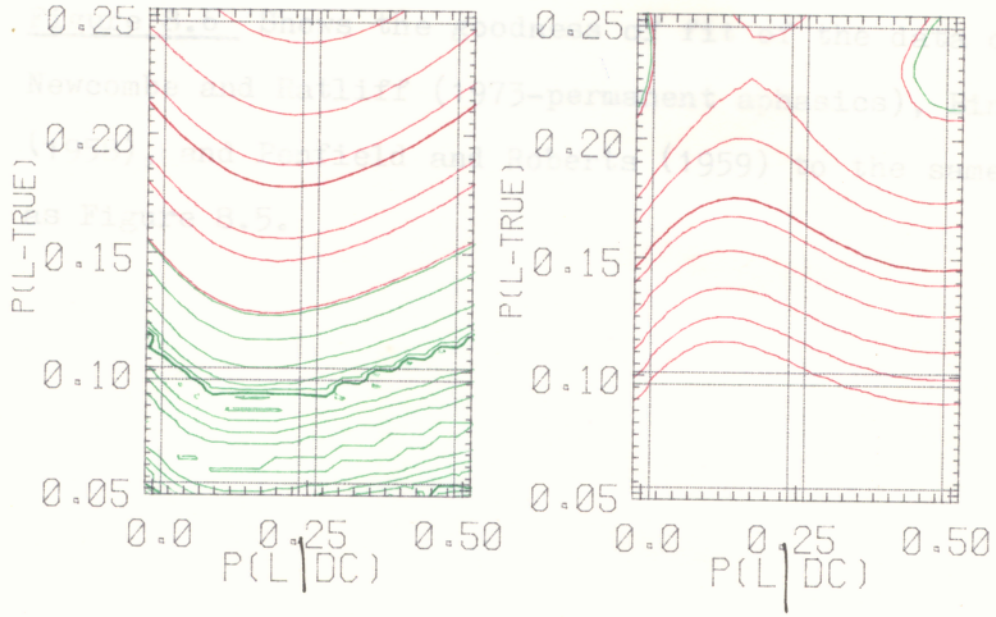

2. Hécaen & de Ajuriaguerra (1964)
3. Newcombe & Ratliff (1973): Temp.
4. Newcombe & Ratliff (1973): Perm.
5. Bingley (1958)
6. Penfield & Roberts (1959)
7. Naumann (1955): Gliomata
8. Naumann (1955): Meningiomata
9. Dennis & Whitaker (1977): 19th C.
10. Dennis & Whitaker (1977): 20th C.
11. Hécaen & Piercy (1956): Total
12. Hécaen & Piercy (1956): Expressives
13. Hécaen & Piercy (1956): Receptives
Aphasia by lesion side
Lesion side
L R
100
80
60
40
20
0
20
30
40
Overall incidence of aphasia

Figure 8.5 Shows the goodness of fit of the data from Conrad (1949), Hecaen and de Ajuriaguerra (1964), and Newcombe and Ratliff (1973 - temporary aphasia) to my own model. The contour maps on the left are for a model predicting acute aphasia, and the contour maps on the right are for a model predicting 'permanent aphasia'. Data fitted is presence or absence of aphasia as a function of right- or left-handed lesion. The goodness of fit has been calculated with two degrees of freedom, Otherwise as for Figure 8.2. In this and the following contour maps, 'temporary model' should be read as being synonymous with 'acute model'.

CONRAD(1949)



HECAEN & DE AJURIAGUERRA (1964)



NEWCOMBE & RATLIFF (1973) :TEMP

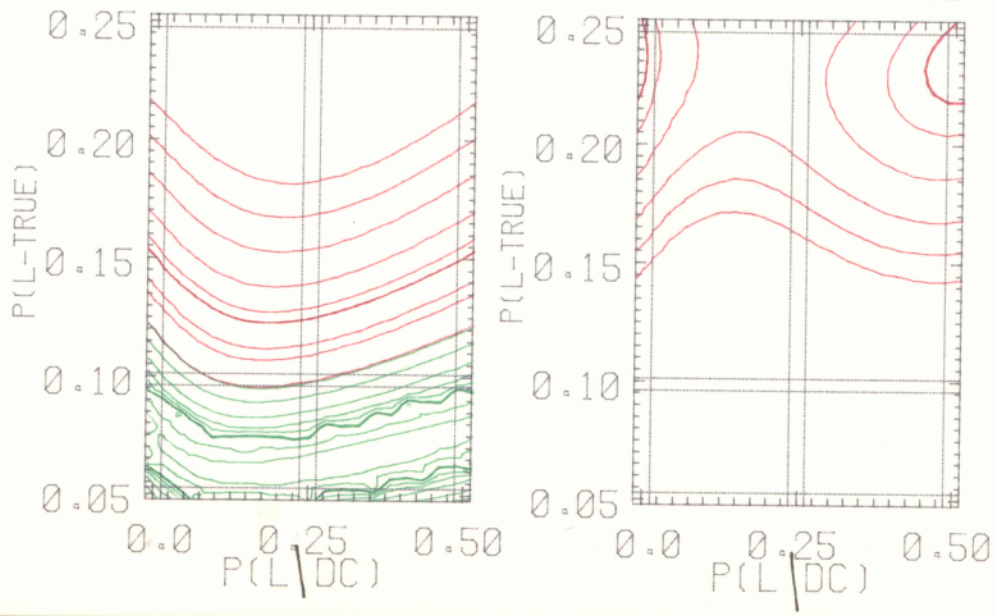
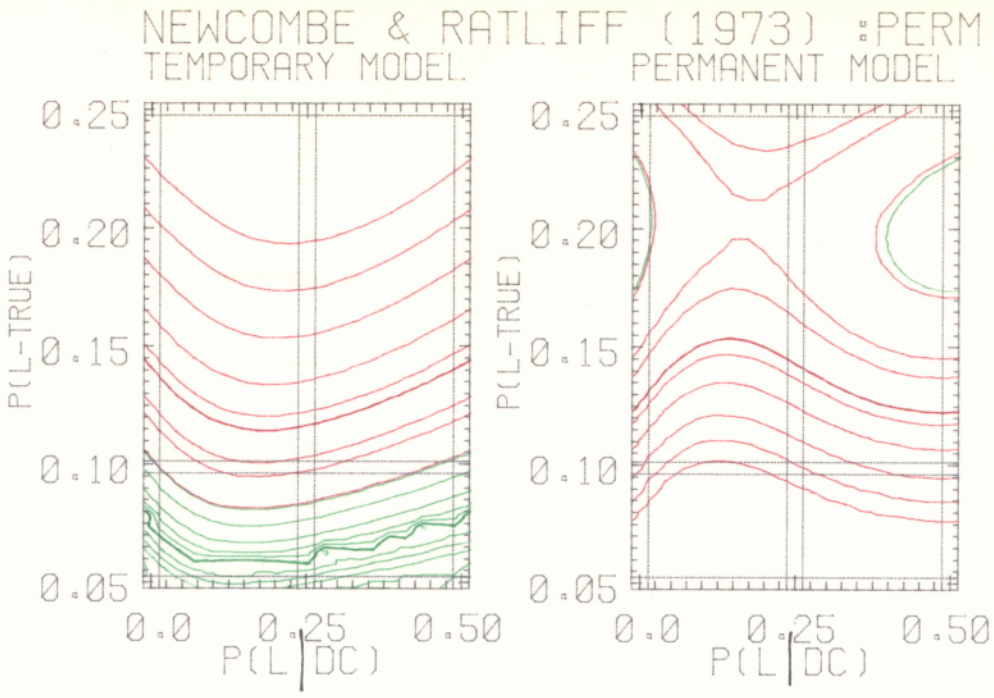
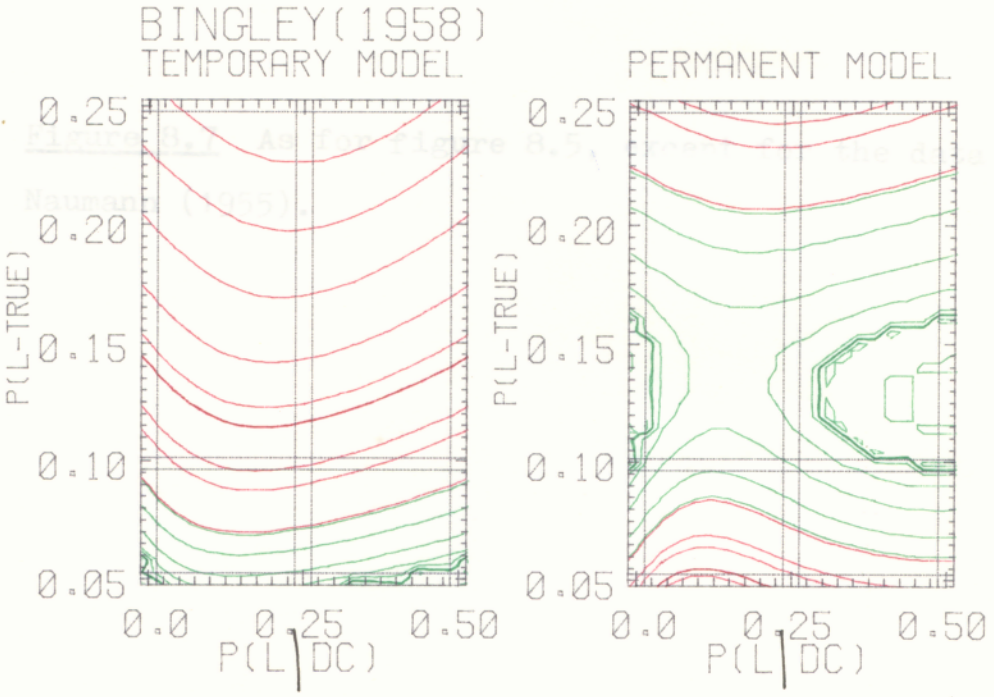


Figure 8.6 Shows the goodness of fit of the data of Newcombe and Ratliff (1973-permanent aphasics), Bingley (1958), and Penfield and Roberts (1959) to the same model as Figure 8.5.


NEWCOMBE & RATLIFF (1973) :PERM
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)


BINGLEY(1958)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

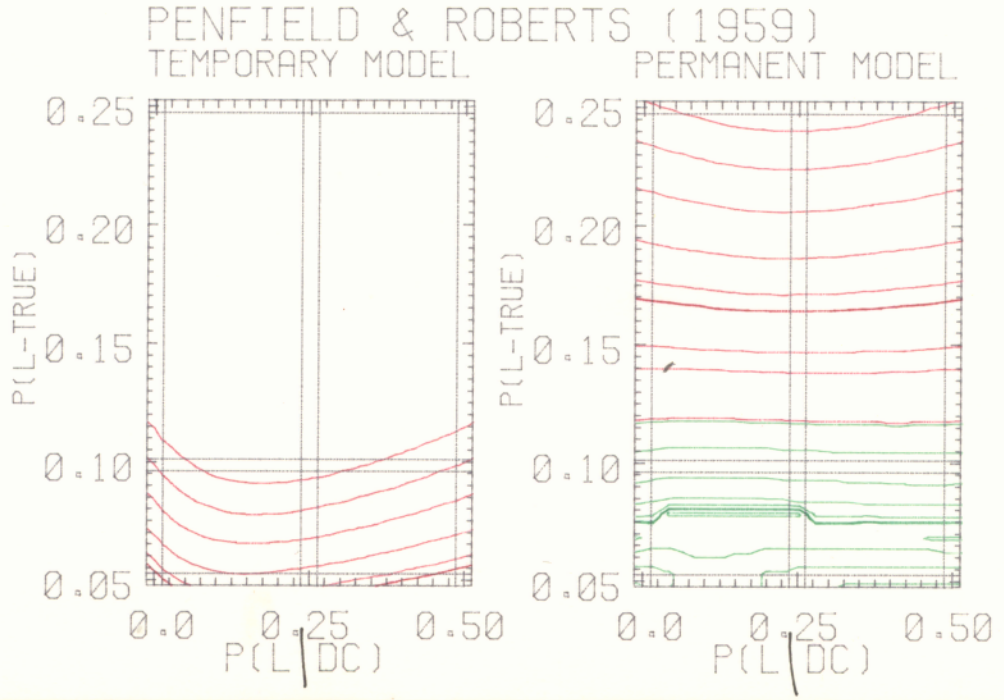
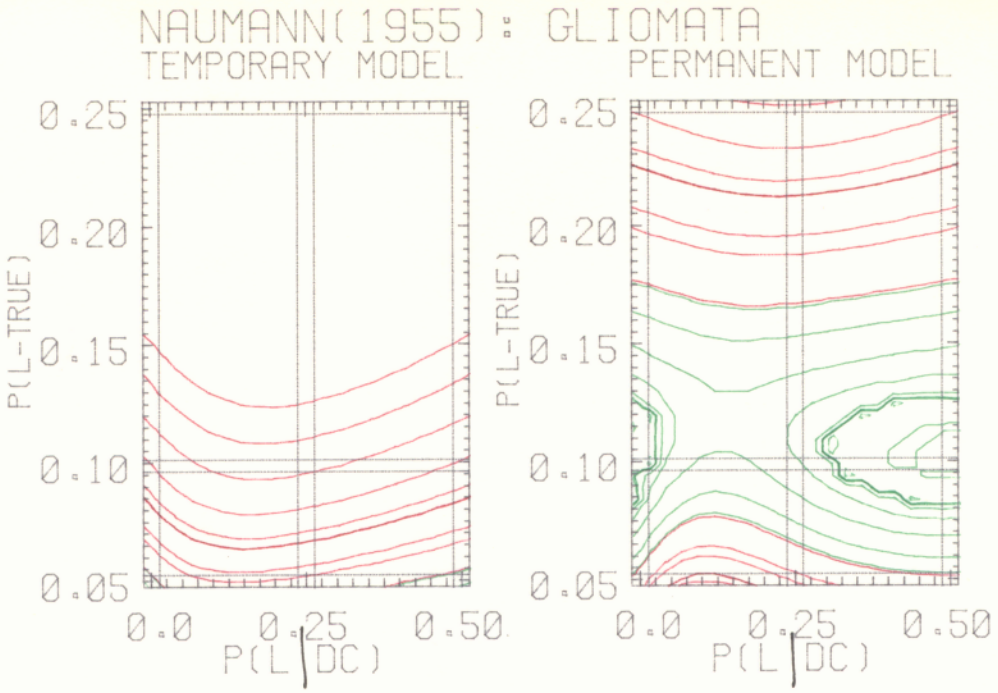

PENFIELD & ROBERTS (1959)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

Figure 8.7 As for figure 8.5, except for the data of Naumann (1955).


NAUMANN(1955): GLIOMATA
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

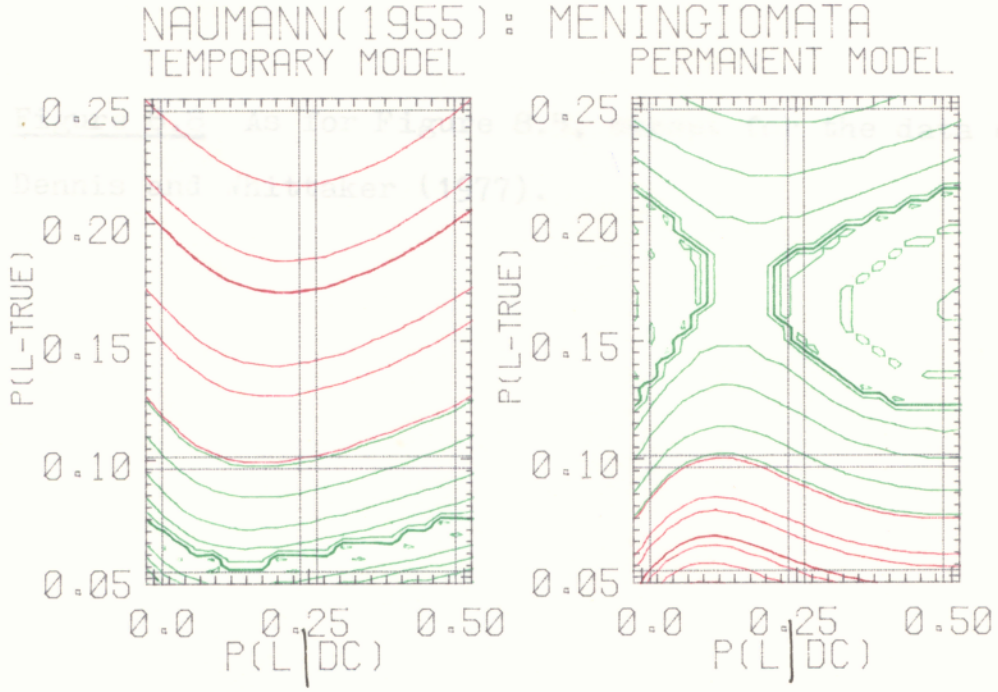
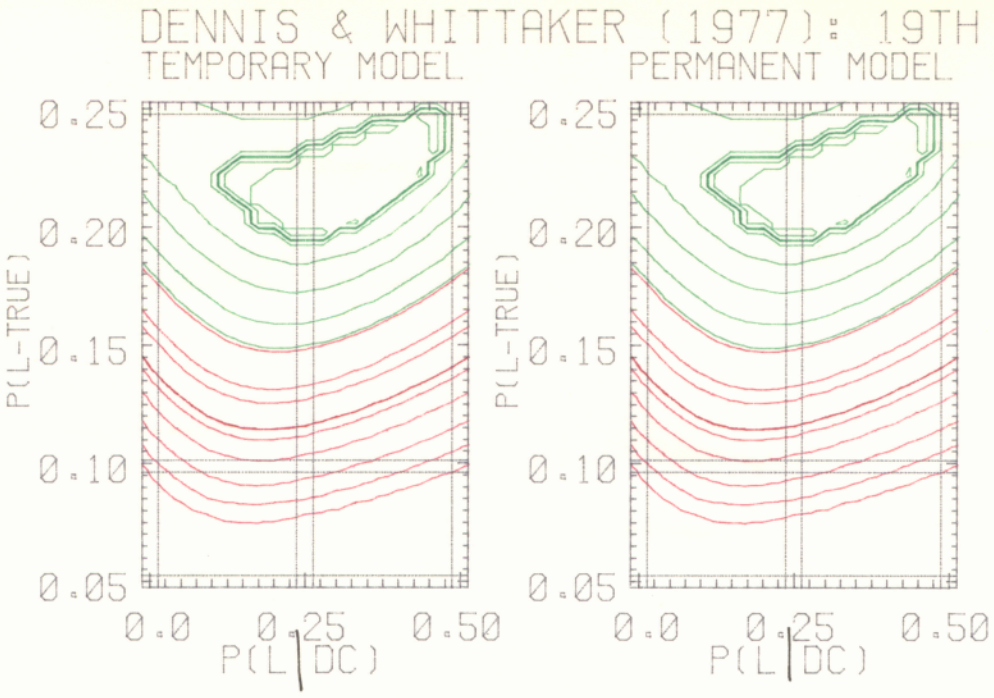

NAUMANN(1955): MENINGIOMATA
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

Figure 8.8 As for Figure 8.5, except for the data of Dennis and Whittaker (1977).


DENNIS & WHITTAKER (1977): 19TH C.
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

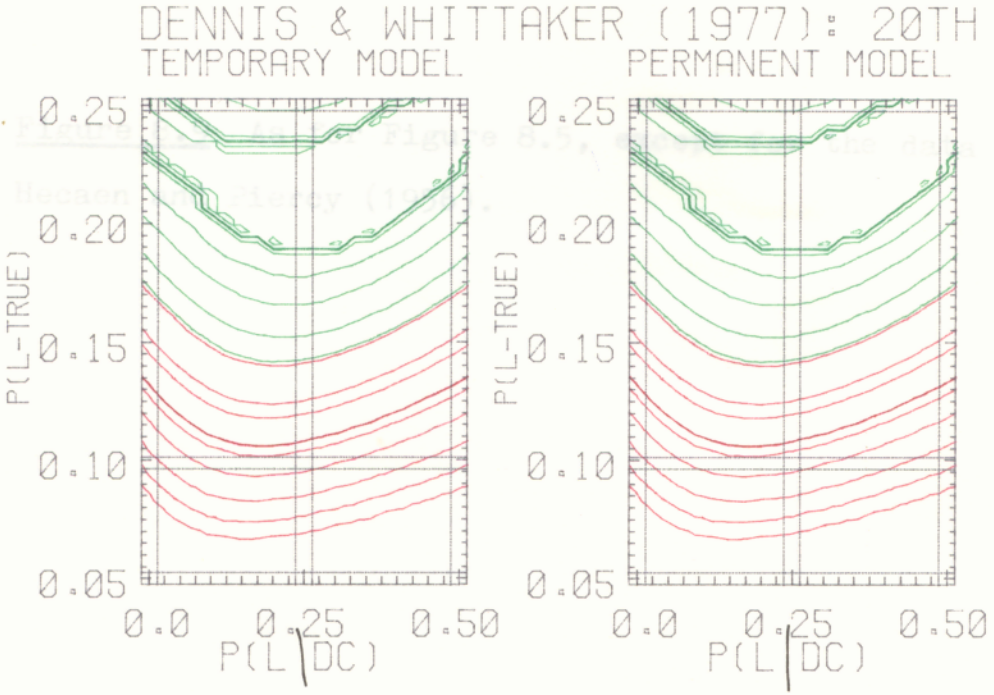
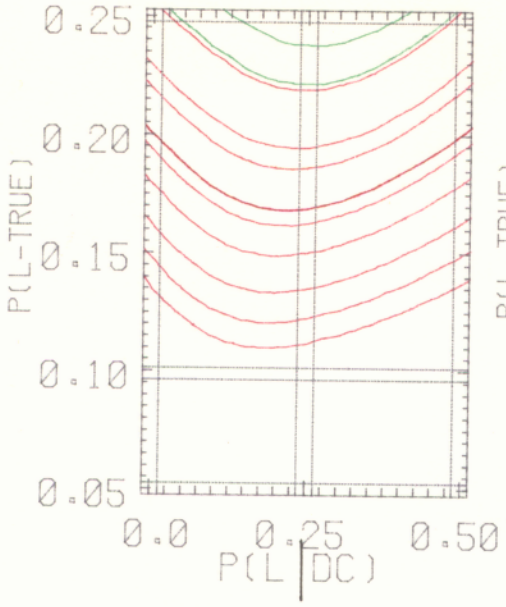

DENNIS & WHITTAKER (1977): 20TH C.
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

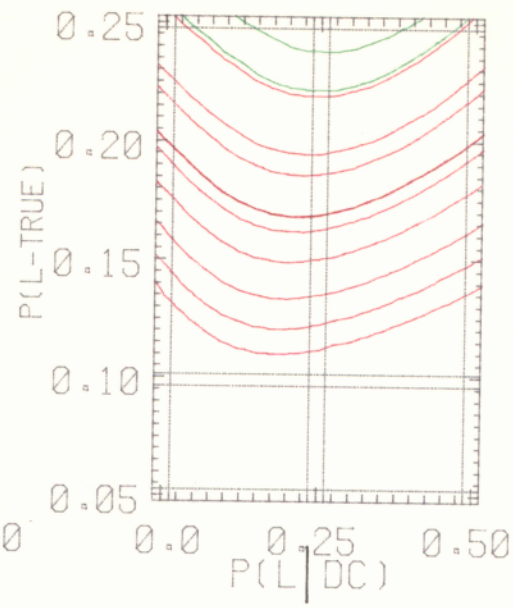
Figure 8.9 As for Figure 8.5, except for the data of Hecaen and Piercy (1956).

HECAEN & PIERCY (1956): TOTAL

TEMPORARY MODEL

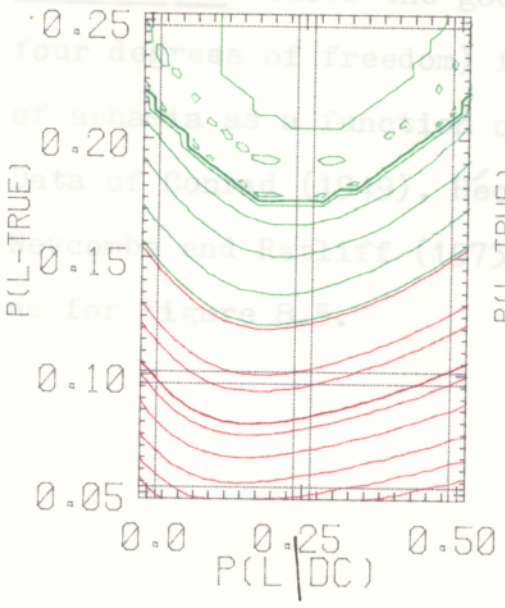


PERMANENT MODEL

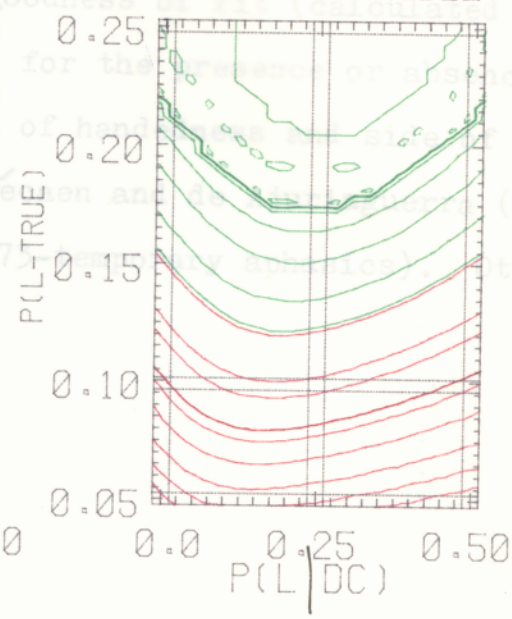


HECAEN & PIERCY (1956): EXPRESSIVES

TEMPORARY MODEL

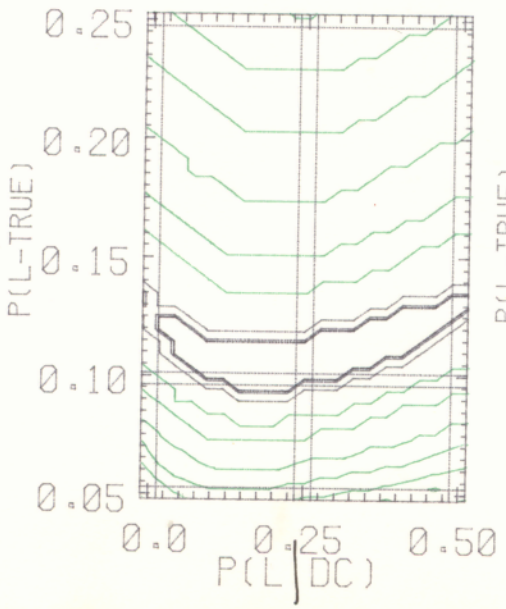


PERMANENT MODEL



HECAEN & PIERCY (1956): RECEPTIVES

TEMPORARY MODEL



PERMANENT MODEL

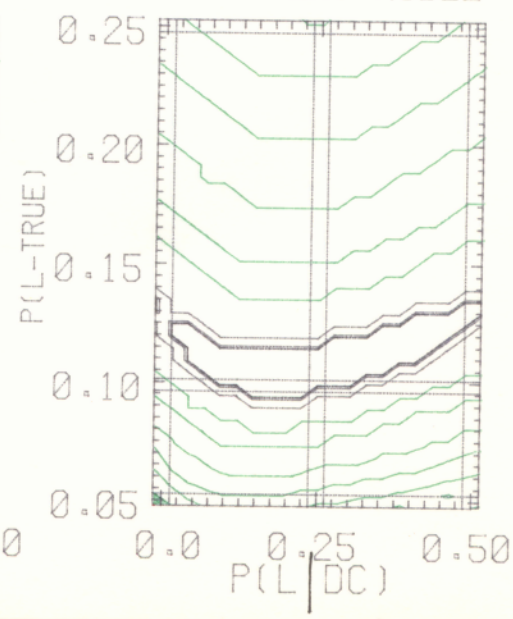
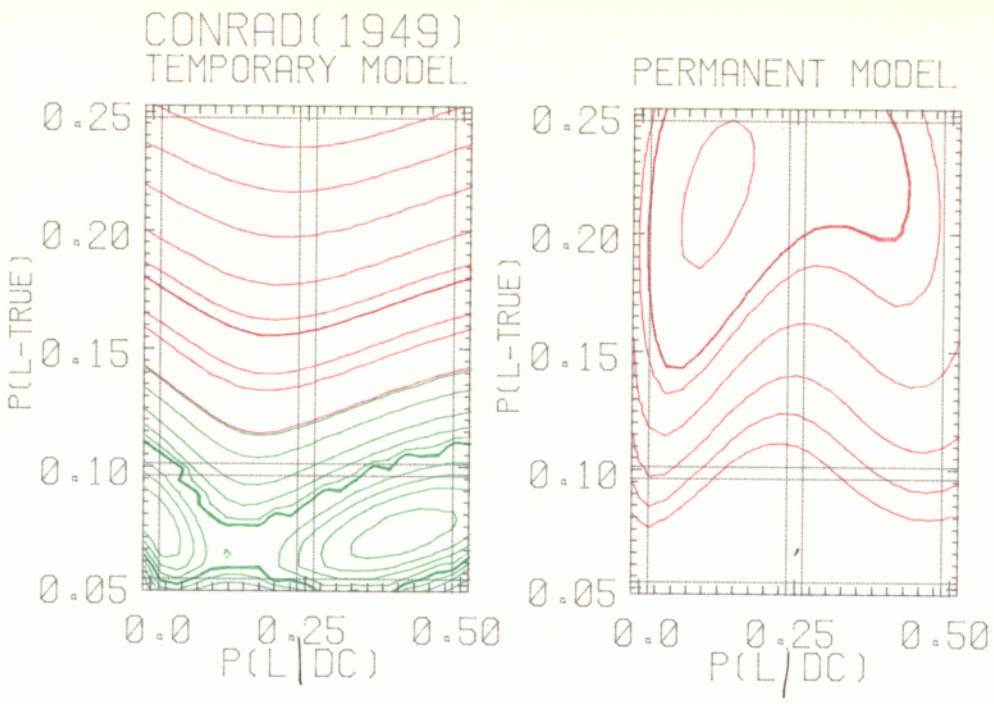
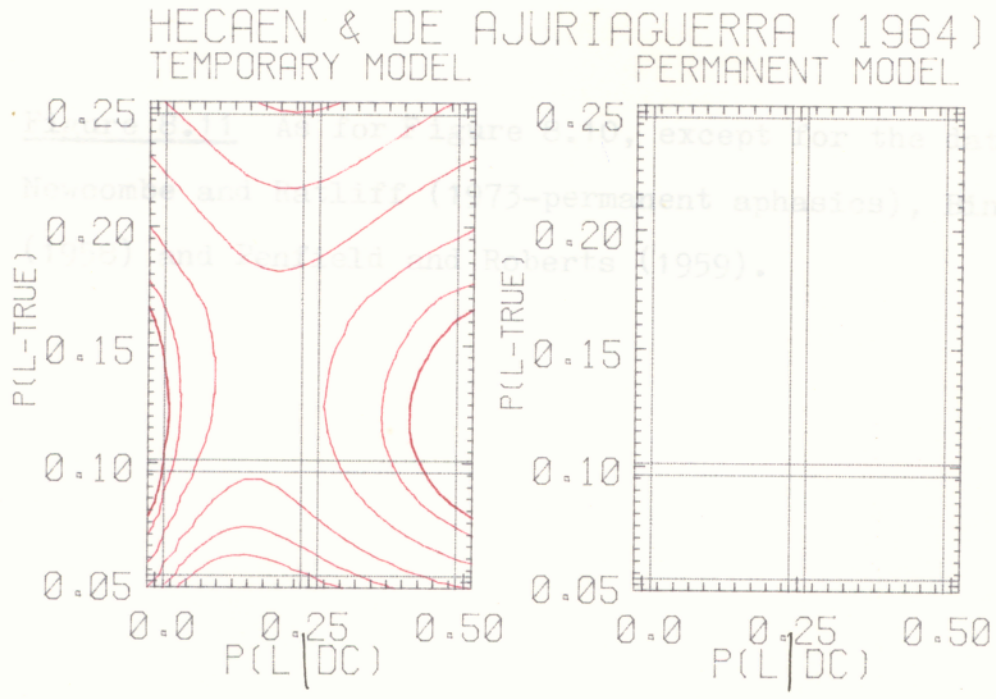
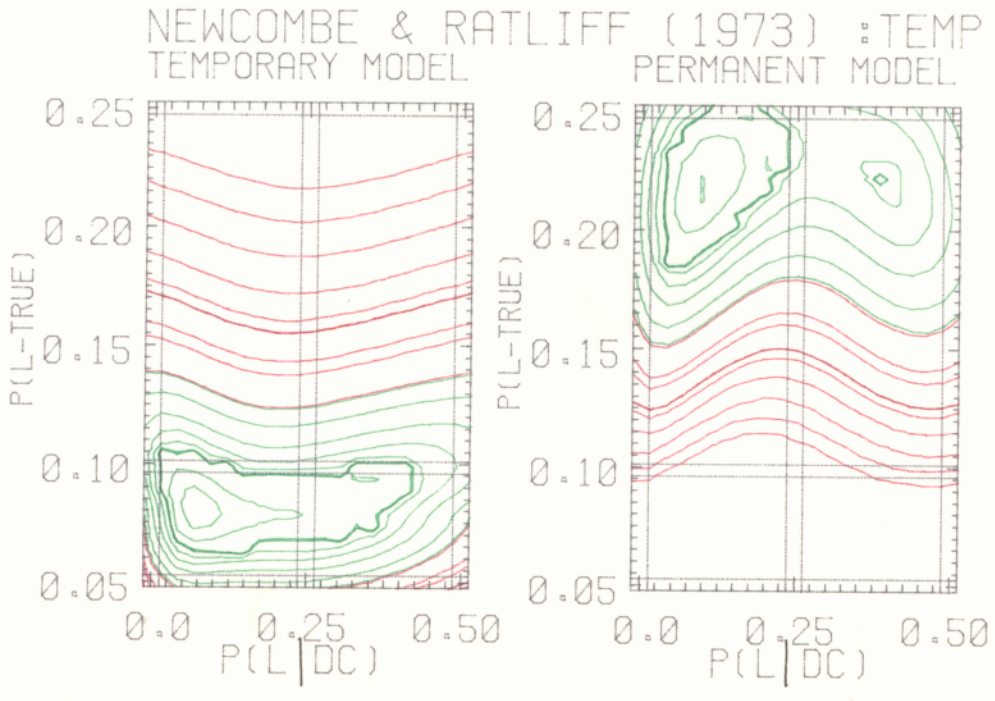


Figure 8.10 Shows the goodness of fit (calculated with four degrees of freedom) for the presence or absence of aphasia as a function of handedness and side of lesion. Data of Conrad (1949), Hécaen and de Ajuriaguerra (1964), and Newcombe and Ratliff (1973-temporary aphasics). Otherwise as for Figure 8.5.


CONRAD(1949)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

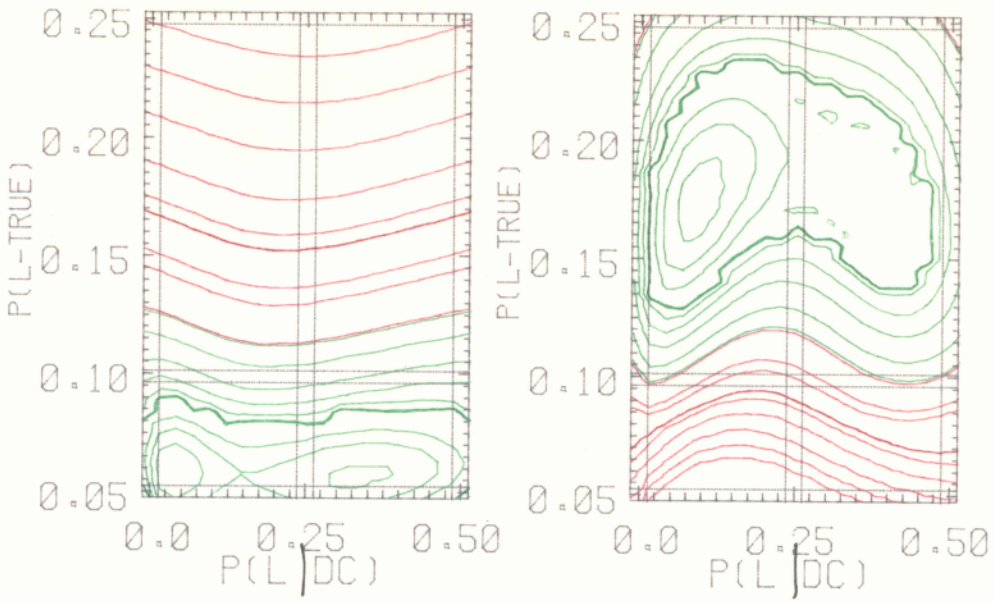

HECAEN & DE AJURIAGUERRA (1964)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)


NEWCOMBE & RATLIFF (1973) :TEMP
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

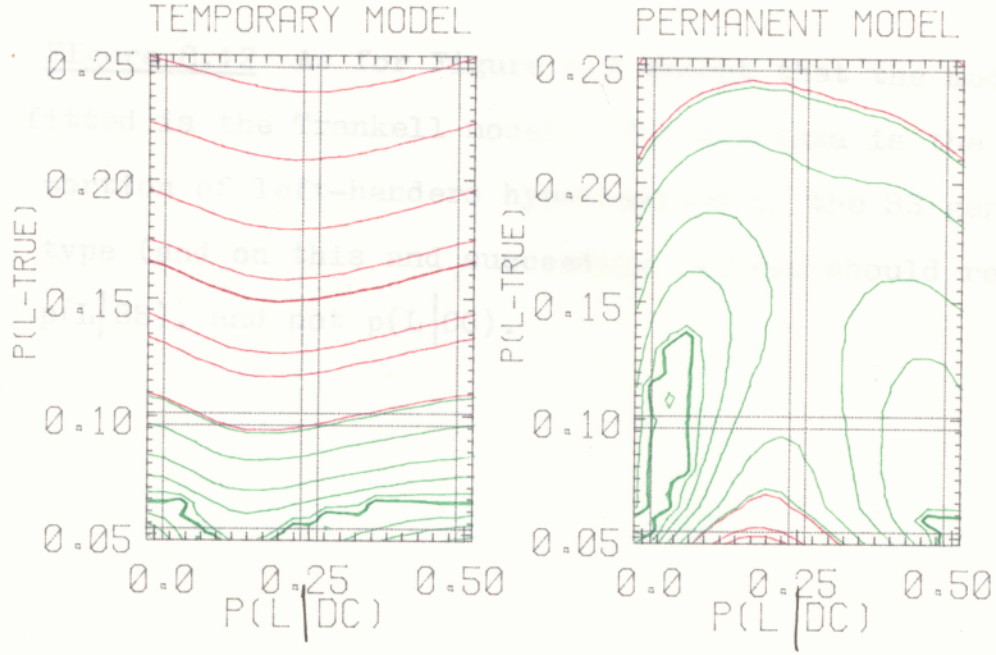
ICMAI

Figure 8.11 As for Figure 8.10, except for the data of Newcombe and Ratliff (1973-permanent aphasics), Bingley (1958) and Penfield and Roberts (1959).

NEWCOMBE & RATLIFF (1973) :PERM



BINGLEY(1958)



PENFIELD & ROBERTS (1959)

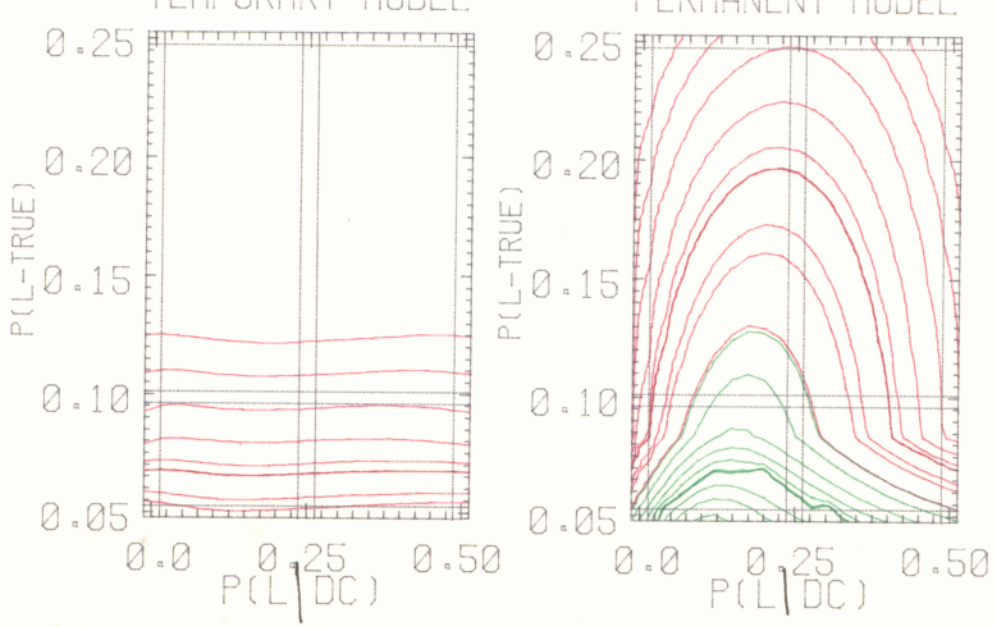
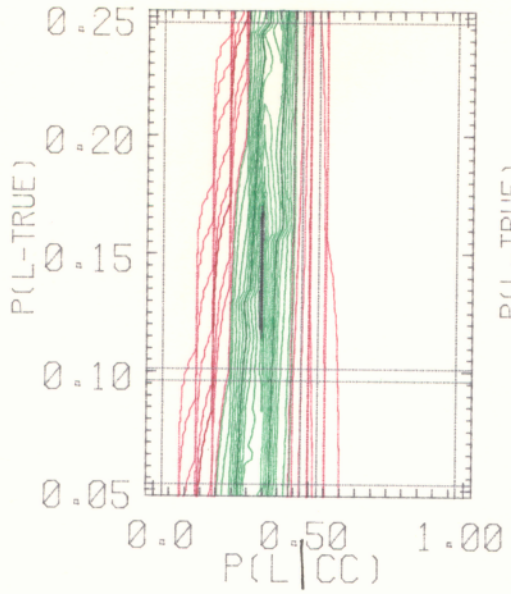
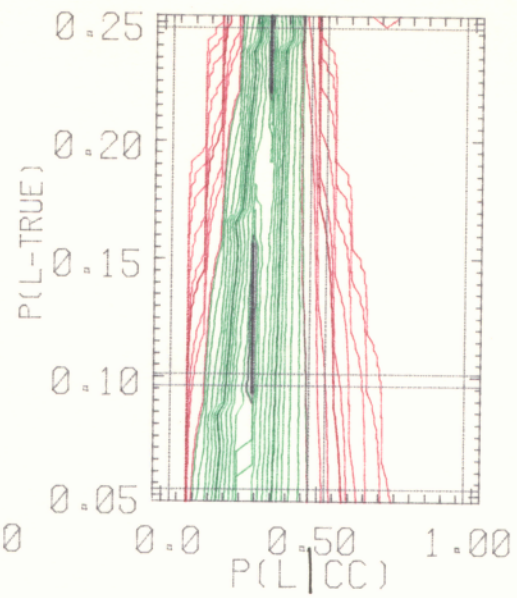


Figure 8.12 As for Figure 8.2 except that the model fitted is the Trankell model. The abscissa is the proportion of left-handers hypothesised in the SS genotype (and on this and succeeding figures should read p(L|SS), and not p(L|CC).

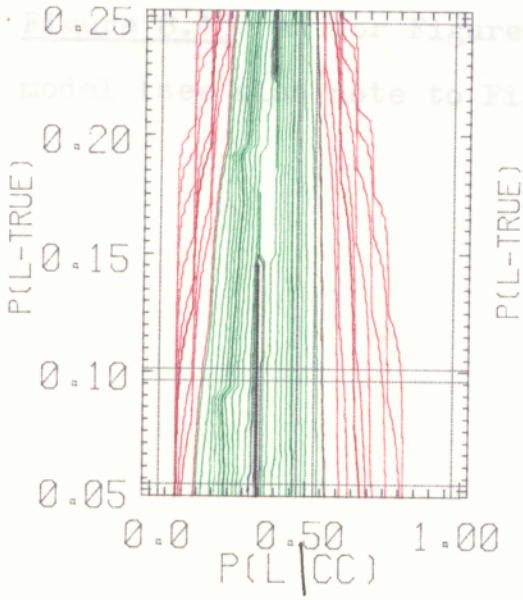
DICHOTIC LISTENING



UNILATERAL ECT



INTRA-CAROTID AMYTAL



HAND-WRITING POSITION

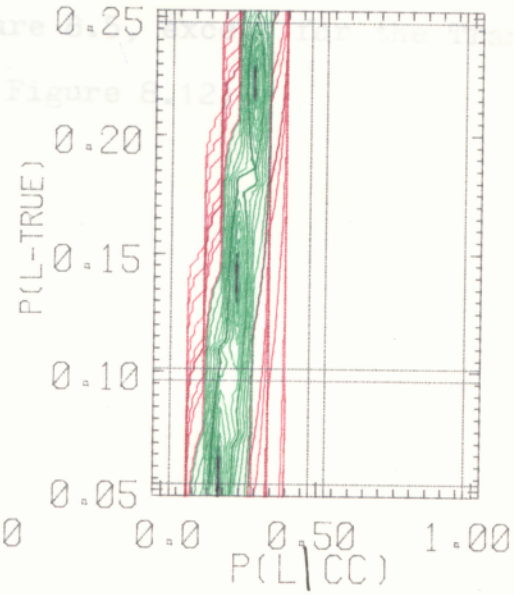
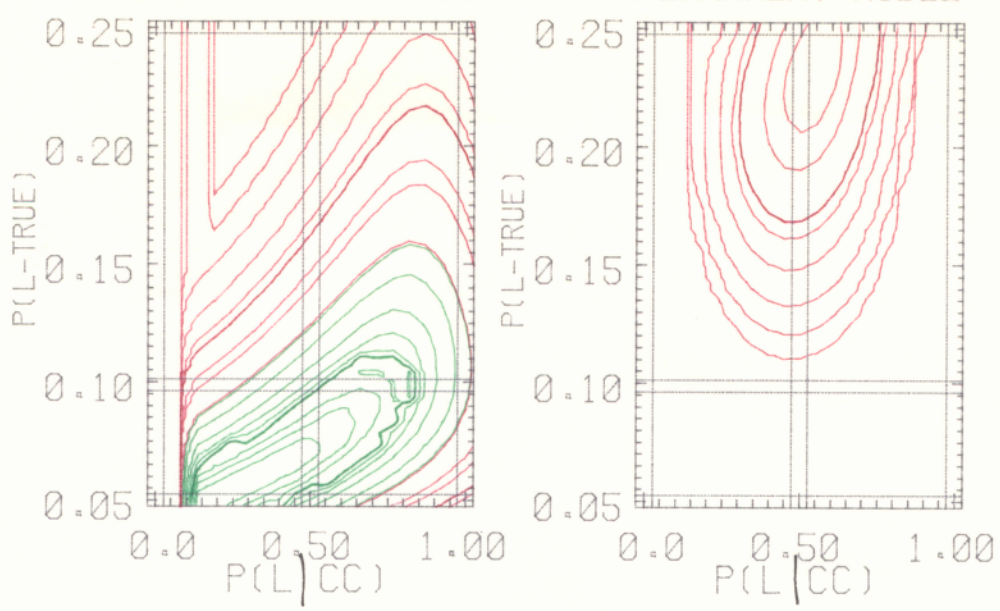
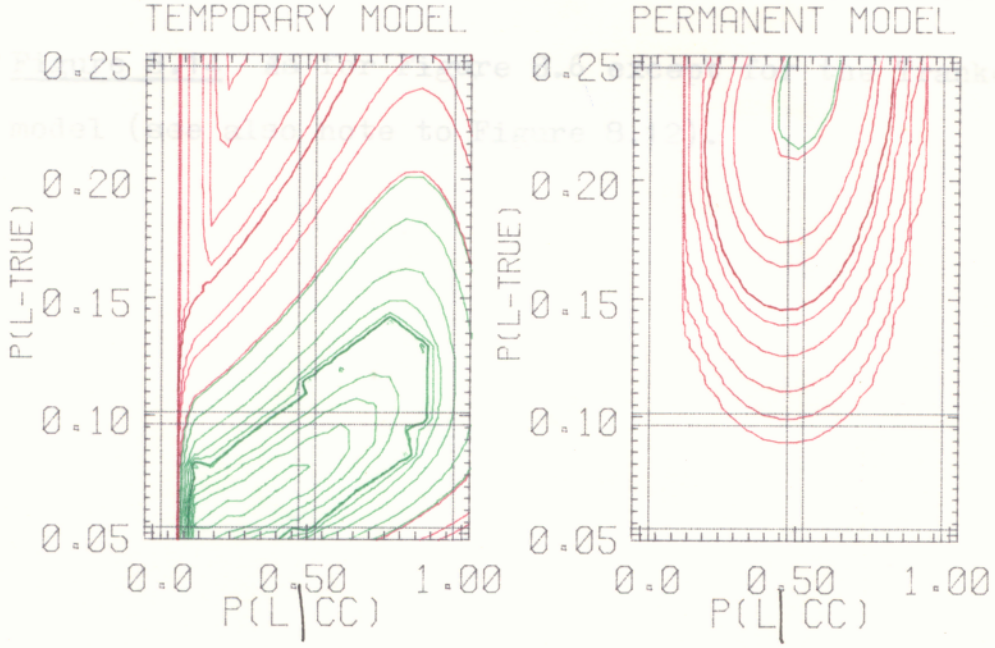


Figure 8.13 As for Figure 8.5, except for the Trankell model (see also note to Figure 8.12).

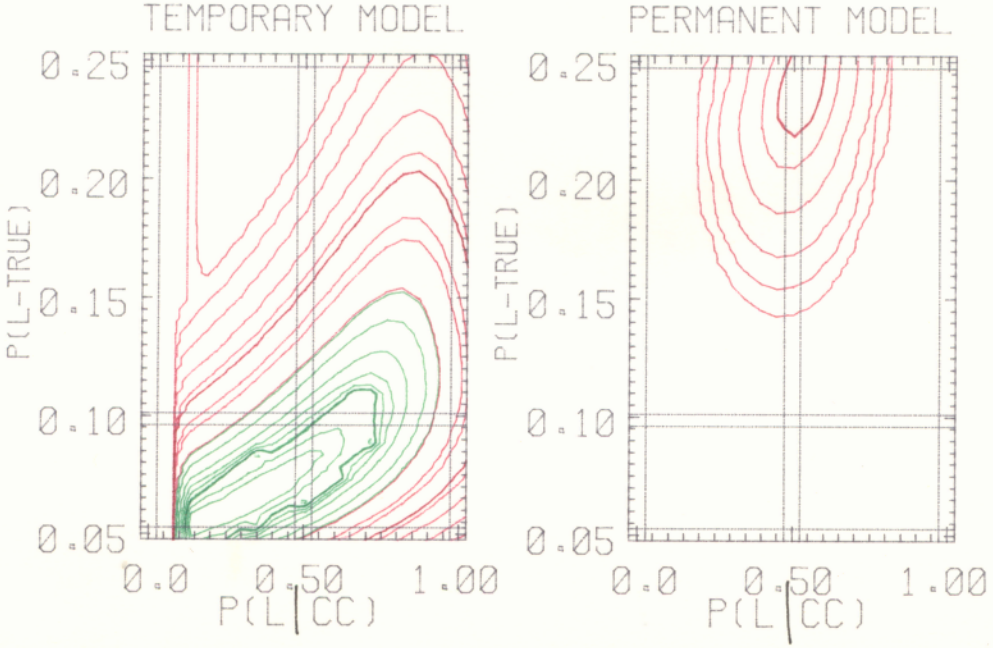
CONRAD(1949)



HECAEN & DE AJURIAGUERRA (1964)



NEWCOMBE & RATLIFF (1973) :TEMP

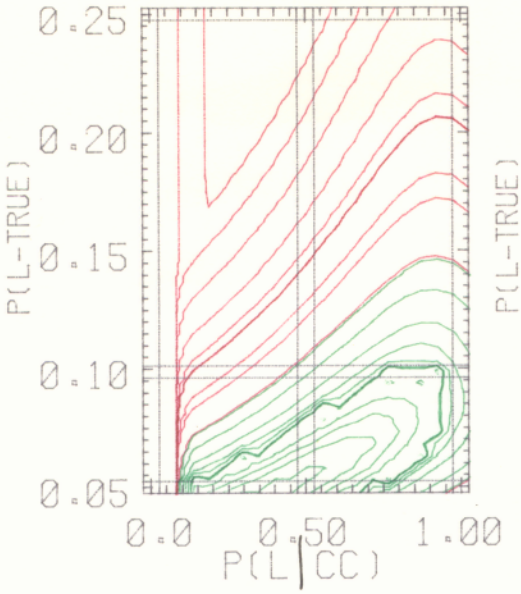


TRNK C1

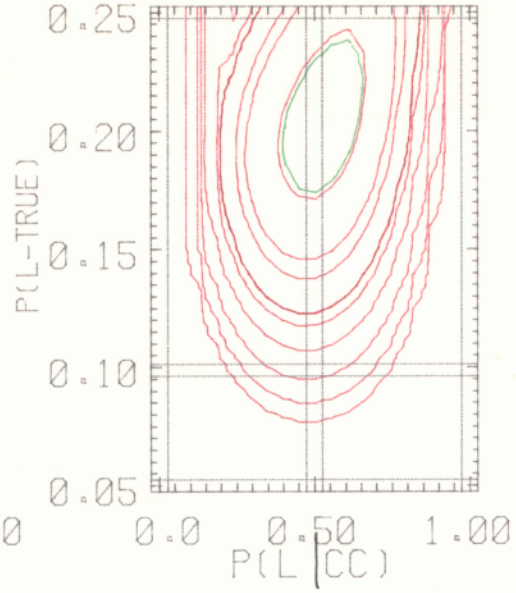
Figure 8.14 As for Figure 8.6 except for the Trankell model (see also note to Figure 8.12).

NEWCOMBE & RATLIFF (1973) :PERM

TEMPORARY MODEL

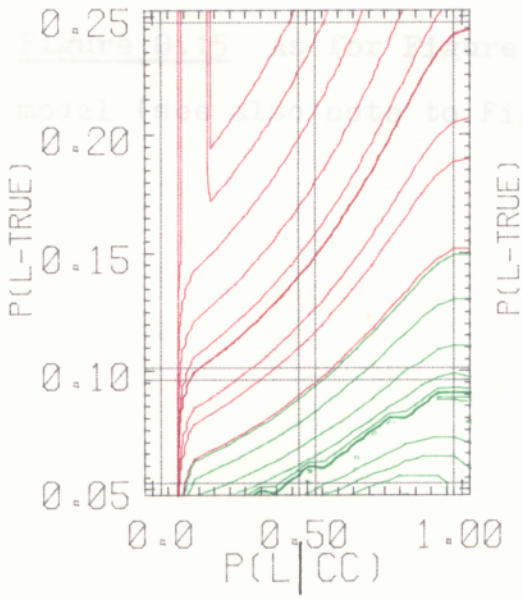


PERMANENT MODEL

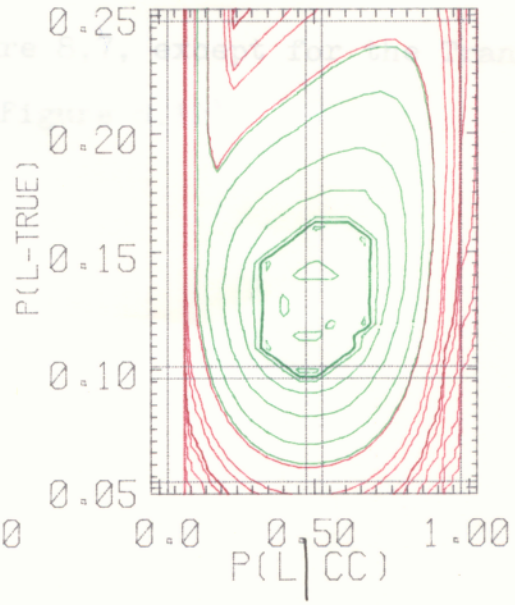


BINGLEY(1958)

TEMPORARY MODEL

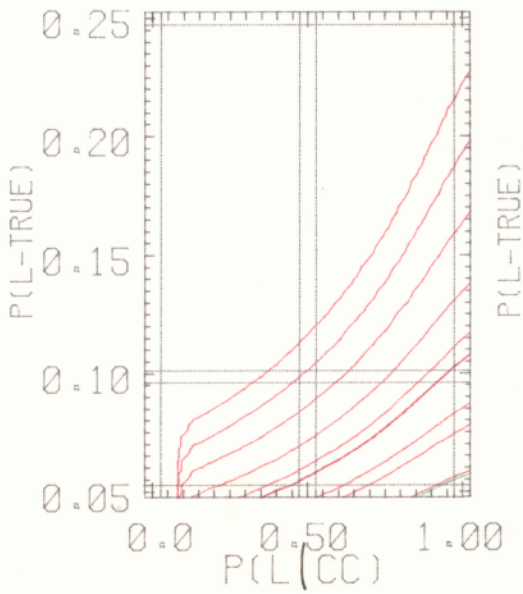


PERMANENT MODEL



PENFIELD & ROBERTS (1959)

TEMPORARY MODEL



PERMANENT MODEL

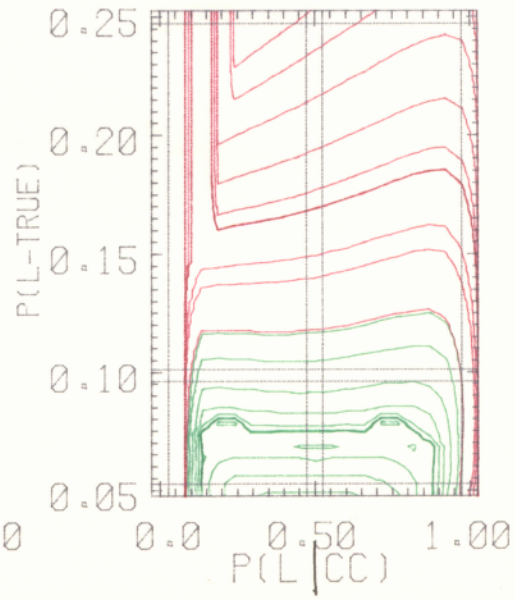
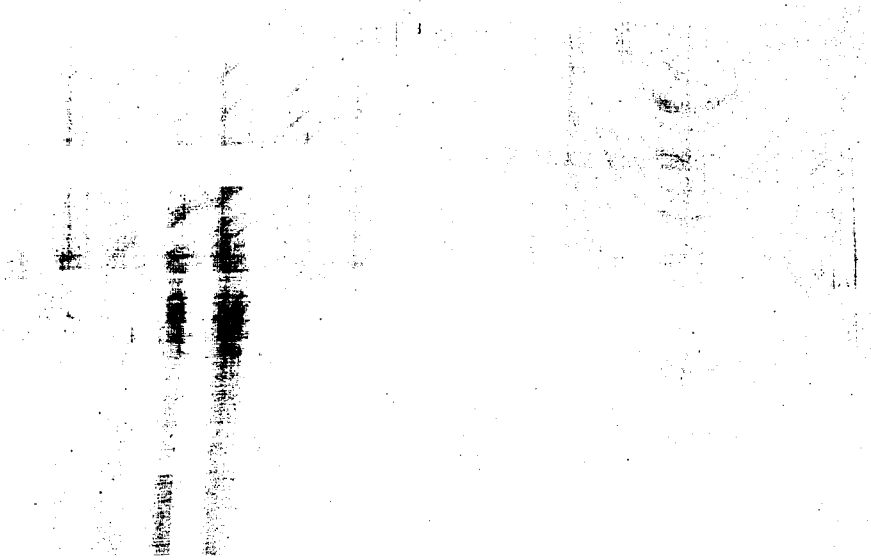
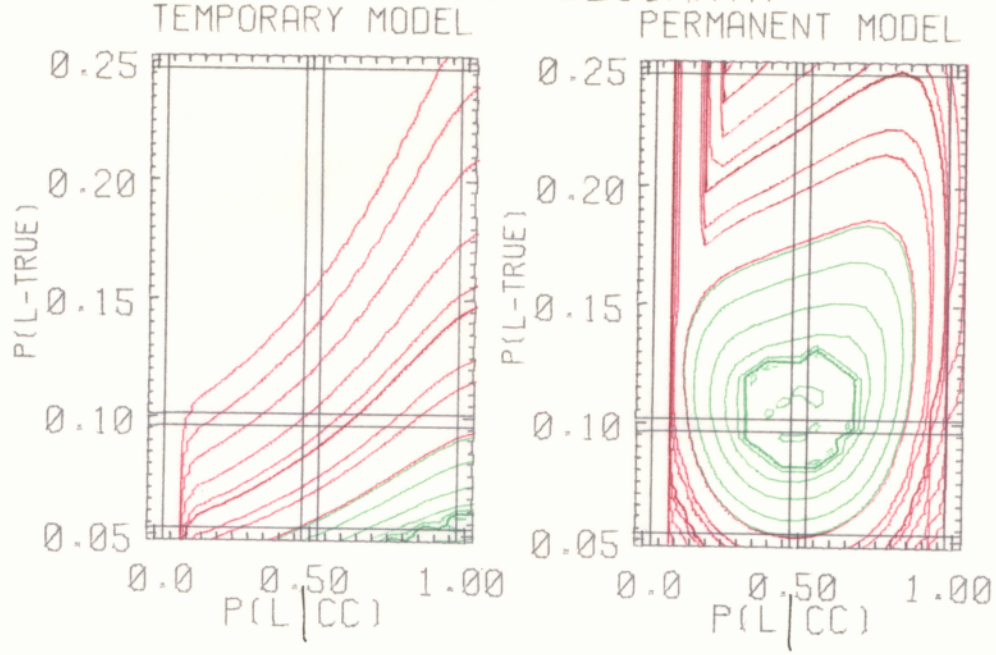


Figure 8.15 As for Figure 8,7, except for the Trankell model (see also note to Figure 8.12).





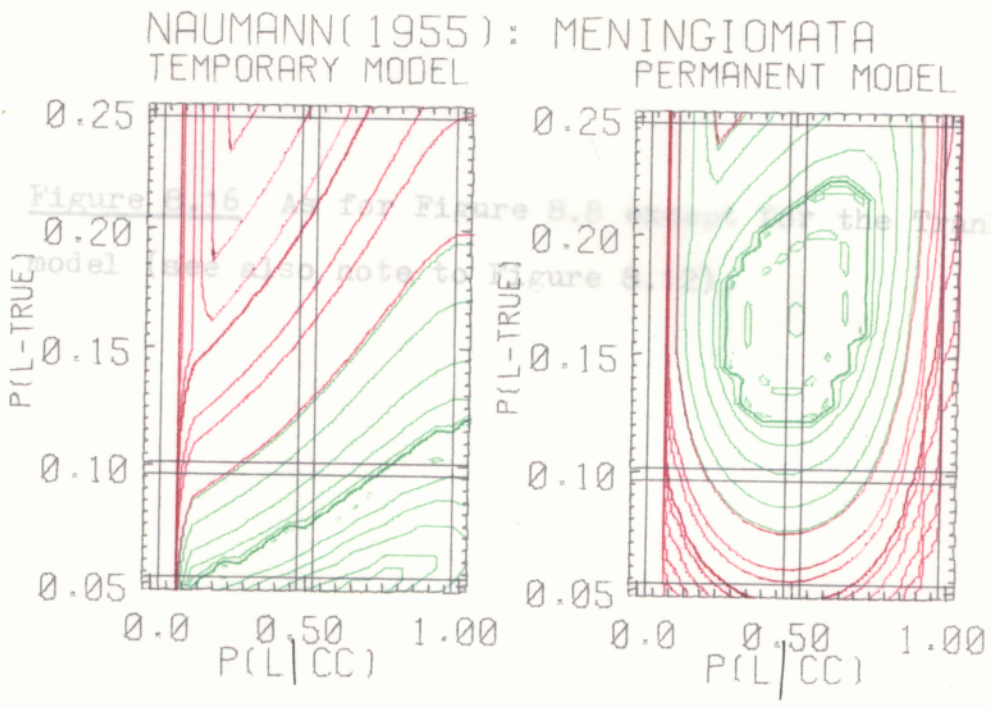


Figure 8.16 As for Figure 8.5 except for the Trunkell model (see also note to Figure 8.15).

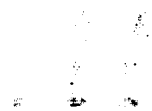
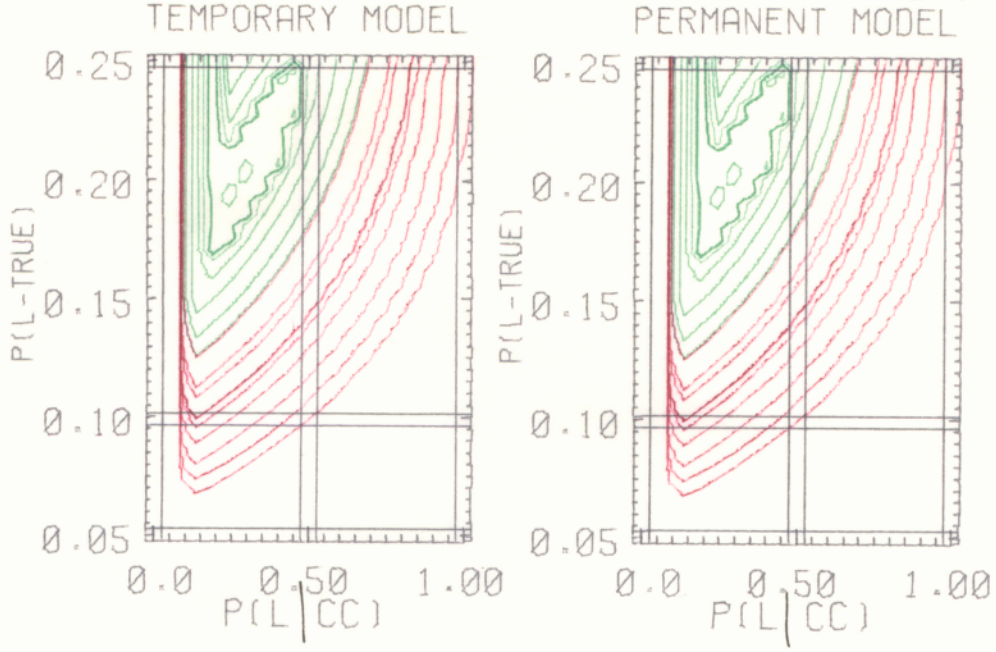


Figure 8.16 As for Figure 8.8 except for the Trankell model (see also note to Figure 8.12).



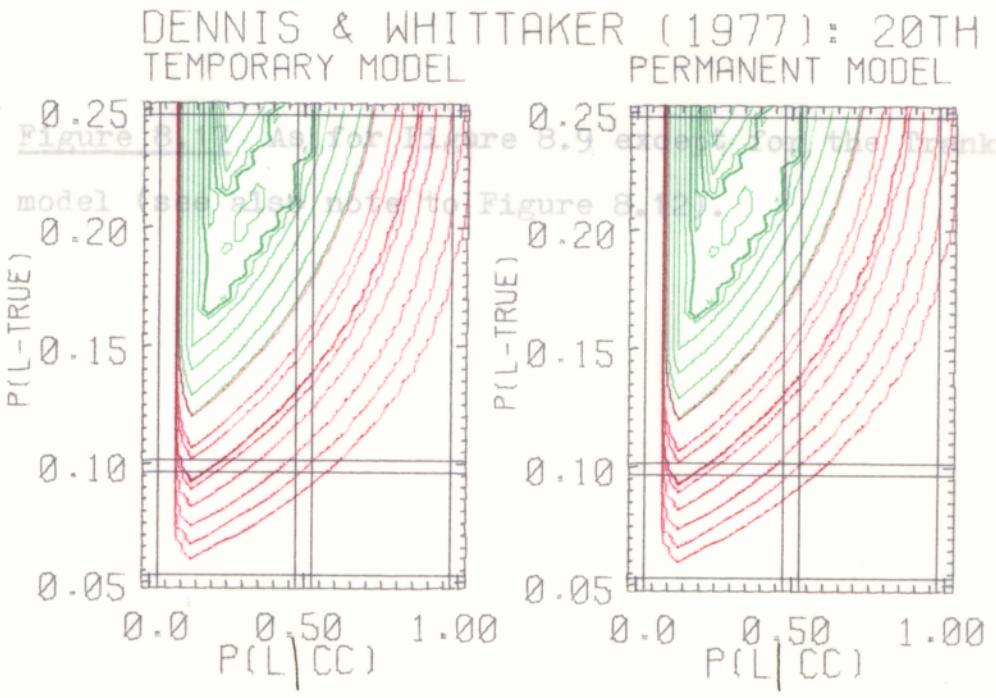
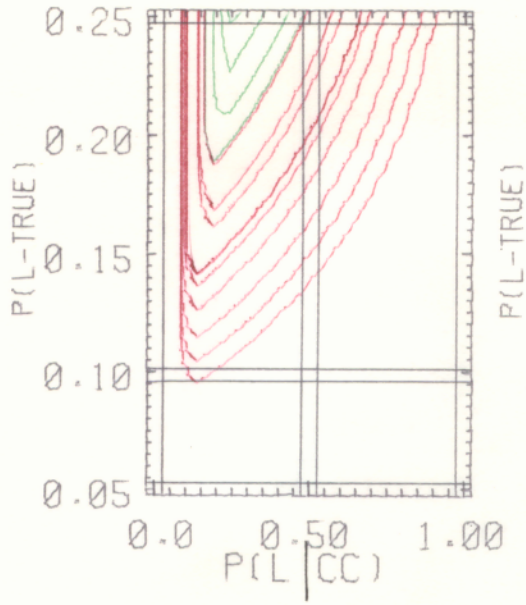


Figure 8.[illegible] As for Figure 8.9 except for the Dunkell model (see also note to Figure 8.[illegible]).

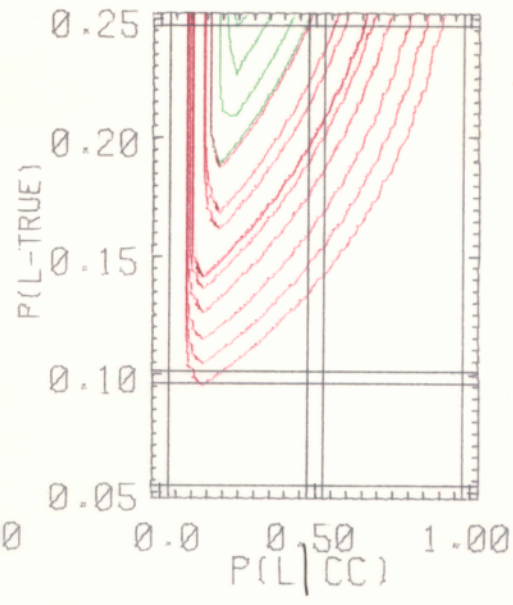
Figure 8.17 As for Figure 8.9 except for the Trankell model (see also note to Figure 8.12).

HECAEN & PIERCY (1956): TOTAL

TEMPORARY MODEL

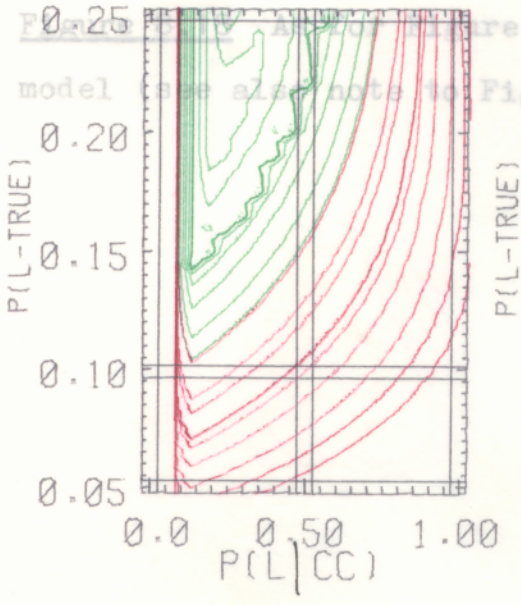


PERMANENT MODEL

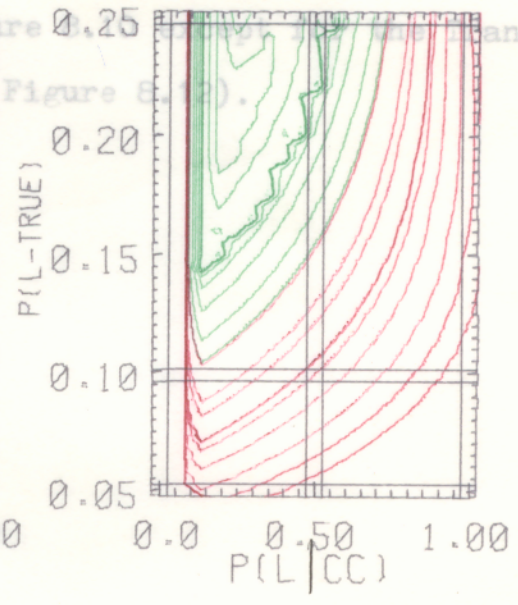


HECAEN & PIERCY (1956): EXPRESSIVES

TEMPORARY MODEL

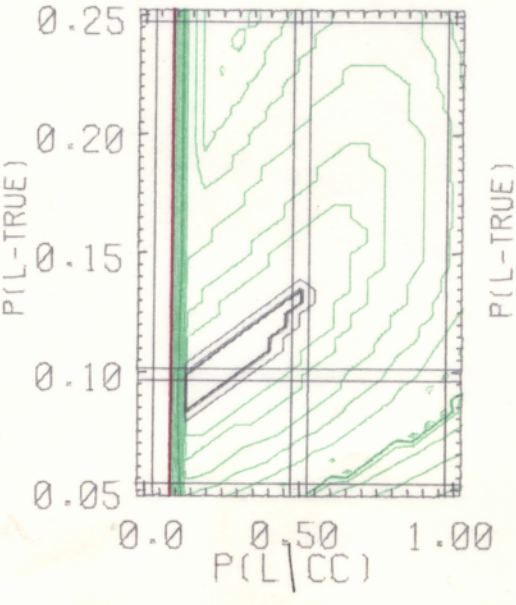


PERMANENT MODEL

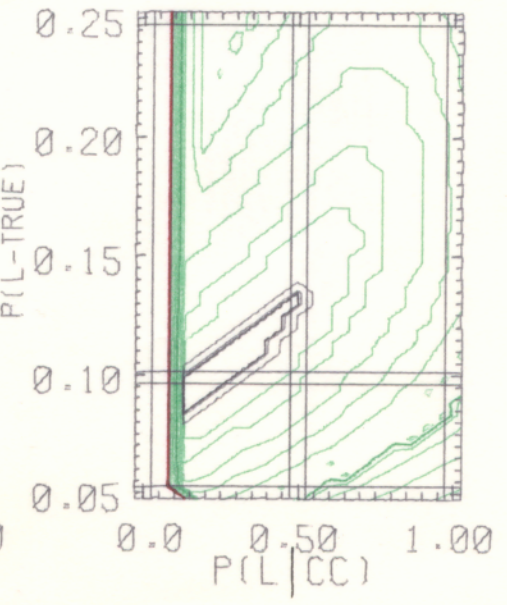


HECAEN & PIERCY (1956): RECEPTIVES

TEMPORARY MODEL

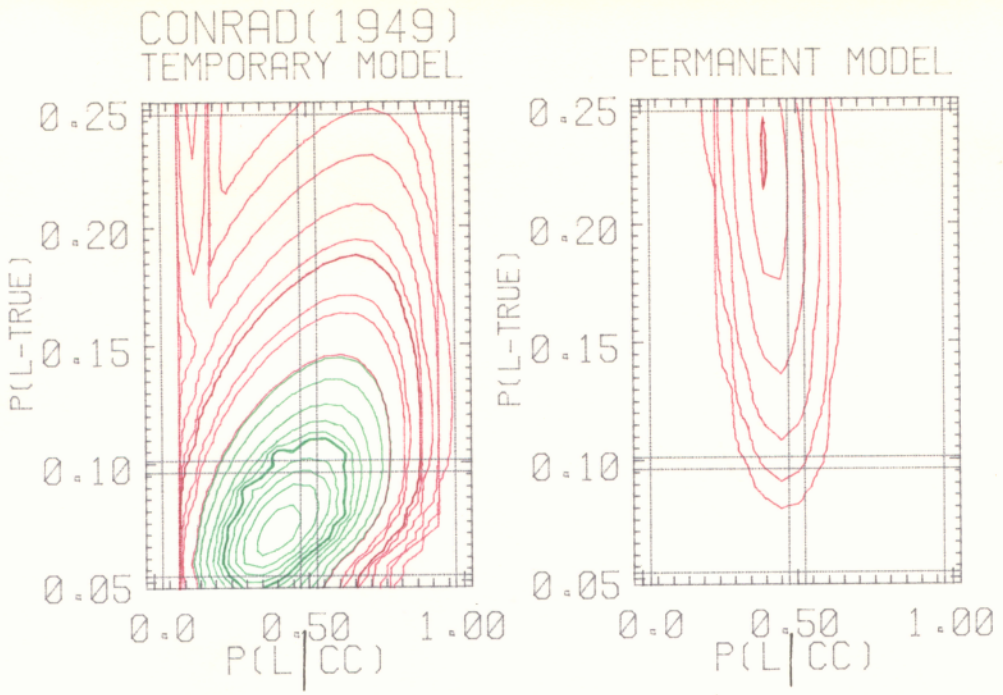


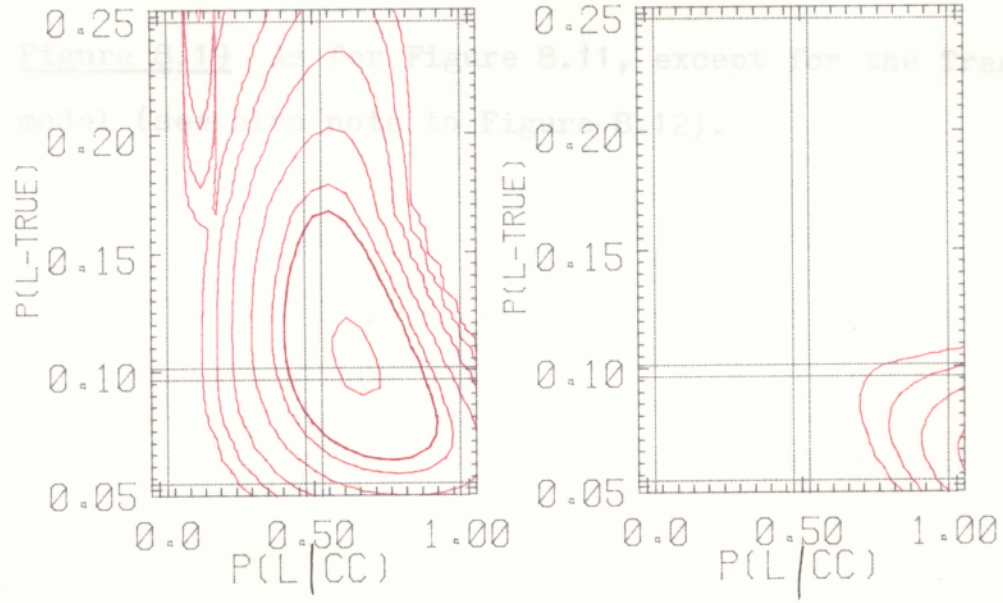
PERMANENT MODEL



TRNK 05

Figure 8.18 As for Figure 8.10 except for the Trankell model (see also note to Figure 8.12).


CONRAD(1949)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|CC)


HECAEN & DE AJURIAGUERRA (1964)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|CC)

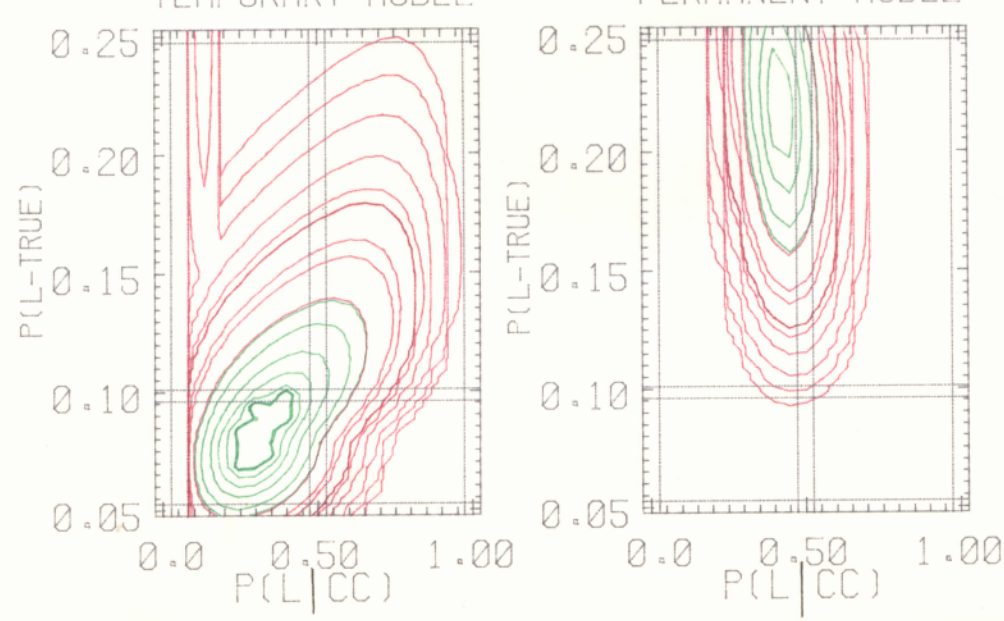
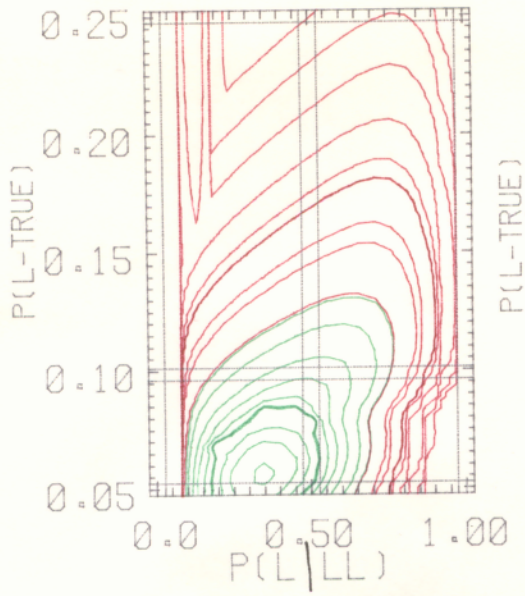

NEWCOMBE & RATLIFF (1973) :TEMP
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|CC)

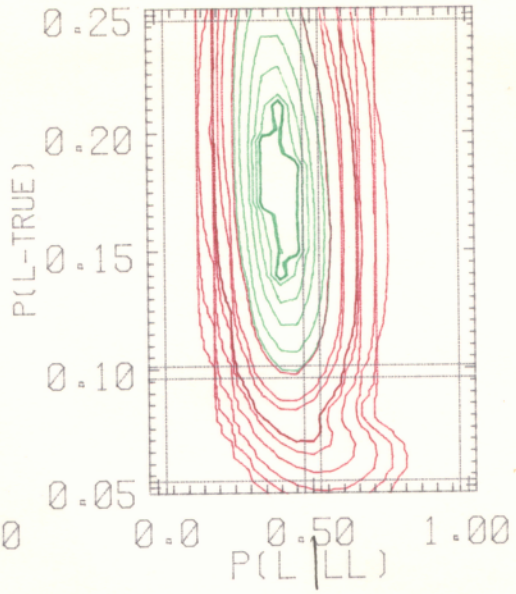
Figure 8.19 As for Figure 8.11, except for the Trandell model (see also note to Figure 8.12).

NEWCOMBE & RATLIFF (1973) :PERM

TEMPORARY MODEL

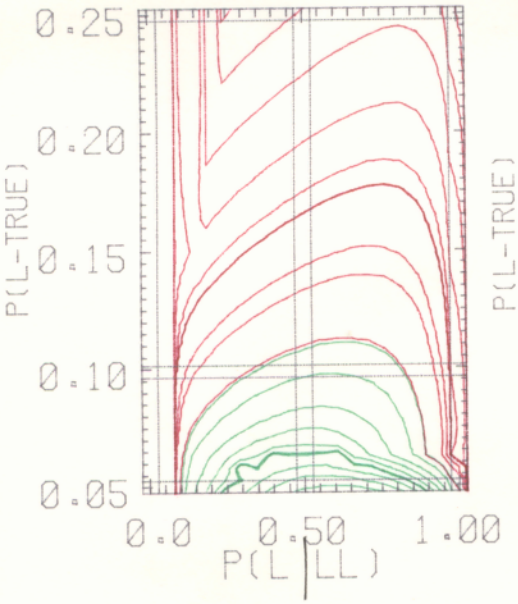


PERMANENT MODEL

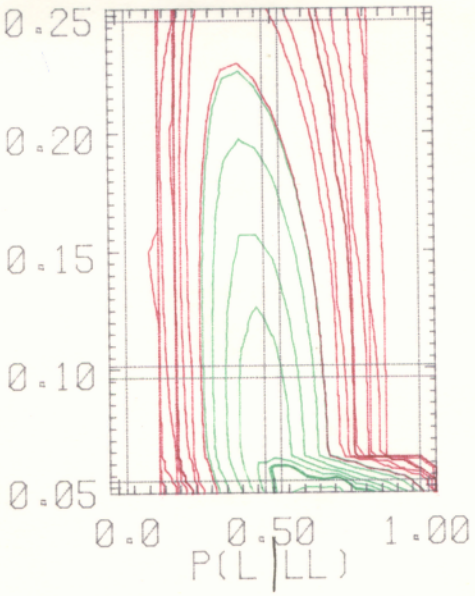


BINGLEY(1958)

TEMPORARY MODEL

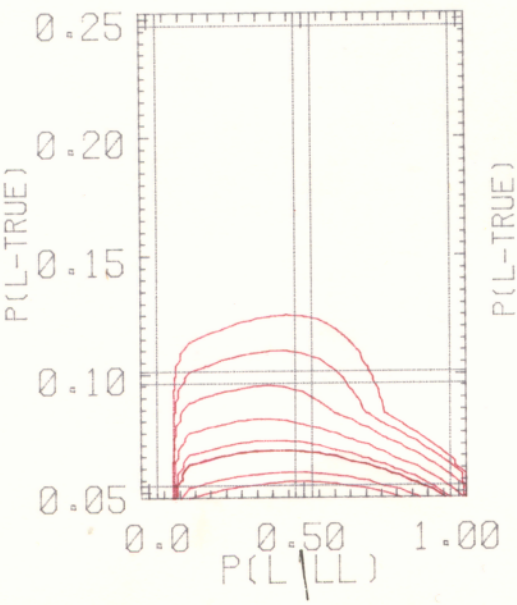


PERMANENT MODEL

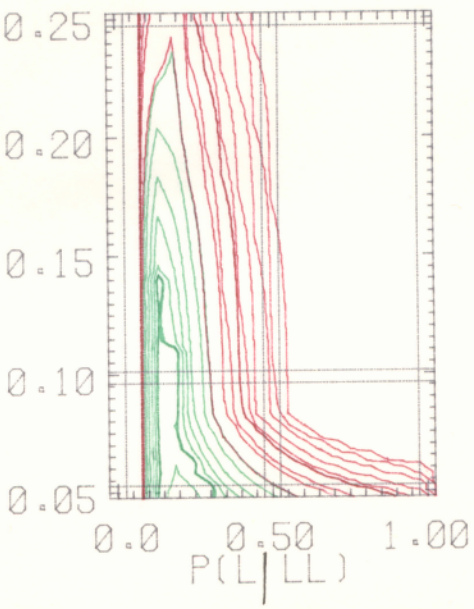


PENFIELD & ROBERTS (1959)

TEMPORARY MODEL



PERMANENT MODEL



TRNK 12

CHAPTER 9: HAND-CLASPING AND ARM-FOLDING

" ... a little folding of the hands ..."

Proverbs, 6:10

Summary

The literature on hand clasping is reviewed and it is shown to be suitable for genetic analysis, there being no classification problems, sex differences, assortative mating or maternal inheritance.

Two-allele genetic models are discussed and shown to be inadequate at explaining the data. A simple three allele model will however fit all the available data adequately, and will also account for the east-west cline in left hand clasping. The limited data on arm folding were also consistent with the three allele model. Hand-clasping, arm-folding and handedness were shown not to be correlated one with another or to show simple genetic linkage.

The genetic model is discussed in relation to the problem of asymmetry as a whole.

9:1 Introduction

In previous chapters I have discussed the inheritance of handedness in terms of a simple genetic model. In this chapter I wish first of all to demonstrate that there are consistent and meaningful familial patterns in hand-clasping and arm-folding, and also to show that this data can be fit by a variant of the simple genetic model described for handedness.

9:2 Types of model of asymmetry inheritance

In asymmetric systems there are usually two phenotypes, right and left, R and L. These are mirror-images, or enantiomorphs. The assumption made in almost all previous models of handedness in particular, and asymmetries in general, has been that enantiomorphic phenotypes must be the product of alleles whose action is symmetric: we may name such alleles as D and S, Dextral and Sinistral. Morgan (1976) has challenged this view and suggested that there is not a single acceptable case in the literature in which enantiomorphic phenotypes can be shown to be derived from symmetric alleles. This forced him to conclude that genes *per se* were not of crucial importance in the inheritance of asymmetry, genes being 'left-right agnosic', and that instead some form of cytoplasmic inheritance was of importance. A further logical possibility, explored earlier in Chapter 7, is

that enantiomorphic phenotypes may be produced by alleles whose actions are asymmetric. Specifically it was proposed that an allele D produced 100% right-handers in its homozygous form, whilst the other allele, C, for Chance, in its homozygous form produced exactly 50% left-handers and 50% right-handers (i.e. a racemic mixture). There are good biological precedents for proposing such a C allele (see Chapter 7).

Annett (1974) has already proposed such a model, although there are differences in detail between her model and the type being discussed here. The distinction between D and C alleles is equivalent to that proposed elsewhere between directional and fluctuating asymmetry (van Valen, 1962).

Considering just two alleles, D and C, we may then consider a general asymmetric model (of the type proposed for handedness):-

	Phenotype	
Genotype	Proportion of type L	Proportion of Type R
DD	0.5 - a	0.5 + a
DC	0.5 - a.x	0.5 + a.x
CC	0.5	0.5

... Model I

'a' controls the rate of manifestation in DD genotypes,
'x' controls the degree of dominance in the heterozygote.

Clearly a has an upper limit of 0.5 (in which case DD produces 100% type R), and a lower limit of 0.0 (in which case there is no detectable inheritance of the asymmetry). The value of x can vary from 0.0 (in which case both CC and DC genotypes produce equal proportions of L and R) to 1.0 (where type R would show some degree of dominance). Handedness can be shown to fit a model in which a = 0.5, and x = 0.5 (i.e. an intermediate model, using the terminology of Wilson (1971a and b)).

Models such as this one (type I) have very strong predictions and limitations. Let p(L) be the proportion of type L in the population as a whole. Clearly p(L) cannot be greater than 0.5 or less than 0.5 - a. In particular, p(L|LxL), the proportion of type L progeny from a mating of two left-type parents, cannot exceed 0.5.

On grounds of symmetry it is also possible to conceive of a model which is the complement of that just described: viz:-

Genotype	Proportion of type L	Proportion of type R	
SS	0.5 + b	0.5 - b	
SC	0.5 + b.y	0.5 - b.y	
CC	0.5	0.5	... Model II

Using the nomenclature of Model I, 'b' represents the rate of manifestation of the SS genotype and 'y' represents

the degree of dominance in the heterozygote. Note that in model II p(L) must be greater than 0.5, and less than 0.5 + b, for all possible sub-populations.

Considering models I and II together, it may be seen that if for any sub-population p(L) is greater than 0.5, then for all sub-populations $p(L) > 0.5$ and vice-versa for $p(L) < 0.5$. This is a very strong constraint and provides a useful method for determining whether a model of type I or II will adequately explain a particular asymmetry. For the particular case of handedness, no single sub-population selected on grounds other than of its asymmetry per se has been shown to have a p(L) greater than 0.5: the discovery of any such population would completely disprove the validity of a model of type I for handedness.

From a consideration of models I and II it will be obvious that there is a third type of two-allele model: this is the case which Morgan (1976) has claimed has never been shown to exist, and which on theoretical grounds would seem to be highly unlikely, the alleles being symmetric in their effects. It will be presented here in its general form, the intention being to show on empirical grounds that it is indeed unsatisfactory for explaining the family data on hand-clasping and arm-folding.

Genotype	Proportion of type L	Proportion of type R	
DD	0.5 - c	0.5 + c	
DS	0.5 + c.z	0.5 - c.z	
SS	0.5 + c	0.5 - c	... Model III

c is formally equivalent to a and b in Models I and II: 'z' represents the dominance and takes values in the range -1 to +1. In this model p(L) can take any value in the range 0.5 + c to 0.5 - c: the values of p(L) in sub-populations may thus 'straddle' the value of 0.5, an important distinction from models I and II.

Model III as stated above is completely symmetric. Clearly it is possible to have an extension of this model in which the penetrance in the genotypes DD and SS is not identical: this type of 'asymmetric-symmetric' model will not be considered in this paper, there being no evidence for even its possible existence. It will be briefly considered as a special sub-set of model IV (below).

For the rest of this paper, "two-allele asymmetric model" will refer to models I and II, whilst "two-allele symmetric model", will refer to model III.

Although thus far only two-allele models have been described, three alleles have been invoked, D, C and S. It is conceivable that all three alleles exist. One may

produce a general model of the form:-

Genotype	Proportion of type L	Proportion of type R	
DD	0.5 - a	0.5 + a	
SS	0.5 + b	0.5 - b	
CC	0.5	0.5	
DC	0.5 - a.x	0.5 + a.x	
SC	0.5 + b.y	0.5 - b.y	
DS	0.5 - a + z(a+b)	0.5 + a - z(a+b)	... Model I

'z' represents the degree of dominance of D over S. By fixing the frequency of allele S at zero, this model becomes model I; by fixing allele D frequency at zero, it becomes model II; and by fixing allele C frequency at zero and making a = b (= c of model III) this becomes equivalent to model III. Whilst this three-allele model is very general it has several problems, one of which is that five independent parameters have to be estimated, i.e. a, b, x, y and z. Furthermore there are three alleles and yet only two phenotypes, and thus knowledge of p(L) alone is not sufficient to give the allele frequencies - one of them has to be independently estimated. It is thus probable that this model will be able to fit almost any data in view of the large number of free variables. In the first instance, therefore, only one very restricted version of this model has been considered. Following on from the evidence that in the case of handedness the mode of inheritance fits a partially dominant

model, the three-allele model has been considered in which all the homozygotes show complete penetrance, and the heterozygotes always manifest mid-way between their respective homozygites. This highly specific model is therefore:-

Genotype	% L	% R
DD	0	100
CC	50	50
SS	100	0
DC	25	75
DS	50	50
SC	75	25

This model as such has no undefined parameters. There are however still only two phenotypes but three alleles, and thus one allele frequency has to be estimated. If one considers not just p(L) but the progeny of the three types of mating pair then this degree of freedom can be removed, and for any particular population the optimal single set of values for allele frequencies may be estimated. This process will be discussed later. For the rest of this paper this highly restricted version of model IV will be known as the "three-allele model".

9:3 Hand-Clasping

9:3.1 Definition

When a person is asked to clasp his hands together, with the fingers interlocking, it is inevitable that one of the thumbs must be on top of the other. For any individual the particular thumb on top is constant, 'Right-Hand-claspers' (RHC) having the right thumb on top. The only long-term follow-up of individuals in order to assess test-retest reliability was by Wiener (1932): in a limited sample of 22 he found 100% agreement after an interval of 18 months.

Although most easily measured by actual inspection, data from survey II (described in Chapter 2) amongst undergraduates of the University of Cambridge, suggests that hand-clasping may accurately be assessed by questionnaire. The Question was,"When you clasp your hands together with the fingers interlaced, which thumb is on the top? Right Left". Of 511 students only 5 (0.98%) did not answer this question satisfactorily. The incidence of left hand-clasping, (LHC) as well as the family data so obtained, was statistically indistinguishable from that obtained by actual inspection of a group of persons living on a housing estate in Cambridge. (I am grateful to Dr. C.G.N. Mascie-Taylor for allowing me to examine this and other of his unpublished data on handed-

ness, hand-clasping and arm-folding); other aspects of the survey by Dr. Mascie-Taylor have been considered elsewhere (Mascie-Taylor and Gibson 1978, 1979; Mascie-Taylor 1979a, and b).

9:3.2 Population Incidence

Estimates of p(L) have been made for a total of fifty-three populations. These give a mean value of 43.85% (SE 1.09%), the distribution being shown in Figure 9.1. It is clear that there are several populations in which p(L) is significantly greater than 0.50; Falk and Ayala's (1971) study of "Caucasians" in New York, "more than 90% (of whom) are descendants of immigrants from Ireland, Italy and Portugal"; Ferronato, Thomas and Sadava's (1974) study of Californians; and two unpublished studies in the United Kingdom, one of which (Dr. Mascie-Taylor) was conducted amongst members of a Cambridge suburb (p(L) = 57.6%, n = 587) and the other of which (Survey II) was conducted amongst graduates of the University of Cambridge, and their parents (p(L) = 56.32%, n = 1453). These data together completely exclude the possibility of a two-allele asymmetric genetic model (Models I and II).

One of the studies seems to give a value of p(L) which is strangely at variance with the other studies; This is the study of Thessalonikan school children by Pelecanos (1969). He found a p(L) of 18.7%, very different

from the value of 47.6% found in Patras by himself only a few years later (Pelecanos, Zacharopoulou and Yannopoulos, 1974). The Thessalonikan data are difficult to explain and need to be independently replicated before being treated seriously.

Figure 9.2 shows a map of the world on which are placed values of p(L) for different populations. Migrant populations are placed at their site of origin; thus Dutch emigrants to Brazil are placed in Holland, and so on. Americans are placed in America only if insufficient information is given to place them elsewhere. Incidences are placed in brackets if the original site cannot be adequately determined. It is apparent that there is a cline extending across the whole of Eurasia and Australiasia, from the United Kingdom in the West (p(L) = 57.4%) to the Solomon Islands in the East (p(L) = 33.7%). Figure 9.3 shows the individual data points for Eurasia/Australasia plotted against degrees of longitude east of Greenwich. The correlation is highly significant. ($r = -0.791$; $n = 40$; $t = 7.97$, $p < 10^{-6}$; analysis weighted for different sample sizes). P(L) in migrants is closer to their sedentes than to indigenous populations, suggesting that the cline might be due to genetic differences. Thus White Australians have p(L) values similar to those of Europe, and very different to those of the Aboriginal Australians.

9:3.3 Generational Differences

Thirteen studies, including our own, present data which allows comparison of the incidence of left hand-clasping (LHC) in different generations, either parent-child data or a transverse study. In 4 studies the difference is significant with $p < 0.05$, and in one study with $p < 0.10$. In all these cases the younger individuals have the higher incidence of p(L). The data points are shown in Figure 9.4. Whilst not conclusive these data might suggest a secular trend towards a greater incidence of p(L).

9:3.4 Sex Differences

Many studies have suggested that there are sex differences in the incidence of LHC: however, others have failed to find such differences, or have found them in the reverse direction. Figure 9.5 summarises the findings of studies on 36 populations. In only one study (Lourie (1972) - Kurds) is the incidence exactly the same in the two sexes. Of the other studies, 12 have found females to have a higher incidence of LHC, whilst 24 have found males to have a higher incidence of LHC: this difference is not significant ($X^2 = 2.85$, 1 df, NS). Neither is there a tendency for either sex to show more extreme lateralisation of hand-clasping (i.e. to be further from the chance expectation of 50%) (see Table 9.1,

$X^2 = 0.45$, 1 df, NS).

9:3.5 Mating Patterns

Figure 9.6 summarises data on mating patterns for hand-clasping. There is no evidence for assortative mating, proportions being accounted for almost entirely by binomial (chance) proportions. The data of Kawabe (1949) is slightly discrepant, but otherwise the fit of the binomial is adequate.

9:3.6 Familial Data

Figure 9.7 shows that there is no evidence for a difference in the proportion of left hand claspers amongst the progeny of L x R matings or of R x L matings. In conjunction with the earlier demonstration of a lack of difference of incidence of LHC in the two sexes, we may probably conclude that whatever the mode of inheritance of hand-clasping, it is unlikely to be sex-linked. For the rest of this paper R x L and L x R groups will be concatenated.

In the literature there are fourteen studies of incidence of LHC in the progeny of various mating types; to these may be added my own data and that of Dr. Mascie-Taylor, shown in Table 9.2. If all these data are plotted as a function of p(L) then it becomes readily apparent that

the 16 samples are not homogeneous, for the four Japanese (J) studies show marked differences relative to the non-Japanese studies,Figure 9.8. In Figure 9.9 are shown the 12 non-Japanese studies: these show a regular relation between the incidence of progeny LHC in R x R, R x L and L x L matings and the overall incidence of LHC. Figure 9.10 shows the four Japanese studies and fitted lines from the non-Japanese studies; clearly there is no overlap between the Japanese and non-Japanese groups.

The distinction between J and NJ studies is of great interest. Consider its implications for a two-allele symmetric model. Given values for c and z then for any value of p(L) the allele frequencies are completely defined. The model may then be used to predict the frequency of LHC in the three types of mating. A computer program has been used fo systematically vary possible values of c and z and to then fit the resulting values of p(L|RxR), p(L|RxL) and p(L|LxL) against empirically derived data, using a X^2 test. There is no single pair of values for c and z for which an adequate fit may be found for all 16 data sets, i.e. J and NJ together. Specifically, for all values of $0.5 \geqslant c \geqslant -0.5$, $0 \leqslant z \leqslant 1$, the minimum X^2_{16} value is 131.61, which is different from chance with a probability $\ll 0.001$; indeed for a completely general 2 allele model (i.e. $0 \leqslant P(L|DD) \leqslant 1.0$, $0 \leqslant p(L|DS) \leqslant 1.0$ and $0 \leqslant p(L|SS) \leqslant 1.0$, where p(L DD), p(L DS) and p(L SS) were all varied independently)

there was no adequate triplet of parameters which would fit all 16 sets of data. However if the 4 J and 12 NJ studies were considered separately then for each group a single c, z pair may be found which fits the data satisfactorily; this is shown graphically in Figure 9.11. The implications of this analysis are clear. A two-allele symmetric model may only be used to fit hand-clasping family data if one is willing to accept that the degree of dominance and penetrance will be greater in Japanese than non-Japanese populations; that is, it is not allele frequencies alone which differ between populations. This conclusion is necessary for any two-allele model, however constructed. Whilst there are precedents for variations in the penetrance of a character between populations, a more parsimonious alternative for the differences between the J and NJ populations is that there are three alleles, and that changes in allele frequency alone determine differences between populations, the penetrance being identical in each case. As mentioned earlier the generalised three-allele model (Model IV) has too many free-floating parameters for it to be useful. We therefore attempted to fit the simple, three-allele model described earlier; this model has a priori acceptability in view of its similarity to a model already shown to be useful in the case of handedness. The model has three alleles and only two phenotypes; a knowledge of p(L) alone is therefore not sufficient for determining allele frequencies. However the extra degree of freedom is lost if one considers

the goodness of fit of family data. To do this a computer program was used which systematically varied the frequency of one allele (say, C). For a particular value of C (where D + C + S = 1) and a known value of p(L) then the values of D and S are uniquely defined. From these may be calculated the values of p(L|RxR), etc., and these values tested against empirical data. Clearly the accuracy of estimation of allele frequencies is entirely dependent upon the number of families included in the data. The process of allele frequency estimation may be seen in Figure 9.12 which shows the Japanese data only. The value of C is systematically altered and the X^2 value calculated, the minimal X^2 value may be found, as also may the 5% range of satisfactory C values. The same process for NJ populations is shown in Figure 9.13. Note that in Figure 9.13 many of the lines terminate at relatively low values of X^2; this is due to an absolute limitation on the frequency of the C allele, for clearly if C becomes too high then it is impossible to find a population of greater than a certain value of p(L). In the extreme case, if C = 1.0, then all populations must have a p(L) of 0.50.

Table 9.3 shows, for all 16 groups of family data, the optimal values of D, C and S, and their 5% ranges, as well as the X^2 value for the optimal fit, all but one of which are acceptable with $p > 0.05$. Note that in many cases an absolute limit of allele frequency is more

important than the 5% limits. The rows of Table 9.3 are arranged in ascending order of optimal D estimates. It will be noted immediately that this shows a moderate correlation with the longitude of the population group; European ('Caucasian') groups have low D values, Japanese groups have high D values. This trend can probably explain the east-west cline demonstrated in Figure 9.3.

A further problem of the two-allele symmetric model discussed earlier, and shown in Figure 9.11, is that although there are several non-Japanese, non-European populations, these show penetrance values more akin to Europeans than to Japanese; this seems counter-intuitive in view of the east-west cline being shown identically for Japanese and for non-Japanese, Non-European groups. Further study of Table 9.3 provides an explanation of this anomaly. As the D values increase as one passes down through Table 9.3, the C values show a concurrent decrease. However the S values appear to show little relation to the D values. This may be demonstrated formally by a Pearsonian Correlation analysis:-

	C	S
D	$r = -0.865$ $p = 1.5 \times 10^{-5}$	$r = +0.266$ $p = 0.317$
C		$r = -0.611$ $p = 0.0118$

There is no correlation between the values of D and S, although highly significant negative correlations exist between C and D, and C and S. (The partial correlation of D and S, $r_{DS.C}$ is of course negative (-0.661) and significant, since D + C + S = 1). From Table 9.3 it is apparent that the S allele is more common in the Japanese populations than in the other European groups. This can be seen more clearly in Table 9.4. We can thus summarise. There is an east-west cline in LHC due to an increased proportion of D alleles in non-European populations. There is a higher frequency of S alleles in European than in non-Japanese, non-European populations, perhaps a reverse cline. However, as in some other ways the Japanese seem to resemble Europeans more than other Eastern groups, they also have high, 'European' levels of S. The Japanese thus have high values of both D and S alleles; other groups have high values of either D or S alleles. Hence the apparent difference demonstrated in Figure 9.9 and 9.10.

9:3.7 Twin Data

Only two studies have provided data on hand-clasping in monozygotic and dizgotic twin pairs. In one study (Martin, 1975) there is no indication of the population incidence of LHC. In neither study is there data from family studies, and thus it is not possible to estimate allele frequencies for the populations from which the twins

were drawn.

For completeness, data from the studies is shown in Table 9.5. It is worth noting that Martin's interpretation of a lack of genetic control, due to a failure to find an increased concordance in MZ rather than DZ twins, is erroneous; as shown in the case of handedness (Chapter 7) lack of concordance in genetic systems in which chance plays a large role is of little consequence. The three allele model, predicts expected frequencies in MZ twin pairs which are close to binomial frequencies.

9:3.8 Family Size

There is no evidence for any differential fertility between right and left hand-claspers (Leguebe,1967): neither do our own data provide any evidence for such a possibility (but see Chapter 10).

9:4 Arm-Folding

9:4.1 Definition

If one asks someone to fold their arms, then one fore-arm crosses over the other. Left arm-folding (LAF) is defined as the left radius and ulna over the right radius and ulna. Test-retest reliability does not seem to be as high as for hand-clasping. Wiener (1932) found

that 3 out of 22 individuals were inconsistent in their preference over an 18 month period. Assessment is usually by visual inspection, and a family study using this method has been carried out by Dr. Mascie-Taylor. Unlike the case of hand-clasping, it was not felt practicable in a questionnaire study (such as my Survey II) to try and assess arm-folding, it being difficult to devise an unambiguous question.

9:4.2 Population Incidence

The incidence of LAF has been determined in 23 population groups. The distribution, shown in Figure 9.14, is very interesting. Unlike the case of hand-clasping, which shows an almost normal distribution, there is a very sharp cut-off, the majority having p(L) values of greater than 50%. However there are 4 populations in which p(L) is less than 50%, all being significantly so ($p < 0.001$ for all groups except the Fali, or Huizinga (1968) for whom $p < 0.05$). Three of these groups (the Fali and Kurumba (Huizinga, 1968)), and the Angolan Negroes (Freire-Maia and de Almeida, J. 1966)) are African whilst none of the groups with $p(L) > 0.50$ are African. The fourth anomalous group is a group of Russian Immigrants to Brazil, who have an extremely low incidence of LAF (8.8%) (Freire-Maia, Freire-Maia and Quelce-Salgado (1960)). Although not explicitly stated it is highly likely that this group is strongly inbred, possibly for several centuries.

Unless the data from these four groups with p(L) < 0.50 can be shown to be methodologically inadequately, it is highly likely that a two-allele asymmetric model will be unable to cope with the arm-folding data.

9:4.3 Generational Differences

There is insufficient data in the literature to investigate this phenomenon, but if effects exist, they are almost certainly small.

9:4.4 Sex Differences

Figure 9.15 shows data from 17 population groups for whom data are given on sex differences. There is no evidence for overall differences being significant.

9:4.5 Assortative Mating

Table 9.6 shows data from 5 studies, two of which are our own. The incidences of LAF in the five studies do not differ significantly, and neither does the 5 x 3 table show any heterogeneity (X^2 = 12.87, 8 df, NS). The data may thus be amalgamated. Of 879 mating pairs, 173 (19.68%) were both right arm-folding, 401 (45.62%) were of different arm-folding types, and 305 (34.69%) both showed LAF. Expected values under a binomial hypothesis are 158.7; 429.6 and 290.7, respectively, which

do not differ significantly from observed values (X^2 = 3.89, 2 df, NS).

9:4.6 Familial Data

There are five studies in the literature which give family data on arm-folding. In addition Dr. Mascie-Taylor has carried out a survey of familial trends in arm-folding (see Table 9.7 for data). The incidences of LAF in these six studies show no significant differences (Dr. Mascie-Taylor, n = 301, LAF = 58.47%; Falk and Ayala, 1971, n = 1196, LAF = 55.93%; Wiener (1932), n = 389, LAF = 56.29%; Leguebe and Martinez-Fuentes (1971), n = 897, LAF = 56.41%; Rhoads and Damon (1973), n = 260, LAF = 58.84%; Ferronato et al (1974), n = 204, LAF = 56.86%; X^2 for homogeneity = 1.64, 5 df, NS). It is thus possible, for the purposes of comparison, to combine the data from the six studies. Of 682 progeny of R x R matings, 51.31% show LAF; of 1553 progeny of R x L matings, 56.53% show LAF; and of 1013 progeny of L x L matings, 60.31% show LAF. These differences are significant overall (X^2 = 13.42, 2 df, $p < 0.005$). The difference between R x R and R x L is significant (X^2 = 5.03, $p < 0.05$) as is the difference between R x R and L x L (X^2 = 13.09, 1 df, $p < 0.001$). The difference between R x L and L x L is almost significant (X^2 = 3.45, 1 df, $p < 0.10$). This is strong evidence for a familial, and probably a genetic, component in the control of arm-folding.

Although the proportions of LAF overall are the same in all six studies, the family data themselves are not homogeneous; thus if one considers the progeny of R x R matings, then there are significant differences between the studies (X^2 = 19.78, 5 df, $p < 0.01$). This situation is analogous to the differences between Japanese and non-Japanese populations in the case of hand-clasping, and for identical reasons it means that it will only be possible to fit a two-allele model (of any type) if one assumes that penetrance and /or dominance may differ between populations. This might be an unreasonable assumption, and thus it was felt more reasonable, as with hand-clasping, to fit a three-allele model, and once again the simple three-allele model described earlier was used. Table 9.8 shows, in an exactly analogous manner to that described earlier for hand-clasping, the optimal estimates and the 5% ranges of allele frequency estimates for the six studies. Five of the six studies are fitted satisfactorily by what is, in effect, a 2-allele asymmetric model, the frequency of D being 0. The study of Falk and Ayala however requires a significant proportion of D allales, which is difficult to explain. If the population had contained a proportion of persons of African derivation these D alleles would have been comprehensible. According to Falk and Ayala their population consists of "all Caucasians, more than 90% are descendants of immigrants from Ireland, Italy and Portugal"; possibly the Portugese contingent contained a proportion of 'African' D alleles

derived perhaps from the Moorish invasions of the Iberian peninsular.

In conclusion the arm-folding data ia satisfactorily fitted by a three-allele model with identical properties to that proposed for hand-clasping.

9:4.7 Twin Data

To my knowledge there is, in the literature, no arm-folding data from twins.

9:5 Inter-Relations between Asymmetries

There is strong evidence that three behavioural asymmetries, handedness, hand-clasping and arm-folding, are familial, and are probably genetic. For this conclusion to be of real worth it is necessary to know whether there is any relationship between the asymmetries; thus if hand-clasping were to be strongly correlated with handedness, the familial trends in hand-clasping would be trivial.

9:5.1 Handedness and Hand-clasping

Seven studies have looked at this. Wiener (1932), Ferronato *et al* (1974) and Beckman and Elston (1962) all found no correlation between handedness and hand-clasping.

My own survey II found no correlation (see Table 9:9.1). The study by Dr. Mascie-Taylor found a significant correlation when all individuals were included in the analysis (Table 9.2.2a; $p < 0.005$). However this seemed to be primarily due to a small number of left-handed families; when the data was re-analysed for the parental generation only (i.e. who were genetically unrelated) the relationship became non-significant (Table 9.9.2b). The study of Pelecanos (1969) claimed to find a "highly significant positive correlation"; however no data is given, and, as explained earlier, this study is open to serious doubt in view of the very low incidence of LHC. Rhoads and Damon (1973) claimed to find a significant correlation ($p < 0.01$) but they give insufficient data to be able to assess this claim adequately. In summary, there is no adequate evidence for a correlation between handedness and hand-clasping.

9:5.2 Handedness and Arm-Folding

Five studies have investigated this relationship. Data from Dr. Mascie-Taylor's study is shown in Table 9.10; no evidence was found for a correlation. Wiener (1932), Ferronato et al (1974), Pelecanos (1969), and Beckman and Elston (1962) all found a similar lack of association. No studies have found a positive association, and thus this result seems fairly secure.

9:5.3 Hand-clasping and Arm-folding

Seven studies have investigated this relationship. Data from a study by Dr. Mascie-Taylor is shown in Table 9.11; there is no significant association. A similar lack of correlation was found by Wiener (1932), Falk and Ayala (1971), Ferronato et al (1974), Pelecanos (1969), Beckman and Elston (1962) and Rhoads and Damon (1973). No studies have found a significant correlation and thus the statistical independence of hand-clasping and arm-folding also seems fairly secure.

9:5.4 Situs Inversus

Situs inversus totalis (either with or without Kartagener's triad) is probably inherited in a similar manner to handedness, a two-allele asymmetric model describing the available data (see Chapter 7). Situs is known to show no correlation with handedness (Torgerson, 1950). No studies, to our knowledge, have looked at hand-clasping or arm-folding in situs inversus; it is conceivable but unlikely that one (or even both) of these behavioural asymmetries is associated with situs, and this topic clearly needs investigation in the future.

9:5.5 Linkage

Whilst there is no simple association between handedness, arm-folding and hand-clasping it is possible that the

genes for their control are located on the same chromosome, and thus would show genetic linkage. Without being able to accurately genotype individuals it is not easy to say with certainty that linkage is or is not present. A crude analysis is however possible. Consider two parents both of whom are right-handed and both of whom are right hand-claspers. If linkage occurs then their children should tend to be either right-handed and right hand-clasping, or left-handed and left hand-clasping more often than chance would predict. And similarly for both parents right-handed and left hand-clasping, etc. Table 9.12 shows, for all pairs in which both parents are of the same handedness and hand-clasping, the proportion of progeny with the same or different hand-clasping and handedness. If linkage occurs, same-same and opposite-opposite should occur more frequently than same-opposite and opposite-same; there is no evidence for this in the present analysis. Tables 9.13 and 9.14 show similar analyses for handedness and arm-folding, and arm-folding and hand-clasping; again, there is no significant linkage.

9:6 Discussion and Conclusions

It is beyond reasonable doubt that hand-clasping and arm-folding are familial, and in the absence of any other theory which can cope with the data, a genetic model seems reasonable. If penetrance values are to remain constant a two-allele model within populations is unacceptable

and it is necessary to postulate a three-allele model. This raises several questions which can perhaps be best discussed by considering the nature and evolutionary development of asymmetries.

In the absence of genetic control, and due to the inevitable chance fluctuations that one finds during development, one must expect that organisms will show a degree of asymmetry, albeit often minimal. 50% of organisms will show one side as 'dominant' and 50% the opposite side as 'dominant' i.e. a racemic mixture, due to fluctuating asymmetry. Directional asymmetry is asymmetry in which a non-racemic mixture of the two phenotypes is found, one enantiomorph predominating over the other. In its most extreme form all of a species are directional in the same way (e.g. in the fiddler crab, and other crustacea, almost all organisms are born with the left claw larger (Vernberg and Gostlow, 1966; Hamilton, Nishimoto and Halusky, 1976)). Directional asymmetry poses distinct problems for genetic systems. Consider the problem of an observer looking at a pair of gloves. He is in radio contact with another individual in a space-ship, who also has a pair of gloves in front of him. The problem for the observer is to tell his galactic colleague which of the two gloves he happens to be pointing at. This problem, first posed by Kant, is impossible to solve unless both the observer and his colleaque have a known and conventional asymmetry. The problem would be unsolvable

if we were communicating with a Martian of unknown asymmetry. (For an ingenious solution dependent upon the failure of sub-atomic parity conservation, see Gardner, 1967). The problem for genes determining asymmetries is essentially similar to that of the observer and the astronaut. The linearly sequenced gene-code (equivalent to the radio message) has to tell a three-dimensional system which of two possible enantiomorphic forms is the correct one. A possible, although highly unlikely, solution, lies in the asymmetry of the helix of the genetic DNA itself; there is however no evidence that this information may be used by cells; and it is extremely difficult to conceive of a possible mechanism.

The only clear, but somewhat unusual, example of the inheritance of an asymmetry, is discussed by Morgan (1976) and found to be of particular interest. The snail, Limnaea peregra, occurs in two forms, a common dextral, and a rare sinistral, form. The asymmetry is determined by a recessive gene which has the strange property of working one generation out of phase: the asymmetry of an individual depends upon the genotype of its 'mother' (they are hermaphroditic), and not at all upon its own genotype (Boycott, Diver, Garstang and Turner, 1920). The effect is due to cytoplasmic inheritance. Thus a gene produces cytoplasm of a particular asymmetric type. However the gene manifests after the asymmetry of the organism has actually been determined, and the cytoplasm produced will

therefore control the asymmetry of the individual's progeny, etc. Morgan proposed that cytoplasmic inheritance might be of importance in determining human asymmetries. Whilst it may have a role, the model clearly cannot cope with several asymmetries all of which appear to be statistically independent. Furthermore evidence for maternal inheritance of these asymmetries is conspicuously lacking in most of the cases, and is, at best, dubious in the case of handedness. Whilst all of asymmetry cannot be explained by cytoplasmic inheritance, it is nevertheless quite possible that there is a cytoplasmic component in asymmetry inheritance, for it is still a problem that the genes are, except for their own helical asymmetry, essentially left-right agnosic.

Earlier, in Chapter 7, a model has been proposed for the inheritance of handedness which depends upon two alleles, D and C (i.e. Model I). The CC genotype shows pure fluctuating asymmetry; the DD genotype is completely directional. The problem remains of how these genes are working. One solution is to suggest that some form of 'asymmetric signpost' is carried over from individual to individual by cytoplasmic inheritance. There is generally only one phenotype for this sign-post and thus all organisms of the species possess it. The action of the DD genotype is to read this signpost and to go in the direction pointed. The CC genotype is effectively blind, and ignores the signpost. With such a system it is possible for the genotype for one asymmetry (say handedness) to be DD, and thus to read the signpost, and for

another asymmetry (say situs inversus) to be CC (and thus to ignore the signpost), thereby producing statistical independence of situs and handedness.

Having proposed such a model it is easy to postulate a mechanism for the S allele. The D allele is essentially left-right agnosic; it does not distinguish left and right, it merely goes in the direction pointed. The SS genotype can read the signpost, but has somehow misunderstood it, and always goes consistently in the wrong direction. The result would be phenotypes enantiomorphic to the DD genotype. It is worth stressing that the D and S alleles are still however left-right agnosic. Consider the rural recruits to the Imperial Russian Army who were supposedly taught to march in step by having a bundle of hay tied to their right foot and a bundle of straw tied to their left foot; the officer thus called out 'Straw - Hay - Straw - Hay' to make the men march in step (Elze, 1924; see Fritsch, 1968, p. 55). The left-right agnosic recruits appear to be able to distinguish left from right. But an urban officer might well have confused the meaning of Straw and Hay, and consistently called Straw 'Hay', and vice-versa (a not unknown error amongst town dwellers). The result would be that the recruits would still act consistently but would now move in the exact mirror-image of the movements produced for the first officer, even though the intentions of the officers were identical. It is thus possible to have just

a pair of left-right agnosic alleles, one of which produces the enantiomorph of the other.

The problem of the inheritance of asymmetry in Limnaea may now be seen in a fresh light. Two alternatives present. One is that the alleles might actually, as it were, turn the signpost around. The other is that they may reverse the reading of the signpost. Whilst the first answer is possible it does tend to produce an infinite regress, for how does the allele know which way to point the signpost? The only solution is by another signpost, and so on. And yet if the genes only alter the reading of the signpost, this also has problems, for the direction is determined before these genes have been read themselves; their action is to read the signpost on behalf of the next generation, and to leave some form of (analogue, presumably enantiomorphic) message to this effect. This latter alternative seems the most reasonable of the two possibilities. It does however leave us in the situation of having no definite example of the alteration of the cellular, cytoplasmic signpost, and hence having no definite evidence for its existence. It is therefore necessary to clearly describe the properties we would expect the signpost to have, so that it will be recognisable when it is found. We may distinguish lesions of the signpost from lesions of the signpost reading system.

Let there be two statistically independent asymmetries producing phenotypes P, $\bar{P}$ and Q, $\bar{Q}$ where $\bar{P}$ is the mirror-image of P, etc. Consider a population of homozygous individuals of phenotype P, Q (i.e. homozygous at each of the loci, which show no linkage). There is also a cytoplasmic signpost, S, which can be reversed to produce its enantiomorph, $\bar{S}$. The signpost may also be 'blocked' or made illegible, symbolised s.

If the signpost is acting typically, i.e. S, then all progeny of the hypothesised population will be of form P, Q. If however the signpost should be reversed, $\bar{S}$, all progeny will be of form $\bar{P}$, $\bar{Q}$. If the signpost should be blocked, s, then $\bar{P}$ and P, and Q and $\bar{Q}$ will each appear randomly, and <u>independently</u>, neither the P nor the Q system having any chirality information of its own. We thus have the situation:-

	Phenotype proportions			
<u>Signpost</u>	<u>PQ</u>	<u>$\bar{P}$Q</u>	<u>P$\bar{Q}$</u>	<u>$\bar{P}\bar{Q}$</u>
S	1.0	0.	0.	0.
$\bar{S}$	0.	0.	0.	1.0
s	0.25	0.25	0.25	0.25

Consider a mutation which meant that the P locus alone was unable to read the signpost, and that the population was homozygous for this mutant; we would then expect, with the three types of signpost:-

Signpost	PQ	$\bar{P}Q$	$P\bar{Q}$	$\bar{P}\bar{Q}$
S	0.5	0.5	0.	0.
$\bar{S}$	0.	0.	0.5	0.5
s	0.25	0.25	0.25	0.25

It is thus possible, in principle, to distinguish lesions of the signpost from lesions of the reading system, by looking at the effects on two or more separate asymmetries. Note that in the above P and Q must both be primary asymmetries (i.e. be statistically independent).

Having discussed the implications of the D and S alleles of hand-clasping and arm-folding for theories of asymmetry inheritance per se, we may briefly discuss the details of hand-clasping and arm-folding.

Unlike handedness, there is no known anatomical or physiological basis for either hand-clasping or arm-folding; they appear to be simple behavioural preferences. There is no evidence, for either asymmetry, of any advantage, or any significant consequence upon behaviour in general. They are essentually trivial behaviours; and yet are controlled genetically. This does not appear to be due to them being 'correlated characters' (Huxley, 1942), for no characters are known to correlate with either asymmetry; furthermore there is no evidence for any reproductive advantage associated with either hand-clasping (Leguebe, 1967) or arm-folding (Leguebe and Martinez-Feuntes, 1971).

Analysis of the allele frequencies for hand-clasping, shows that the frequency of C correlates inversely with both D and S, but D and S show no significant inter-correlation. A parsimonious explanation of this would be that initially all individuals were of CC genotype. The alleles D and S arose by mutation, and each spread by genetic drift and migration procuding the east-west cline demonstrated earlier. The importance of genetic drift in determining the allele frequency is shown by entering the measured values into the model of Watterson and Perlow (1978).

Initially a model was tested in which only heterozygotes of intermediate penetrance were allowed. Later a further 26 models were tested in which various combinations of penetrance were allowed. Table 9.15 gives these results; it may be seen that the purely intermediate model described earlier is an excellent fit, with only one other model obtaining a slightly lower X^2 value. It may also be seen that the heterozygote SC must show intermediate penetrance, whilst the degree of the dominances of DC or DS is not critical, all allowing good fits. Parsimony would suggest that in the first instance all three heterozygotes may be regarded as of intermediate penetrance.

Animal models of hand-clasping would be useful for testing the hypothesis proposed in this paper. Behavioural analogues might be the manner in which insects fold their wings, for if the wings are of sufficient length it is

inevitable that one wing must be laid over the other, and for many insects this would seem to be constant for any individual. 50% of Drosophila melanogaster fold left over right, and 50% vice-versa. An attempt has been made in this species to selectively breed for one of the two phenotypes; this was claimed to be successful for a few generations and then the effect was lost (Purnell and Thompson, 1973). Possibly the wild type is mostly CC. Selection might have encouraged lethals associated with homozygous D or S, and hence the loss of the character after a few generations. Appendix 9.1 presents evidence to suggest that all of these flies may well be of the CC genotype. Not all insects are racemic for wing-folding; less than 0.1% of Gryllotalpa gryllotalpa and Acheta domesticus fold the left wing over the right, whilst this same phenotype is found in about 5-10% of Acheta assimilis, A. rubens, and Teleogryllus commodus (Neville, 1976). We may presume that the majority of these insects have S alleles.

Non-behavioural analogues of hand-clasping may be found in the fish. In teleost fish the nerves from the eyes decussate completely at the optic chiasma, either the entire left nerve passing dorsal to the right nerve, or vice versa. In the majority of teleosts the chiasm shows a dimorphic racemic mixture (Parker, 1903). The flatfish (heterosomata) represent an interesting group for they have a gross asymmetry, one eye migrating round

so that it is on the same side of the body as its opposite number. In the most primitive heterosomata, the Psettodes, the optic chiasm is also racemically dimorphic, and is unrelated to the eye which migrates. The other flatfish are classified into families according to whether the chiasma is racemically dimorphic (the soles, Soleidae) or is monomorphic, all species showing either right over left or left over right (the flounders, Pleuronectidae). The soles may be further divided into two types, according to whether the left or the right eye is the one that migrates (the tongue soles, Cynoglossidae, are all sinistral, whilst the true soles, the Soleidae are all dextral). The flounders may be similarly sub-divided according to the migrating eye (the turbots, Bothidae, are sinistral and have the right optic nerve over the left, whilst the flounders proper, the Pleuronectidae, are dextral, and have the chiasma with the left optic nerve over the right (Norman, 1934)). Whilst there are almost no known exceptions to the monomorphisms of the chiasma amongst the pleuronectidae, the migrating eye is not quote so constant and occasionally, in both flounders and soles, one finds reversed individuals, and in a few cases the majority are actually reversed. Thus it would seem that the chiasma and the migrating eye are independent asymmetries. There is sometimes a geographical variation in the migrating eye (but never in the chiasma). A Japanese flounder, Kareius bicoloratis, is typically dextral, all of 83 specimens from one series being

so. The American flounder, Platichthys stellatus, although with a typically dextral chiasma, manifests with about 50% of reversal of the migrating eye, in those specimens found off the coast of California. As one travels up the Pacific coast of America the proportion of eye reversal type increases until off the Alaskan peninsular, 68% are reversed, and those variants off the Japanese coast, have 100% reversal (all of 476 specimens in one series). K. bicoloratis and P. stellatus are both 'dextral' flounders, although the latter species manifests, in the waters off Japan, as a typical reversed dextrals (i.e. sinistrals). We may suggest therefore that the Japanese variants of these species are all DD and SS genotypes respectively. The two species occasionally hydridise to produce the 'species', Pseudoplatichthys oshorensis; this is of particular interest as of 27 specimens caught, 14 were reversed and 13 non-reversed, i.e. exactly intermediate between their parental groups (Hubbs and Kuronuma, 1941). This may be presumed to be formally equivalent to the genotype DS which, in the case of human hand-clasping, and arm-folding, we have proposed forms a racemic mixture of the two phenotypes; the flatfish provide strong justification for this assumption.

A further example from the flatfish also helps in justifying our conceptual model of a cytoplasmic signpost. Whilst reversed flounders are found fairly frequently in some species, these reversed individuals always show a

typical arrangement of the chiasma, and a typical asymmetry of the viscera; it is only the eye migration which is reversed. Hubbs and Hubbs (1944) describe a single flatfish of species Tanakius kitahaeae which is regarded as exceptionally rare, since not only was its eye-migration atypical for its species, but also its optic chiasma was reversed, as also was the asymmetry of the viscera, i.e. it was a true mirror image of the typical form. It thus showed reversal of three independent asymmetries. This is the only reported case of a reversed chiasma in a flounder, and the only known case of situs inversus. The coincidence of all three asymmetries thus seems highly implausible in terms of mutation of three separate genetic loci. A more parsimonious explanation might be that the cytoplasmic signpost had been reversed, thus reversing all of the other asymmetries in one fell stroke. Certainly this remarkable case is otherwise very difficult to explain.

The heterosomata are therefore of exceptional interest. They show the manner in which the CC genotype can differentiate into both pure DD and pure SS genotypes, and thereby produce completely different families of fish; they support the case for the DS genotype producing a racemic mixture of the phenotypes; and they provide a possible case of reversal of the 'cytoplasmic signpost'.

APPENDIX 9.1 WING-FOLDING IN DROSOPHILA MELANOGASTER

A9:1.1 Introduction

Asymmetries in any organism are of interest for the light that they may throw upon human asymmetries, especially that of the preferential specialisation of one cerebral hemisphere for speech (Morgan and Corballis, 1978). It is of importance that no example has ever been described of an asymmetry being inherited according to a simple Mendelian mechanism (Morgan, 1976). Purnell and Thompson (1973) examined wing-folding in a wild strain of Drosophila melanogaster and concluded that it was possible to select, at least for a number of generations, for either of the two possible phenotypes (Right wing over left wing, R/L, or Left wing over right wing, L/R). Wing-folding is of further interest in that it might be regarded as bearing a similarity to human hand clasping, the inheritance of which can be explained by a simple genetic model (see Chapter 9).

It is clearly of little use to try selecting for one of the two phenotypes if those phenotypes are themselves unstable in time. Purnell and Thompson tested for consistency of wing-folding by examining 20 individuals on ten separate occasions and finding that "each individual fly didshow a strong tendency to close the same wing first each time"; detailed statistics were not however given.

As a preliminary to an attempt to replicate the selection experiment of Purnell and Thompson we looked at the consistency of wing-folding, and, having found no evidence of consistency, did not continue with the experiment proper. Purnell and Thompson gave no indication of the method of assessment of wing-folding, or of whether the flies were mobile or anaethetised; we looked specifically for a possible difference between unanaethetised and etherised flies.

A9:1.2 Method

Specimens of Drosophila melanogaster were obtained from a commercial animal suppliers (T. Gerrard, Sussex), and kept in bottles containing 'blue Formula 4.24' culture medium. Two strains were used, white-eyed and wild-type, but no differences were found between them, and results have thus been combined. Virgin flies were collected within 16 hours of ecdysis and the flies etherised. Flies were then placed individually into 50 mm x 12 mm culture tubes, and their wing-folding assessed before the effects of the ether had disappeared. The next day wing-folding was re-assessed in the unanaethetised flies, the individual culture tubes being placed on the stage of a low-power binocular microscope, lighting being laterally from above, and at an almost glancing angle. Each fly was assessed in this way on at least five separate occasions at intervals of at least 24 hours. Some of the flies were then assessed

whilst etherised. For this the flies were kept in their individual culture tubes and ether vapour injected into the tube through the cotton wool plug by means of a hypodermic syringe. After assessment, as above, air was injected into the tube in order to dispel the ether vapour. Each fly was assessed on five separate occasions of at least 24 hours.

A9:1.3 **Results**

140 individual flies were examined. 97 survived sufficiently long to be assessed on five distinct occasions whilst unanaethetised. Table A9.1.1 shows that the vast majority (92/97) showed some inconsistency in wing-folding, and that the distribution was indistinguishable from the expected values for a binomial distribution with p(L/R) = 0.5, (X^2 = 1.96, 4 df, NS). 40 flies were tested whilst etherised, and of these 33 survived for five successive assessments. Table A9.1.2 shows that once again the majority of the flies were inconsistent (27/33), and that the distribution was indistinguishable from a binomial with p(L/R) = 0.5 (X^2 = 0.51, 2 df, NS; groups 0 and 1 and 4 and 5 combined to give expected values of greater than 5.0).

The 33 flies tested whilst etherised had also been tested earlier whilst unanaethetised; the correlation between the number of times a fly folded L/R whilst

unanatthetised and the number of times the same fly folded L/R whilst etherised was only 0.187, which is not significantly different from chance (n=33).

Thirty-seven flies were also assessed for wing-folding whilst unanaethetised and then immediately etherised and re-assessed. Table A9.1.3 shows that even with an interval of only 2 or 3 minutes between the assessments there was no consistency of wing-folding (X^2 = 1.65, 1 df, NS).

A9:1.4 Discussion

There is no evidence from the present analysis for consistency of wing-folding in Drosophila melanogaster. It is thus difficult to know how selection for wing-folding type could be effective. It is possible that the Eversden-14 wild type stock used by Purnell and Thompson shows genetic differences from the present strain, but this seems unlikely. An alternative explanation of the apparent selection found by Purnell and Thompson is that their statistical analysis may be in error. Consider their Table 2. For L/R bias there is a correlation of 0.962, significant at the 0.01 level, between generation number and proportion of L/R flies. However the decision to analyse only generations o-4 out of the 15 possible generations, is clearly ad hoc. It is therefore artificially selecting only one out of 12 possible consecutive groups of five from fifteen (e.g. 1-5, 2-6, etc.). Moreover it is apparent that the decision to analyse five successive

generations was also ad hoc, for in the R/L analysis, eight successive generations were analysed. Under such conditions the significance level quoted is seriously over-estimated, and is almost certainly insignificant in reality This problem is compounded by the impossibility of re-constructing the data in order to re-analyse it, since there is no indication of the number of flies assessed in order to plot each of the points on Figure 1. The hypothesised selection for R/L bias is of the order of 8% over 8 generations, i.e. 1% per generation. To detect such a difference between one generation and the next would require at least 1000 progeny per generation in order to produce a statistically significant difference between successive generations.

A9:1.5 Conclusions

Wing-folding in Drosophila melanogaster is not consistent. The statistical analysis of Purnell and Thompson (1973), who claimed to find evidence of selection for wing-folding type, is probably in error, and hence there is neither evidence for the inheritance of wing-folding, nor for its modification by selection.

APPENDIX 9:2

Code numbers for data points on figures 9.3, 9.4, 9.5, 9.6, 9.7, 9.8, 9.9, 9.10, 9.11 and 9.12.

Code	Study population
1.	Beckman and Elston (1962).
2.	Beiguelmann (1964).
3.	Bonné (1966).
4.	Chattopadhyay (1968).
5.	Dahlberg (1926).
6.	Downey (1926).
7.	Falk and Ayala (1971).
8.	Ferronato *et al* (1974).
9.	Forrai and Bankovi (1969).
10.	Freire-Maia and de Almeida (1966).
11.	Freire-Maia *et al* (1960).
12.	Freire-Maia *et al* (1958) 'Caucasians'.
13.	Freire-Maia *et al* (1958) 'Mongoloids'.
14.	Freire-Maia *et al* (1958) 'Mulattoes'.
15.	Freire-Maia *et al* (1958) 'Negroes'.
16.	Freure-Maia *et al* (1958) 'Indians'.
17.	Frisancho *et al* (1977) Lowland Mestizos.
18.	Frisancho *et al* (1977) Lowland Quechuas.
19.	Frisancho *et al* (1977) Highland Quechuas.
20.	Huizinga (1968) Fali.
21.	Huizinga (1968) Kurumba.
22.	Kawaube (1949).

APPENDIX 9:2 CONTINUED

23. Lai and Walsh (1965) Aborigines.
24. Lai and Walsh (1965) White Australians.
25. Lai and Walsh (1965) Chinese.
26. Lai and Walsh (1965) Indians.
27. Lai and Walsh (1965) Japanese.
28. Lai and Walsh (1965) New Guinea.
29. Lai and Walsh (1965) Phillipines.
30. Leguebe (1967).
31. Lourie (1972) Kurds.
32. Lourie (1972) Yemenites.
33. Lutz (1908).
34. McManus (Survey II).
35. Malhotra (1968).
36. Mascie-Taylor (1978) Personal communication.
37. Pelecanos et al (1974).
38. Pons (1961).
39. Rhoads and Damon (1973) Baegu.
40. Rhoads and Damon (1973) Kwaio.
41. Rhoads and Damon (1973) Lau.
42. Rhoads and Damon (1973) Nasioi.
43. Saldanha et al (1960).
44. Singh and Goel (1975) Khatri.
45. Singh and Goel (1975) Bhaniya.
46. Srnivasan and Mukherjee (1975).
47. Tiwari and Bhasin (1968) Brahmin.
48. Tiwari and Bhasin (1969) Rajput.

APPENDIX 9:2 CONTINUED

49. Wiener (1932).

50. Yamaura (1940) Japanese.

51. Yamaura (1940) Koreans.

52. Yoshiwara (1957)

53. Kawabe (1953).

In the figures a letter 'p' after a number indicates parental population, and a letter 'c' means the child population.

APPENDIX 9:3

As for Appendix 9:2 but for Arm-folding of Figures 9.14 and 9.15.

Code	Study population
1.	Beckman and Elston (1962).
2.	Beiguelmann (1964).
3.	Bonné (1966).
4.	Chattopadhyay (1968).
5.	Falk and Ayala (1971).
6.	Ferronato et al (1974).
7.	Forrai and Bankovi (1969).
8.	Freire-Maia and de Almeida (1966).
9.	Freire-Maia et al (1960).
10.	Huizinga (1968) Fali.
11.	Huizinga (1968) Kurumba.
12.	Leguebe and Martinez-Fuentes (1971).
13.	Lourie (1972) Kurds.
14.	Lourie (1972)YYemenites.
15.	Mascie-Taylor (1978) Personal communication.
16.	Pelecanos (1969).
17.	Pelecanos et al (1974).
18.	Rhoads and Damon (1973).
19.	Srnivasan and Mukherjee (1975).
20.	Wiener (1932).

Table 9:1 Shows, for hand-clasping studies, the number of populations in which males have a higher incidence of LHC, by the population incidence of LHC (greater or less than 50%).

		Pop'n incidence of PHC		
		<0.50	>0.50	
Sex differences in LHC	Male > Female	11	12	23
	Female<Male	3	9	12
		14	21	35

$X^2 = 0.89$, 1 df, NS.

Table 9:2 Family tables for hand-clasping, by sex.

i. McManus: Survey II

		Progeny			
		Right HC		Left HC	
Father	Mother	Male	Female	Male	Female
Right	Right	32	11	31	16
Right	Left	27	11	44	20
Left	Right	30	16	56	18
Left	Left	28	22	54	24

ii. Dr. Mascie-Taylor's Data

Right	Right	14	16	16	15
Right	Left	10	13	21	17
Left	Right	19	19	28	28
Left	Left	8	18	31	28

n.b. For my own study only one child was analysed per parental pair, whilst for Dr.Mascie-Taylor's study all children were included who were old enough for assessment.

TABLE 9.3 Shows optimal allele frequency estimates for 16 populations. Optima were estimated by observing goodness of fi altering C allele frequency at intervals of 0.01. 5% ranges are also shown, except where an asterisk is given, in which the limit is absolute (see text). Data are arranged in order of ascending D allele estimates. All allele estimates are x

Study	D			S			C			goodness of fit of optimal values (2 df)	P	N (chi
	Optimal	5% range		Optimal	5% range		Optimal	5% range				
		Max	Min		Max	Min		Max	Min			
Survey II	0	13.6	0*	21.8	35.3	21.8	78	78*	51	1.150	0.562	44
Terronato et al (1974)	0	14.9	0*	14.5	29.0	14.5*	85	85*	56	0.965	0.617	20
Falk and Ayala (1971)	6.0	15.57	0*	15.9	25.4	9.9*	78	90*	59	0.128	0.938	118
Dr. Mascie-Taylor	8.3	25.8	0*	30.6	48.1	22.6*	61	77*	26	0.224	0.894	301
Wiener (1932)	10.1	22.5	6.59*	3.9	16.4	0*	86	93*	61	2.037	0.361	469
Mai & Walsh (1965): White Australians	11.0	35.0	1.5*	9.9	33.9	0*	79	98*	31	1.229	0.540	198
Neguebe (1967)	17.7	**	**	17.2	**	**	65	**	**	6.311	0.042	912
Cons (1961)	24.1	48.1*	0*	27.8	51.8*	3.8*	48	96*	0*	0.991	0.609	106
Mai & Walsh (1965): New Guineans	23.9	23.9*	30.4	0	6.5	0*	76	76*	63	2.631	0.268	321
Rhoads & Damon (1973)	35.7	52.5	35.7*	0	16.7	0*	64	64*	31	0.099	0.951	257
Freire-Maia et al (1958)	35.9	48.9	24.9*	11.0	24.05	0*	53	75*	27	0.363	0.834	466
Lutz (1908)	41.5	53.0	29.03	21.4	32.9	8.9	37	62	14	0.038	0.981	598
Yamaura (1940)	42.7	55.7*	28.2	31.2	44.2*	16.7	26	55	0*	0.722	0.696	436
Yoshiwara (1957)	42.9	56.4	27.9	28.0	41.5	13.0	29	59	2	1.562	0.457	294
Kawabe (1953)	57.6	62.1	52.6	14.4	18.8	9.3	28	38	19	2.679	0.261	1498
Kawabe (1949)	59.1	60.6	57.1	29.8	31.3	27.8	11	15	8	5.526	0.063	1392

*Limits absolute rather than due to 5% values of X^2

**5% limits not calculable due to optimal fit with $p > 0.05$

Note: C values are in integer units (see text). Values of D and S less than 1 have been rounded down to 0.

TABLE 9.4 Mean allele frequency estimates for population groups.

Analysis of variance after arcsin transformation ($p^1 = \sin^{-1}(\sqrt{p})$).

Differences between geographical groups:- D Allele; $F(2,13) = 7.79$, $p = 0.006$; E vs NJNE $F(1,10) = 4.28$, $p = 0.065$; E vs J $(F1,11) = 12.05$, $p = 0.0052$ J vs NJNE $(F(1,5) = 3.87$, $p = 0.106$ S Allele $F(2,13) = 6.93$, $p = 0.0089$ E vs NJNE $F(1,10) = 7.16$, $p = 0.0232$ E vs J $(F1,11) = 2.35$, $p = 0.152$ J vs NJNE $F(1,5) = 14.44$, $p = 0.0126$ C Allele $F(2,13) = 13.63$, $p = 0.0006$ E vs NJNE $F(1,10) = 0.10$, $p = 0.75$ E vs J $F(1,11) = 24.40$, $p = 0.0004$ J vs NJNE $(F(1,5) = 24.76$, $p = 0.0042$

Allele	European (E) n = 9	Non-Japanese Non-European (NJNE) n = 3	Japanese (J) n = 4
D	0.1319	0.318	0.506
S	0.1811	0.036	0.258
C	0.687	0.645	0.236

Table 9:5 Data on hand-clasping in twins.

Twin type	Study	Pair Type R-R		R-L		L-L		n
Monozygotic	Martin (1975)	9		12		7		28
	Dahlberg (1926)	18		34		17		69
	Total	27	(27.8%)	46	(47.4%)	24	(24.8%)	97
Dizygotic	Martin (1975)	1		11		7		19
	Dahlberg (1926)	34		56		33		123
	Total	35	(24.6%)	67	(47.2%)	40	(28.2%)	142

Table 9:6 Mating patterns for Arm-folding

Mating Type	Ferronato et al (1974)	Leguebe & Martinez-Fuentes (1971)	Dr. Mascie-Taylor	Rhoads & Damon (1973)	Wiener (1932)	Tota
Right-Right	11	70	39	28	25	173
Right-Left	33	140	100	84	44	401
Left-Left	31	109	54	77	34	305
Total	75	319	193	189	103	879

Table 9:7 Arm-folding: Family tables, by sex, of a study by Dr. Mascie-Taylor

		Right AF		Left AF	
Father	Mother	Male	Female	Male	Female
Right	Right	9	12	23	19
Right	Left	14	17	19	14
Left	Right	18	22	22	29
Left	Left	19	14	23	27

For each parental pair, all eligible children were included.

Table 9:8 As for Table 9:3 but for Arm-folding

	D			S			C				
	Optimal	5% limits Max	Min	Optimal	5% limits Max	Min	Optimal	5% limits Max	Min	goodness of fit of optimal values	
Dr. Mascie-Taylor	0	2.5	0*	16.9	19.4	16.9*	83	83	78	4.84	0.0
Wiener (1932)	0	4.2	0*	12.7	16.7	12.7*	87	87*	79	3.43	0.1
Ferronato et al (1974)	0	4.1	0*	13.8	17.8	13.8*	86	86*	78	4.11	0.1
Rhoads & Damon (1973)	1.1	17.6	0*	18.8	35.3	17.8*	80	82*	47	0.89	0.6
Leguebe & Martinez-Fuentes (1971)	1.1	9.5	0*	13.9	22.4	12.9*	85	87*	68	2.02	0.3
Falk & Ayala (1971)	18.1	27.1	8.1	29.9	38.9	19.9	52	72	34	0.16	0.9

Table 9.9 Relationship between handedness and hand-clasping. Including both parents and progeny

i. Survey II

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	545	688	1233	55.80%
	Left	70	99	169	58.58%

$X^2 = 0.36$, 1 df, NS.

ii. Dr. Mascie-Taylor's data

a. All Individuals

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	290	342	632	54.1%
	Left	13	42	55	76.3%

$X^2 = 9.28$, 1 df, $p < 0.005$.

b. Parents only

		Hand-clasping			
		Right	Left	n	%LHC
Handedness	Right	172	181	353	51.27%
	Left	11	22	33	66.66%

$X^2 = 2.28$, 1 df, NS.

Table 9:10 Relationship between handedness and arm-folding (Dr. Mascie-Taylor's data).

		Arm-Folding			
		Right	Left	n	%LAF
Handedness	Right	276	357	633	56.39
	Left	29	25	54	46.29

$X^2 = 1.66$, 1 df, NS.

Table 9:11 Relationship between arm-folding and hand-clasping (Dr. Mascie-Taylor).

		Arm-Folding			
		Right	Left	n	%LHC
Hand-clasping	Right	130	171	301	56.81%
	Left	174	212	386	54.92%

$X^2 = 0.173$, 1 df, NS.

Table 9:12 Linkage of handedness and hand-clasping

The tables consider only mating pairs for which both parents are of the same handedness and of the same hand-clasping type. The Table shows number of progeny in which hand-clasping and/or handedness are the same as the parents.

Survey II

		Hand-clasping		
		Same	Opposite	
Handedness	Same	79	63	142
	Opposite	16	7	23
		95	70	165

$X^2 = 1.047$, 1 df, NS

Dr. Mascie-Taylor's data

		Hand-clasping		
		Same	Opposite	
Handedness	Same	50	65	115
	Opposite	2	4	6
		52	69	121

$X^2 = 0.004$, 1 df, NS.

N.B. For my own survey only one progeny for each parental pair; for Dr. Mascie-Taylor's Survey all progeny included.

Table 9:13 Linkage of Handedness and arm-folding

Data from Dr. Mascie-Taylor's study; otherwise as for Table 11.

		Arm-Folding		
		Same	Opposite	
Handedness	Same	37	72	109
	Opposite	2	4	6
		39	76	115

$X^2 = 0.17$, 1 df, NS.

Table 9:14 Linkage of Arm-Folding and Hand-Clasping

Data from Dr. Mascie-Taylor's study; otherwise as for Table 11.

		Arm-Folding		
		Same	Opposite	
Hand-clasping	Same	9	23	32
	Opposite	21	29	50
		30	52	82

$X^2 = 1.08$, 1 df, NS.

Table 9.15 Shows the goodness of fit of a group of three-allele models to the 16 sets of hand-clasping data. For all models penetrance of D and S is complete (i.e. a = 0.5 and b = 0.5 in Model IV). Each of the three heterozygotes can express in three different ways; as either of the relevant homozygotes (thus in a particular model DC might manifest as if it were DD (or alternatively as if it were CC) or as an intermediate degree of penetrance (symbolized by I in the Table). For intermediate penetrance $p(RHC|DC) = \frac{1}{2}(p(RHC|DD)+P(RHC|CC))$, etc. The next 16 columns show the X^2 value of the optimal fit of the particular model for each of the data sets. The right-hand column gives the total of the 16 X^2 values. Each individual dataset has 2 d.f. and the total has 32 d.f. For the individuals columns any X^2 less than 5.99 is an adequate fit with $p > 0.05$, and for the total column a X^2 of less than 56.66 is an adequate fit with $p > 0.05$. Optimal goodness of fits were determined by finding the minimal X^2 values for all models with the C allele frequency varied in 0.01 steps between 0.01 and 0.99.

Allele Pair Genotype DC	SC	DS	Falk & Ayala (1971)	Ferronato et al (1974)	Freire-Maia et al (1958)	Kawabe (1949)	Kawabe (1953)	Lai & Walsh (1965): New Guineans	Lai & Walsh (1965): White Australians	Leguebe (1967)
Penetrance										
		DD	278.9	53.8	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.9	26.3	10.0	35.8	58.6	19.1	96.4
		SS	120.7	25.8	47.8	78.4	158.4	60.5	24.8	95.6
		DD	0.1	1.0	0.6	3.0	0.1	6.5	1.1	6.9
DD	I	I	0.1	1.0	0.5	5.0	0.7	6.5	1.2	6.3
		SS	0.2	1.0	0.4	12.0	1.8	6.5	1.4	5.6
		DD	28.2	8.3	20.0	34.3	42.4	33.4	2.0	9.1
	CC	I	41.7	9.9	16.6	10.0	30.0	30.9	6.3	21.7
		SS	147.1	12.1	33.7	59.9	116.8	40.1	7.7	89.2
		DD	278.9	63.7	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.9	26.3	9.6	58.6	58.6	19.1	95.7
		SS	199.6	6.1	63.6	78.4	158.4	56.7	23.3	208.4
		DD	0.1	1.0	0.6	3.3	0.2	2.6	0.9	7.3
I	I	I	0.1	1.0	0.4	5.5	2.7	2.6	1.2	6.3
		SS	0.2	1.0	0.3	14.2	6.5	2.6	1.5	5.1
		DD	0.3	0.4	18.0	24.1	24.4	20.5	2.0	9.1
	CC	I	26.3	6.5	9.9	10.0	20.3	18.2	4.2	14.9
		SS	93.0	14.5	44.0	78.4	158.4	42.8	9.0	49.0
		DD	278.9	63.7	58.4	65.3	87.2	87.6	35.0	160.5
	SS	I	123.2	37.1	26.3	9.7	35.8	58.6	19.1	90.8
		SS	0.4	0.9	22.4	74.3	158.4	36.6	2.0	8.9
		DD	1.8	2.6	0.5	2.5	3.9	11.8	1.1	9.0
CC	I	I	0.1	0.9	0.4	6.6	6.5	4.6	1.4	5.6
		SS	0.2	0.9	0.4	17.4	9.8	4.6	1.7	4.4
		DD	0.3	0.4	13.5	19.3	19.3	17.0	2.0	9.1
	CC	I	20.0	5.1	8.3	9.9	19.9	16.5	3.6	11.7
		SS	76.2	16.4	36.6	78.4	132.0	36.8	10.6	37.9

Lal & Walsh (1965): White Australians	Leguebe (1967)	Lutz (1908)	Dr. Mascie-Taylor's data	Survey II	Pons (1961)	Rhoads & Damon (1973)	Wiener (1932)	Yamaura (1940)	Yoshiwara (1957)	TOTAL
35.0	160.5	50.0	64.7	116.1	16.2	35.1	112.4	34.1	23.9	1280.0
19.1	96.4	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	584.7
24.8	95.6	57.2	21.2	39.6	7.3	35.1	67.4	27.9	28.8	896.3
1.1	6.9	0.8	0.2	1.2	1.3	1.7	2.0	1.0	0.4	27.8
1.2	6.3	0.1	0.2	1.1	1.1	1.7	2.0	0.7	1.4	29.9
1.4	5.6	0.1	0.2	1.1	1.0	1.7	2.0	1.5	3.0	39.5
2.0	9.1	19.0	16.0	28.0	4.1	17.2	4.0	7.2	7.2	280.4
6.3	21.7	6.8	7.4	13.5	4.5	14.2	27.6	4.7	5.0	250.8
7.7	89.2	32.9	13.4	36.3	5.3	13.3	50.0	14.9	13.1	685.7
35.0	160.5	50.0	64.7	116.1	17.9	35.8	112.4	34.1	23.9	1291.7
19.1	95.7	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	593.7
23.3	208.4	72.7	0.2	1.4	6.8	34.0	96.8	29.6	25.6	1061.6
0.9	7.3	1.0	0.3	1.2	1.4	0.1	1.9	1.3	0.2	23.5
1.2	6.3	0.0	0.2	1.1	1.1	0.1	2.0	0.7	1.6	26.7
1.5	5.1	0.3	0.2	1.1	1.1	0.1	2.1	1.8	3.5	41.7
2.0	9.1	12.6	12.7	17.2	0.3	10.3	4.0	7.2	4.3	167.4
4.2	14.9	3.7	5.1	9.5	3.5	8.0	17.5	3.5	3.8	164.7
9.0	49.0	44.5	15.9	25.1	4.5	21.4	54.8	20.4	15.9	691.6
35.0	160.5	50.0	64.7	116.1	18.6	35.8	112.4	34.1	23.9	1292.4
19.1	90.8	16.1	20.2	50.0	5.3	23.6	60.3	5.6	6.5	588.0
2.0	8.9	32.3	0.2	1.4	0.2	33.7	7.3	6.8	9.7	395.5
1.1	9.0	0.1	0.4	2.3	1.4	3.6	5.7	0.8	0.7	48.2
1.4	5.6	0.0	0.2	1.2	1.1	1.2	2.3	0.9	2.1	35.0
1.7	4.4	0.7	0.2	1.1	1.2	1.3	2.4	2.2	4.3	52.8
2.0	9.1	9.2	0.3	1.9	0.3	8.0	4.0	7.2	3.2	115.1
3.6	11.7	2.9	3.9	7.3	2.9	7.4	14.7	3.1	3.6	141.0
10.6	37.9	36.1	10.2	19.9	4.5	15.9	45.5	19.4	17.2	599.7

Table A9:1.1 shows, for 97 flies, the number of times out of five that each fly was observed to fold its wings L/R. Flies were not etherised. Expected values under a chance (binomial) hypothesis are shown beneath the observed values.

	n(L/R)					
	0	1	2	3	4	5
Observed	2	17	27	35	13	3
Expected	3.0	15.1	30.3	30.3	15.1	3.0

Table A9:1.2 As for Table A9:1.1, but for 33 etherised flies

	n(L/R)					
	0	1	2	3	4	5
Observed	3	4	9	9	5	3
Expected	1.0	5.1	10.3	10.3	5.1	1.0

Table A9:1.3 For 37 flies the wing-folding type was assessed immediately before and just after etherisation

		After	
		R/L	L/R
Before	R/L	11	5
	L/R	10	11

Figure 9:1 Shows the incidence of Left Hand-Clasping in fifty-three separate studies of different populations.

Hand-Clasping: Population incidences

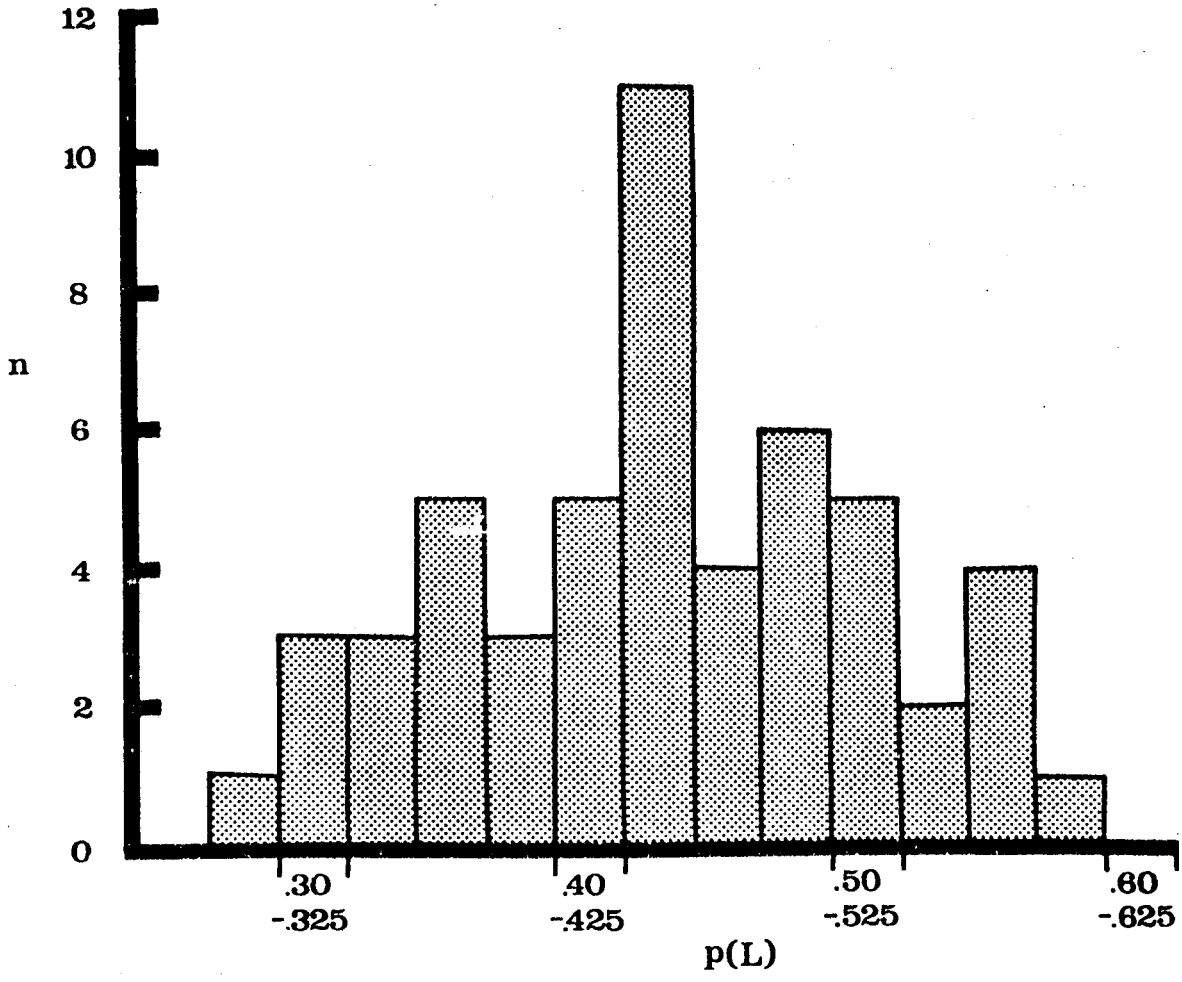


Figure 9:2 Shows a map of the world on which are placed the individual incidences of left hand-clasping in different populations. Values are expressed as percentages. All points are plotted as of the subjects' original home, migrants being plotted as of their sedentes. In a few cases this was not possible for American studies and they have just been plotted at their present homes. Data-points plotted in small brackets are those which cannot be accurately placed due to a poor description in the original apapers. 'WA' refers to White Australians who could not more adequately be plotted elsewhere. The profusion of Indian studies have been plotted in the ocean beneath their country, due to lack of space.

Hand Clasping: per cent Left

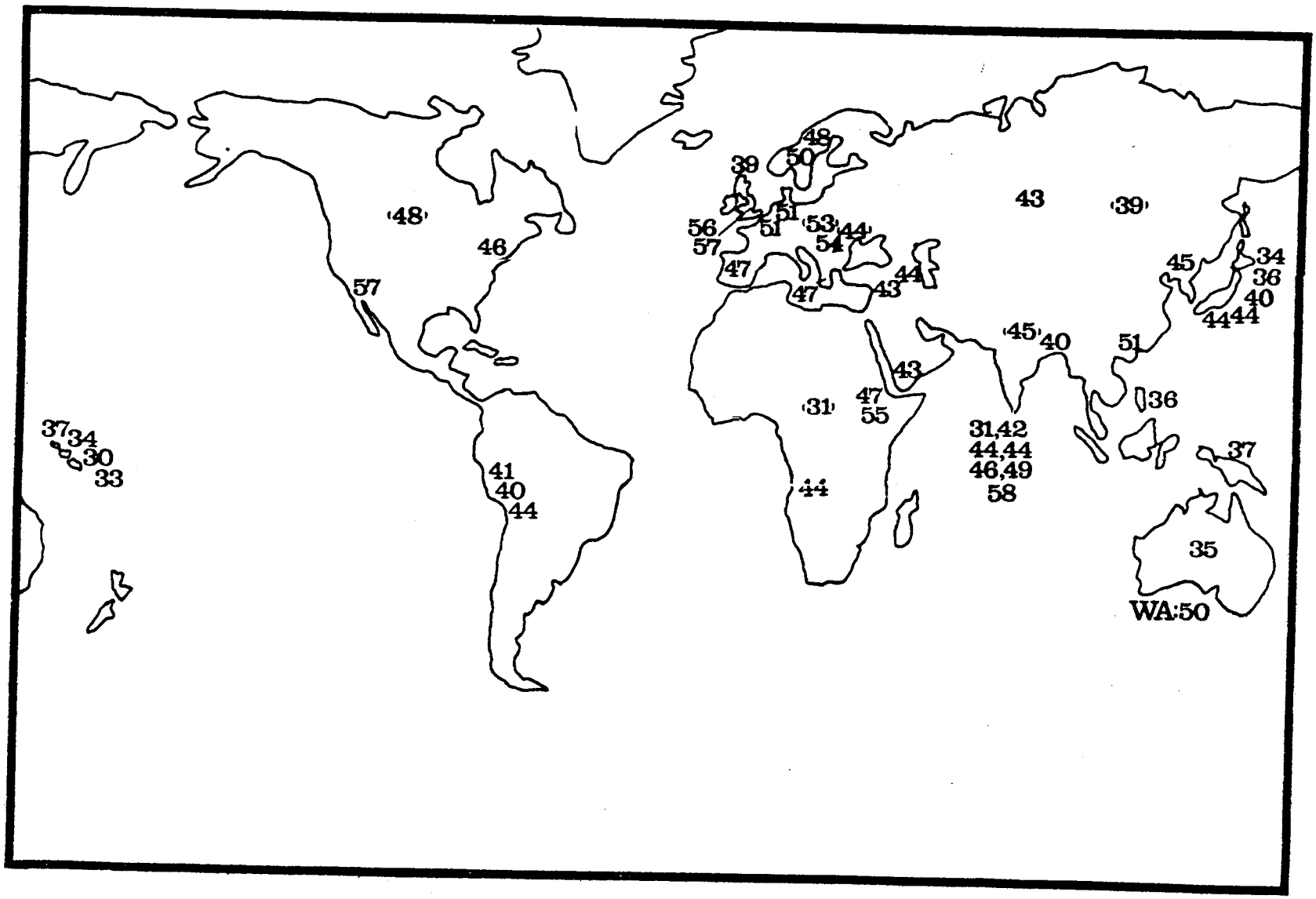


Figure 9:3 Shows for all Eurasian and Australasian studies the incidence of left hand-clasping as a function of the degrees east of Greenwich. Data points are plotted ± 1 standard error. Numbers alongside each point refer to the study from which it was taken (see Appendix 9.2 for key).

Hand-Clasping: Eurasia/Australasia

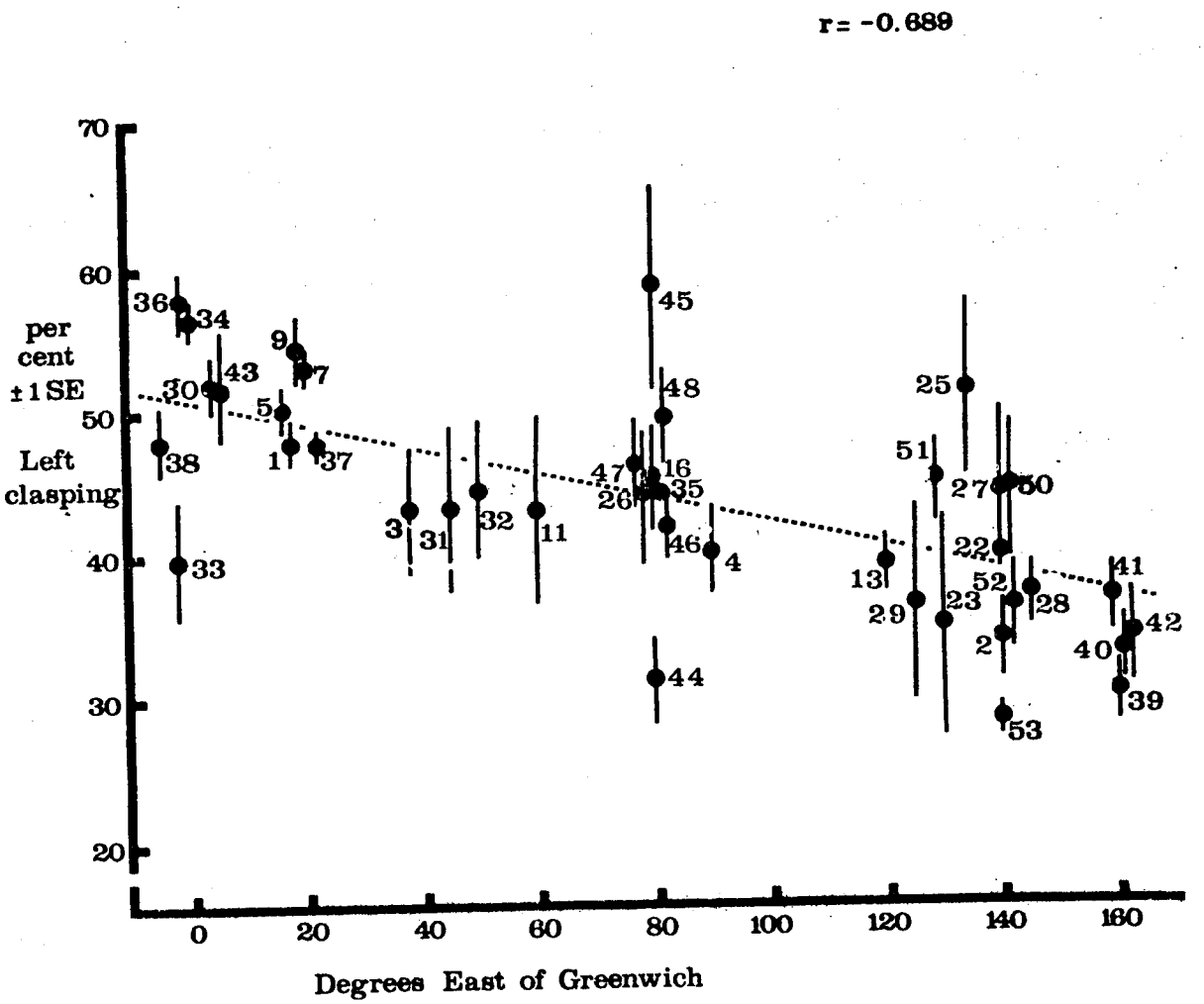


Figure 9:4 Shows the incidence of left-hand clasping in children as a function of the incidence in parents, for 14 studies. Numbers alongside each point indicate the source (see Appendix 9.2 for key). Significance values are placed alongside points in which the incidence in parents and children differs with a p less than 0.10. For some of the studies the incidences are not those in related individuals, but in generations of different age.

Hand-Clasping: Generational differences

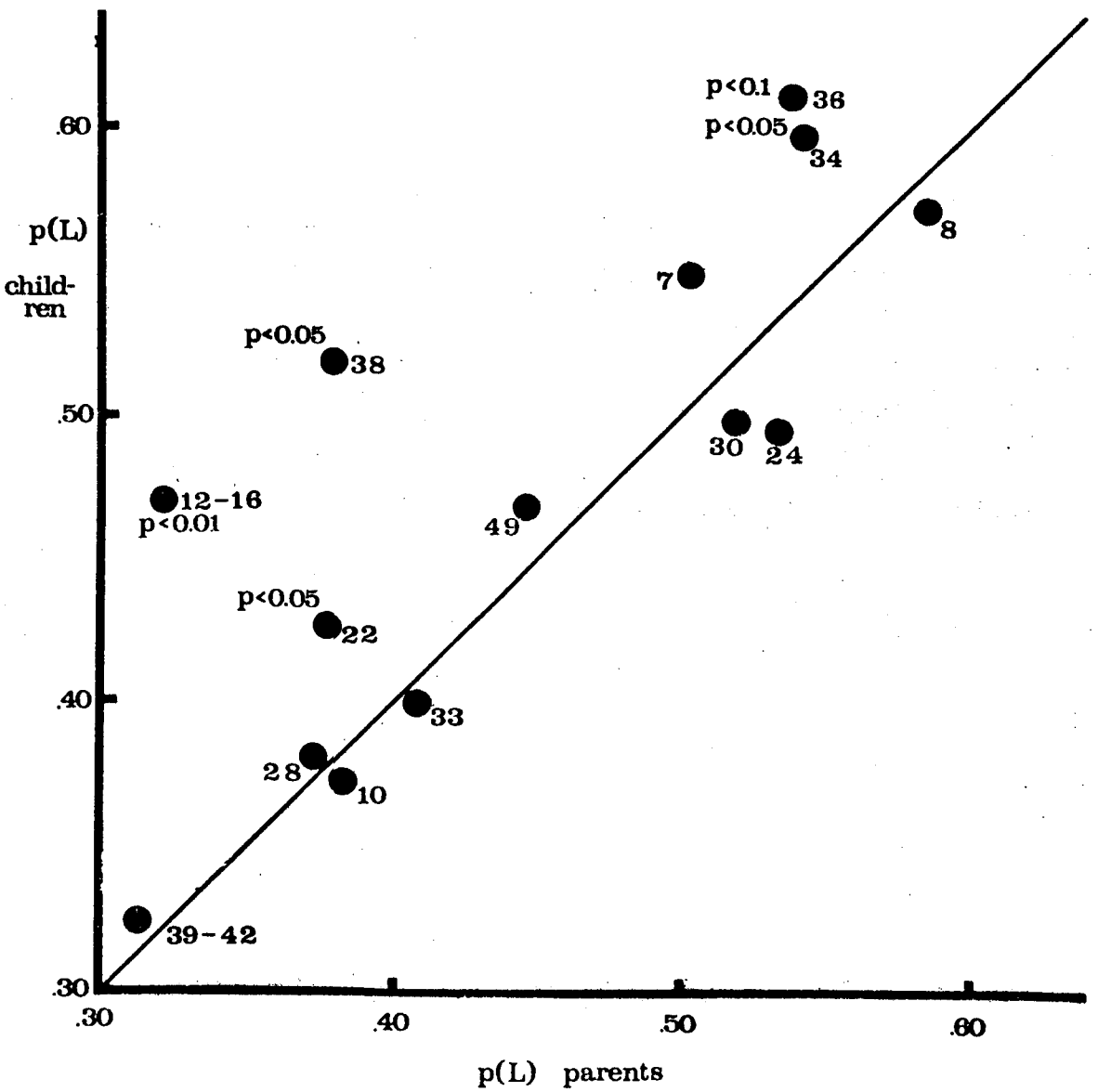


Figure 9:5 Shows the incidence of left hand-clasping in females as a function of the incidence in males, for 36 separate populations. Numbers alongside data points indicate their source (see Appendix 9.2 for key); a subscript 'p' or 'c' indicates parental or child generation respectively. The diagonal line represents the line of equality for male and female incidences

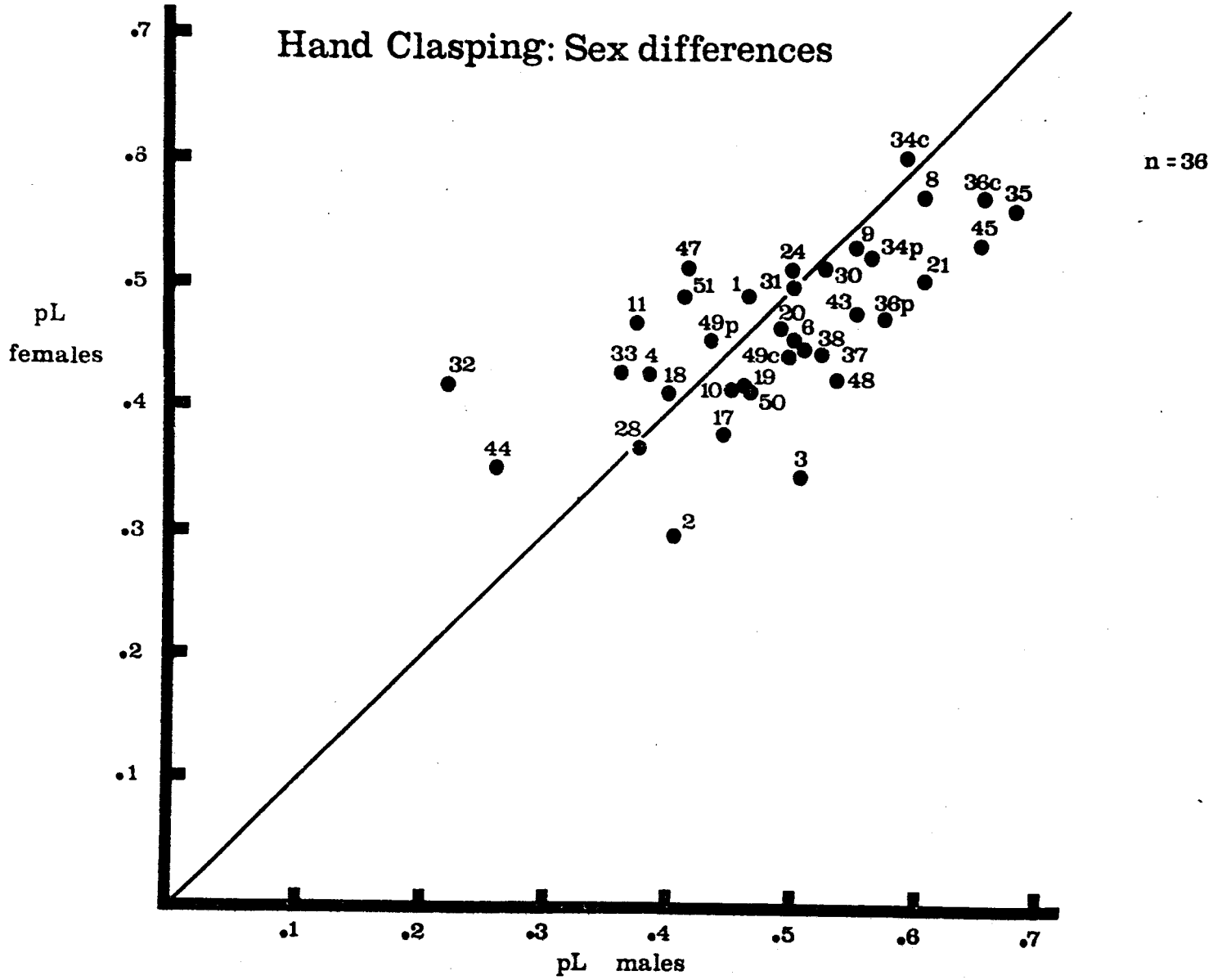

Hand Clasping: Sex differences
n = 36
pL
females
pL males
.1
.2
.3
.4
.5
.6
.7
34c
8
36c
35
45
9
34p
21
47
24
30
51
1
31
43
36p
11
20
49p
6
38
33
4
49c
37
32
18
19
48
10
50
17
28
44
3
2

Figure 9:6 Shows data from fourteen studies on the pattern of parental mating, with respect to hand-clasping type. For clarity only R-L and L-L pairs are shown. Dotted lines indicate the expected incidences under a chance (binomial) hypothesis. Numbers alongisde each point indicate the source (see Appendix 9.2 for key). Data points are plotted ± 1 standard error.
(■) L-L pairs; (▲) R-L pairs.

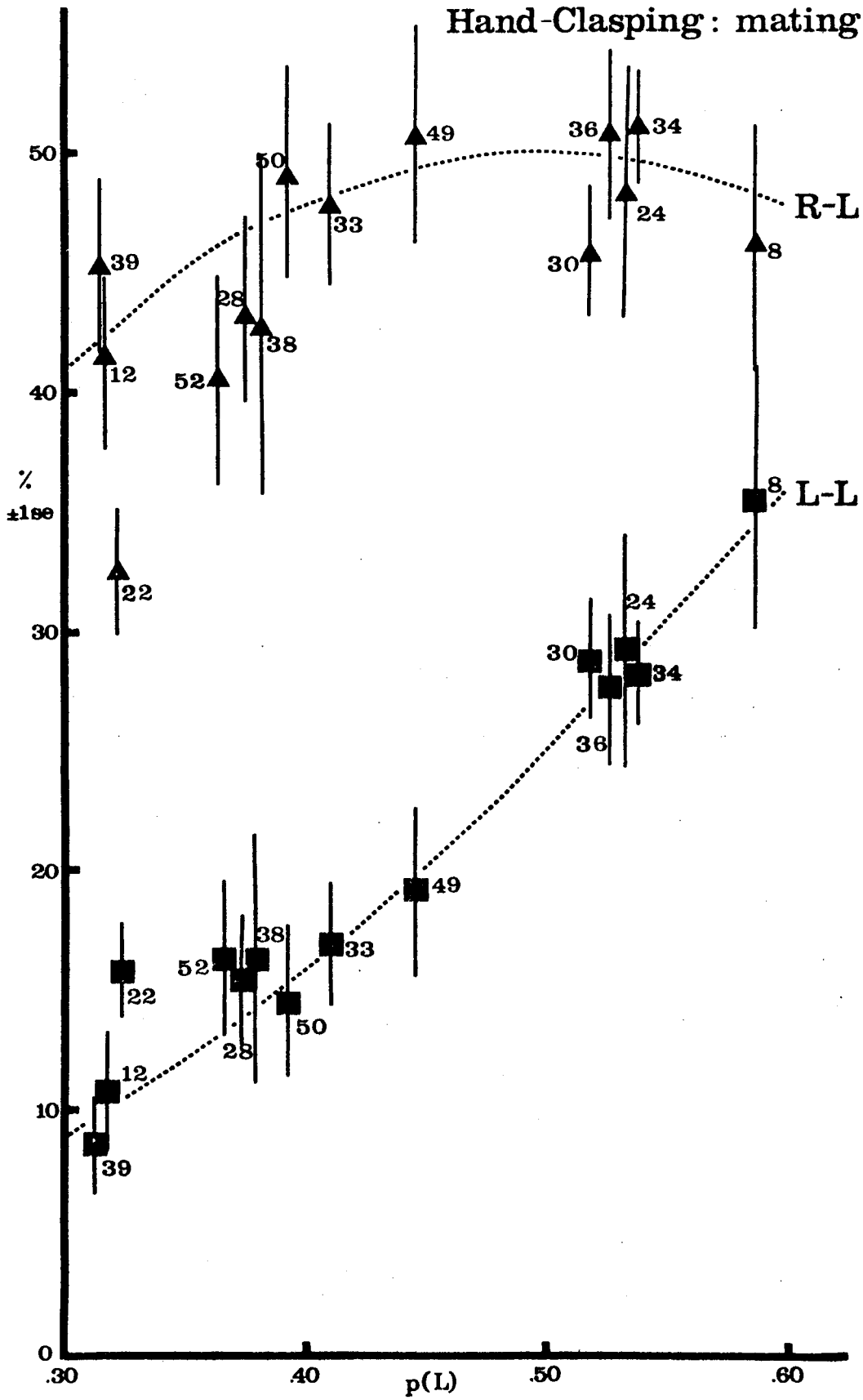

Hand-Clasping: mating
R-L
L-L
%
±1se
p(L)
0
10
20
30
40
50
.30
.40
.50
.60
39
12
22
52
28
38
50
33
49
36
34
24
30
8

Figure 9:7 Shows, for eleven studies, the incidence of left hand-clasping in the progeny of L x R matings as a function of the incidence of left hand-clasping in the progeny of R x L matings. Numbers alongside data points indicate source (see Appendix 9.2 for key). None of the points are significantly different from equality, the line of equality being drawn as solid.

Hand-Clasping: RxL vs. LxR

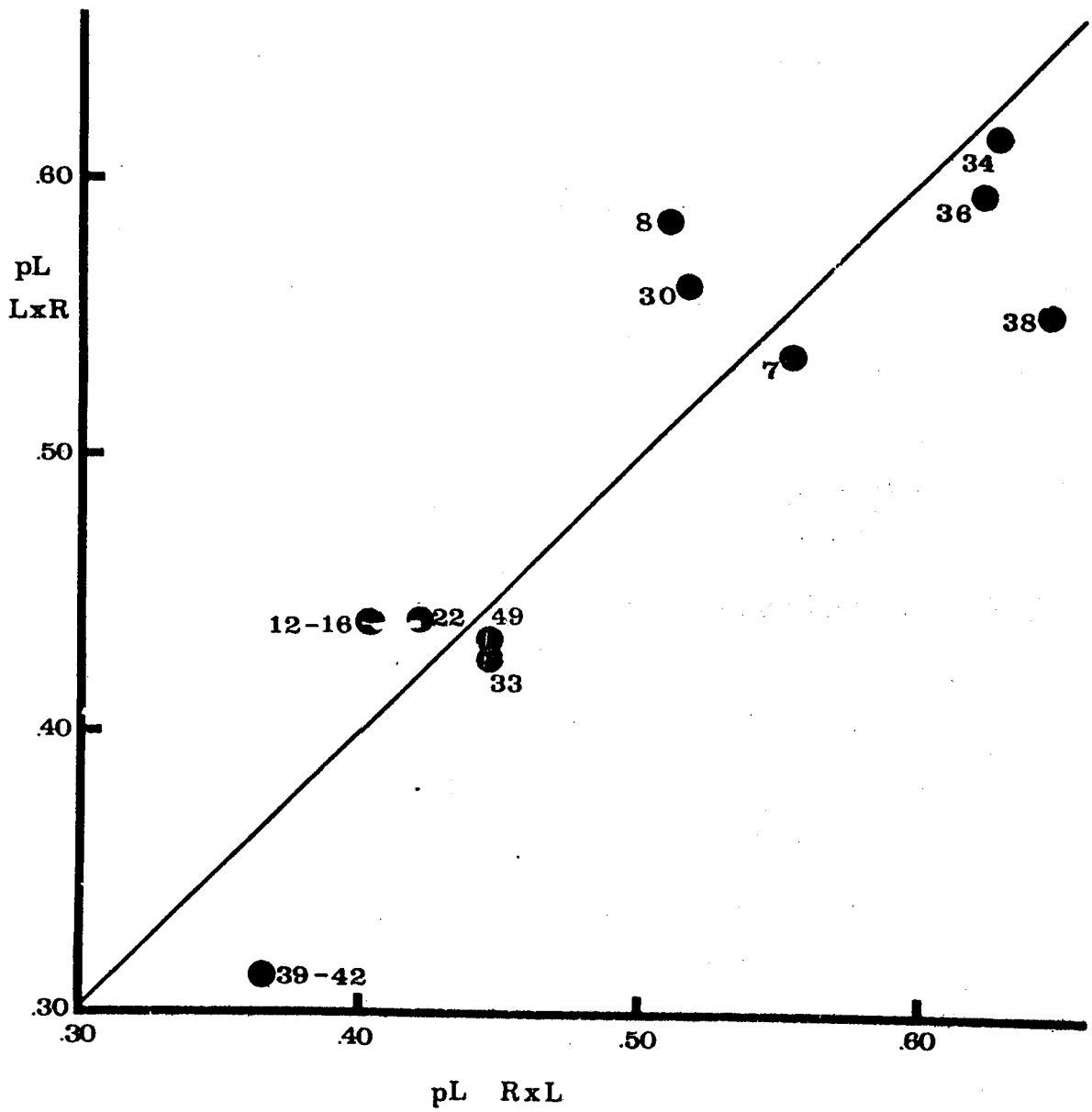


Figure 9:8 Shows the proportion of LHC progeny from RxR (○,●) and LxL (△ ,▲) matings, ± 1 standard error; some standard error bars have been omitted where there is confusing overlap. Japanese studies are shown as solid data points (● ,▲) and non-Japanese studies as open datapoints (○ ,△). p (L) represents the proportion of LHC in the progeny (which does not differ significantly from parental p(L)). For clarity RxL matings have been omitted; in most cases however they are between LxL and RxR, and they have been included when determining the fit of the models. Lines represent the expected values of the best fit two-allele symmetric models described in the text; ------ and ..... for RxR and LxL respectively of Non-Japanese populations, and ——— and _ _ _ _ _ for RxR and LxL respectively of Japanese populations. For key to data-points see Appendix 9.2.

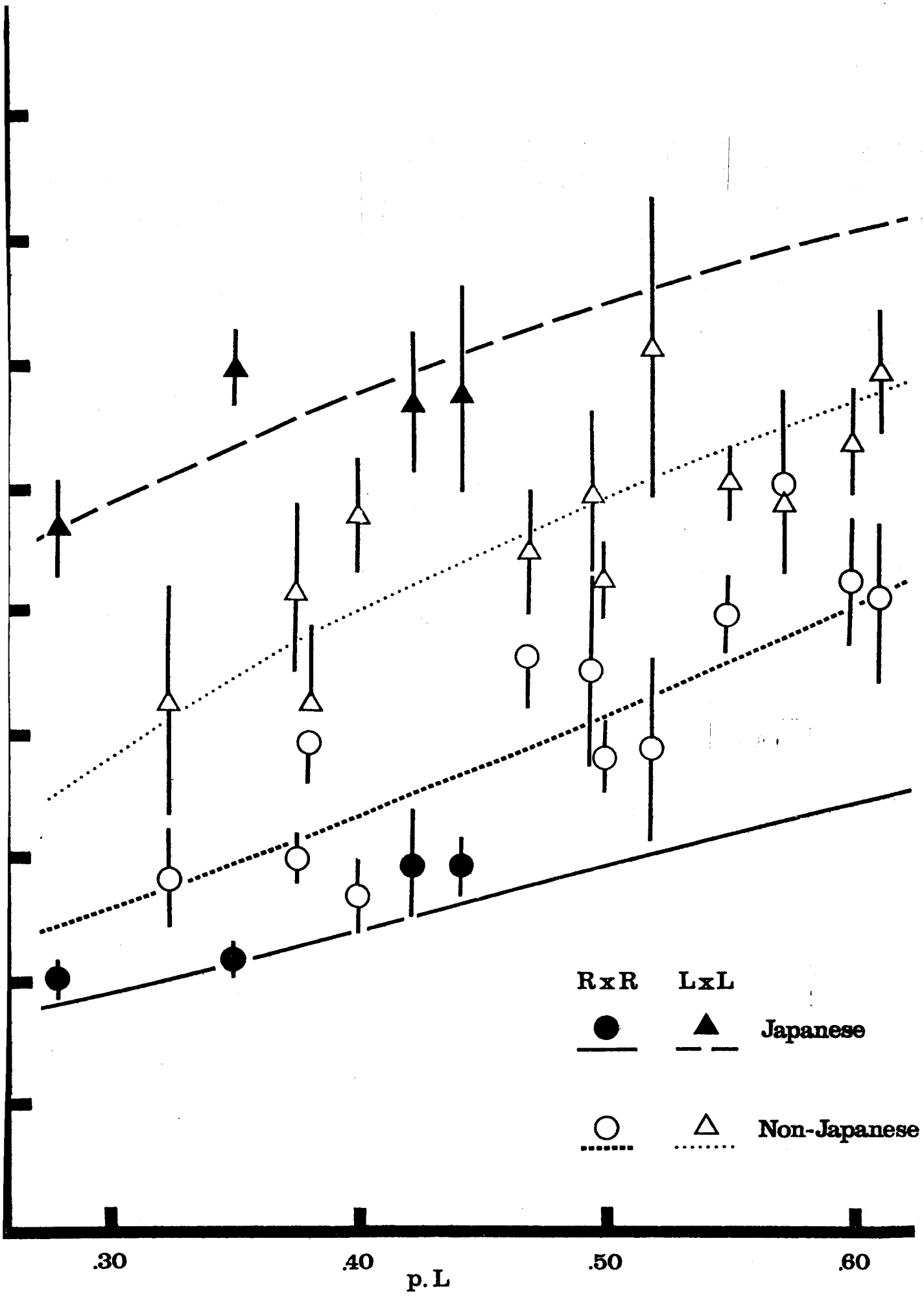

RxR
LxL
Japanese
Non-Japanese
.30
.40
.50
.60
p. L

Figure 9:9 Shows, for twelve non-Japanese studies, the incidence of left hand-clasping in the progeny of RxR matings, R x L matings, and L x L matings. Numbers alongside data points indicate source (see Appendix 9.2 for key). Points are plotted ± standard error. In order to avoid confusing overlap of standard error bars the data for R x L and L x L progeny have been displaced upwards by a scale value of 20%. Solid lines indicate predicted values using a two-allele symmetric model (see text for details). For a clearer perspective of the true relationship between R x R, R x L and L x L see Figures 9.8 and 9.10.

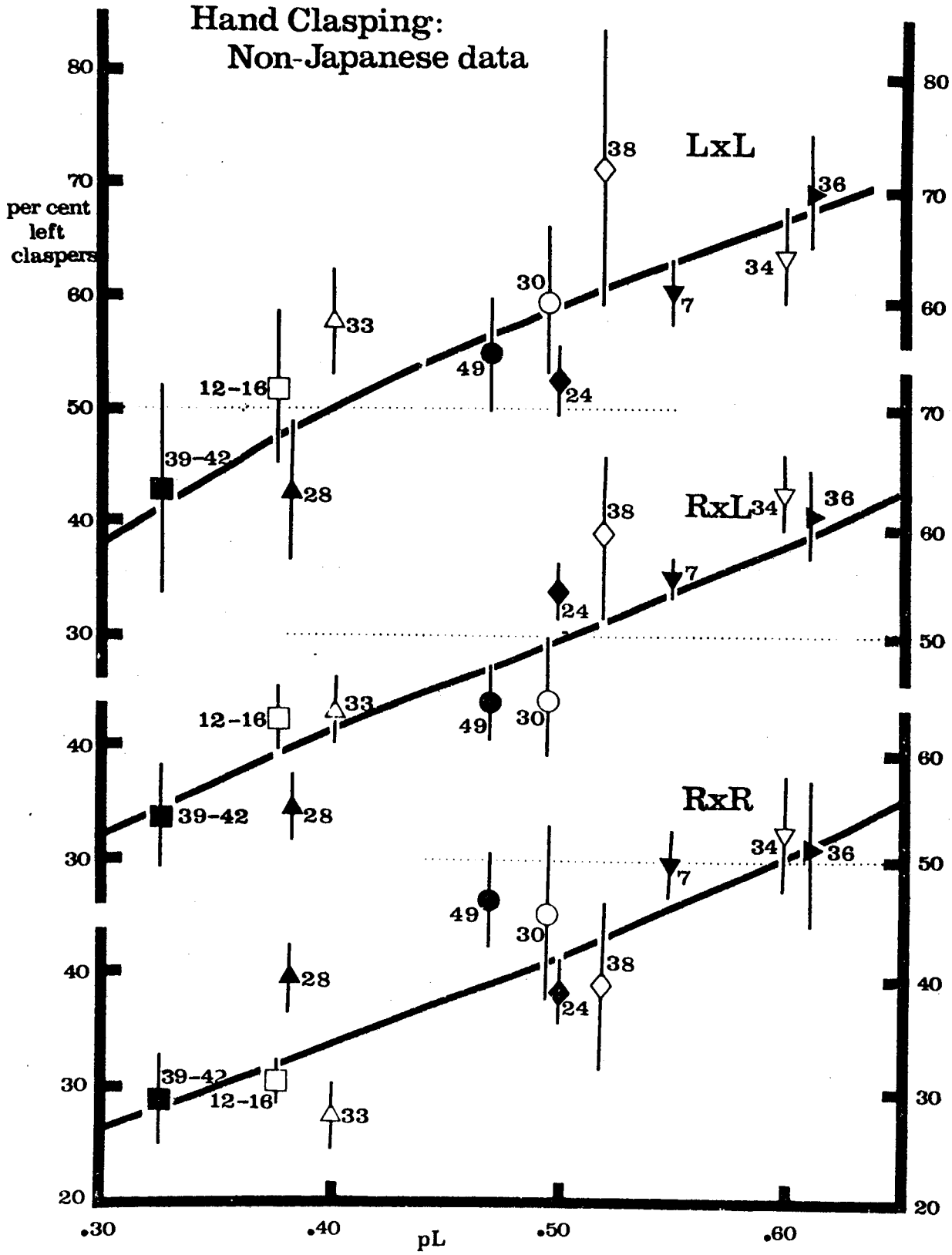

Hand Clasping:
Non-Japanese data
per cent left claspers
LxL
RxL
RxR
pL
80
70
60
50
40
30
20
.30
.40
.50
.60
38
36
34
30
7
33
49
24
12-16
39-42
28

Figure 9:10 shows, for the four Japanese studies, the proportion of left hand-clasping progeny from RxR, RxL and LxL mating pairs. Data points are plotted ± 1 standard error. Numbers alongside datapoints indicate source (see Appendix 9.2 for key). (■) L x L; (●) R x L; (◆) R x R. Solid and dotted lines marked L x L, R x L and R x R represent the fitted lines for non-Japanese studies shown in Figure 9.9 and are present for comparison. Note: unlike figure 9.9 all datapoints in this figure are plotted on the same scale and axes.

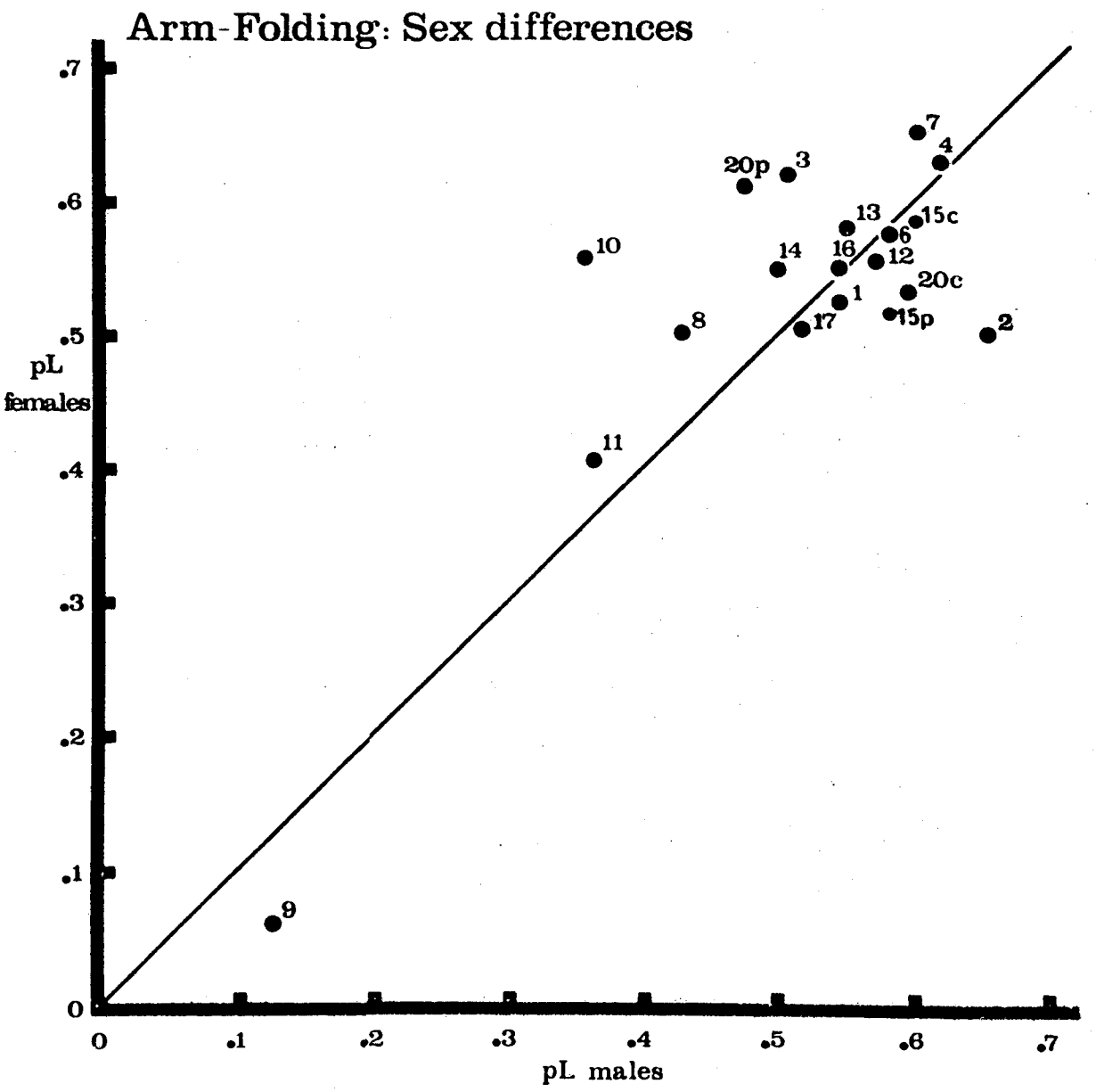
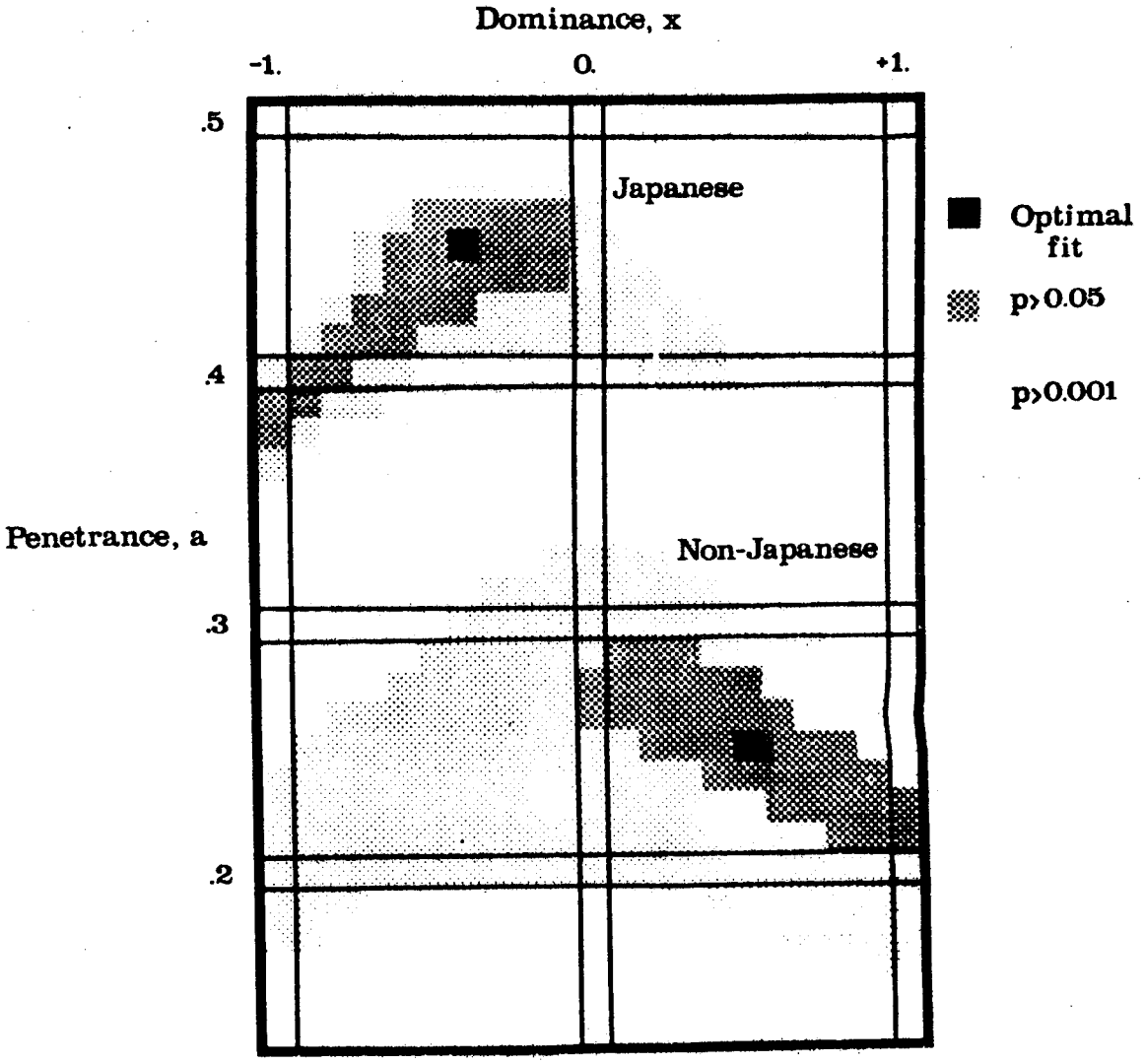

Arm-Folding: Sex differences
.7
.6
.5
.4
.3
.2
.1
0
pL
females
7
4
20p
3
13
15c
10
6
14
16
12
20c
1
8
17
15p
2
11
9
0
.1
.2
.3
.4
.5
.6
.7
pL males

Figure 9:11 shows, for Japanese and non-Japanese data analysed separately, the values of c and z in a two-allele symmetric model which will satisfactorily fit the data. See text for further details

Hand-Clasping



	L	R
DD	.5-a	.5+a
DS	.5+ax	.5-ax
SS	.5+a	.5-a

Figure 9:12 shows the goodness of fit of a three-allele model for the Japanese data with various values of C allele incidence. Numbers alongside curves indicate data source (see Appendix 9.2 for key). A solid circle at the end of a curve means that an absolute limit has been reached (see text for details).

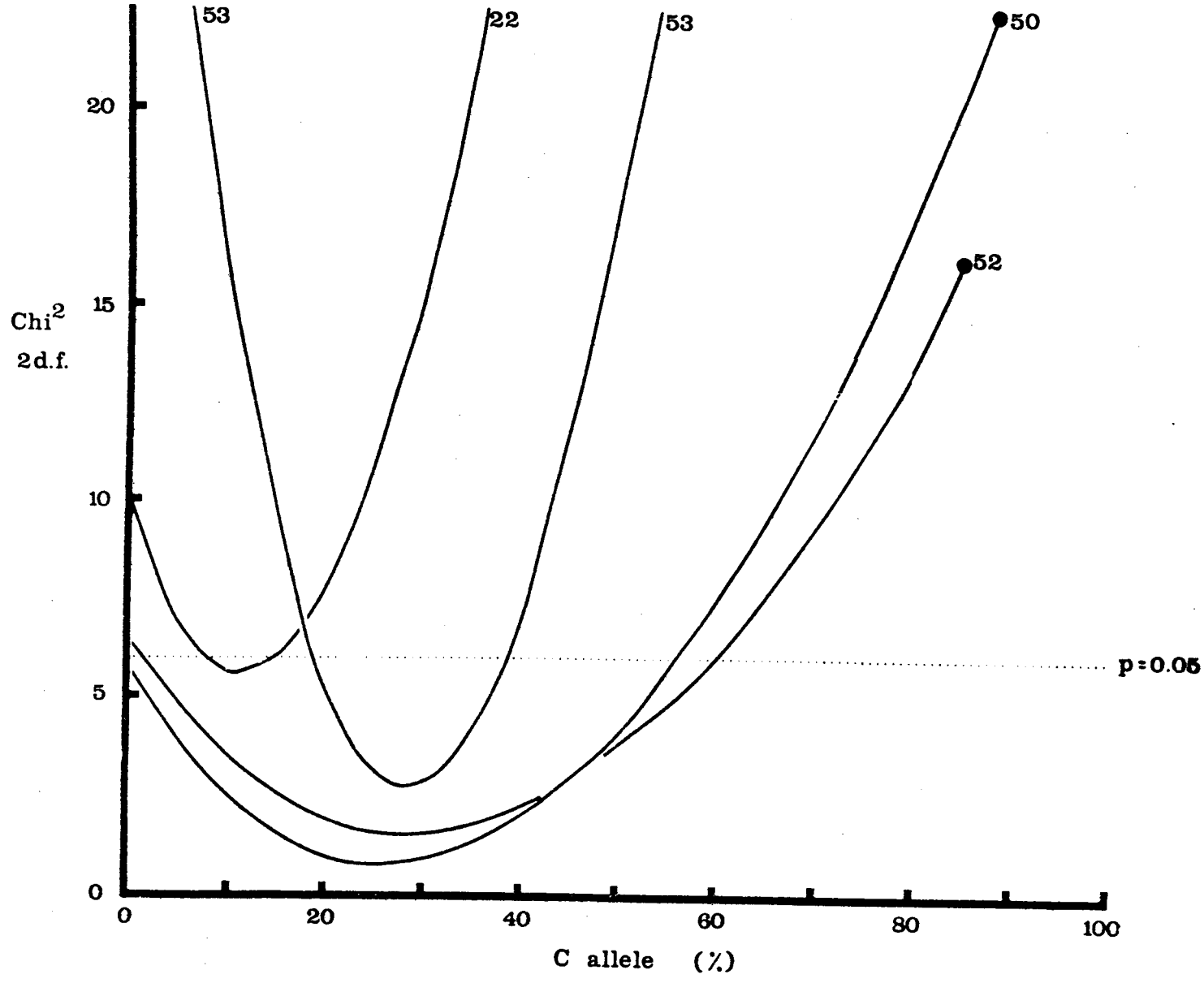

53
22
53
50
52
20
15
10
5
0
Chi2
2d.f.
p=0.05
0
20
40
60
80
100
C allele (%)

Figure 9:13 As for Figure 9:12 but for non-Japanese studies.

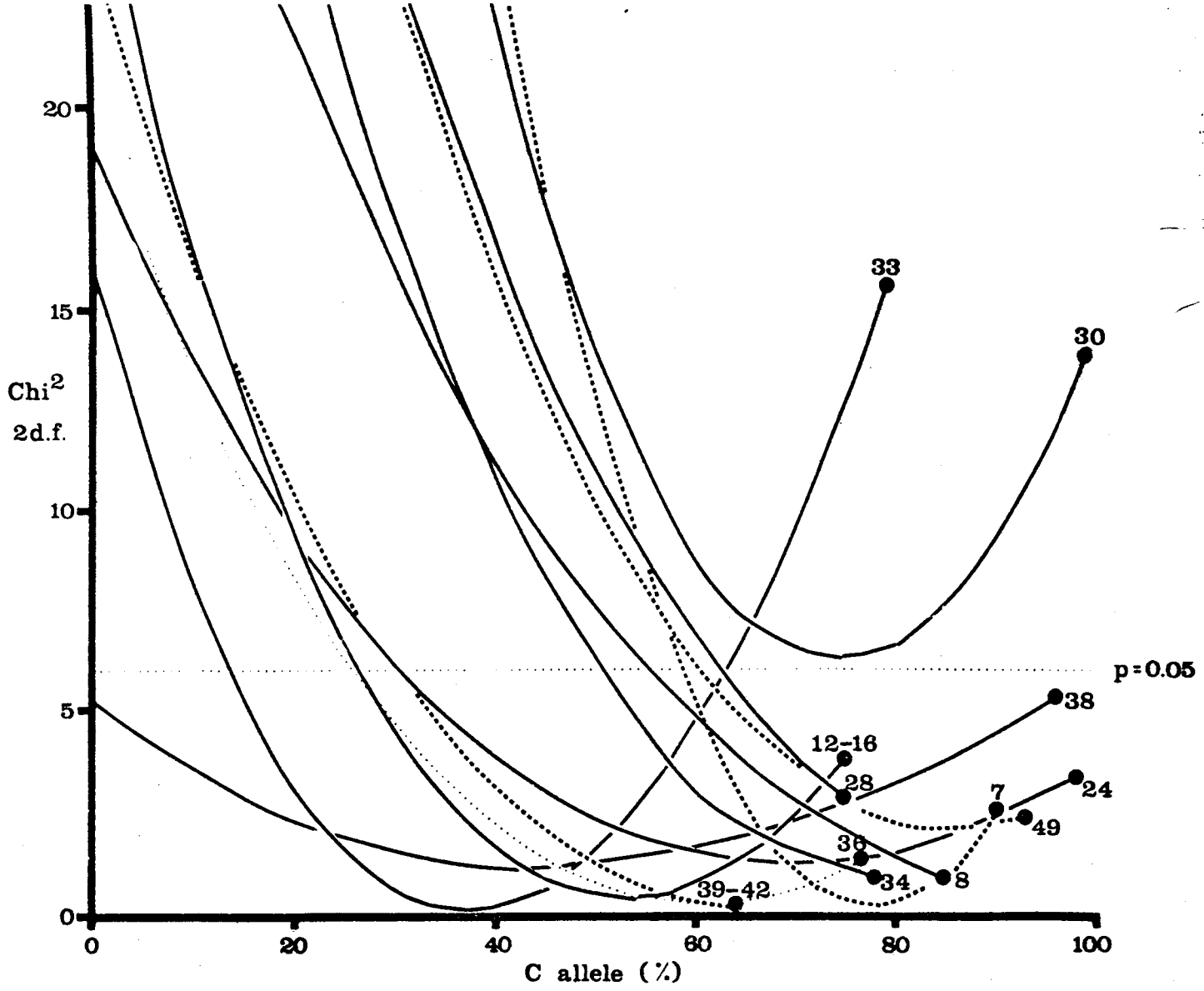

20
15
10
5
0
Chi2
2d.f.
0
20
40
60
80
100
C allele (%)
p=0.05
33
30
38
12-16
28
7
24
49
36
34
8
39-42

Figure 9:14 shows, for twenty-three populations, the incidence of left arm-folding.

Arm-Folding: Population incidences

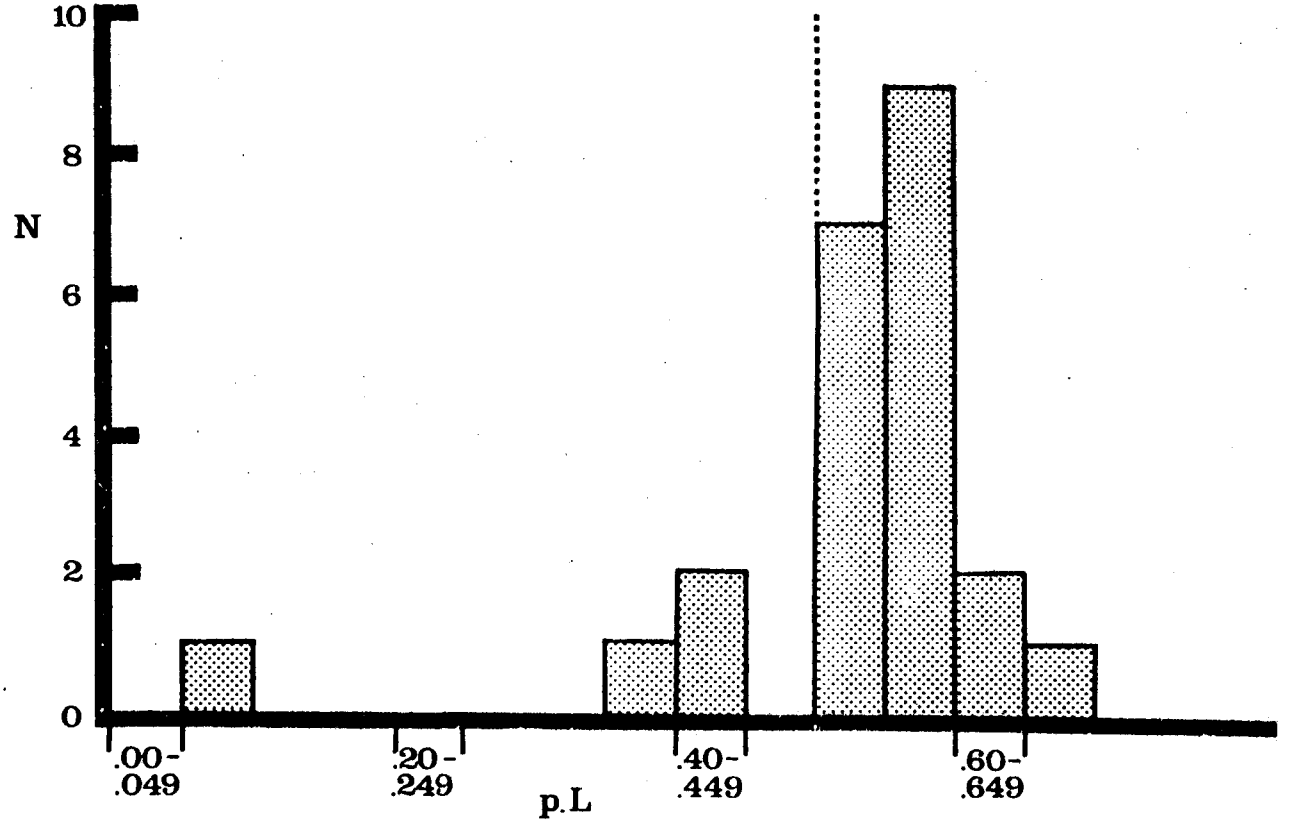
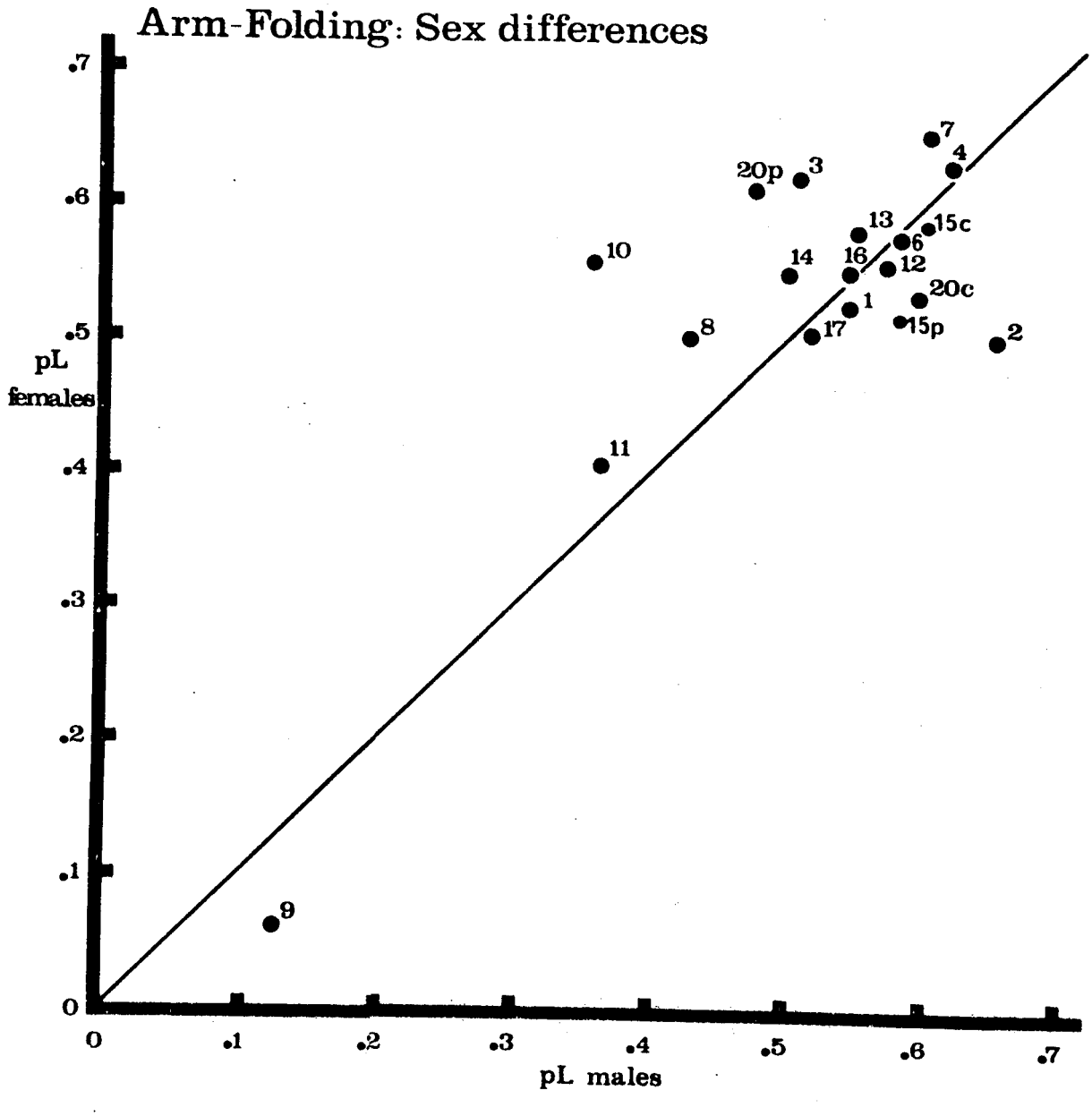


Figure 9:15 shows, the incidence of left arm-folding in females as a function of the incidence of left arm-folding in males, for data from 19 populations. Numbers alongside data points indicate the source (for key see Appendix 9.3).


Arm-Folding: Sex differences
.7
.6
.5
.4
.3
.2
.1
0
pL
females
0
.1
.2
.3
.4
.5
.6
.7
pL males
7
4
20p
3
13
15c
6
10
14
16
12
20c
1
8
17
15p
2
11
9

CHAPTER 10: WHAT IS THE ADVANTAGE OF LEFT-HANDEDNESS?

"His left hand is under my head, and his right hand doth embrace me"

Song of Solomon, ii, 6.

"Birth, and copulation, and death
That's all the facts when you come to brass tacks"

T.S. Eliot, Sweeney Agonistes

10:1 Introduction

In previous chapters I have argued that handedness is determined by a simple genetic system with two alleles at a single locus. There are, I hypothesise, just two phenotypes, right and left-handedness. Given the supposed frequencies of the two phenotypes (about 9.5% and 90.5%), and of the underlying alleles (D = 0.81; C = 0.19) then by necessity we are dealing with a genetically controlled polymorphism. What little evidence there is would suggest that these handedness proportions may well have been constant for fairly long periods of time. Certainly from Ramaley (1913) to the present day, the data may be fitted by an identical genetic model. On a larger time-scale, Coren and Porac (1977) have pointed out that the incidence of left-hand usage in art has remained constant at 7.4% over a five millenia period. Dennis (1958) found a similar proportion of left-hand usage in two Egyptian tombs dated at 2500 BC and 1500 BC. Harrower (1928) and Thomas (1929) discuss similar, although less than reliable, evidence. Prior to the Ancient Egyptian period there is less good evidence of the predominance of right-handedness. Uhrbrock (1973) has reviewed the evidence of Palaeolithic art and concluded that there was an excess of right-handers, although the evidence is far from adequate. Abler (1976) has examined a relatively small number of Palaeolithic skulls and concluded that they show a similar asymmetry to that found by Geschwind and Levitsky (1968) in the planum

temporale of modern brains.

Taken together the above evidence suggests that there has been a balanced polymorphism for handedness for at least five millenia, and possibly for five hundred or more millenia.

Genetic theorists (see e.g. Cavalli-Sforza and Bodmer, 1971) have carefully examined the necessary conditions for the maintenance of balanced polymorphisms. "For a polymorphism to be stable there must be at least two opposing forces acting upon it, and they must be balanced in their effects upon it" (Cavalli-Sforza and Bodmer, p 123). Polymorphisms may be maintained by mutation; this however requires far higher mutation rates than are conventionally accepted and this hypothesis will not be considered further in the present case (although local 'hot-spots' might maintain the occasional polymorphism). The usual explanation of polymorphisms is therefore in terms of heterozygote advantage, and this adequately explains such polymorphisms as sickle-cell anaemia in which although the homozygote, SS, dies at an early age, the heterozygote SA is actually more resistant to malaria than the 'normal' individual, AA; in an area for which there is endemic malaria and hence a selective pressure for SA, the S allele will be maintained in the gene-pool. However there are almost no other human polymorphisms for which the maintaining forces are understood. What does however seem to be clear is that the balancing

forces need bear no obvious relation to the main phenotypes. Thus it has been suggested that schizophrenia is maintained by an increased fecundity of the grand-parents; and phenylketonuria by an increased IQ of the heterozygote (the homozygote of course having a very low IQ). In at least one genetically inherited asymmetry in man, Kartagener's triad (see Chapter 7), there is a major reproductive disadvantage due to sperm immotility, an anomaly which seems to bear little obvious relation to the side of the heart. It is thus quite possible that the relative advantages of the two forms of handedness may bear no relation at all to brain function.

In trying to explain the balanced polymorphism of handedness we must therefore seek at least two selective forces which together maintain the D and C alleles. This search would be simpler if we could identify the genotypes of individuals; this we cannot do as yet, and we must therefore search for selective advantages for phenotypes. To put it crudely, there must be an advantage to being left-handed and an advantage to being right-handed. To simply have both phenotypes (or particularly, to have all three genotypes)of equal fitness would ultimately result, by genetic drift, in the loss of either allele from the gene-pool (chance determining which of the two it should be).

For alleles to have an increased fitness they must

ultimately affect reproductive capacity: that is, the persons concerned must eventually contribute a greater proportion of their genes to the gene-pool than the prior probabilities would suggest. (For a more detailed discussion of quite what is meant by fitness see the Appendix to this chapter).

The literature on handedness is replete with suggestions that right-handers are of greater IQ than left-handers. The issue is however vexed, with several studies failing to find effects (see e.g. Hardyck et al (1976) for a review). I do not wish to go into this "advantage" here, although it may well be that increased intellectual abilities cause the increased fitness of the DD genotype (although such an hypothesis is difficult to reconcile with, for instance, the total lack of social class differences in sinistrality (see Table 3.7 above), for social class has a prima facie correlation with IQ).

Of far greater interest is the potential advantage which must be shown by left-handers (or probably by the DC genotype), which will maintain the C allele . (Note that if the fitness of both DD and CC are greater than that of DC then the polymorphism is unstable, and will ultimately result in fixation of either the D or the C allele). Thus the DC genotype should be the fittest of the three genotypes; since the majority of left-handers

will be DC rather than CC (see Chapter 7), then there must be an advantage of left-handers over right for some characteristics. These fitnesses need only be very small; Cavalli-Sforza and Bodmer point out that fitnesses of the order of 10^{-5} (i.e. the heterozygote is only 0.001% better at reproducing than the homozygote) will result in balanced polymorphisms after several thousand generations.

Several investigators (e.g. Roux, 1977; MacCluer, 1978; Mayo et al, 1978) have pointed out the extreme difficulty, with such potentially small fitness differentials, of actually demonstrating differences in reproductive fitness. Mayo et al (1978) have also pointed out that differences in mean number of progeny may be of less evolutionary consequence than control of the variance of progeny number.

In the rest of this chapter I wish to look directly at data concerning the reproductive fitness of right- and left-handers; the causes of any differences will be left until differences have been demonstrated.

The stimulus to the present study was the close scrutiny of a paper by Hicks and Kinsbourne (1976). In a somewhat confused attempt to discriminate between genetic and environmental hypotheses for the origin of left-handedness, they examined the handedness of

a large number of their students, dividing the students (mean age 20.18 years) into those who had two biological parents, those with only one biological parent (due to death, etc.) and those with one or more step-parents. Handedness correlated better with biological parents handedness than with step-parental handedness. The result is however of little worth since:

a. the ANOVA used was invalidated by the gross bimodality of the Oldfield handedness questionnaire (see Figure 2.3).

b. the students had, on average, lived with their step-parents for only 7.24 years, i.e. they were on average 12.94 years old at the time of the re-marriage; one may presume that adult handedness is fixed by the age of 12, and probably many years before that.

Of far greater interest in the Hicks and Kinsbourne study are the incidences of sinistrality in the parents of various groups. The table below summarises the data:

Parental type	Mothers				Fathers			
	Right	Left	N	% Left	Right	Left	N	% Left
Two biological parents	998	103	1101	9.35%	998	103	1101	9.35%
One biological parent and no step-parent	11	5	16	31.25%	23	4	27	14.81%

Parental type	Mothers				Fathers			
	Right	Left	N	% Left	Right	Left	N	% Left
Step-parent and a biological parent:-								
a. Biological parent	16	11	27	40.74%	56	25	81	30.80%
b. Step-parent	15	12	27	44.44%	60	21	81	25.92%

There is an increased incidence of sinistrality in both mothers and fathers who do not come from a family with two biological parents (Mothers, $X^2 = 62.21$, 1 df, $p < 0.001$; Fathers, $X^2 = 45.11$, $p < 0.001$).

Whilst such data is indirect evidence, I would like to suggest, on the strength of this rather surprising result, that there might be an improved reproductive efficiency in left-handers. This could, perhaps, manifest in many ways: for instance, in terms of a tendency to have more sexual partners, and hence to divorce and re-marry more often; to have more children, and perhaps less miscarriages; (but possibly more therapeutic abortions); to have children at an earlier age, to reach puberty earlier, or to have more diseases associated with sexual activity. The rest of this chapter will critically assess some of the above notions in view of the evidence from data in several surveys. To my knowledge there is almost no relevant data in the literature; although there is a limited suggestion that left-handers tend to be

illegitimate rather more often than might be expected (Pringle 1961).

The little that is known about hand usage in sexual behaviour (Oldfield, 1970) seems to further our knowledge little beyond that of the Song of Songs.

10:2 The number of children in left-handed families

If there is a reproductive advantage in being left-handed then we might well expect to find that left-handers come from larger families than do right-handers; alternatively left-handed matings (i.e. in which one or both parents are left-handed) might have more children than non-left-handed matings. Data to test both of these hypotheses can be obtained from Surveys I and II, and the first hypothesis can be tested using the NCDS data.

Table 10.1 shows the results of two-way analyses of variance of the data of surveys I and II. For the NCDS data it is necessary, in view of the heterogeneity of the sample, to include social class as a controlling variable. Also, since the relevant dependent variable is the household size when the propositus is seven years odl, it is necessary to include propositus parity as an independent variable since many families may not be complete by that stage. Finally, in view of the different incidences of left-handedness in the two sexes in the NCDS

study, sex of propositus is included as a further independent variable. Table 10.2 shows the results of this fourway analysis of variance.

Table 10.3 shows a summary of the relevant analyses of Tables 10.1 and 10.2; the significance levels of the main effects, and the size and variation of the fitted constants for the analysis of variance equation (Multiple Classification Analysis - see Nie et al, 1975). From this latter table it is clear that in Surveys I and II there is no significant tendency for left-handers to come from larger families. The NCDS data does not show a significant effect; however if a one-tailed test is used (since the direction of the effect was specified in advance) then left-handed propositi have a tendency ($p = 0.078$) to come from larger families (an average of 0.05 children greater). Whether this latter effect is truly significant is a moot point. Figure 10.1 shows the NCDS data plotted as a function of social class, parity, and sex. Not surprisingly there is a massive parity effect, and also a social class effect, and a parity and social class interaction, none of which are unexpected.

Examination of the data of Table 10.3 in terms of parental handedness shows a rather different result. In five of the six studies families with at least one left-handed parent have a significantly greater family size, there being a simple unweighted mean effect size of 0.37

children more in left-handed matings, the combined one-tailed probability being 0.0016.

In summary, the NCDS provides limited support for the possibility of left-handed propositi coming from larger families. The data from surveys I and II provides good support for the possibility that left-handed parents have slightly larger families than do non-right-handed parents. Regrettably this latter hypothesis cannot be further tested on the large NCDS data.

10:3 Parental age and handedness

A reproductive advantage may be manifested in several ways other than a simple excess of progeny; if maternal age is relatively less then the generation time will be reduced, and hence relatively more progeny will be produced per unit of time.

Table 10.4 summarises data from the NCDS and survey II on maternal age at the birth of the child. Once more analysis of the NCDS development variable (maternal age) must be in terms of the independent variables social class, parity, and sex of propositus (Table 10.5). There are massive effects of social class and parity, and possibly a small interaction between them, none of which are unexpected. The effect of handedness is not quite significant (two-tailed probability = 0.116), but is in the expected

direction, and thus the one-tailed probability = 0.058. Figure 10.2 shows a graph of the NCDS data as a function of class, parity and sex.

Table 10.6 shows the analyses of variance for Survey II, the mother's age at the birth of the first four children being analysed separately (note that in this analysis, each mother may re-appear in each of the four tables, whereas in the NCDS analysis each mother can only appear once, at a single parity).

Table 10.4 summarises the data of Tables 10.5 and 10.6. It is clear that there is probably no effect of parental type, although the data is only from survey II, and hence relatively small. For both survey II,and II combined with NCDS, there is a highly significant effect of propositus handedness on maternal age; on average the mother of a left-handed propositus will have a child some 0.309 years earlier than the mother of a right-handed properitus (weighted mean of effect sizes).

In summary, there seems to be moderately good evidence that left-handed propositi come from families in which the mother is relatively younger.

Table 10.7 examines, for the NCDS only, the paternal age as a function of properitus handedness. However since normally maternal and paternal age are highly correlated

(r = 0.873 in the NCDS data) is is not surprising that in a simple four-way analysis of variance there is a similar effect as to maternal age. The more interesting question is whether when the relative contribution of the maternal-paternal correlation is partialled out, there is still a paternal age effect. Table 10.7 shows an analysis of variance with maternal age as a co-variate. Now there is not a hint of a paternal age effect; and indeed the class effect is much-reduced and appears to be quadratic in form, a surprising effect. The parity effect remains similar although much reduced in size.

10:4 Interval between marriage and first birth and between successive births

A reproductive advantage might also manifest as a decreased interval either between marriage and the first birth, or between successive births. The NCDS data allows one to examine this question as a function of the propositus handedness (but regrettably, not as a function of parental handedness). Once more, it is the large sample size of the NCDS which justifies this rather oblique approach to the problem.

Table 10.8 shows an analysis of variance in which the interval between marriage and the first birth is the dependent variable, and social class, parity of the present birth, sex of propositus and handedness of propositus are

the independent variables. There is a large class effect. There is also a parity effect, which may probably be interpreted partly as a maternal cohort effect, and partly as an interaction with social class. There is however not a hint of a main effect due to parity, and the fitted constant is actually in the wrong direction. The highly significant H x C x P interaction is probably an artefact of non-normality coupled with a large sample size.

Table 10.9 shows an analysis of variance in which the interval between the previous birth and the present birth is the dependent variable, and class, parity, handedness and sex are the independent variables. Once more there is a highly significant effect of social class, although this seems to be quadratic in form; there is also a parity effect which has a quadratic component (and may well be interpretable in part as a maternal cohort effect). The handedness effect is rather near significance, and, although in the expected direction, is very small.

In summary, neither of these studies show any convincing link with handedness.

10:5 The incidence of miscarriages/abortions/ectopics by handedness

This question may be examined in survey II, and in the NCDS data. Table 10.10 shows a four-way analysis of variance

in which the dependent variable is the number of abortions, miscarriages or ectopics suffered by the mother (a square-root transformation has been used to stabilise variance) and the independent variables are social class, propositus handedness, parity and sex. The parity effect is highly significant (as would be expected). There are no class or sex effects. Handedness shows no significant main effect. The highly significant four-way interaction is almost certainly an artefact of non-normality and may be ignored.

In survey II the mothers were asked to report how many miscarriages they had had. Mothers of right handed propositi reported a mean of 0.458 miscarriages (N = 417, SD = 0.957), whilst mothers of left-handed propositi reported a mean of 0.5020 miscarriages (N = 81, SD = 1.127), an effect in the opposite direction to that predicted, and for which the probability is non-significant.

The data of survey II (but not the NCDS) may be re-analysed in terms of maternal handedness. Right-handed mothers had a mean of 0.504 miscarriages (N = 428, SD = 1.050) whilst left-handed mothers had a mean of 0.428 miscarriages (N = 49, SD = 0.677). The difference, although in the expected direction, is not significant. 28.03% of the right-handed mothers, and 32.65% of the left-handed mothers had had no miscarriages, a non-significant difference.

In summary there is no evidence for a difference in history of miscarriages as a function of handedness.

10:6 Onset of puberty as a function of handedness

A reproductive advantage could also manifest itself as an earlier onset of puberty, and hence an increased (or at least earlier) reproductive span. This may be investigated in several ways in the NCDS data. In all cases it is necessary to analyse by social class, parity (or strictly maternal cohort in some cases) and propositus handedness.

Table 10.11 shows the age of the mother's menarche. There are massive class effects (probably a function of nutritional status) and parity effects (probably a cohort effect, the age of menarche having fallen successively this century). There is not a hint of a handedness effect.

Table 10.12 to 10.15 shows the assessment of the developmental stage of the secondary sexual characteristics of boys and girls as a function of their own handedness. Note that since the survey was carried out at the age of 11, most children are not far into puberty, if at all. Consequently, the distributions are rather skewed, and hence non-normalities may produce spurious interactions in the analysis of variance.

Table 10.12 shows the stage of development of the boys' genitalia. There is a highly significant effect of handedness, although it is in the opposite direction to that predicted. I suspect that this result is spurious.

Table 10.13 shows the stage of development of the boys' pubic hair. None of the main effects are significant although there may be a significant trend in the class data. In particular, in contra-distinction to Table 10.12 there is no effect of handedness (hence my suggestion that the effect in Table 10.12 is spurious).

Table 10.14 shows the stage of development of the girls' breasts. There is a significant parity effect, which is probably not spurious (see below). There is not a hint of a handedness effect.

Table 10.15 shows the stage of development of the girls' pubic hair. As in Table 10.14 there is a significant effect of parity. Whether this main effect is truly one of parity is an interesting question. Do high parity girls really reach puberty earlier? A possible artefact is that these high parity girls inevitably come from older mothers, and since parental income and hence child nutrition, correlate with parental age, these girls might receive substantially better diets. Alternatively there might be interaction effects between children (pheromonal perhaps?). Nevertheless the effect is small. Of

more relevance in the present study is that there are no effects of handedness of girl's sexual development.

In summary, pubertal development does not relate to handedness.

10:7 Summary

There is evidence that left-handers have relatively younger mothers, and that left-handed matings produce more progeny. There is also some evidence that left-handers are more likely to divorce. These differences represent possible advantages for left-handers which may help to maintain the existence of the phenotype.

APPENDIX 10:1 FITNESS AND HANDEDNESS

Lest it is not quite clear what advantages are being proposed, let us consider a formal model, using the notation of Cavalli-Sforza and Bodmer (1971; p 125 *et seq*).

Let the fitnesses of the three genotypes, DD, DC and CC be 1-s, 1 and 1-t respectively. For a balanced polymorphism s and t must both be greater than zero. If $s = 0$, and $t = 0$, then drift alone will eliminate one or other allele; if s and t are > 1, then an unstable equilibrium will eventually result in either one or other allele being lost. If $s > 0$ and $t < 0$, then D will rapidly become fixed; and vice-versa for $s < 0$ and $t > 0$.

Let the frequency of the C allele at equilibrium be 0.19 (i.e. 9.5% true sinistrality). We may then write:

	DD	DC	CC
Fitness:	1-s	1	1-t
p(Genotype|right-handed)	0.724972	0.255083	0.019945
p(Genotype|left-handed)	0.	0.81	0.19

Weighting the genotype frequencies by their fitnesses we obtain:

$$\text{Fitness}_{\text{Right-handers}} = F_r = 1. - 0.7249s - 0.0199t$$

$$\text{Fitness}_{\text{Left-handers}} = F_l = 1. - 0.19t$$

Relative fitness of right-handers with respect to left-handers

$$= RF = \frac{F_r}{F_l} = \frac{1. - 0.7249s - 0.0199t}{1 - 0.19t}$$

It may readily be shown (Cavalli-Sforza and Bodmer, 1971, Eq'n 4.5) that in a stable polymorphism at equilibrium:-

$$\frac{t}{s} = \frac{p(D)}{p(C)}$$

If this equation is substituted into the previous equation then it rapidly becomes clear that RF is 1.0. This is not surprising however, and indeed it is the reason that there is a polymorphism. However this overall fitness is comprised of two components, s and t. From one generation to the next, RF = 1.0. But this does not mean that at particular stages of each generation that RF = 1.0. Only the net effect must be 1.0. If, for instance, we consider a pair of alleles in which, say, AA produces a doubling of the fertility of an organism, but a produces a doubling of the probability of the progeny surviving to reproductive age, then if there were only two phenotypes, these would have an identical net RF; but at particular stages of the reproductive cycle their RF's would vary dramatically.

If we consider the situation when s = 0, i.e. there is a disadvantage to being CC, then it becomes clear that:-

$$\frac{F_r}{F_1} = \frac{1. - 0.0199t}{1. - 0.19t} > 1$$

conversely for t = 0, i.e. a disadvantage to being DD, then:

$$\frac{F_r}{F_1} = 1. \; 0.7249s > 1$$

Thus by isolating components of the reproductive process from one generation to the next, we might possibly find differences between the two phenotypes. But it must be remembered that overall such differences will be statistically difficult to discern.

TABLE 10.1 Shows, for the 6 groups of surveys I and II, the analysis of variance of total family size upon handedness of 'propositus' (i.e. student in propositus generation, or student's mother in the maternal generation, or student's father in the paternal generation).

	I:propositus (student) generation			I:maternal generation			I:paternal generation			II:propositus (student) generation			II:maternal generation			II:paternal generation		
Main Effects	df	F	p	df	F	p	df	F	p	df	F	p	df	F	p	df	F	p
Propositus handedness (right vs left)	1	0.091	0.763	1	1.393	0.241	1	0.885	0.349	1	0.075	0.784	1	0.061	0.805	1	0.091	0.7
Family type (RxR vs 'RxL,LxR & LxL)	'1	0.165	0.685	1	1.106	0.296	1	4.951	0.028	1	6.764	0.010	1	0.291	0.590	1	0.651	0.420
Interaction	1	0.576	0.448	1	0.002	0.966	1	0.580	0.448	1	4.799	0.029	1	0.003	0.954	1	0.097	0.756
Residual	815			97			111			432			419			405		
Total	818			100			114			425			422			408		

Breakdown by sub-groups

Propositus	Family	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N
R	RxR	2.827	1.231	601	2.802	1.769	71	2.847	1.789	85	2.596	1.028	307	2.822	1.601	344	2.853	1.718	342
R	RxL,LxR,LxL	2.741	1.112	116	2.315	2.001	19	3.692	1.974	13	3.114	1.279	61	2.970	1.731	34	3.069	1.907	29
L	RxR	2.718	1.147	64	3.555	3.045	9	2.000	0.925	8	2.860	1.059	43	2.891	1.173	37	2.735	1.399	34
L	RxL,LxR,LxL	2.842	1.127	38	4.000	2.828	2	3.667	3.082	9	2.680	1.600	25	3.000	2.563	8	3.250	1.893	4

Table 10.2 Analysis of variance of total children in household when propositus aged 7 (NCDS I), as a function of father's social class (C), and propositus parity (P), sex (S) and handedness (H).

	df	F	P
Main Effects			
H	1	2.004	0.157
C	4	43.518	$\ll$ 0.001
P	3	1158.427	$\ll$ 0.001
S	1	0.042	0.837
Interactions			
H x C	4	1.077	0.366
H x P	3	1.086	0.354
H x S	1	0.066	0.797
C x P	12	2.531	0.003
C x S	4	0.727	0.573
P x S	3	1.711	0.162
H x C x P	12	0.769	0.683
H x C x S	4	1.656	0.157
H x P x S	3	3.656	0.012
C x P x S	12	0.846	0.603
H x C x P x S	11	0.913	0.527
Residual	10241		
Total	10319		

Multiple Classification Analysis Grand Mean = 2.80

H		C		P		S	
Right	- 0.01	I	- 0.09	0	- 0.53	Male	- 0.001
Left	0.04	II	- 0.12	1	- 0.09	Female	0.001
		III	- 0.03	2	0.69		
		IV	0.11	3	1.47		
		V	0.50				

Multiple R = 0.519

TABLE 10.3 Shows, for surveys I and II and NCDS, the results of a multiple classification analysis of family size as a function of propositus handedness (right or left) and of parental type (R x R or 'R x L, L x R or L x L"). Exact two and one-way probabilities are given for these main effects. The full ANOVA tables are given in Table 10,1 . For the NCDS data, only the p value and MCA result is given for propositus handedness, although a complete 4-way ANOVA table is given in Table 10.2 . (Note that in the NCDS, parental handedness was not known). Combined probabilities have been calculated using the method of Kendall (1951). All MCA results have been adjusted for independents.

	Propositus handedness					Parental handedness				
Survey	Right	Left	Direction	p_{two}	p_{one}	R x R	RxL,LxR,LxL	Direction	p_{one}	p_{two}
I-propositus family	0.001	-0.03	-	0.763	0.618	0.01	-0.04	-	0.683	0.658
I-maternal family	-0.08	0.66	+	0.241	0.121	-0,11	0.40	+	0.296	0.148
I-paternal family	0.07	-0.43	-	0.349	0.825	-0.20	0.86	+	0.028	0.014
II-propositus family	-0.01	0.03	+	0.784	0.392	-0.07	0.28	+	0.010	0.050
II-maternal family	-0.01	0.06	+	0.805	0.402	-0.01	0.13	+	0.590	0.295
II-paternal family	0.01	-0.08	-	0.763	0.618	-0.02	0.23	+	0.420	0.210
NCDS	-0.01	0.04	+	0.157	0.0785					

Combined one-tailed probabilities

Surveys I and II $X^2 = 10.22$, 10 df $p = 0.421$ $X^2 = 24.75$, 10 df, $p = 0.00584$

Surveys I, II and NCDS $X^2 = 15.31$, 12 df $p = 0.224$

TABLE 10.4 Shows, for surveys II and NCDS, the results of an analysis of variance of maternal age at the birth of a child. Results are expressed as a multiple classification analysis, and for surveys II there are in terms of propositus handedness, and family type (RxR vs RxL, LxR, LxL) whilst for the NCDS just the effect in terms of propositus handedness is given; the full analysis of variance is given in Table 10.5 (for the NCDS) and Table 10.6 for survey II. All MCA results are adjusted for all other independents.

Survey	Propositus handedness					Parental handedness				
	Right	Left	Direction	p_{two}	p_{one}	R x R	RxL,LxR,LxL	Direction	p_{two}	p_{one}
II- Child 1	0.08	-0.42	+	0.404	0.202	0.11	-0.46	+	0.364	0.182
II - Child 2	-0.02	0.09	-	0.853	0.573	0.07	-0.29	+	0.626	0.313
II -Child 3	0.46	-3.62	+	0.020	0.010	-0.05	0.16	-	0.095	0 952
II - Child 4	0.49	-2.37	+	0.172	0.086	-0.11	0.22	-	0.289	0.855
NCDS	0.02	-0.20	+	0.116	0.051					

Combined one-tailed probabilities

Survey II	x^2 = 18.42, 8 df,	p =0.0182	x^2 = 6.142, 8 df, p = 0.631
Survey II & NCDS	x^2 = 24.37, 10 df,	p =0.00667	

TABLE 10.6 Shows, for the four parities of survey II, the analysis of variance of maternal age upon handedness of propositus and family type.

Main Effects	II: Child 1			II: Child 2			II: Child 3			II: Child 4		
	df	F	p	df	F	p	df	F	p	df	F	p
Propositus handedness (right vs left)	1	0.697	0.404	1	0.034	0.853	1	5.497	0.020	1	1.894	0.172
Family type (RxR vs RxL, LxR & LxL)	1	0.824	0.364	1	0.238	0.626	1	2.807	0.095	1	1.137	0.282
Interaction	1	0.779	0.378	1	0.113	0.737	0	-	-	0	-	-
Residual	420			383			228			84		
Total	423			386			230			86		

Breakdown by sub-groups		Child 1			Child 2			Child 3			Child 4		
Propositus	Family	Mean	SD	N	Mean	SD	N	Mean	SD	N	Mean	SD	N
R	RxR	28.23	4.53	299	31.07	5.99	270	33.26	5.87	148	34.06	3.73	46
R	RxL,LxR,LxL	27.47	3.55	59	30.82	6.50	57	33.56	7.52	41	35.86	6.96	23
L	RxR	27.38	3.96	42	31.41	5.95	41	34.16	6.97	25	8.11	9.99	9
L	RxL,LxR,LxL	27.75	4.87	24	30.52	6.80	19	33.83	8.53	12	31.66	4.03	6

Table 10.5 Analysis of variance of mother's age at birth of child as a function of child's parity (P), handedness (H) and sex (S), and of husband's social class (C).

	df	F	P
Main Effects			
H	1	2.473	0.116
C	4	75.917	$\ll 0.001$
P	3	973.932	$\ll 0.001$
S	1	0.001	0.989
Interactions			
H x C	44	1.127	0.342
H x P	3	1.864	0.133
H x S	1	1.970	0.160
C x P	12	1.180	0.291
C x S	4	2.922	0.020
P x S	3	0.395	0.757
H x C x P	11	1.499	0.124
H x C x S	4	0.941	0.439
H x P x S	3	4.713	0.003
C x P x S	12	1.336	0.190
H x C x P x S	12	0.995	0.450
Residual	11190		
Total	11268		

Multiple Classification Analysis Grand Mean = 26.87

H		C		P		S	
Right	0.02	I	1.52	0	-2.52	Male	0.0
Left	- 0.20	II	1.20	1	0.35	Female	0.0
		III	-0.33	2	2.92		
		IV	-0.41	3	4.65		
		V	-1.47				

Multiple R = 0.465

TABLE 10.7 Analysis of variance of father's age at birth of child as a function of his own social class (C), and of propositus handedness (H), sex (S) and parity (p). Analysis of covariance is also given, taking maternal age into account.

	Without co-variate			With co-variate		
Main Effects	df	F	p	df	F	p
H	1	0.401	0.526	1	0.328	0.567
C	4	48.298	<< 0.001	4	4.995	0.001
P	3	724.232	<< 0.001	3	45.927	<< 0.001
S	1	1.557	0.212	1	1.503	0.220
Co-Variate	-	-	-	1	1.59×10^5	<< 0.001
Interactions						
H x C	4	2.260	0.060	4	2.237	0.063
H x P	3	2.893	0.034	3	1.460	0.223
H x S	1	4.734	0.030	1	1.949	0.163
C x P	12	1.255	0.238	12	1.679	0.065
C x S	4	1.629	0.164	4	0.560	0.692
P x S	3	0.602	0.614	3	0.621	0.602
H x C x P	11	1.535	0.112	11	1.762	0.055
H x C x S	4	0.934	0.443	4	0.890	0.469
H x P x C	3	4.014	0.007	3	1.286	0.277
C x P x S	12	1.837	0.037	12	1.594	0.086
H x C x P x S	11	1.433	0.151	11	1.049	0.399
Residual	10855			10853		
Total	10932			10931		

Multiple Classification Analyses (adjusted for independents and for co-variate) Grand Mean = 29.94

H	C	P	S
Right - 0.01	I 0.19	0 -0.48	Male -0.04
Left + 0.06	II 0.16	1 -0.001	Female 0.05
	III -0.14	2 0.61	
	IV 0.08	3 0.97	
	V 0.40		

Multiple R = 0.772

Table 10.8 Analysis of variance of interval between marriage and the birth of the mother's first child, as a function of parity of present child (P), social class (C), the sex of the child (S), and the writing hand of the child (H).

	df	F	P
Main Effects			
H	1	0.235	0.628
C	4	27.350	$\ll 0.001$
P	3	126.247	$\ll 0.001$
S	1	1.474	0.225
Interactions			
H x C	4	0.419	0.795
H x P	3	1.364	0.252
H x S	1	0.037	0.848
C x P	12	0.856	0.583
C x S	4	1.207	0.306
P x S	3	2.027	0.108
H x C x P	12	7.535	< 0.0001
H x C x S	4	1.417	0.228
H x P x S	3	0.118	0.949
C x P x S	12	0.887	0.560
H x C x P x S	11	0.641	0.795
Residual	10079		
Total	10157		

Multiple Classification Analysis Grand Mean = 2.20 years

H		C		P		S	
Right	-0.001	I	0.30	0	0.42	Male	-0.03
Left	0.03	II	0.38	1	-0.09	Female	0.03
		III	-0.08	2	-0.53		
		IV	-0.21	3	-0.83		
		V	-0.35				

Multiple R = 0.220

Table 10.9 Analysis of variance of interval between birth of pre child and present child, as a function of husband's social class (C), and propositus parity (P) and sex (S) and handedness (H).

	df	F	p
Main Effects			
H	1	0.187	0.665
C	4	8.306	<0.001
P	3	13.871	≪0.001
S	1	0.554	0.457
Interactions			
H x C	4	0.806	0.521
H x P	3	0.801	0.493
H x S	1	0.514	0.474
C x P	12	0.598	0.846
C x S	4	0.656	0.622
P x S	3	0.707	0.548
H x C x P	11	1.056	0.398
H x C x S	4	2.077	0.081
H x P x S	3	1.026	0.380
C x P x S	12	1.047	0.402
H x C x P x S	10	0.874	0.557
Residual	7123		
Total	7199		

Multiple Classification Analysis Grand Mean = 3.53 years

H		C		P		S	
Right	0.001	I	-0.30	1	0.11	Male	-0.01
Left	-0.03	II	0.01	2	0.15	Female	0.01
		III	0.07	3	0.15		
		IV	-0.04	4	-0.04		
		V	-0.26				

Multiple R = 0.103

Table 10.10 Analysis of variance of number of abortions /miscarriages/ectopics (after square-root transformation to stabilise variance) as a function of husband's social class (C), parity (P), and propositus handedness (H).

	df	F	p
Main Effects			
H	1	0.001	0.981
C	4	1.847	0.100
P	3	33.571	$\ll$0.0001
S	1	0.001	0.978
Interactions			
H x C	4	1.652	0.158
H x P	3	0.365	0.778
H x S	1	1.749	0.186
C x P	12	0.748	0.705
C x S	4	0.087	0.987
P x S	3	0.164	0.921
H x C x P	11	1.520	0.117
H x C x S	4	1.009	0.401
H x P x S	3	3.032	0.028
C x P x S	12	0.901	0.545
H x C x P x S	12	2.808	0.001
Residuals	11195		
Total	11273		

Multiple Classification Analysis Grand Mean = 1.05
(NB. All variables are expressed as square-roots)

H		C		P		S	
Right	0.001	I	0.01	0	-0.02	Male	0.001
Left	- 0.001	II	0.00	1	0.00	Female	-0.001
		III	-0.001	2	0.02		
		IV	0.01	3	0.03		
		V	-0.01				

Table 10.11 Analysis of variance of age at mother's menarche by social class of husband (C), parity of present child (P), handedness of present child (H) and sex of present child (S)

	df	F	p
Main Effects			
H	1	0.018	0.893
C	4	5.588	$\ll 0.001$
P	3	9.121	$\ll 0.001$
S	1	0.062	0.803
Interactions			
H x C	4	1.541	0.187
H x P	3	0.666	0.573
H x S	1	0.379	0.538
C x P	12	1.067	0.384
C x S	4	0.378	0.825
P x S	3	1.582	0.192
H xC x P	12	0.848	0.600
H x C x S	4	0.543	0.704
H x P x S	3	0.405	0.749
C x P x S	12	0.981	0.465
H x C x P x S	11	1.706	0.066
Residual	7667		
Total	7745		

Multiple Classification Analysis Grand Mean = 13.26 years

H		C		P		P	
Right	-0.001	I	-0.16	0	-0.11	Male	0.01
Left	0.01	II	-0.11	1	0.05	Female	-0.001
		III	0.00	2	0.05		
		IV	0.15	3	0.21		
		V	0.06				

Multiple R = 0.083

Table 10.12 Analysis of variance of developmental stage of boys' genitalia by social class of father (C), parity (P) and own handedness (H).

	df	F	p
Main Effects			
H	1	8.702	0.003
C	4	0.236	0.918
P	3	1.044	0.372
Interactions			
H x C	4	1.191	0.313
H x P	3	0.467	0.705
C x P	12	1.309	0.205
H x C x P	12	0.810	0.641
Residual	5311		
Total	5350		

Multiple Classification Analysis Grand Mean = 1.80

H		C		P	
Right	0.01	I	0.01	0	0.01
Left	-0.08	II	0.02	1	-0.001
		III	-0.01	2	-0.04
		IV	-0.01	3	0.02
		V	-0.00		

Multiple R = 0.050

Table 10.13 Analysis of variance of stage of development of boys' pubic hair, as a function of father's social class (C), own parity (p) and own handedness (H).

	df	F	p
Main Effect			
H	1	0.369	0.543
C	4	1.659	0.157
P	3	1.097	0.349
Interactions			
H x C	4	0.539	0.707
H x P	3	1.921	0.124
C x P	12	0.406	0.962
H x C x P	12	0.429	0.953
Residual	5279		
Total	5318		

Multiple Classification Analysis Grand Mean = 1.41

H		C		p	
Right	0.001	I	0.00	0	0.01
Left	-0.01	II	0.01	1	0.00
		III	0.00	2	-0.03
		IV	-0.001	3	-0.00
		V	-0.09		

Multiple R = 0.045

Table 10.14 Analysis of Variance of developmental stage of girls' breasts as a function of father's social class (C), own parity (p) and own handedness (H).

	df	F	p
Main Effects			
H	1	0.085	0.770
C	4	0.351	0.844
P	3	4.043	0.007
Interactions			
H x C	4	0.918	0.452
H x P	3	1.531	0.204
C x P	12	1.363	0.176
H x C x P	12	0.643	0.806
Residual	5041		
Total	5080		

Multiple Classification Analysis Grand Mean = 2.01

H		C		p	
Right	0.001	I	-0.02	0	0.03
Left	-0.01	II	0.03	1	0.02
		III	-0.01	2	-0.06
		IV	-0.01	3	-0.11
		V	0.02		

Multiple R = 0.052

Table 10.15 Analysis of variance of developmental stage of girls' pubic hair as a function of father's social class (C), own parity (p) and own handedness (H).

	df	F	p
Main Effects			
H	1	0.734	0.392
C	4	0.223	0.926
P	3	4.000	0.007
Interactions			
H x C	4	0.742	0.563
H x P	3	3.346	0.018
C x P	12	0.618	0.829
H x C x P	12	0.951	0.494
Residual	5006		
Total	5045		

Multiple Classification Analysis Grand Mean = 1.91

H		C		p	
Right	0.001	I	0.02	0	0.03
Left	-0.03	II	0.01	1	0.02
		III	-0.001	2	-0.06
		IV	0.00	3	-0.10
		V	-0.04		

Multiple R = 0.053

Figure 10.1 Shows the family size (at propositus age 7, i.e. NCDS I) as a function of social class, sex of propositus and parity of propositus.

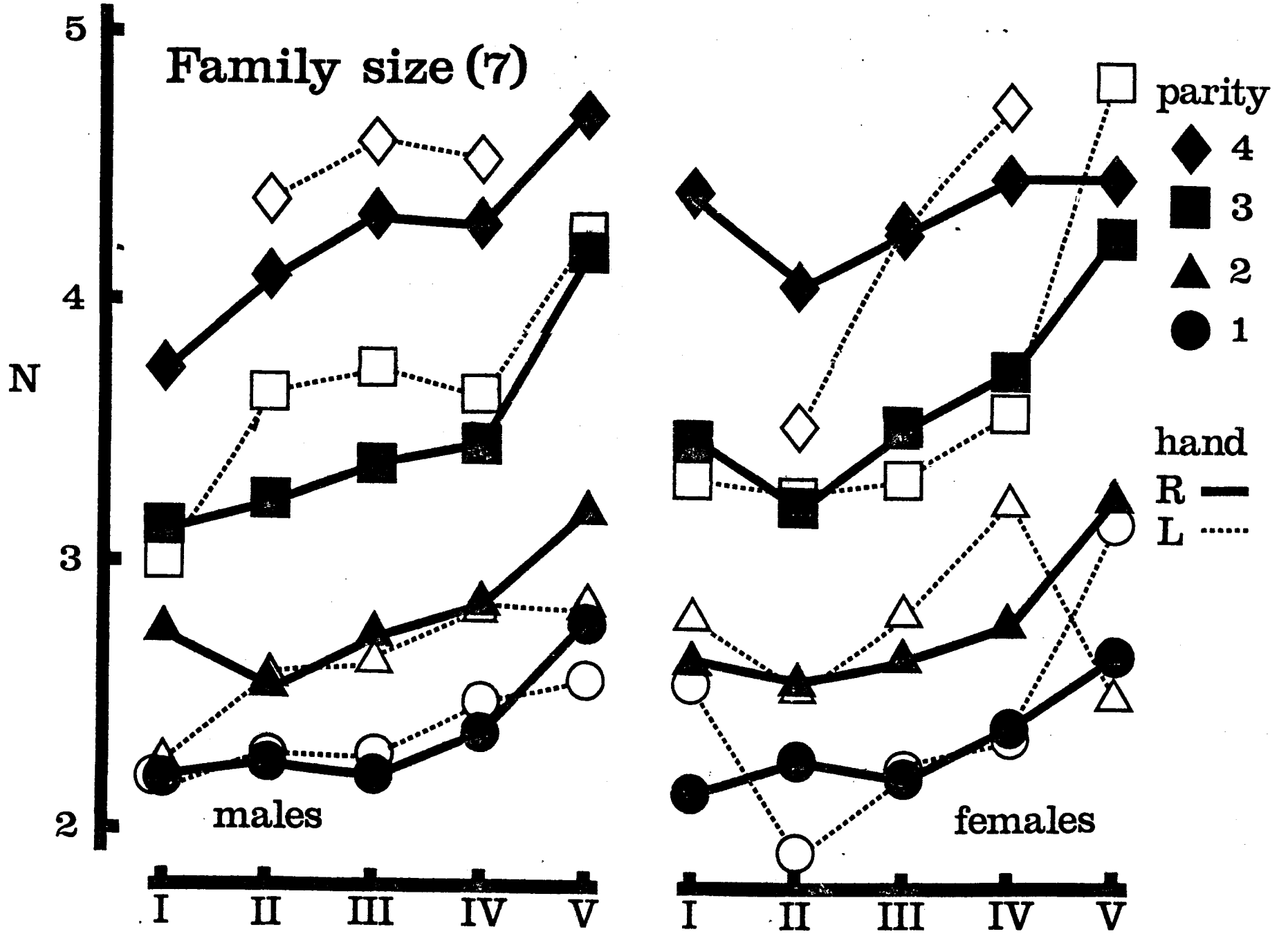
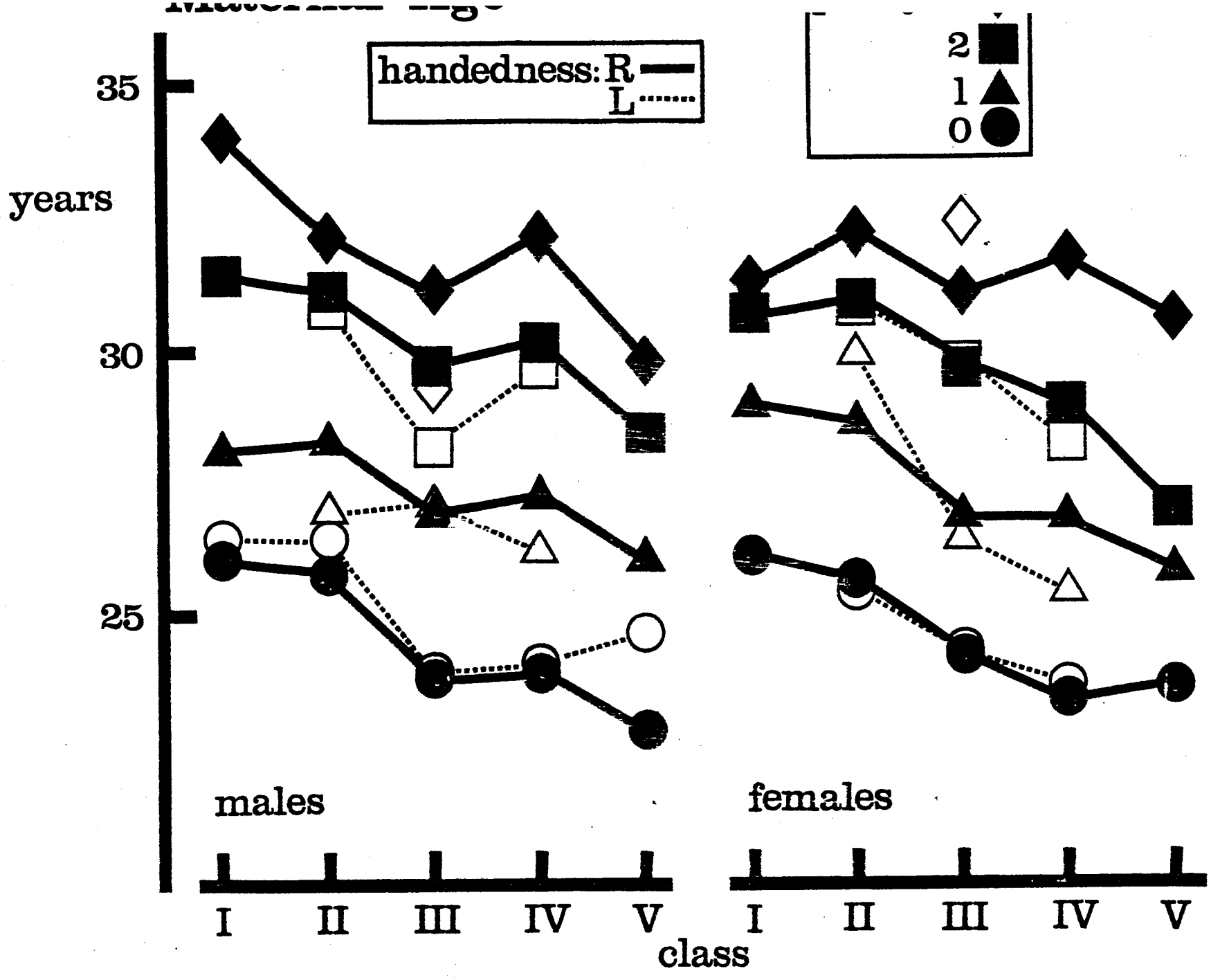

Family size (7)
N
5
4
3
2
males
females
I
II
III
IV
V
parity
4
3
2
1
hand
R
L

Figure 10.2 Shows the mother's age at birth of the propositus as a function of social class, parity, and sex of propositus.


handedness: R
L
2
1
0
35
30
25
years
males
females
I
II
III
IV
V
class

CHAPTER 11: MISCELLANEA ET ESOTERICA CHIROLOGIA

"Nothing oppresses the heart as much as symmetry"

Victor Hugo (quoted Fritsch, 1968, p.87)

"The (narwhale) is some sixteen feet in length, while its horn averages five feet, though some exceed ten, and even attain to fifteen feet ... it is only found on the sinister side, which has an ill-effect giving its owner something analogous to the aspect of a clumsy left-handed man".

Melville, Moby Dick

"Siegfried: And is that heart
in the usual place,
at the left of his breast?

Mime: Of course; dragons
have hearts just like men".

Siegfried, II, 2 (translated by Andrew Porter)

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*Note that in the original German text there is no reference to right or left.

11:1 Introduction

In this chapter I wish to discuss some disparate aspects of the biology of asymmetry. No attempt will be made to link the separate topics together in a logical order, nor to expound any of them in the completeness which they perhaps deserve - they are merely interesting data, or interesting theoretically, and should not be passed by with out mention in a thesis on the biology of laterality.

11:2 Primary and secondary asymmetries; direct and indirect secondary asymmetries.

Man is a bilaterally symmetric organism. However this symmetry is not even skin deep for skinfold thickness is greater on the right side of the body than the left (Damon, 1965). Amongst paired organs there are large numbers of asymmetries, a summary of which appear in Table 11.1. Diseases of the paired organs also show a distinct asymmetry of occurrence, some of which are summarised in Table 11.2. It is hardly necessary to point out that the unpaired organs, particularly the thoracic and abdominal viscera, are grossly asymmetric. All such asymmetries cry out for an explanation; it is simply not satisfactory to say, as did Hass (1951) after reviewing 22,895 cases of congenital dislocation of the hip, of which 14,011 (61.19%) were on the left side ($X^2_1 = 114.8$, $p \ll 0.001$), that "It seems very probable that coincidence plays a

large part".

For some diseases there might well be particular mechanical factors which predispose one side rather than the other. The haemodynamics of the pulmonary circulation (Boyd, 1961) and the particular anatomy of the bronchial tree, (Brock ,1954) probably explain much of the right-sided predominance of primary (Blacklock, 1932) and secondary (Boyd, 1961) tuberculosis, carcinoma of the bronchus (Bignall, 1958), lung abcess (Brock, 1954) and lobar pheumonia (Tugwell and Williams, 1979). Similarly the relation of the iliac vessels to the loaded colon explains the left-sided predominance of ilio-femoral thrombosis (Negus, 1970). The cultural and metaphyiscal associations of 'right' (good, true, clean) and 'left' (bad, false, dirty, sinister) (Needham, 1973) might account for the predominance of hysterical paralysis (Brown-Sequard, n.d.) and conversion reactions (Stern, 1977) on the left, and perhaps also for other conditions such as mastalgia (Preece et al, 1976) as well as for patients' preference for treatments applied to the right side of the body (Ronn, 1976). The mechanics of right-handedness might explain the right-sided presentation of what is actually bilateral Parkinson's Disease (Klawans, 1972). Such explanations can however only form part of an infinite regress and one has ultimately to find an explanation for the asymmetry causing the asymmetries. As a general principle, asymmetric output must be a function

of asymmetric input. We must therefore examine the ways in which asymmetries may be related one to another, in order to find a succinct hypothesis to account for their inter-relations.

Firstly I would like to differentiate between primary and secondary asymmetries. Primary asymmetries are all uncorrelated one with another; secondary asymmetries are correlated. This simple distinction is of the utmost importance as if two asymmetries are uncorrelated it is extremely difficult to explain them by the same genetic mechanism. The asymmetries of handedness and viscera are primary in this sense as they are completely uncorrelated (Torgersen, 1950); hand-clasping and arm-folding may probably be added to this list (although as pointed out in chapter 9:5.4 the relationship with situs is unclear). Handedness and speech dominance are <u>not</u> both primary asymmetries, since they are clearly correlated with one another.

Secondary asymmetries may be direct or indirect. A direct secondary asymmetry is one in which there is a direct causal relation between one asymmetry and another; thus callouses on the hand may be a direct secondary asymmetry to handedness, the more frequently used hand developing the greater callouses. Indirect secondary asymmetries however would be linked only via their genetic control; thus both asymmetries would be consequent to an

earlier cause. The best example of this would be cerebral speed dominance; handedness and speech dominance, in the model described earlier, in Chapter 8, are linked only in so far as they are determinate in the DD genotype and have a random component in the DC and CC genotypes. Neither causes the asymmetry of the other.

Consider the viscera. The heart is almost always on the left side. It is one of the earliest viscera to develop and hence it is probably the primary asymmetry. In situs inversus the position of the heart is reversed; presumably here, as with left-handedness, all directional asymmetry has been removed and fluctuating asymmetry with canalisation forces the tissues one way or the other. In situs inversus totalis not only the situs of the heart but also of the liver, intestines, etc., is reversed. The implication is that these asymmetries are a direct consequence of the right-sided heart, for otherwise we would expect to find 50% of cases of situs inversus with a left-sided liver, etc. Presumably in this particular case there is a haemodynamic or other organising effect which causes the liver to be contralateral to the heart.

Within the details of the embryology of the heart, we may find evidence of direct and indirect asymmetries (see de la Cruz et al, 1969, 1971 for excellent accounts of the embryology). The direction of the apex, the position of the ventricles, and the trunco-conal morphology are

all indirect asymmetries since all possible combinations can and do occur. The position of the atria is directly linked to situs and apex position, since atrical inversion never occurs.

In order to see the relationship of direct and indirect asymmetries more clearly, let us consider the case of varicocoele, which almost always occurs on the left side.

The condition of varicocoele almost always occurs on the left side (Campbell, 1928). If this were directly related to cardiac asymmetry we would expect the incidence of left-sided varicocoele in situs inversus to be 0%; if it were indirectly related we would expect the left-sided incidence to be 50% (fluctuating asymmetry and canalisation); and if it were not secondary to cardiac asymmetry at all we would expect 100% on the left, as in situs solitus. In fact varicocoele in situs inversus is invariably on the right (Grillo-Lopez, 1971), and we may say therefore that there is a direct secondary asymmetry. Conversely consider osteoarthrosis of the hands. This is about 12% worse in the right than the left hand of right-handers, mean scores for the hands being 2.555 and 2.274 respectively (Acheson et al, 1973). Let x be L/(L+R), = 0.4709. If this left-right asymmetry were a result of mechanical stresses we would expect it to be directly linked to handedness and thus the value of x in left-handers should be 1 - 0.4709 = 0.5291, i.e.

12% worse in the left hand. If the asymmetry were unrelated to handedness we would still expect the right hand to be the more severely affected, by 12%. If the asymmetry were indirectly related to handedness then using an additive model of inheritance we may calculate an expected value of R/(L+R) of 0.49005 (i.e. 4.1% worse in the right hand) (for calculations see the table 11.3 where 0.49005 is the value of y). The actual figures for left-handers are right-hand 1.887, left-hand 1.835, a difference of 2.8% with the right hand being more severely affected. Osteoarthrosis appears therefore to be indirectly linked to handedness, perhaps a surprising finding.

We may thus produce a general method of determining the type of control of asymmetry. Let x represent the degree of asymmetry in right-handers with situs solitus. The expectations under the various types of symmetry control are then shown in Table 11.3.

To summarise this section. All tissues show fluctuating asymmetries. Some also show directional asymmetries which may be primary, or directly or indirectly secondary to either of the known primary asymmetries of heart and brain. Tissues may be asymmetric at the organ level (e.g. in overall size) or even at the physiological level (e.g. in the asymmetric ability of sweat glands to concentrate sodium; Gibinski et al, 1971). A method is described for determining the type of the

asymmetry. Regrettably it is not possible to assess most of the known asymmetries in terms of this mathematical model since data on handedness and situs are simply not available. Similarly there is a lack of data on the inheritance of the asymmetries; one notable exception to this is carcinoma of the breast, for which there is evidence that the asymmetry is heritable (see McManus, 1977).

11:3 Hemihypertrophy

A fascinating condition which has not been discussed earlier in this paper is hemihypertrophy (Ringrose et al, 1965). Commonly associated with Wilm's tumours of the kidney, (Fraumeni et al, 1967) it presents simply as an accelerated growth of one side of the body over the other, usually to a degree which is immediately obvious on examination. It is commonly stopped by removal of the associated tumour. The mechanism of the condition raises several interesting questions for theories of symmetry. It implies that it is possible to make the tissues of one side of the body grow faster than those of the other, and that, as a result, tissues of the two sides may not be identical. Since the hypertrophy involves all of the tissues of one side we must assume that a systemic effect is occurring, and that possibly the tumour itself is secreting a 'dextrotrophic' or 'sinistrotrophic' substance; Wilm's tumours have been demonstrated to secrete growth promoting substances (Beierle et al, 1971).

A related condition, found usually in birds (Crew and Munro, 1937), but at least once in man (Brachetto-Brian et al, 1943), is that of hemi-hermaphroditism, in which once side of the body shows male and the other female secondary sexual characteristics. Once more it is tempting to speculate that perhaps cell receptors of one side are more sensitive to testosterone or oestrogen (an hypothesis which should be empirically testable). It also throws some light on the relatively high incidence of conditions in Table 11.2 related to the sexual organs.

11:4 The evolution of biological asymmetry

There are three distinct problems here; the evolution of molecular asymmetry; the evolution of morphological asymmetry in the form of situs; and the evolution of functional asymmetry in the form of handedness and speech dominance.

11:4.1 Molecular asymmetry

The well-known fact that the constituents of organic matter are usually only one of a pair of possible stereo-isomers, was first demonstrated by Pasteur in 1848. In general biological systems use L-amino-acids and D-ribo-sides and D-sugars. Why this should be has provoked much speculation, often with a metaphysical tinge (e.g. du Noily's (1947) claim that it is apparent that God must have

made the first asymmetric molecule - see Gardner, 1967).

Two separate problems arise. Why only one type? And why that particular type?

The evolutionary advantages of building complex systems out of only one of a pair of stereo-isomers are obvious. Imagine the inefficiency of a production line for cars in which each man assembling had beside him a racemic mixture of right and left-hand thread screws which he had to put into pre-threaded holes. Even if a 'wrong' screw were to be forced into a hole with the opposite thread, this would radically weaken the structure. The chemical analogues are readily found in synthetic polymer formation. Blout and Idelson (1956) showed that the formation of Poly-γ-benzyl-(L-/D)-glutamate was far quicker if the substance contained only L or D isomers. Furthermore the polymer became far longer if composed of purely one isomer.

Blout et al (1957) subsequently showed that pure L or pure D polymers were far more stable than polymers composed of both isomers.

The advantages therefore of having only one or other stereo-isomer seem clear. But the advantages of, say, just L-amino-acids are not so clear. One popular argument is that chance alone is the sole determining factor. Thus

one would start off with a racemic primordial soup; the first type of isomer to produce a self-replicating molecule (presumably a rare event) would then have an enormous advantage and would eliminate its competitor. The argument is put nicely by Wald (1957):)

> "I once had the pleasure of discussing this matter with Albert Einstein. He had asked my opinion, and then said, "You know, I used to wonder how it comes about that the electron is negative. Negative-positive- these are perfectly symmetric in physics. There is no reason whatever to prefer one to the other. Then why is the electron negative? I thought about this a long time, and at last all I could think was, 'It won in the fight'". I said, "That's just what I think of the L-amino acids. They won in the fight"" (Wald, 1957).

All such theories however were conceived during that period when it was thought that the physical universe was completely symmetric. In 1957 Lee and Yang received a Nobel Prize for their demonstration of the non-conservation of parity. Recently attempts have been made to suggest that the primordial soup itself might have been asymmetric due to the asymmetric effects of the incident radiation. Garay et al (1974) have recently found that D-amino acids were more sensitive to destruction by β-particles. Kovacs and Garay (1975) showed that the precipitation rates of the two isomers of tartaric acid were different in the presence of non-polarised β-irradiation. Bonner et al (1975) found an asymmetric degradation of the stereo-isomers of leucine with radiation. Norden (1977) showed that a racemic 'soup' of amino-acids became asymmetric

after only 60-80 hours of exposure to polarised light.

The implication of these experiments seems clear. The primordial broth may well have been asymmetric. But since, in no sense, could it be construed as consisting entirely of one isomer, or the other, all that happens is that we must set the a priori probabilities of, say, L-amino-acids, not at 0.5 but at some higher value less than 1. Nevertheless whilst chance may well still play a large role, it is possible as Morgan (1976) has suggested, that our basic asymmetries are a consequence of the inherent asymmetries of sub-atomic matter.

11:4.2 The evolution of Situs

The predominance of morphological asymmetries in the higher animals implies that there must be an advantage to such asymmetries. But as with optical asymmetry, we can conceive two separate questions; why is the system asymmetric, and why does it have this particular asymmetry?

The need for asymmetry of some sort is probably simple in the higher animals. When an organism is bilaterally symmetric then there reaches a stage of complexity at which it is no longer practical to control symmetric effectors. Thus imagine a vertebrate with two mirror-image hearts driving blood through symmetric arteries and veins. Before long, unless there were perfect synchrony between

the sides, the system would become out of phase, and one would find, for instance, that the systole of one heart would be opposing the diastole of the other, and exceptional pressure fluctuations would occur. Of course this could be avoided by having totally separate blood circuits; but then the immunity of one circuit would not transfer to the other side, with an obvious decrease in resistance to infection. The problem of running two hearts would be akin to the modern problem of knowing how to run computers with more than one central processing unit; the CPU's interact in awkward, unpredictable and unpleasant ways.

Once one accepts that it is preferable to have only a single, asymmetric heart it is obvious that it should, if possible, always be on the same side in different individuals. To return to the motor-car argument of the previous section, imagine a production-line producing both right and left-hand drive cars, the order of appearance being random. Each assembler will have two boxes of mirror-symmetric parts, each to be welded on to mirror-image symmetric positions. Clearly it would not be long before cars began to appear which had some right and some left-components; and before long such cars would reach a point in the production line beyond which they could proceed no further. The biological analogy of this process is what Waddington (1957) has called 'selection for repeatability'. It is better than the system has only

one known, and predictable, asymmetry, which it may then modify, and build upon, than that it should have the potentially increased variance due to two mirror-image phenotypes, but which will allow far more disasters to occur during development and modification. To sometimes have to turn right at an un-sign-posted junction, sometimes left, is a sure manner of losing one's way; better always to turn left, even if it means a detour, before certainly arriving at one's destination.

But such arguments do not justify a particular asymmetry. It is difficult to know what are the advantages of, say, a population with left-sided hearts, as opposed to a population with right-sided hearts. Once more, chance may be the major factor: the left-sided heart 'got there first'. The only moderately clear case of one of enantiomorphic phenotypes having an advantage, is that of the snail, Fruticola lantzi, in which the more common dextral form withstands starvation conditions far better (Gause and Smaragdova, 1940). It must of course be remembered that the selective advantage of an asymmetric phenotype may well have nothing to do with the asymmetry per se. Consider the case of Kartagener's Triad, in which as well as situs inversus (or rather, fluctuating asymmetry) there is cilial dysfunction resulting in sinusitis and bronchiectasis. There is however also immotility of the spermatozoa, which results in infertility; clearly the latter produces sufficient of a reproductive

disadvantage to lead to the almost total loss of the gene from the gene-pool (Afzelius, 1976). In uncomplicated situs inversus there also appears to be a tendency to infertility (Arge, 1960). The predominance of a left-sided heart as opposed wto the predominance of a right-sided heart may well have as much to do with mitochondrial bio-synthesis as with the haemodynamics of the cardio-vascular system.

11:4.3 The evolution of functional asymmetries

From sections 11:4.1 and 11:4.2 it should be clear that for any complex bilateral system there are clear functional advantages to developing some form of consistent lateral dominance, for then control will be easier. In the case of the functional as opposed to the morphological asymmetries this argument seems less strong, and, as has been argued, earlier in Chapter 10, it may well be that there are reproductive advantages to fluctuating asymmetry, whilst for hand-clasping and arm-folding I have argued, in Chapter 9,6 that what genetic factors there are can be explained simply in terms of genetic drift (that is, a total lack of advantages).

As to the question of why it is the left hemisphere that is dominant for speech, rather than the right hemisphere, there is simply no answer, and we may only return once more, to the null hypothesis that "it won the fight".

11:5 The season of birth of left-handers

Leviton and Kilty (1979) have recently suggested that in a survey of school-girls there was a significant excess of left-handers born around the month of November. Regrettably it is not easy to assess Leviton and Kilty's claim, since they give no actual data, merely the result of a statistical significance test. Nevertheless, since there were only 33 left-handed school-girls in their sample, each month-category can only have contained an average of 2.75 persons. Here I will describe results from my own larger study, other aspects of which have been reported in earlier chapters (i.e. surveys Ia, Ib and II).

In survey I a total of 948 students gave detailed information about their handedness. In survey II not only did 511 students give handedness information, but also their parents completed a handedness questionnaire. In the present report I have concatenated data from the two surveys and from parental and student generations.

Table 11.4 shows the sample size, for males and females, and for both sexes combined, for each particular month of birth. Before testing for seasonality of births of left-handers it is necessary to test for seasonality of overall births. Using the method of Edwards (1961), there are no seasonal trends in either males, females or the two sexes combined ($X_2^2 = 1.15$; $X_2^2 = 5.08$; $X_2^2 = 3.40$

respectively). Of the 1049 males, 134 (12.77%) were left-handed, whilst of the 706 females, 73 (10.34%) were left-handed. In the males there was no evidence of a seasonal trend in sinistral births ($X^2_2 = 2.17$, NS). Amongst the females there was no significant seasonal trend ($X^2_2 = 3.73$, NS), and in contrast to the data of Leviton and Kilty, the maximum incidence of sinistrality was around the month of July/August. Combining the male and female data, there was no evidence of a seasonal trend ($X^2_2 = 0.34$, NS).

In summary, the present analysis fails to support the hypothesis of Leviton and Kilty of a seasonal trend in sinistral births.

Table 11.1 Asymmetries in the paired organs of man and animals.

Organ/Tissue	Species
Humerus	Man
Humerus	Human Fetus
Radius	Man
Bone mass in hand	Man
Femur	Man
Clavicle	Man
Scapula	Man
Pelvis	Man
Skull: frontal and parietal bones	Man
: malar bone	Man
: Antero-posterior length internally	Man
: Position of falx	Man
: Mandible	Man
: Mandibular condyle	Man
: Jugular foramen	Man
: Incomplete pterygo-alar foramen	Man
Transverse process of cervical vertebrae	Chicken
Dystopia caudalis tuberculi anterioris	Mouse
Septal aperture of humerus	Rat
Rate of skeletal maturation	Man
Palmaris longus muscle	Man
Foramina transversaria imperfecta	Mouse
Polydactyly	Chicken
Polydactyly	Mouse
Heterodactyly with diplopodia	Chicken
Motor Cortex Betz cells	Man
Planum temporale of brain	Man
Areas 41 and 42 of auditory cortex	Man
Audiogenic seizure threshold	Mouse
Brachium of inferior colliculus	Man
Lateral lemniscus	Man
Gliae of lateral lemniscal nuclei	Man
n. mesencephalis lateralis pars dorsalis	Birds

Asymmetry	Reference
Larger on the right	Latimer & Lowrance (1965)
Larger on the right	Schultz (1926)
Larger on the right	Latimer & Lowrance (1965)
Greater on the right	Garn et al (1976)
Larger on the left	Latimer & Lowrance (1965)
Heavier on the left	Lowrance & Latimer (1957)
Heavier on the right	Lowrance & Latimer (1957)
Heavier on the right	Lowrance & Latimer (1957)
Larger on the right	Woo (1930a)
Larger on the left	Woo (1930a)
Greater on the right	Hoadley & Pearson (1929)
Shifted to the right	Inglessis (1925)
Longer on the left	Morant et al (1936)
Longer on the right	Morant et al (1936)
Larger on the right	Woo (1930b)
More common on right	Chouke (1947)
Longer on the left	Kawahara (1974)
More common on the right	Grüneberg (1950a)
More common on the left	Riessenfeld et al (1975)
Greater on the left	Torgersen (1951)
More common on the right	Thompson et al (1921)
More common on the left	Grüneberg (1950b)
More common on the left	Bond (1920)
More common on the left	Fortuyn (1939)
More common on the right	Taylor & Gunns (1967)
More cells on the left	Lassek (1940)
Larger on the left	Geschwind & Levitsky (1968)
Larger on the right	Campain & Minckler (1977)
Lower on the left	Collins & Ward (1970)
More fibres on the left	Ferraro & Minckler (1977a)
More fibres on the left	Ferraro & Minckler (1977b)
Greater number on right	Ferraro & Minckler (1977b)
Larger on the left	Cobb (1970)

Table 11.1 continued/.....

Organ/Tissue	Species
Habenular Nucleus	Frog, newt, eel
Hypoglossal nucleus	Man
Optic Nerve and eye	Rat
Maturation of optic nerve	Rabbit
Unilateral microphthalmia	Rat
Cutaneous tactile sensitivity	Human neonate
Unilateral thyroid aplasia	Rat
Testis	Man
Testis	Human Fetus
Ovary	Human Fetus
Testicular artery arching over renal vein	Man
Displacement of pelvic ureter	Man
Intra-uterine position of fetus	Man
Polythelia	Mouse
Sodium concentration of sweat	Man
Skinfold thickness	Man
Digital dermatoglyphics: total ridge count	Man
Palmar dermatoglyphics: ridge count	Man
Palmar dermatoglyphics: ridge count	Man
Side of scalp hair-whorl	Man
Direction of scalp hair-whorl	Man
Length and area of striate cortex	Man
Noradrenaline concentration in the thalamus	Man
Height of the external ear	Owl
Depth of lingual fossa of 1st upper incisor	Man

Asymmetry	Reference
Double on left only	Braitenberg & Kemali (1970)
Larger on the left	Tomasch & Etemadi (1962)
Larger on the right	Wang (1927)
More rapid on the right	Narang (1977)
More common on the right	King (1929)
More sensitive on the right	Hammer & Turkewitz (1974)
More common on the right	King (1929)
Larger on the right	Chang et al (1960)
Larger on the right	Mittwoch & Kirk (1975)
Larger on the right	Mittwoch & Kirk (1975)
More common on the left	Notkovitch (1955)
More common on the right	Kabakian et al (1976)
Left occiput more common	Steele & Javert (1942)
More common on the left	Little & McDonald (1945)
Greater on the left	Gibinski et al (1971)
Greater on the right	Damon (1965)
Greater on the right	Holt (1953)
Greater on the left	Floris (1975)
Greater on the right	Pateria (1973)
More common on the right	David & Osborne (1976)
More commonly clockwise	David & Osborne (1976)
Greater on the right	Cohn & Papez (1930)
Greater on the left	Oke et al (1978)
Higher on the right	Norberg (1968)
Deeper on the left	Aas & Risnes (1979)

Table 11.2 Disease asymmetries in Man

More common on the Right side	Reference
: Conditions present at birth	
Congenital unilateral third nerve palsy	Victor (1976)
Helical pit of the ear	Stannus (1914)
Side of testis in hermaphroditism	Polani (1970)
Congenital inguinal hernia, in both males and females	Packard & McLaughli (1953); Atwell (196
Club foot	Schnall & Smith (197
Radial aplasia	Schnall & Smith (19
Infantile hemiplegia	Dennis & Whitaker (1977)
: Conditions presenting during childhood	
Adolescent gynaecomastia	Nydick et al (1961
Retinoblastoma	Duke-Elder (1967)
Varicocoele	Campbell (1928)
Hemihypertrophy	Schnall& Smith (19
: Conditions presenting during adulthood	
Carpal tunnel syndrome	Birkbeck & Beer (1
Mallet finger	Abouna & Brown (19
Meningioma	Cushing & Eisen-Hardt (1958)
Nephroptosis	de Zeeuw et al (1977)
Ovarian tumour	Seegar (1958)
Post-encephalitic Parkinsonism	Reynolds & Locke (1971)
Adenocarcinoma of the kidney	Riches et al (1951)
Spontaneous pneumothorax	Crimm (1948)
Testicular tumours	Ferguson (1965)
Trigeminal Neuralgia	Rothman & Wepsic (1974)
Third molar impaction	Henry & Morant (1936)

Table 11.2 continued/.....

More common on the Left side	Reference
: Conditions present at birth	
Cleft lip, with or without cleft palate	Sanders (1933)
Congenital absence of a limb	Simmel (1961)
Congenital dislocation of the hip	Hass (1951)
Renal agenesis	Schnall & Smith (1974)
Polythelia	Schnall & Smith (1974)
Polydactyly	Schnall & Smith (1974)
Erb's Palsy	Bennett & Harrold (1976)
Hairy pinnae	Stern et al (1968)
Infantile hemiplegia	Dennis & Whitaker (1977)
: Conditions presenting during childhood	
Neuroblastoma	Schnall & Smith (1974)
Hemifacial microsoma	Schnall & Smith (1974)
: Conditions presenting during adulthood	
Acute optic neuritis	Bradley & Whitty (1967)
Carcinoma of the breast	McManus (1977)
Benign breast tumours	Ciambellotti (1961)
Adenoma of the epididymis	Beccia et al (1976)
Intra-cranial glioma	Denman & Smith (1954)
Osteoarthrosis of the hands	Acheson et al (1970)
Booerhaave's syndrome	Curci & Horman (1970)
Sprained ankle	Basur et al (1976)

Table 11.2 continued/.....

More common on the left side
: conditions presenting during adulthood

Renal artery stenosis	Schwartz & White (1964)
Subclavian steal syndrome	Santschi et al (1966)
Wallenberg's syndrome	Ask-Upmark & Bickerstaff (1976)
Herniated lumbar inter-vertebral disc	Kelsey (1975)
Atherosclerotic gangrene of the leg	Frohn (1976)
Aorto-Iliac artery thrombosis	Spiro & Cotton (1970)
Phantom breast following mastectomy	Weinstein et al (1970)
Conversion reactions	Stern (1977)
Hysterical paralysis	Hoadley & Pearson (1929)
Carcinoma of the Ethmoid sinus	Robin & Shortridge (1979)
Deviation of the Nasal septum	Robin & Shortridge (1979)

TABLE 11.3

Handedness		Right		Left	
Situs:		Solitus	Inversus	Solitus	Inversus
Primary asymmetry		x	x	x	x
Secondary to cardiac asymmetry	- Direct	x	1 - x	x	1 - x
	- Indirect	x	0.5	x	0.5
Secondary to handedness	- Direct	x	x	1 - x	1 - x
	- Indirect	x	x	y	y

Note: i. In the last line of the table $y = (5 + 4x - \sqrt{16x^2 - 8x + 9}$

ii. If x =0.5then of course the situation is simply that of fluctuating asymmetry. Fluctuating asymmetry does not imp that other asymmetries cannot be directly secondary, but tl cannot possibly be indirectly secondary.

iii. It is not necessary to know the incidence of a condition i left-handers with situs inversus to determine asymmetry ty this is fortunate since such individuals represent about 4 per million of the population.

iv. It is possible that the model above will suggest that an asymmetry is indirectly secondary to handedness; this of course could simply mean that it is directly secondary to cerebral speech dominance.

v. It has been assumed that the inheritance of situs is recessive and that of handedness is additive. These assumptions might require modification at a later date. Alternatively it is theoretically feasible that the expression of an indirect asymmetry may be additive for one particular asymmetry and recessive for another

Table 11.4 Shows, for males, females and males and females combined, the total number (N), and the number of left-handed (nL) subjects, born in each month of the year.

	Males		Females		Males & Females	
Month	N	nL	N	nL	N	nL
January	103	15	53	1	156	16
February	81	14	47	5	128	19
March	82	12	67	6	149	18
April	98	12	61	6	159	18
May	99	14	64	6	163	20
June	81	9	75	7	156	16
July	84	7	53	6	137	13
August	94	12	66	10	160	22
September	74	10	54	7	128	17
October	84	10	61	4	145	14
November	88	10	57	9	145	19
December	81	9	48	6	129	15
Total	1049	134	706	73	1755	207

PART II: THE SYMBOLISM OF LEFT AND RIGHT

"As for Odysseus, this is the fifth year
since he took leave and left my country.
Unhappy man - and yet the bird-omens
were favourable, wholly on his right,
as he went away".

Homer, The Odyssey, Bk XXIV; translated by
T.E. Lawrence.

CHAPTER 12: RIGHT-LEFT AND THE SCROTUM IN GREEK SCULPTURE

"Zoe: How's the nuts?

Bloom: Off side. Curiously they are
on the right. Heavier I suppose.
One in a million my tailor,
Mesias, says".

James Joyce, Ulysses.

In man the scrotum is clearly asymmetrical, the right testicle usually being placed higher than its opposite number[1]. (For footnotes, please see the end of the Chapter). The cause of this asymmetry is not clear. We may however reject a simple mechanical explanation which would say that the heavier of the two organs is pulled to the lower position by the action of gravity, for in both adults and fetuses it is clear that the right testicle is both the heavier and also the greater in volume[2]; thus the larger and heavier is also the higher. Such a relationship is counter-intuitive, and we may expect that it would present difficulties to artists, and to sculptors in particular.

Table 12.1 shows the observed relationships in 187 sculptures, the majority of which are from ancient Greece, the data being pooled from two separate studies[3]. In the single largest group the right testicle is placed higher (and thus correctly), but simultaneously the left testicle is made larger, the reverse of the correct anatomical situation. Winckelmann was partly correct when he observed of Greek sculpture that "Even the private parts have their appropriate beauty. The left testicle is always the larger, as it is in nature; so likewise it has been observed that the sight of the left eye is keener than the right"[4]: his observations of nature were less accurate than those of sculpture. A further examination of Table 12.1 shows that the second most frequent asymmetric group consists of those cases in which the left

testicle is higher and the right testicle is larger, that is, the mirror-image of the most common type. This would imply, as I have suggested elsewhere[3], that the Greeks were, in part, using a simple mechanical theory to account for the relation of scrotal size and position. But this hypothesis alone cannot account for the predominance of cases in which it is the right testicle which is the higher. There is also a further asymmetry within the Table which requires explanation, namely that there are far more entries in the cells below the main bottom-left/top-right diagonal than in those above it. This results in an excess of figures in which the right testicle is higher but the two testicles are equal in size. The implication is that the asymmetry in height is prior to the asymmetry in size. A further examination of the data will give more information on this point, but firstly it is necessary to examine the difficult question of left-right symbolism in Greece, and its relation to theories of reproduction.

Right and Left are of fundamental symbolic significance in many cultures and have been much studied by anthropologists[5]. As G.E.R. Lloyd has emphasised[6], this is no less true in Classical Greece (and as others have suggested, perhaps also in our own culture[7]). Most interestingly for our present purposes, this dichotomy was of fundamental importance to Greek theories of the determination of the sexes. Anaxagoras proposed that the male was the active principle in determining sex (as modern science holds). He

suggested (unlike modern science) that the male seed comes from the right testis and the female from the left[8]. Furthermore that the male fetus grew on the right side of the uterus and the female on the left[9]. This theory was extended by Leophanes (or possibly Cleophanes) who proposed that a man may determine the sex of his offspring by copulating with either the right of the left testis tied off[10]. Empedocles suggested that the sex of the child was determined entirely by the female, the principle feature being the heat of the womb, which was controlled by the degree of flow of the menses[11]. Later theories stressed the importance of the female but suggested that noy only the side of implantation of the fetus, but also the side of origin of the ovum were of importance, once again the right side producing the male, and the left the female[12].

The Hippocratic authors attributed great significance to the differences between left and right. They proposed that diseases of the right side of the body were more severe[13] (particularly in the case of pleurisy), that the milk from the right breast was stronger and more suitable for male infants[14], and perhaps most interestingly of all, that if the right testicle was cold and retracted then this was a sign of death[15].

The most elaborate form of left-right symbolism found its origins in the Pythagoreans, who associated right

with male and left with femals, as well as with many other paired opposites[16]. This was extended by Aristotle into a general theory of right and left in biological systems, and he proposed that it was the right side which initiated movement[17], which was warmer and less watery than the left[18], and also stronger[19]. Aristotle rejected all of the previous theories of sex determination and instead concluded that the critical variable was the amount of innate heat produced by the fetal heart, before any of the other organs were differentiated[20]. Most interestingly he proposed that the testes themselves were not directly concerned with reproduction per se. Their functions were two-fold: firstly to act as weights whose action was to keep open the ducts whereby the seed is discharged[21], and secondly to act to tension the entire body, thereby causing the deepening of the voice and the changing of form which occurs in the male at puberty[22]. A further consequence of Aristotle's theory of right and left, although not stated explicitly, is that the right testicle ought to be higher than the left, for 'in as much as motion commences on the right, and the organs on this side are in consequence stronger than those on the left, they must all push upwards in advance of their opposite fellows'[23]. He also claims that the parts on the right are 'naturally more solid and more suited to motion than those on the left'[24]: and we may thus expect that the right testicle ought to be smaller (or at least denser) than its counterpart.

Whilst this Aristotelian viewpoint suggests several good reasons why the sculptor ought to portray the right testicle as higher and smaller, there are also contradictory implications of the theory. Perhaps the heavier and lower testis would be tensioning the left side of the body more than the right, but the left side is the female side. Similarly perhaps the stronger testis ought to be the larger, not the smaller. Pre-Aristotelian theory may be linked with Aristotelian in that the hotter (right) testis ought to produce male seed (or greater innate heat); indeed in terms of modern physiology we may expect it to be hotter since it is nearer to the abdomen.

The question now arises as to the relationship between Greek right-left theory and the portrayal of the scrotum. Stewart[3] has divided his data into three historical periods between 600 and 480 BC; my own data may be broadly regarded as 'classical' (480-320 BC). In Figure 12.1 we can see the historical development of the left-right asymmetry of the scrotum. The percentage of works in which the testes are equal in both size and height (that is the middle cell of the table) decreases significantly with time ($X^2 = 8.52$, 3 df, $p < 0.05$), possibly as a result of sculptors becoming more concerned about details of anatomy. To assess the development of the asymmetry, an asymmetry score was calculated for each of the four periods. To do this a score of +1 was given for a right testicle which was larger, -1 for a left testicle which was larger, and 0 for a case in which the two organs were equal in size. The final

total was divided by the number of cases to give an average asymmetry score which would lie between +1 and -1. A similar process was used for the asymmetry scores for height, +1 being given for a higher right testicle, and -1 for a higher left testicle. These scores may be seen in Figure 12.1 (calculated in two ways according to the inclusion of the 'equal' groups). The method of calculation produces little substantial effect upon the scores. Although Stewart[3] has suggested that there appears to be a trend whereby the right testis becomes higher somewhat before the left testis becomes larger, this is not clear in the present analysis, differences between groups being within the limits of chance variation ($X^2 = 4.11$, 3 df, NS; after exclusion of the equal groups and merging of groups where necessary to produce expected values greater than 5). We may thus conclude that in all probability there was little change in the nature of the asymmetry over the period 600 - 320 BC, although its usage became more frequent.

In Figure 12.1 are also shown the approximate dates of the principle left-right theorists and it seems fairly clear that the asymmetry was in existence before the theorising. The actions of the sculptors probably represent the utilisation of either a formal theory, or possibly a set of folk-beliefs, which had been extant in the centuries before it was codified by the later philosophers. The actions of the philosophers may well have encouraged the

portrayal of detailed left-right asymmetry, but they were unlikely to have been the origin of it. Certainly the Greek mind would appear to be no less vulnerable to left-right speculation than any other culture, either at anthropological or philosophical level. Evidence from Homer suggests the existence of a pre-Classical right-left symbolism[6].

Notes and References

1. Chang et al. (1960) found that the right testis was the higher in 62.1% of 486 men, and the left testis higher in 27.4%, the two being equal in height in the remaining 10.5%. Antliff and Shampo (1959) found an essentially similar result in 386 men, the right testis being higher in 65.1% and the left higher in 21.9%. The two sets of authors differ in their findings as to the effects of handedness, Chang et al. claiming that the relationship is reversed in left-handers, whilst Antliff and Shampo found no such reversal. There is also evidence that in the bull the right testis tends to be the higher of the two.

Astley Cooper (1830) was well aware of the differences in height of the testes, although he did not comment on differences in size. Elsewhere, in his book on the breast, Cooper (1840) criticised the errors of sculptors. "I have, in my work on the testes, pointed out the errors of those who paint or chisel from imagination, and not from observation of nature, in placing (the testes) of equal height, although the left is usually much lower than the right; and the same remark may apply to the breasts ....." I am unable to find any reference to sculptural representations of the scrotum in Cooper (1830), or in the 2nd or 3rd editions of that work.

2. Chang et al. (1960) found that the average weights of the right and left testes were 9.95 and 9.36 grams respectively, and the volumes 9.69 and 9.10 ccs, the differences being highly significant statistically. The densities are thus 1.0268 and 1.0286, a difference which is unlikely to be significant. Mittwoch and Kirk (1975) found a similar relationship in human fetuses, and showed that the right ovary also tends to be the larger. This difference is also found in other animal species (Jost, A. et al. (1972).

3. McManus, I.C. (1976); Stewart, A.F. (1976).

4. Winckelmann, J.J. ('History of Ancient Art', trans. A. Gode, New York, 1968, page 296 (Book V; VI, 11)). There is no evidence of any systematic acuity difference between the two eyes (McGuiness, D. 1976); there is however a trend for the right eye to be the dominant eye (Porac and Coren 1976), a feature possibly noted in Hippocrates (Littré, E. (1840), Book V. p. 137, para. 15).

5. See Needham, R. (1975) for a survey. For biblical and Quranic examples, see Walsh and Pool (1942; 1943), and for Chinese thought see Granet (1934).

6. Lloyd, G.E.R. in Needham (v.s.); see also Lloyd (1966) and Braunlich (1936).

7. See for instance Domhoff, G.W. (1969). Thus for instance in James Joyce's 'Ulysses' there are references to the left breast being more sensitive (Joyce, 1969, p. 377), the left hand being nearer to the heart (p. 345) and to the asymmetry of the scrotum (p. 454). He also repeats the Empedoclean theory of conception (p. 415). There is some evidence that indeed the left breast is more sensitive for pressure discrimination, although the right breast is more sensitive to two-point discrimination (Weinstein; 1962).

8. Aristotle, De Gen. An., 763, b. 31. (All references from Aristotle are taken from the translations of Farquharson (1912), Ogle (1912), Platt (1910), Thompson (1910) and Warrington (1956)). See also Freeman (1949), p. 272.

9. It was commonly assumed that the human uterus was bicornuate, as it is in many animal species (De Gen. An., 716, b. 33). Also attributed to Parmedides (Tarán, 1965; p. 263), and as such quoted by Galen (Epid., VI, 48); see also De Gen. An., 763, b. 21 and Kember O. (1971) and Lloyd, G.E.R. (1972) for difficulties in the interpretation of Parmenides. Galen also claimed that "everything in the reproductive organs on each of the two sides, I mean the right and the left, (is) quite alike" W.H.L. Duckworth (1962;

p. 130), although elsewhere he points out that the left testis is more varicose than the right (see note 13) and that the scrotum around it is looser (De Usu Partium, May(1968); Vol. II, p. 308). He also noted the asymmetry of the testicular vein insertions, but erroneously described them as arteries, and therefore suggested that the left testis received inferior blood and was therefore cooler than the right (ibid, p. 306).

10. De Gen. An., 765, a. 22. Also proposed by the Hippocratic authors (Littré, VIII, p. 501, para. 31), who also suggested that if the right testicle developed first the child would be male, and if the left, the first child would be female (Littré, V, p. 313, para 21). The possibility of controlling sex by a ligature was also espoused by Giles of Rome in the late Middle Ages (Hewson, 1975), who also claimed, following the Hippocratic Corpus (Littré, VI, 291), that the male fetus tended to be on the right side of the uterus. As late as 1891 Mrs.Ida Ellis in her 'Essentials of Conception' (see Pearsall, 1971, p. 303) stated "It is the male who can progenate a male or a female child at will, by putting an elastic band round the testicle not required. The semen from the right testicle progenates male, whilst that from the left female children; men who have only one testicle can only beget one gender, but sometimes they do not descend, remaining in the body,

in which case a child of either gender may appear". In 1914 Prof. A. Fischer-Dückelmann was still repeating the Aristotlean story of the bull who could sire either sex at will, according to the side of entry on copulation (Fischer-Dückelmann, 1914), probably deriving the story from Pliny's Natural History, VIII, LXX, where the use of ligatures is also described (ibid, VIII, LXXII, Loeb edition). That the side of origin of the sperm had any bearing upon the sex of the child was rejected by Sir Thomas Browne, although on the basis of a somewhat dubious physiology (Pseudodoxia Epidemica, Book IV, v; Wilkin 1852); (in another context however he also repeats the story of the significance of the side of entry of a bull during copulation(ibid, Bk. V, XX)). Likewise de Graaf also rejected the possibility of any differences between the testicles in their ability to produce males (Tract. de Vir. Org. Gen. Ins. (1668), see H.D. Jocelyn, B.P. Setchell (1972)). The theory was rejected on experimental grounds by H.D. King (1911), and S.M. Copeman (1919), although neither experiment would be regarded as acceptable by modern criteria of statistical proof (see also Crew, 1952).

12. De Gen. An., 763, b. 31. In its strong form the suggestion that sex is dependent upon the side of the uterus at implantation may be rejected by Aristotle's own observation that a female fetus has been observed

in the right part of the uterus and a male in the left (De Gen. An., 765, a. 18). The modern version of the theory suggesting that the side of origin of the ovum is important has been suggested by Rumley Dawson (1909). Experimental disproof of the hypothesis may be found in H.E. King (1909; 1911), L. Doncaster & F.H.A. Marshall (1910) and Copeman (1919). As with the experiments mentioned in Note 10, they are probably not acceptable by modern standards in rejecting a weak version of the theory, although they indubitably reject a strong version, that is that males come only from the right side. In passing it is perhaps worth noting that there is indeed asymmetry of the fetus in utero but this does not seem to be sex-related: the fetus at term far more often occupies the position known as Left Occiput Transverse, than it does Right Occiput Transverse (Steele & Javert 1942).

13. Littré, VII, p. 155. Whilst there is no definite evidence that diseases on one side of the body are more severe than those on the other side (with the possible exception of carcinoma of the breast, see McManus (1977)), there is excellent statistical data showing that some diseases are more common on one side than the other; in this context, of some relevance are that carcinoma of the breast, varicocoele, and tumours of the testicle occur more

commonly on the right side (Busk & Clemmeson, 1947; Campbell, M.F. 1928; Beccia, 1976; Ferguson, 1965).

14. Littré, V., p. 137, para 15. This theory was transmuted in the middle ages so that it was proposed by Trotula of Salerno in the 11th century, that "women bearing male children have the right breast larger, those female children the left" ('The Diseases of Women' trans. Mason-Hohl (1940)).

15. Littré, VIII, p. 669.

16. Aristotle, Metaphys., A, 5 986. a. 22.

17. De Inc. An., 705. b. 14, 705. b. 30, 706. b. 5.

18. De Part. An., 493. b. 19.

19. Hist. An., 493. b. 19.

20. De Gen. An., 766. a. 25. He argues that more female children are produced by the young and by those verging on old age since in these groups the amount of vital heat is important (De Gen. An., 766. b. 29). Modern research suggests that the proportion of male children does decrease with the age of the father (but not with the mother) although the effect is small, the change in the secondary sex ratio (at birth) being from 51.65% male children to fathers aged 16,

to 51.10% male children to fathers of age 45 (Novitski & Sandler, 1958). A further consequence of Aristotle's theory of increased vital heat in male children is that they ought to tend (but by no means completely as Aristotle himself admits) to start moving earlier in the womb (Hist. An., 583. b. 3).

21. De Gen. An., 717. a. 34; and thus castration is effective because it closes the internal ducts. That a castrated bull may fertilise a female for a few days after castration was attributed to a delay in the ducts closing off (instead of to the inevitable storage of semen in the seminal vesicles for a few days or weeks). An identical theory was held also by Giles of Rome (Hewson, 1975; p. 91). The role of the seminal vesicles was correctly appreciated by Sir Thomas Browne, (Wilkin, 1852; IV, v).

22. De Gen. An., 788. a. 10.

23. De Part. An., 671. b. 28. Aristotle bases his theory on his (erroneous) observation that the right kidney is placed higher than that on the left, which is true in some animals, but not in man.

24. De Part. An., 672. a. 22. Based upon the differences in the amount of fat around the right and left kidneys: see also note 1 regarding the density of

the testicles.

Table 12.1

Distribution of asymmetry shown in the portrayal of the scrotum in 187 Greek sculptures (or, in a few cases, Renaissance copies); data pooled from McManus (1976) and Stewart (1976).

Side of Larger Testicle	Side of Higher Testicle			
	Left	Equal	Right	Totals
Left	5 (2.6%)	7 (3.7%)	60 (32.1%)	72 (38.5%)
Equal	10 (5.3%)	43 (23.0%)	23 (13.4%)	78 (41.7%)
Right	26 (13.9%)	1 (0.5%)	10 (5.3%)	37 (19.8%)
Totals	41 (21.9%)	51 (27.3%)	95 (50.8%)	187 (100%)

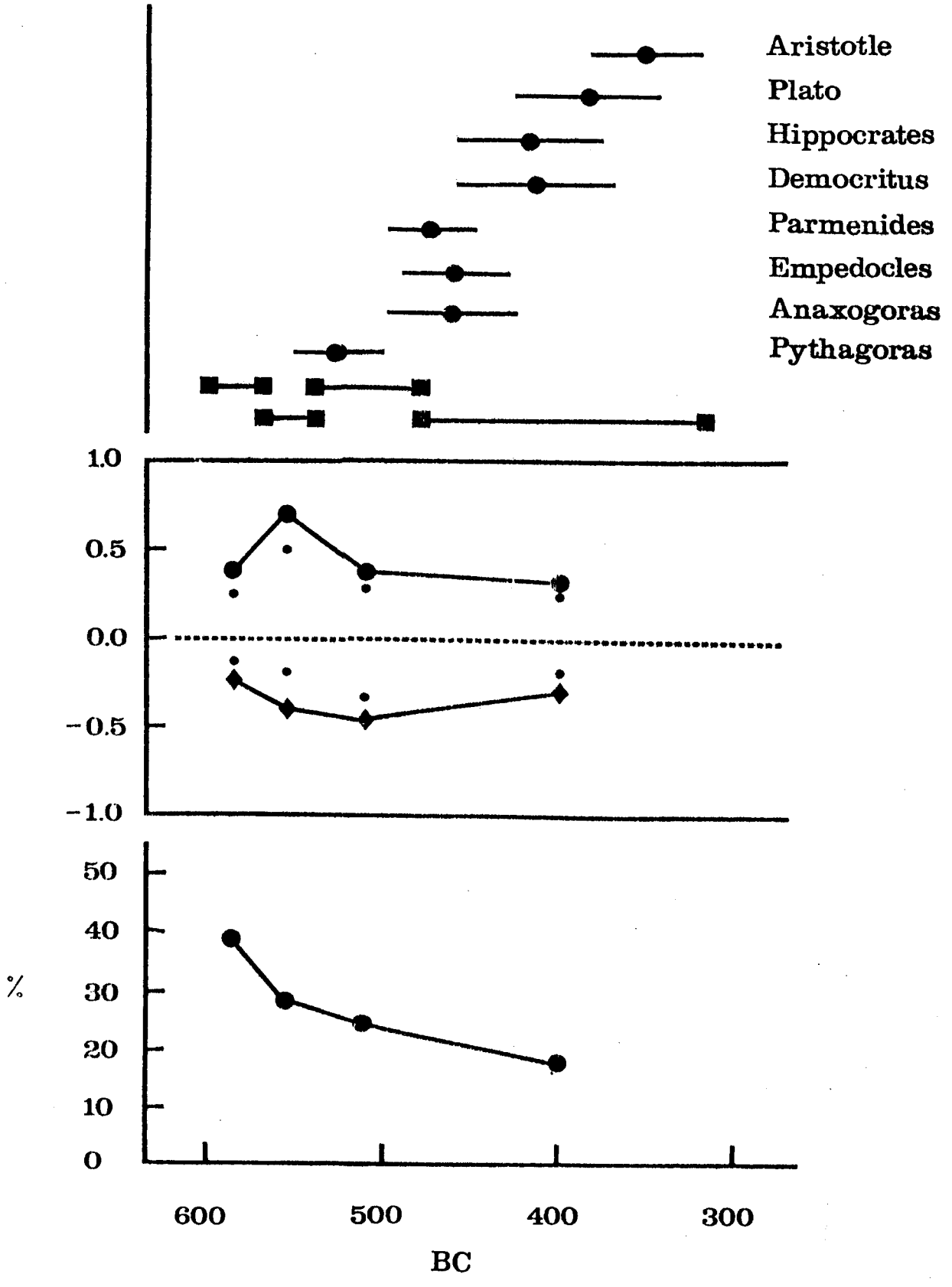
Figure 12.1

Abscissa is calibrated in years BC. Data points are plotted at the mid-point of their relevant period. The range of the points is shown graphically by the squares at the bottom of part C. The first three points represent Stewart's data for the periods 600-570, 570-540 and 540-480 BC. The fourth point represents my own data which may be described as 'classical', and is represented by the period 480-320 BC.

a. Shows the percentage of figures in each period in which the testicles were both of the same height and the same size. The decline is statistically significant (see text).

b. Shows the 'asymmetry scores' calculated as described in the text. These may be calculated in three ways according to the treatment of the 'equal' groups. The solid circles and triangles joined by solid lines show the scores when all of the 'equal' groups are excluded (i.e. just the corners of the table remain). The small dots represent the results when all of the data is included. If only the middle cell of the table is excluded then results are obtained mid-way between the other two points, and have been excluded in the interests of clarity. The changes in the score are statistically not significant.

c. Shows the lives of some relevant philosophers for comparison with the data points below.


Aristotle
Plato
Hippocrates
Democritus
Parmenides
Empedocles
Anaxogoras
Pythagoras
1.0
0.5
0.0
−0.5
−1.0
50
40
30
20
10
0
%
600
500
400
300
BC

CHAPTER 13: THE SIGNIFICANCE OF THE LEFT CHEEK

Summary

A detailed statistical account is given of the use of right and left cheeks in Western art, concentrating particularly on works from the Italian Renaissance and the Mediaeval period. It is suggested that choice of right or left cheek is not just a result of mechanical constraints or chance processes, but represents the expression of a left-right 'symbolism', the left cheek being used to portray individuals who are different or distant from the artist.

The possible origins of this phenomenon (as well as other left-right symbolisms in the iconography of the Crucifixion) are considered and it is suggested that an underlying brain asymmetry might be the fundamental asymmetry and that the particular orienting event of Christ's actual crucifixion, and its record in the form of the Shroud of Turin, might have 'crystallised' the phenomenon in the Western artistic tradition.

"There is no obstacle but the inertia of tradition to prevent aesthetics from undertaking an extensive program of direct comparative observations ... It would imply an effort to make all general theories grow directly out of detailed analyses of works of art, instead of being merely illustrated with occasional examples as in most past and present writing on aesthetics".

Munro (1956)

"The value of research depends upon the field where it is carried out ..... The most meagre adept may make elaborate statistics of the number of times in the art of the middle ages our Lord blesses with three fingers, how many times with two and a half, and how many times with two only; or how frequently St. Catherine has her wheel, or St. Andrew his cross, to right or again to left".

Berenson (1950)

13:1 Introduction

The research in this paper represents the final product of an initial chance observation, followed by several years of exploration, discovery and much interim speculation and theorising, mostly acting upon intuition, allowing the data to develop in its own way. To present the results other than chronologically and autobiographically would be once more to compound the lie which Medawar (1963) tells us is implicit in most scientific writing. It would also destroy the excitement of the covert detective story which much science represents.

To quote the master of that genre, "The world is full of obvious things which nobody by any chance ever observes" (Conan Doyle, 1902). By chance alone I was fortunate to observe one such obvious thing: the rest fell into line with a certain inexorable logic of its own.

13:2 Portraits

In 1971 I was carrying out some work on the shapes of pictures and required for completeness a large series of several hundred portraits. The college library had just acquired a copy of Roy Strong's 'The English Icon', which I borrowed and tabulated its contents. For reasons which I am unable to explain, possibly for completeness also, I noted down whether the subject of each portrait was facing to the left or the right (or to put it less ambiguously, whether the subject was showing his left or right cheek to the observer). To my surprise I found that there was a significant excess of left cheeks. More importantly however this excess was significantly greater in female subjects than in male. Our first reaction to this data was to think in terms of some simple mechanical factor, possibly related to the right-handedness of the artist, but such explanations were confounded by the sex difference. I spent the next few months in collecting a larger series of data, containing amongst other things, the entire public collection of the National Portrait Gallery, London. The results of this study are shown in Figure 13:1. In our paper (McManus and Humphrey, 1973) we

analysed the data in terms of the amount of the body visible in the portrait and found that if the whole body was portrayed then the left cheek was portrayed significantly more often. This result we had found difficult to explain and had suspected that it might well be artefactual. A few years later I re-analysed this data (and included a little more) in terms of the degree of turn of the head, distinguishing between 'full-face' (in which the whole of both eyes was distinguishable) and 'profiles' (in which both eyes were not completely visible). The dependance upon the amount of body visible is indeed artefactual and is secondary to the turn of the head. Figure 13:2 shows that only those portraits in which both eyes are visible show a left-cheek tendency. This confounds any mechanical effect (which we would expect to be greater in the profile group), and suggests that a more psychological explanation is required, the salience of full eye-contact clearly being of importance.

Other more recent analyses of this data show several features of note. Figure 13:3, as well as Figures 13:4 and 13:5, show that the left-cheek tendency in portraits is stable with time (in contradistinction to the data to be presented later). Several other studies have confirmed the reality of the left-cheek predominance. Of the 390 portraits of the Medici in the sculpture gallery of the Uffizi, Florence, 216 (55.4%) show the left cheek ($X^2_1 = 4.3$, $p < 0.05$). In a book of American primitive paintings (Lipman, 1969) 22 out of 29 (75.9%) showed the left cheek

($X^2_1 = 6.72$, $p < 0.01$). Uhrbrock (1973) found that of 199 American commemorative medals, 136 (68.3%) showed the left cheek. Gordon (1974) showed that Goya's portraits tend to show the left cheek, and LaBar (1973) found that modern photographic portraits showed the same tendency. Coles (1974) found in a study of 188 portraits by Sir Joshua Reynolds that 58.6% showed the left cheek, and in a miscellaneous group of Western paintings that 53.8% showed the left cheek. Of 93 portraits by Titian (Wethey, 1971), 59 (63.4%) showed the left cheek ($X^2_1 = 6.1$, $p < 0.02$) (the two self-portraits having been excluded).

13:3 Self-portraits, Rembrandt, Rosetti and van Gogh

By early 1973 we had reached the point where in attempting to answer the deceptively simple question, Why is there a predominance of left cheeks in portraits?, we had found all mechanical explanations neither convincing nor satisfactory (McManus and Humphrey, 1973), and had come round to the remaining alternative, originally suggested by Nick Humphrey, that the cheeks had a symbolic significance. A review of possible mechanical artefacts in the left-cheek excess is given in Appendix A13:1.

A few weeks after our paper in Nature, Professor Landauer of University College, London, was kind enough to send the data shown in Figure 13:1 concerning self-portraits. He had found that of the 302 self-portraits in Goldscheider

(1950), only 119 (39.4%) showed the left cheek, a significant trend towards the right. This was consistent with our developing hypothesis that a symbolic explanation was necessary, but could also be interpreted mechanically. Inspired by Professor Landauer's result I then looked for a further series of self-portraits, with the intention of studying individual artists in more detail. The obvious artist to choose was Rembrandt for he probably painted more self-portraits than any other artist. It is worth noting in passing that we know that Rembrandt had at least two large mirrors in his studio, which he used frequently, and thus we may assume that he was aware of and had explored the nature of mirror-images (Konstom, 1977).

Figure 13:6 shows that indeed Rembrandt's self-portraits do show a strong tendency to show the right cheek. The data was collected from Bredius' catalogue of Rembrandt. This catalogue classifies the portraits both by sex and by kinship. It was thus simple to look at the results in terms of these two parameters (Figure 13:6). It is clear that not only do the proportions showing the left cheek depend upon the sex of the subject, but also upon the kinship relation, non-kin subjects showing the left cheek significantly more often ($X^2_2 = 8.91$, $p < 0.02$) (Humphrey & McManus, 1973). This result seems to dispose entirely of any mechanical hypothesis for the left cheek predominance in portraits, since the critical variable is seen to be the artist's relationship with his subject (or

rather the artist's perception of the relationship). When we look at Figure 13:6 in these terms we can equate the dimension of left cheek-right cheek with a parallel dimension of 'unlike myself - like myself'. Whether this is an accurate description of the dimension is difficult to tell at this stage and alternative dimensions of 'self as I would like to be perceived - self as I really am', or 'public - private' might be equally appropriate. This point will be returned to later after more evidence has been presented. With this hypothesis we were able to reconsider some other sets of data and provide explanations for them. Van den Hoeff (1973) has discussed Rembrandt's engravings.

In 1973 Dr. Edwin Clarke had pointed out to me that illustrations from mediaeval descriptions of neuroanatomy showed a strong tendency to show the left cheek. Thus of the 49 figures in Chapter Three of Clarke & Dewhurst (1972) 41 (83.6%) showed the left cheek. That this is not a mechanical artefact is shown by the fact that both drawings and woodcuts (which were probably drawn in reverse) both show the same left cheek tendency (18/21 and 23/28 respectively), implying that the artists intended to produce a finished product showing the left cheek. It is difficult to think of any form of portrayal of a human being which is less 'like oneself' than a dissected head; hence we may reasonably expect a strong left cheek predominance.

LaBar (1973) found that 63.4% of 628 photographs in a school year-book showed the left cheek. These photographs

were chosen by the students themselves, from four possible poses, as the most suitable for the year-book. The students were thus being asked to choose a 'public face', one which was possibly not too revealing. The preference for the left cheek thus becomes comprehensible. If we look at LaBar's data in a little more detail then a further trend becomes apparent. Figure 13:7 shows the percentage of left cheeks as a function of seniority: as one becomes more senior so the right cheek portrayal, the more personal, becomes more prominent. Perhaps status allows one to expose the otherwise vulnerable right cheek. LaBar had noted that his data showed no difference in left cheeks between the sexes. This may be explained since in each case it was the student himself/herself who chose the appropriate pose: this contrasts with the usual situation with painted portraits in which a male artist paints a male or female subject. It is perhaps also worth pointing out that LaBar's data disposes of any simple mechanical explanation dependent upon painting techniques themselves.

LaBar's work raises the possibility that individual artists might differ in their use of the left and right cheek. In general we might predict that women artists would reverse the portrayal of males and females but would still persist in producing self-portraits showing right cheeks: thus the dimension would be Left Cheek - Males - Females - Self - Right Cheek. Regrettably this hypothesis is not a simple one to test due to a lack of

readily available data on women artists.

Individual artists might also be idiosyncratic in their usage of the left-right dimension in a manner related to their own world perceptions. Let us consider van Gogh and Rosetti.

Figure 13:8 shows data on Rosetti's portraits. Whilst the position of the (very limited) number of self-portraits is normal, the males show significantly more left cheeks than do the females. The implication is that Rosetti felt women to be closer to him than men, a suggestion which is not incompatible with biographical data.

The Rosetti data show that the male and female groups may be reversed in their proportions of right and left cheeks. Data from van Gogh shows that the most extreme right cheek group need not be the self-portraits. Here only the male portraits are considered (Figure 13:9) and are classified by social class. We know, both from direct and indirect evidence, that van Gogh, though of middle-class origins, felt a strong identification with peasants and workers. When we consider the heads and self-length portraits (Figure 13:9) we find that self-portraits are placed between the lower and middle classes: this we may interpret as the middle classes being 'unlike self' whilst the lower classes may be interpreted as representing almost an idealised self, more like self than self itself.

This picture is further complicated when we consider the male full-length portraits, all of which are of the lower-classes, usually of peasants working in the fields. In this group there is a strong left cheek predominance: we may speculate that it was only the concept of the noble peasant which van Gogh found attractive, rather than the (working) reality itself. It is worth pointing out that we know that van Gogh was certainly concerned with the effect of left-right reversal upon his work (Humphrey and McManus, 1973).

The discussions of the last few paragraphs, whilst interesting, raise severe methodological problems. The theories inevitably have an ad hoc quality about them which is due to the inevitable lack of data on any one artist. If the entire oeuvre has been used to create a hypothesis what may be used to test it? This dilemma possibly represents a special case for Popper's falsifiability theory of science, for whilst the hypotheses described above are in principle testable (by collecting more data) this is in practice impossible (where may one find another 300 Rembrandt paintings?).

For these reasons my research drifted away from the study of individual artists towards large groups of artists where more data was available and thus categorisation still left samples of sufficient size to be statistically analysable. By choosing an historical period in which we

would expect a greater degree of unity of thought amongst artists we may also reduce the problem of inter-artist variation. For these reasons (amongst others) I looked next at the art of the Italian Renaissance, a very prolific period in which large numbers of works were produced on a limited range of thematic material, thereby allowing detailed study.

Most of the data so far reported had been partly influential in constructing the 'like self - unlike self' hypothesis. The data from the Italian Renaissance, and Mediaeval paintings, are however temporally *a posteriori* to the hypothesis and thus may be regarded as a test of its usefulness.

13:4 The Crucifixion and the Annunciation

It is not necessary for the hypothesis to consider just typical portraits, but in principle any human representation should show the phenomenon to a greater or lesser extent.

Whilst on holiday in Tuscany in September 1973 it became readily apparent to me that Renaissance portrayals of the Crucifixion and the Annunciation both showed strong, regular asymmetries. This observation was put on a surer footing on my return home by studying the pictures illustrated in Bernard Berenson's eight-volume survey of the Italian Renaissance (1957, 1963, 1967, 1968).

The crucifixion portrays the horrible and unusual death of a man who was quite unlike any other man. We may expect that Christ will therefore be perceived as different from the artist, and thus ought to show the left cheek. Of the 147 crucifixions in Berenson, 146 (99.3%) portray Christ showing the left cheek. The one exception was painted by Titian when a very old man. That an iconological interpretation is valid is suggested when we consider those pictures which also show the crucifixion of the two thieves. Of the 25 thieves portrayed in Berenson, 20 (80%) showed the right cheek. By contrast alone we might expect this result, for the thieves are the archetype of the ordinary, fallen, sinning man, and are thus close to the artist. The traditionally 'good' thief (placed on Christ's right hand) shows the right cheek in all thirteen cases, whilst the 'bad thief shows the right cheek in only 50% of 10 cases. This is within the compass of the theory: the good thief, representing an idealised form, whom the artist hopes to imitate, is more to the right than self (vide van Gogh, supra), whilst the unrepentant thief is exactly mid-way between the two. The reasons for the thief on the right being the 'good' thief are also considered in Appendix A13:4.

The Annunciation represents a slightly more complex situation, for which it is necessary to introduce a slight modification of the hypothesis. The rider to the theory simply states that the hypothesis is applied firstly

to the most important figure in the picture, and then to those of lesser importance in turn. Of the 209 Annunciations in Berenson, 202 (96.7%) show the angel entering from the left side, and, since the Angel Gabriel and the Virgin Mary are usually shown in part or complete profile, this means that Gabriel is usually showing the right cheek, and Mary the left cheek. A simple interpretation of this data would say that the angel enters from the left simply because the eye reads movement from the left to the right and thus this conveys the greatest sense of movement (e.g. Witmer, 1902; Keller, 1942). Using the same principle it has been claimed that figures entering on to a theatrical stage are far more prominent if they enter from the audience's left than from the right (Dean, 1946). This explanation is however untenable. First it is only this particular Annunciation which shows this gross asymmetry: of 8 other paintings in Berenson in which an angel enters to make an announcement, in 5 he enters from the <u>right</u> side (Fisher's exact test, $p = 2.13 \times 10^{-5}$; two-tailed test) (see also section 6, para 6 of this Chapter). Secondly, in an experiment in which art students were simultaneously shown pictures and their mirror-images there was no tendency for the students to choose those Annunciations with the angel coming from the left as the original or correct version, suggesting that the implied direction or movement is of little significance. (see Appendix A13:5).

From the point of view of 'like-self, unlike-self'

the Annunciation presents a minor problem. Both figures are very unlike self, and thus ought to be portrayed showing the left cheek; but this is pictorially impossible. The solution is to ask, Which is the more important of the two figures? In the Renaissance there is no doubt that the answer would have been the Virgin Mary: hence she shows the left cheek, and the angel inevitably, but unsatisfactorily, has to show the right cheek. When the angel becomes the most important figure in the composition (as in Plate 13:1 for instance) then indeed he does show the left cheek. Of the other figures in this particular picture, all but one of the shepherds are showing their right cheeks as may be expected. Only the least important figures, the fourth shepherd and the women, are anomalous in the cheek shown.

13:5 The Madonna and Child

Whilst the application of the 'like-unlike' hypothesis to the Madonna and Child portrait came after that of the Crucifixion and the Annunciation my interest in its asymmetries had been aroused far earlier: it is therefore necessary to have a slight digression.

Salk (1966, 1973) claimed that about 80% of real mothers carried their child on the left side of their body, and that this proportion was unrelated to the handedness of the mother. As such this basic result has been confirmed

(Rheingold & Keene, 1965; de Chateau & Anderson, 1976; de Chateau et al., 1978; Lockard et al. (1979). Salk suggested that children underwent intra-uterine auditory imprinting to the mother's heart-beat, and that his result was a consequence of the mother being aware that the proximity of the child to her heart would result in the soothing of the child. Whether this explanation is correct is not relevant to the purposes of this study (and indeed other factors such as differential breast sensitivity to touch and pressure, may well be relevant (Weinstein, 1962)). Salk suggested that the tendency to hold the child on the left side was a fundamental biological feature and supported this statement by producing data to show that in Italian Renaissance paintings of the Madonna and Child, the child was held on the mother's left side in 80% of cases. Uhr-brock (1973) also found a similar but weaker tendency and suggested that it demonstrated the artists' sensitivity to the natural biological relationship between mother and child. It is noteworthy that Burt (1937) had also described the tendency of real mothers and Madonnas to hold their child on the left side.

Whilst in Italy I noticed that Salk's observation seemed to be true at the beginning of an art gallery, but that as one proceeded through the gallery its verity seemed less and less likely. When I looked through Berenson's works I found that only 492 (53.9%) of the 913 Madonna and Child portraits showed the child on the left. After several

false starts the reason for this discrepancy became apparent. Figure 13:10 shows the percentage of children held on the left side by the date of production of the painting. At the beginning of the Renaissance just about 100% of the pictures of the Madonna and Child showed the child on the left: by the first quarter of the sixteenth century a significant proportion were holding the child on the right. The growing disbelief in Salk's theory as one proceeded through the galleries was a direct result of their chronological arrangement. Salk's error was in using as a data source a book entitled 'The Christ Child in devotional images during the XIV century'/(Shorr, 1954). i.e. the years 1300-1399 when indeed four-fifths of pictures do have the child held on the left side.

Such a highly significant shift in the mode of portrayal of the Madonna and Child is unlikely to be due to simple artistic conventions for one may demonstrate that there are no important differences either between schools (Figure 13:11), or, more importantly, according to whether the artist was great, good or simply indifferent (Figure 13:12). Why is there an asymmetry in the first place? and more importantly, Why is there a very significant temporal shift? Let us consider the application of the 'unlike-self, like-self' hypothesis. For convenience Madonna and Child portraits will be described by a three letter code (e.g. LRL) where the first letter represents the side of the child, the second the cheek shown by the

Madonna and the third the cheek shown by the Child.

At the beginning of the Renaissance the majority of Madonna and Child pictures were of the LRL type: the reasons for this will be discussed in the next section. From a Renaissance point of view however the LRL picture is unsatisfactory; unlike the case of the annunciation one cannot simply say that one or other figure is unimportant: both Madonna and Child were probably of equal importance during the Italian Renaissance. I would suggest that the transfer of the child from the Madonna's right to left side is a part of the solution to this problem. What is required is that both Christ and Mary should show their left cheeks. If Mary shows her left cheek as well as Christ then the result is an LLL portrait, which is unacceptable, for the mother is looking away from her child. If though the child is switched to the other side then a solution becomes possible. We now have an RLL picture. The Madonna is now looking at her child, but unfortunately this is countered by the child looking away from the mother, also pictorially unacceptable. This cannot be corrected by the child then looking to the right for this would render the child inferior in status to the mother. The final solution involves the artistic device of introducing another person or object into the RLL picture. If the child is holding an object such as a goldfinch, or book, in his outstretched right hand, then it is pictorially acceptable for him to look away from his mother. Alternatively a well placed saint

or donor in the left-hand foreground will produce the same result. If we look at the data then we may see this process occurring. In Figure 13:13 is shown the percentage of cases in which the child is showing the left cheek: as is to be expected this shows little shift through the period concerned. However the Madonna's cheek shows a very different pattern: there is a highly significant shift towards portrayal of the left cheek. The net result may be assessed by the 'total left cheeks' shown. This is an index of the artists' success at portraying both figures showing the left cheek. Figure 13:13 shows that there is a significant increase in the total left cheeks. That these shifts are occurring throughout Renaissance Italy, and for all classes of artists simultaneously, is shown in Figures 13:14 and 13:15. If we look in detail at the eight possible types of picture (Figure 13:16) then it is clear that there is a great shift away from the LRL and LRR portraits of the early Renaissance, toward the RLR, and more importantly, the RLL portraits of the Late Renaissance. It is also noticeable that in the final period considered, 1500-1549, there is a general tendency towards randomness in the mode of portrayal, the eight types becoming more equal in distribution. This may possibly represent part of the early decline into Mannerism, with its emphasis upon painterly qualities at the expense of the more profound meanings of the picture. (Blunt, 1973).

13:6 Mediaeval Pictures

The analysis of the previous section leaves a major problem for the hypothesis of 'like-self, unlike-self', Why does the Renaissance Madonna and Child start out, in 1250, with a predominance of LRL pictures? This question may be answered by considering mediaeval painting and theology. The crucial point is that during the period 600 to 1350 AD attitudes within the Catholic Church towards the Virgin Mary underwent a radical shift, the development of the Cult of the Virgin Mary (O'Connor, 1967; Lagarde, 1915).

A fundamental tenet of Catholicism involves the doctrine of original sin, which all persons may be regarded as having from the moment of conception onwards; furthermore, all may be considered to have shown actual sin during the course of their life. Initially the only exception to this rule was Christ himself, since he was conceived of the Holy Ghost. In particular, as St. Augustine (354-430) emphasised, the Virgin Mary was not an exception to the rule. During the 6th and 7th centuries the churches of Rome and Constantinople began to split further and further apart. About the year 700 AD the Eastern church began to celebrate a festival of the conception of the Virgin Mary: this festival did not in any way suggest that the conception of Mary was different from an ordinary conception as far as the doctrine of original sin was

concerned. During the early 11th century the Normans were in Sicily, where there were large numbers of Greeks celebrating the rites of the Eastern Church. A consequence of this was that the festival of the conception of Mary was brought to England, from whence it spread to France, and eventually to Germany and Italy. The presence of this festival caused some embarrassment in Rome, since by now it had become modified such that it was celebrating the Immaculate Conception of Mary, i.e. that Mary, from conception onwards, was exempt from sin, both original and actual. The church vigorously opposed this change and St. Bernard (c. 1140), in an attempt to prevent the development of the new doctrine, found justification for the elevation of Mary to the status of Saint. However, by now the doctrine was gaining a populist momentum and despite all the efforts of the thirteenth century doctors of the church, including St. Thomas Aquinas, it would not be stopped. In an attempt to neutralise the view, it was therefore argued that whilst subject to original sin, Mary was purified from the stain during her intra-uterine life. This was insufficient however, and by now mass opinion was being supported by some of the scholars, the most influential of these being Duns Scotus (1265-1308). The belief was then officially taken up by the Franciscans, and from then onwards it was only a matter of time before the Immaculate Conception became an official church dogma; this eventually occurred in 1854.

The effects of the Cult of the Virgin upon the production of works of art may easily be seen. Initially the Crucifixion was the corner-stone of religious art, being by far the single most common image during the Middle Ages. In Figure 13:17 is shown the ratio of the numbers of representations of the Crucifixion and of the Madonna and Child. It can be seen that during the period 500-1250 there were two to three times as many versions of the Crucifixion as of the Madonna and Child. Yet at the end of the thirteenth century, in the life-time of Duns Scotus, this changes dramatically and very many more Madonnas were painted than Crucifixions. These results can be seen as representing 'consumer demand' of the time, and the demand was clearly for the Virgin Mary.

The analysis of the development of the Madonna and Child during the Renaissance has depended upon the equal status of the Madonna and of the Christ-child: but up until the year 1250 this was not generally considered to be the case. We might therefore expect significant differences in the portrayal of the left and right cheeks during this earlier period. The data to be described here have all been obtained from the collections of the British Museum, London, and the Victoria and Albert Museum, London, and from three fairly comprehensive books on mediaeval art (Beckwith, 1970; Lasko, 1972; Rickert, 1954). It must be remembered that data from this period is quite rare due to the success of the iconoclastic movement during the 6th

to 8th centuries.

Let us consider first the crucifixion. Here, as during the Renaissance, the situation is simple. Christ is clearly the dominant, and often the only, figure, and is also very 'unlike self': he should therefore be portrayed showing the left cheek, and in 138 crucifixions of the period 500-1349 AD this is indeed so in all but four cases three of which show Christ full-face. Furthermore in those cases where the thieves are also portrayed (of which 10 examples were found) of the 19 thieves portrayed, 18 show the right cheek; this is similar to the Renaissance situation, although there is no evidence of differentiation between good and bad thieves.

In the Annunciation the dominant figure is Mary, and as in the Renaissance, since only one figure may show the left cheek, it has to be Mary. Of 46 cases of the Annunciation, the angel enters from the left side in 36 (84%) of cases. When we look at this data chronologically (Figure 13:18), it appears that the situation has taken a while to develop into its final form, suggesting that initially the resolution of the problem of two figures represented a difficulty, which by the Renaissance, had been well resolved. As in the Renaissance, for Annunciations other than to the Virgin Mary, there is no significant tendency for the angel to come in from the left (see Plate 13:2): of 8 such pictures, only 2 show the angel

entering from the left side (Fisher's exact test, p = 0.00399 one-tailed test).

The Madonna and Child again represents a more complex case. As in the Renaissance we have the problem of requiring, if possible, the child to look in the direction of the mother, and vice-versa. It is also necessary for the Christ child, who is theologically the more dominant figure, to show the left cheek. However unlike the Renaissance situation, the role of Mary is far less critical: whilst it would be desirable to have her showing the left cheek, this is not essential. An initial solution to the problem was to avoid all asymmetry completely, thereby producing the classic Byzantine type of Madonna and Child, as shown in Plate 13:3, in which the child is held centrally and both Madonna and Child are full-face (see Figure 13:15). However as the Middle Ages progressed this portrayal became less and less popular and instead the classic LRL pose developed. This pose solved the problem of showing the child's left cheek, and in consequence since the child ought to be looking at his mother, he was held on the left side and thus Mary was forced to show her right cheek. That Mary was showing her right cheek was of minor consequence since at this stage she was much less important than Christ: whilst it would be preferable if the situation could be avoided, it did not really matter. It is noticeable that in many cases Mary was painted full-face whilst the other asymmetries were retained, presumably to avoid

as far as possible showing Mary's right cheek.

It is ironic that at the very time the LRL solution to the portrayal of the Madonna was being reached, the Festival of the Immaculate Conception was just reaching England.

This data from the Middle Ages is therefore of exceptional interest. Not only can it explain the occurrence of the LRL type of Madonna and Child, at the beginning of the Renaissance, but it also shows how the portrayal of a left or right cheek may be sensitive to a change in the perceived status of an individual.

If my present interpretation of the crucial role of the elevation of the Virgin Mary is correct, then one ought to be able to make several predictions. Firstly in those branches of the Christian church which did not elevate the Virgin Mary should continue to show a predominance of LRL type Madonnas into the middle and late 2nd millenium AD. The Greek Orthodox Church never elevated Mary; scrutiny of the collection of paintings of the Byzantine Museum in Athens shows that almost all Madonna and Child paintings of this period are LRL, even though painted in the 16th to 18th centuries (the four notable exceptions to this rule were all of the Cretan-Venetian school - implying a strong Italian influence). The Coptic Church, and the Russian Orthodox Church, like

the Greek Church, never elevated the Virgin Mary: the collections of Icons in the Coptic Museum, Cairo, and in a series of books on Russian icons (no exact numerical data) all seem to suggest that the vast majority of such pictures are LRL in type.

A second prediction would be that in branches of the Catholic Church which denied the elevation of Mary, the mode of portrayal should revert to the more primitive form. Carey Wolfe (1978), in a project carried out under my supervision, investigated this possibility very carefully, in the paintings of Northern Europe produced before, during and after the Protestant Reformation. Before the Reformation there is no doubt that the trends in the mode of portrayal are very similar to those in Italy. However at the very moment of crucial interest, the number of pictures of the appropriate type, declines rapidly, due to the rise of humanism, and of a move away from the decoration of churches, so that the hypothesis becomes almost impossible to test properly.

Despite the lack of confirmation of an effect during the Reformation, the combined impression of these somewhat brief studies of the Greek, Coptic, Russian and Protestant Churches is that they give further support to the hypothesis of an important role played by the theological status of the Virgin Mary.

13:7 The Nature of the Left-Right Dimension

Whilst there are many ad hoc mechanical factors which might account for some of the left cheek phenomenon (see Appendix A13:1 for a resumé), I would suggest that no mechanical explanation can be entirely satisfactory because it is unable to account for differences in proportions of left cheeks dependent upon social or theological variables. Appendix A13:3 discusses a further case for which it is difficult to conceive of any mechanical explanation, but for which the 'like-self', unlike-self' hypothesis can deal moderately well. It is necessary therefore to try and decide quite what it is that is conveyed by the left and right cheek, and how.

Renaissance symbolism is very complex and became greatly hypertrophied with the passage of time. There are however no well-documented examples of left and right being of primary symbolic significance (although as shown in Appendix A13:4, there are interactions with other symbolisms). I would argue that the left cheek phenomenon is not a symbolism per se. On iconological grounds this would be due to the absence of a clear-cut, one-to-one relationship between signifier and signified (Barthes, 1967). We are not dealing therefore with a symbolism of the form exemplified by the well-known Lion of St. Mark, which represents either St. Mark, or, by derivation, the State of Venice: whenever the Lion (the signifier) is

present, one or other of these 'signifieds' must also be implied. No such relationship can be discerned with the left cheek phenomenon.

The possibility of a conventional symbolism may perhaps also be rejected on psychological grounds. The relation between signifier and signified is usually available to the conscious awareness of the subject, and even if it is not immediately available may usually be obtained relatively easily by simple experimental means. Thus, if we consider the case described by Barthes (1972) of the 'Roman fringe' or the 'Roman curl', a single, almost circular lock of hair conventionally placed on the forehead of Romans in modern films, a symbol which Barthes points out was not actually used by the Romans themselves. Whilst perhaps not directly aware of this sign, it soon became readily apparent to us when it is pointed out. If we were to be shown a series of faces, some with and some without Roman curls, and were asked, as naive observers, and with feedback, to divide them into Greeks and Romans, we would rapidly realise the cue we were using.

No evidence of such a process can be found in the case of right and left cheeks. In an experiment reported in Chapter 14 it was found that when subjects applied the semantic differential to portraits showing the left or right cheek, there was no evidence at all of differentiation in response according to the cheek shown. Similarly with neither

ordinary students (Blount et al, 1975), nor with art students (see Appendix A13:5) is there any evidence that subjects are able to correctly discriminate the original of a picture from its mirror-image. Neither is there evidence that subjects use either the cheek shown in portraits, or the direction of light in portraits, (see Appendices A13:2 and A13:5) as cues to the correct left-right orientation.

When we look in more detail at the results of the semantic differential experiment reported above we find that whilst there are no differences between the subjects' interpretations of left and right cheeks, we find that if we now look at the pictures in terms of the artists' use of left or right cheek in the original, as a function of the subjects semantic differential judgements, there is a clear differentiation of left and right cheeks, implying that the artist is attaching a different meaning to the two cheeks, a meaning which is not apparent to the subject of the experiment. Such a result immediately raises the question as to whether the artists, if acting as subjects in a similar experiment, would have recorded different responses to right and left cheeks; to this we have no answer.

The logical status of the left-cheek, right-cheek dimension is therefore rather difficult. It may be compared in some ways with grammatical rules, which very often are

not apparent to the users of a language but are used continually by them, and yet are readily available for inspection once a simple rule has been formulated: e.g. 'In English in sentences of the form Noun Phrase - Active verb phrase - Noun phrase, the first noun phrase is almost always the subject and the second noun phrase is almost always the object of the verb phrase'. And yet the analogy is not complete for there is evidence that ordinary subjects do not perceive the left cheek rule at all, only artists. It is perhaps more akin to a stylistic rule such as that which condemns the use of the split infinitive: split infinitives may be commonly observed in the speech and writing of ordinary people, and these people are often very bad at perceiving them,even when they are pointed out. Professional writers on the other hand rarely produce split infinitives, and thus our hypothesis of a rule governing the split infinitive would only apply to their literary output. Is the left cheek phenomenon simply then a stylistic feature (akin perhaps to a particular way of painting cupids floating in a Baroque heaven?). Probably not, for the simple reason that none of the artists appear to have perceived the rule themselves. The condemnation of the split infinitive is most vociferous from the writers themselves, and it is they who have recognised it: no evidence for such an insight on the part of the painters is apparent. My interpretation of the left-cheek, right-cheek hypothesis is that it is describing a rule which is usually sub-conscious, but frequently applied,

is in no way a simple stylistic convention, and may perhaps be understood in terms of a 'deep structure', akin to those which Chomsky proposes in language, and Levi-Strauss proposes in anthropology, not being directly accessible psychologically, but having to be inferred from the works produced. Whilst such an interpretation has many problems it can at least account for the stochastic nature of the 'symbolism' of the left cheek, it helps to explain the non-appearance of the phenomenon in ordinary subjects in an experimental situation, and also accounts for the occurrence of the phenomenon in artists of all grades of competence. Whether the presence of the 'deep structure' differentiates artists from non-artists, or whether perhaps the sense is merely more highly tuned in the artists is a question capable of empirical analysis. Thus one might for instance give feedback to experimental subjects according to their semantic differential responses to paintings and determine whether, without being explicitly told, they could detect, either consciously or sub-consciously, the existence of a left-cheek, right-cheek dimension.

Whilst I would argue that the left-cheek, right-cheek dimension possibly represents some form of 'deep structure', it is worth adding that this must inevitably be subject, in its expression, to the ordinary laws of psychology. Thus I would expect that artists would, as Gombrich (1960) suggests be actively experimenting to produce the desired effects,

the particular effects at a particular moment distinguishing the style of the period. We may find an example of this in the sketches of Michelangelo, sketch books being the artists' equivalent of the scientists' laboratory bench, a place where experiments may take place. We have seen earlier that right cheeks are very rare indeed in Italian Renaissance crucifixions, the incidence being about 0.68%. If we take an estimate of this two standard errors higher we obtain a figure of 2.03%. In Michelangelo's sketchbooks there are 10 drawings of Crucifixions, of which 3 show the right cheek. The probability of such a high occurrence of right cheeks, 3 out of 10, is very unlikely (exact $p = 0.0007$). The implication is that the artists are testing and sometimes rejecting the use of one cheek or the other. Regrettably Michelangelo produced no finished versions of the Crucifixion, for comparison. The failure of ordinary subjects on the semantic differential judgements applied to right and left cheeks might be primarily a result of a lack of experience of utilising the two cheeks to convey meaning. A further prediction of this 'experimentation' model of the use of cheeks, is that when artists decide to copy works by other artists they should preferentially choose those works which are more appropriate for the time: thus an Italian artists copying a Madonna and Child in 1300 should copy pictures of the type LRL significantly more often than would be expected.

Having discussed the formal nature of the left-cheek,

right-cheek dimension it is necessary to consider in a little more detail what is is that is being conveyed. Earlier on the definition was kept purposely vague, and the question deferred. The best insight into the dimension is perhaps given by the semantic differential experiment of Chapter 14. Here we find that portraits painted originally showing the left cheek tend to be more evaluatively positive, more dynamic and more spiritual (these three factors being extracted by factor analysis of the 20 dimensions of the original semantic differential). This is not however a satisfactory description. Firstly it is unlikely that three orthogonally separate dimensions should be reduced to a single dimension, and secondly these dimensions do not correspond in any useful way with the dimensions proposed earlier, 'like-myself, unlike-myself', and 'public-private'. Any further description of the dimensions must await further experimental investigation. Certainly from the difficulties encountered in finding the dimension and then probing it experimentally we may expect that the labels to be attached to it are not simple dichotomous terms such as good-bad, but are semantically more complex.

13:8 The Origin of the Left-Right dimension

What is the origin of the left cheek - right cheek dimension? Two major alternatives confront us: either it is inherent in the make-up of the human brain (as Chomsky would argue are the deep structures of human language), or

else it is acquired, presumably as the result of cultural influences. The origin of the dimension in no way alters its logical interpretation: thus it may have all the formal properties of a deep structure and yet still be dependent upon strong cultural influences for its expression (the existence of portrait painting being one such necessary cultural pre-condition).

If the dimension is in some real sense innate then we ought to be able to find wide-spread evidence of it. In this paper I have so far shown evidence for the differential usage of right and left cheeks over the period 600 AD to the present day. If the dimension were truly innate we might expect to find evidence for it in earlier historical periods, and also in different cultures. In the limited number of Roman portraits I have managed to examine, 10 showed the left cheek, 14 the right cheek, and 3 were completely symmetrical. Possibly with a larger sample size we might find evidence of a difference in cheek usage: alternatively, in view of Quintillian's comment that "in a portrait the full-face is the most attractive" (Inst. Orat., II, xiii, 12) we may not find anything. Too small an amount of Greek painting has survived to make any useful conclusions. In one area of left-right asymmetry which I have studied in Greek sculpture, the scrotum, there is evidence of left-right symbolism, but only in the ordinary sense of symbolism, being clearly related to the Pythagorean mystic traditions (see Chapter 12). Earlier still there is

evidence that the Egyptians differentiated between left and right in their wall-paintings, although whether this is symbolic is not clear (Dennis, 1958).

Cross-cultural evidence on the portrayal of left and right cheeks is almost totally lacking. Of 48 portraits of single Moghul emperors in the Victoria Memorial, Calcutta, 24 (50%) showed the left cheek, the proportions of left cheeks in paintings (n = 32) and engravings (n = 16) being identical; this result is however not large enough to be conclusive.

One piece of experimental evidence is however interesting. Children, if asked to draw a profile, draw it facing to the left in a very large proportion of cases. This effect is probably independent of handedness and also is shown as strongly by Japanese as well as American children (Jensen 1952 a,b).

If an innate interpretation of the phenomenon were required then one obvious candidate would be the right-left asymmetry of the brain itself, an account of which is briefly given in Appendix A13:1.3. At present the evidence is not strong enough to either accept or reject any innate origin of the left cheek phenomenon.

Even if there is an innate structure upon which the dimension depends we might also expect strong cultural

influences to be important. In Appendix A13:4 I have gathered together evidence on the possibility that the details of the Shroud of Turin, a cloth which purports to be that in which Christ was wrapped after his death, are the primary origin of all asymmetries in Western art, particularly those in the religious painting of the Italian Renaissance. In Appendix A13:5 I have collected evidence that shows that there is no reason to believe that the direction of light in paintings is a fundamental asymmetry to which all others are secondary, contrary to the suggestion of Coles (1974).

13:9 Conclusions

In this Chapter we have come a long way from the initial, deceptively simple, observation that portraits are more likely to show the left cheek than the right. In studying the phenomenon I have used methods which are unusual in studying art history, and these can probably find their origins in my medical training. The traditional approach to art history, the detailed study of the individual painting (or of the individual artist), can be likened to the doctor's study of the symptoms and signs of the individual patient. Medicine however found that one could not always make useful generalisations by studying the individual patient, and thus epidemiological methods developed; the manner in which I have studied painting in

this paper I would wish to call 'the epidemiology of art', attempting to sketch out the natural history of a style, and to follow its evolution. Later in medical history, and also in this study, we find the application of experimental methods, the manipulation of stimuli and subjects in order to clarify issues, 'experimental aesthetics' in this study. Whether such methods are justified in the study of art is a function of the results produced and the insights the methods provide into the subject-matter. Whether or not the left-cheek right-cheek hypothesis is itself a correct or useful approach to this data there is no doubt that an explanation of some sort is required for effects which are of enormous statistical significance, and cannot be interpreted just as the results of chance.

APPENDIX A13:1 MECHANICAL ARTEFACTS AND THE PREDOMINANCE OF THE LEFT CHEEK

In section 13:2 I briefly raised the possibility that the predominance of the left cheek might be a result of a mechanical artefact, perhaps related to the right-handedness of the artist. In section 13:3 I concluded that such an interpretation was probably untenable due to the impossibility of accounting for the dependence of the phenomenon upon social and theological variables. In this Appendix I will briefly consider some of the possible artefacts which must be borne in mind when analysing any left-right asymmetries in paintings, but which in my opinion cannot account for the main differences between left and right cheeks. The vexed question of the direction of light in pictures is considered separately in Appendix A13:2.

A13:1.1 The Right-handedness of Artists

Whilst a few famous artists such as Leonardo, Michelangelo and Holbein are often quoted as being left-handed, there is no reason to doubt that the majority of painters were probably right-handed and indeed more persons in pictures are shown as right-handed (Coren and Porac, 1977). As a result we might expect certain asymmetries to arise from their unimanual use of palette and brush. It has often been proposed that a right-hander will be able to draw

a left-cheek profile more easily than a right-cheek profile (e.g. J.S., 1870; Schapiro, 1969), presumably as a result of the sweep of the curve of the face coinciding with the locus of movement of the hand. Martin (1906) with adults and Jensen (1952 a,b) with children, have both found that when asked to draw a profile, the majority of persons will draw a left profile, Jensen's results showing this to be also true in both Egyptian and Japanese children, both of whom have very different styles of writing from American children. Jensen's results on left-handers were somewhat equivocal due to a small sample size; only 42% of 33 left-handed children drew a left-profile, as compared with 65% of 355 right-handed children. This difference is highly significant ($X^2 = 7.41$, $p < 0.01$, 1 df), although 42% is not significantly different from chance expectations of 50%. Whether therefore the tendency of children to draw left-profiles is determined by handedness per se is not clear.

The profile-painting tendency cannot explain the left-cheek predominance in paintings since the phenomenon is found only in full-face paintings and not in half-profiles or full-profiles (see Figure 13:2).

A13:1.2 A visual preference for the left-side of the face

If a preference existed, for whatever reason, for the left side of the face, we might expect a predominance of

left cheek paintings. The human face is not completely symmetric, the direction of asymmetry being dependent upon the handedness of the individual as far as the nose is concerned (Sutton, 1963), but being independent of handedness for the direction of hair-whorl (and hence the side of parting of the hair) (Lauterbach and Knight, 1927). The nasal asymmetry can probably be explained by the differential pull of the facial muscles on the two sides of the head during early development (Washburn, 1946). That faces are psychologically asymmetric was shown by Wolff (1933), and McCurdy (1949), who asked subjects to say whether the right or the left side of the face was more representative of the whole face; the right side (i.e. the right cheek) was regarded as more representative overall. Whilst thought by many individuals to be a reflection of the actual asymmetry of the face of the subject, Gilbert and Bakan (1973) have demonstrated that the asymmetry is in fact primarily in the eye of the beholder, the half of the face in the experimental subject's left visual field (i.e. the portrait's right cheek) being regarded as more representative overall. This tendency is independent of reading habits, and Gilbert and Bakan propose that the asymmetry is fundamentally of the cerebral hemispheres. Hilliard (1973) has clearly demonstrated that facial recognition is superior in the left visual half-field, and thus in the right cerebral hemisphere in the majority of individuals; similar results were found in children from 5 years upwards (Young and Ellis, 1976).

Whilst all these reasons might be excellent for assuming that an asymmetry might exist in the perception of paintings there does in fact appear to be no such asymmetry. Blount et al (1975) showed there to be no tendency to choose left cheeks in actual paintings as the preferred version, and indeed there was a slight tendency to prefer right cheeks, a feature also shown in a study using art-students (Appendix 13:5). The difference between these experiments and the ones described earlier is that our experiments used actual portraits which are invariably quite asymmetric, whilst those of Wolff et al used completely full-face pictures, a type very rare in painting.

In conclusion there is no evidence of a tendency to prefer left-cheeks in painted portraits, although there might be excellent a priori reasons for supposing such an asymmetry.

A13:1.3 Asymmetry of Visual Space

Perceptual space is not completely left-right symmetric. The left-half of the visual world projects predominantly to the right cerebral hemisphere, and the right-half to the left cerebral hemisphere. In the right-handed majority of the population these hemispheres subserve clearly different functions. The right hemisphere is primarily concerned with visuo-spatial analysis, whilst the left-hemisphere is specialised for verbal and language functions.

More specifically the right hemisphere has been shown (almost always in tests using tachistoscopic presentation of stimuli separately to the two half-fields) to be superior in the enumeration of dots, the localisation of dots, the detection of line slant and depth, and in the perception of some geometric forms (Kimura and Durnford, 1974), the fusion of Julesz-type stereoscopic figures (Carmon and Bechtoldt, 1969), and the discrimination of both hue and saturation of colours (Davidoff, 1976). Perhaps also of relevance to aesthetics is that the right hemisphere would appear also to show different emotional responses to the left hemisphere, tending to rate films as more unpleasant or horrific (Dimond et al, 1976). Some authors have also suggested that the two hemispheres differ in their affective states, the right hemisphere tending to be depressive, and the left to be hypomanic or euphoric (Gainotti, 1972).

Whilst most of these effects have been obtained by using tachistoscopic presentation of stimuli we might also expect some of the phenomena to be demonstrated during free, unrestricted vision, particularly in the edges of the visual fields. Of particular relevance to the perception of paintings are the findings of Adair and Bartley (1958) that objects in the left foreground (i.e. viewer's left) of pictures are phenomenally closer than those in the right fore-ground. That a psychological rather than a physiological explanation of this effect is required is shown by the finding of Bartley and Dottardt (1960) that objects

in the left and right backgrounds appear phenomenally equi-distant. That objects in the left fore-ground are also more salient is shown by Nelson and MacDonald's (1971) finding that when asked to choose a title for a picture, subjects tended to choose that one referring to the objects placed in the left half rather than the right half of the picture.

The relationship of all these perceptual asymmetries to the left-cheek, right-cheek phenomenon is not clear. They are possibly the fundamental asymmetry underlying the whole process, which determines its whole direction, although no positive evidence for this statement can be produced. Certainly none of these results can be used to explain away the left-cheek, right-cheek phenomenon; at best they can only explain its direction. Preliminary results suggest that there are no laterality differences in the aesthetic responses of right and left-handed subjects, but the result requires further confirmation.

The possibility also exists that when looking at faces individuals may use different scanning strategies. Yarbus (1967) in his Figure 115 shows beautifully that one particular individual scanned only the left side of a face. Walker-Smith et al (1977) found that of three subjects, one showed a strong tendency to concentrate on the right eye of pictures, one to look at the left eye, and the third to concentrate on the nose and mouth. Whether a directional asymmetry of such

gaze strategies exists is not clear, but may probably be discounted (Argyle and Cook, 1976; p 51). Lord and Haith (1974) found no systematic differences in the perception of gaze with either right or left eye.

A13:1.4 Asymmetry of the subject

Most of the discussion so far has centred around asymmetries of the artist and his perception (or perhaps conceivably of the asymmetries of the eventual viewer of the painting). However the subject of the painting also has his own asymmetries. The possibility of a facial asymmetry being of importance in determining the cheek shown has already been discounted (see A13:1.2 above) (see also Footnote 1).

Footnote 1.

An interesting and apparent exception to this statement is shown in the famous portraits of Federigo, the Duke of Montefeltro. He lost his right eye and badly disfigured the right side of his face in a jousting accident. In order to increase his nasal field of vision from the remaining eye he had the bridge of his nose partially cut away. The result is a very characteristic profile which, in the portraits by Berruguete and Justus of Ghent (in Urbino) and Piero della Francesca (in Florence and Milan) all show the left profile, with the characteristic hooked nose clearly visible. However the large portrait of Federigo by Piero, in the Uffizi, has, on its reverse, a painting of The Triumph of Federigo da Montefeltro, showing the instantly recognisable profile with the hooked nose, but this time showing the right cheek, which is apparently intact, although the picture was presumably painted at the same time as the painting on the converse. The implication is clear: Piero wanted, for compositional reasons, to show Federigo's right cheek in this picture, and so he simply produced a mirror-image.

An asymmetry of motor function is conceivable as a factor in portrait asymmetry. Gesell and Ames (1950) found that the tonic neck reflexes are very well formed in neonates, the tendency being for most of the infants to move their head so that they were looking over their right shoulder (i.e. to show their left cheek). This tendency has been further analysed recently and appears to be due to both motor and sensory asymmetry of the neonate (Turkewitz, 1977). How far this spontaneous turning tendency extends into adult life is not clear. Gesell and Ames (1947) found that the direction of turning correlated with later handedness. Motor asymmetries in adults are relatively common. Lynn and Lynn (1943) claimed that about 40% of individuals show a distinct asymmetry whilst smiling, one corner of the mouth rising earlier than the other. Kimura (1973) described how right-handed individuals use their right-hand far more for making free expressive gestures whilst talking, although this asymmetry does not apply for making self-touching movements. Gur and Gur (1977) have studied the phenomenon reported by Kinsbourne (1972) in which subjects tend to turn their eyes to the right if performing verbal tasks and to the left if performing visuo-spatial tasks. They found this to be only a useful test of hemisphere dominance if subjects were facing away from the experimenter, and thus in a non-anxiety provoking situation. If eye-movements were recorded whilst subjects faced the experimenter then the direction of gaze indicated not the mode of cognitive task, but rather a personality difference

between subjects, left-movers being for instance less susceptible to hypnosis and more prone to psychosomatic symptoms.

Whilst it is conceivable that some of the variation in left and right cheeks shown in pictures might be attributed to subject motor asymmetries, this really does seem somewhat unlikely. A confusing factor which must be remembered in these studies is that if one postulates that, say, in right-handers, the muscles of the right side of the body are all just a little stronger than those on the left then the pattern for head-movements is difficult. The main muscle for making sideways movements of the head is the sterno-cleido-mastoid. However stimulation of the right sterno-cleido-mastoid results not in movement to look over the right shoulder but to look over the left shoulder (i.e. to show the right cheek to the world).

In summary it is difficult to see how these motoric asymmetries could reasonably account for the left-cheek, right-cheek data.

A13:1.5 Asymmetries of conventions in painting style.

The main asymmetry to be discussed here is that proposed by Professor A. Green, and discussed in Corballis and Beale (1976). Professor Green has pointed out that there was a definite tendency for pairs of portraits to be painted,

so that, typically, the husband would be hung on the left, the wife on the right, and the two would look inwards, towards each other. Since they thus show opposite cheeks, it is reasoned that we might expect a difference between male and female portraits in their proportions of right and left cheeks.

I have re-examined the Rembrandt data to look at the paired portraits. Of those which are known to have been paired, the vast majority are indeed in the manner described by Green (Table A13:1.1). When we now consider the data previously described in Figure 13:6 we find that the main trends are not substantially altered by removal of the portraits known to have been paired (Table A13:1.2). The difference between kin and non-kin is not quite significant at the 5% level, but this is primarily due to a reduced sample size. The main trends are still apparent. Corballis and Beale suggest thatthe trends shown in Table A13:1.1 are 'carried over even to portraits hung singly, and almost certainly applied to the portraits painted by Rembrandt . This hypothesis is however not sufficient to explain all of the left cheek data. It would predict that there would be a trend for female portraits to show the left cheek (as is the case) but would also have to predict that there would be a <u>right</u> cheek predominance for male portraits: this is not the case for Rembrandt's male non-kin portraits, 45.7% showing the left cheek, which is not significantly different from a chance expectation, $X^2 = 0.78$, 1 df).

It is certainly not the case for most male portraits by other artists (see section 13:2). Further evidence against the hypothesis (which comes close to being impossible to disprove), is that the production of paired portraits probably developed in the mid-Renaissance (Francesca's portraits of the Duke and Duchess of Urbino (see Footnote 1), possibly being one of the earliest pairs); the differential usage of right and left cheeks probably occurred at a far earlier stage than this, going back at least to the middle ages. The only portrait precedents for the mediaeval artists were classical sculptures, and possibly some of the Graeco-Roman and Coptic wax portraits used for the lids or coffins and sarcophagi. Whilst it was possible these were painted in pairs (one example being in the Naples museum, inv.9058, and which incidentally shows the man showing his left cheek and the woman her right), it is highly unlikely , on stylistic grounds, that the mediaeval artists were aware of these portraits.

A13:1.6 Summary of Appendix A13:1

Most of the available evidence on possible left-right asymmetries which could act as artefacts to confuse the left cheek phenomenon have been considered. None of them are capable of explaining away the data presented in the main part of this paper, except perhaps with an unreasonable degree of ad hoc elaboration and multiplication of hypotheses. It is however possible that the asymmetries of

the cerebral hemispheres in some way relate to the underlying asymmetry which originally gave rise to the left-cheek, right-cheek dimension.

APPENDIX A13:2 THE DIRECTION OF LIGHT IN PICTURES

Coles (1974) documented the tendency for light in Western pictures to come from the left-hand side (that is, as if the source of illumination were placed to the left of the viewer), although the tendency had been recognised earlier (e.g. Gombrich, 1960). Coles suggested that the direction of the light was the primary asymmetry in portraits and that the cheek shown was only secondary to this, the left-sided light acting as a reference point against which the turn of the subject's head could, as it were, be titrated to give different degrees of 'openness'.

Of the finding that light tends to come from the left there seems to be little doubt. Thus in the 340 English Icon paintings from Strong (1969) I found that 208 (61.2%) showed the light from the left, 9 (2.6%) the light from the right, and in 123 (36.2%) the direction of the light was either indeterminate, or in a few cases, actually contradictory. This last group of portraits, with indeterminate or contradictory light, represent a test of Coles' theory. If the direction of light were indeed truly primary then in this group we would expect 50% left cheeks, and 50% right cheeks. Table A13:2.1 shows that this is not the case, a definite left cheek tendency still being apparent.

The group of Italian Renaissance portraits (Berenson and Pope-Hennessey, 1966) also show a strong predominance of

left-sided light, but have a far lower proportion of indeterminate pictures: of 272 portraits, 219 (80.4%) showed light from the left, 45 (16.5%) light from the right, and only 8 (2.9%) were straight on, indeterminate or contradictory. When we analyse these data further we find, in Table A13:2.2 for both male and female subjects, a significant interaction between the cheek shown and the direction of the light; this direction is in the same direction as that suggested by the limited number of pictures for the English Icon. The interaction is however in the opposite direction to that shown by Coles: far from being less common than might be expected by chance, the right-cheek, right-light group are more common. In these portraits therefore the light is arranged so that it tends to shine directly into the face of the subject rather than passing across the face at a glancing angle, and when we analyse the data in these terms we find no interaction with the direction of the light A13:2.3. The pictorial utility of such a convention is obvious.

The difference between this data and that of Coles is very important. If we take the combined data of myself and Coles (Table A13:2.4) then we find that whilst there is still a significant interaction between the direction of the light and the cheek shown (albeit in the direction described by Coles), this interaction is only just significant with probability less than 5%. Considering the sample size of 1245 this suggests a fairly

mirror effect and I would suspect that the addition of more data to it might well make it non-significant. Coles' hypothesis, that the direction of light is the primary assymetry, would thus appear to be untenable. That however the direction of light is important in some way is indisputable for the differences between my results and those of Coles are themselves highly significant. For this I can at present offer no explanations.

The problem remains of why the light in pictures should come from the left side. Gombrich (1960) claims that "Psychologists have found that in the absence of other clues, Western observers have settled for the probability that the light falls from high up and from the left-hand side". Whilst accepting that there is *prima facie* evidence for his statement (e.g. from the typical colouring of maps, the mis-interpretation of hills and valleys in inverted moon photographs, etc.) I can find no reference in the psychological literature to support the claim, certainly not in Gombrich's quoted reference (Gibson, 1950). I intend to carry out experiments to test the hypothesis.

Gombrich's hypothesis suggests that the reason for light coming from the left is an innate perceptual tendency. It is however possible that there are also cultural influences, or perhaps just simple stylistic differences, associated with different schools of art. Thus Rembrandt painted a far higher proportion of paintings with light from the left

(95.9%) than did Reynolds (64.3%), the difference being statistically highly significant (X^2 = 70.9, 1 df, $p < 0.001$; data taken from Coles, 1974). When we consider data from the Italian Renaissance we find a similar effect. Figure A13:2.1 shows for the four Italian Schools, the percentage of pictures with light coming from the left. There is no difference between the Venetian and Northern Schools, and the Central and Florentine schools are marginally different, but the difference between the combined Central and Florentine schools and the combined Venetian and Northern schools is highly significant. For the case of the Madonna and Child pictures, Figure A13:2.2 shows the historical changes for each of the four schools separately; the differences between the schools are not sampling artefacts due to the inclusion of more pictures of a later date for the Venetian school, although no simple relationship is discernable.

Figure A13:2.3 shows the data on the direction of light for the mediaeval crucifixions, the only group for which sufficient mediaeval data could be obtained. The majority give no intimation of a source of light, and it is clear that it is a Renaissance innovation, associated presumably with the drive to greater realism. Within the Renaissance itself there is a distinct trend to produce less pictures in which the light is indeterminate, for both crucifixions (Figure A13:2.3) and also for the Madonna and Child (Figure A13:2.4). The use of left sided light at the

beginning of the Renaissance is however immediate, there being no transitional phase in which a racemic mixture of left and right sided light was produced (e.g. in Figure A13:2.3). Figure A13:2.4 shows that with the Madonna and Child figures there is a significant trend towards using light from the right more as the Renaissance progresses. This is not however the case with the Crucifixions (Figure A13:2.3). Since with the Crucifixions we find no long-term trend in cheek shown, left being shown for the entire time, and yet with the Madonna and Child we find significant variation of portrayal, we might expect that the two phenomena might be linked, the direction of light being secondary to the cheek shown. The correlation is not however simple, for with the increasing proportion of left cheeks in the Madonna and Child portrait during the Renaissance we find a decreasing proportion of left-sided light. Figure A13:2.5 explores the relationship between light and the various facets of the Madonna and Child, in more detail. For Madonna and Child pictures with light coming from the left we find the highly significant chronological changes already described (section 13:5). For those Madonna and Child portraits with light coming from the right we find statistically significant chronological trends only in the case of the Madonna's cheek: nevertheless there is a distinct trend in the case of both Madonna's cheek, and the side of the child, for the chronological change to be in the opposite direction to that of the pictures with the left light. Such data are not easy

to interpret. If indeed the trends shown in 'Side of Child' and 'Madonna's Cheek' for right light are the reverse of those shown for left light then this would be good evidence that the light is responding secondarily to the cheeks shown in the painting. In the absence of convincing statistical proof we are left in a limbo. Certainly these data do not seem to strongly support, in any way, the contention that the direction of light is the primary asymmetry in such pictures.

In view of the remarkably rapid left-light predominance at the beginning of the Renaissance it is necessary to consider one other possible reason for the origin of the light direction. In Appendix A13:4 I have considered some of the symbolism of the Crucifixion and suggested that much of this might be secondarily related to the actual wound received by Christ at his Crucifixion and recorded in the form of the Shroud of Turin. From this asymmetry, of the face and chest wounds, we might also expect a tendency for light to come from the left side. We know that Christ died late in the afternoon and thus we might expect (particularly given the views of Molanus, quoted in Appendix A13:4)that Christ would look towards the setting sun in the west, the west traditionally being placed to the left-hand side. Alternatively it could be argued that since Christ had to show his left cheek (either for iconological reasons or due to a bruise on the right cheek) then the light had inevitably to come from the left

side, for the light represented the Sun, and hence good, the Church, etc. Given the existence of such an asymmetry in Crucifixions we might expect it to rapidly generalise to all picture types.

In summary therefore I would propose that light tends to come from the left in Western paintings for one (or possibly all three) of the following reasons:

i. Because of a basic perceptual asymmetry which assumes that light will come from the left.

ii. Secondarily to other asymmetries such as the portrayal of cheeks, and of the side of holding of the child in Madonna and Child figures.

iii. As a consequence of the symbolism attached to the Crucifixion, which might find its origins in the actual events of the Crucifixion, as transmitted through the Shroud of Turin.

APPENDIX A13:3 ASYMMETRY IN ITALIAN RENAISSANCE POLYPTYCHS

An extremely popular type of picture in the early Renaissance was the large polyptych altar peice; in the later Renaissance this transmuted into a large single picture in which there was a central item (Madonna and Child, local saint, etc.) with pairs of figures, usually saints, symmetrically placed to either side of the main figures: the archaetypal picture is the 'Madonna and Child with Saints'. Of the approximately 6000 pictures in Berenson, 605 consist of this type of picture, in most cases with the Madonna as the central figure. To any theory of left/right symbolism the polyptychs pose an immediate challenge in that they have a very strong inherent symmetry. Thus for instance in Plate A13:3.1, whenever a figure to one side of the mid-line shows one cheek, the corresponding mirror-image figure shows the opposite cheek.

Within this system asymmetry does however occur. Plate A13:3.2 shows an example which is not totally symmetric as far as the cheeks are concerned. Figure A13:3.1 shows the total number of figures within such polyptychs. 27.4% of polyptychs show a symmetry 'error' of some sort in the cheeks, the vast majority showing one, or occasionally two, errors. (It is not of course possible, on logical grounds, to say which of the two figures is in error). 14.28% of saint-pairs show an error (Figure A13:3.2).

The theory of 'like self-unlike self' has difficulties with polyptychs for its small effects will tend to be masked by the stronger effects induced by simple symmetry considerations. Nevertheless we may find evidence for its existence. We may regard the saints as different from self in so far as they represent an idealised self, and we may expect a tendency for saints to show the left cheek, although in so far as the saints are still human we would not expect 100% left cheeks. The symmetry of the polyptychs, if applied strictly, would prevent this predominance of the left cheek. Symmetry is not however strict, for we have found frequent errors. I would postulate that these errors are attempts to portray more left cheeks and thus to correctly place the saints on the spectrum of 'like-unlike self'. If this is so we would expect more errors resulting in two left cheeks being shown than those resulting in two right cheeks. Figure A13:3.3 shows that this is indeed the case, although it is only so for the Florentine and Central Italian Schools (a finding which resembles the difference in schools described in Appendix A13:2). The difference between schools may possibly be artefactual, being simply a function of the chronological distribution of the works. Whilst this result is not strong evidence for the theory of 'like self-unlike self', it is at least compatible with it: an explanation in any other terms would be very difficult to produce. The errors themselves, of either type, are equally distributed between polyptychs of all sizes.

The polyptychs also provide a useful role in testing the theory further and for demonstrating that the hypotheses it creates are capable of empirical disproof. In view of the symbolism of all sorts which is rife in Mediaeval art, we may expect that strict rules would exist for the painting of the saints to the left or right of the centre of a polyptych; in terms of my own hypothesis it woudd seem likely that the most important saints (perhaps defined operationally as those portrayed most often) would be perceived, and hence depicted, as being closer to Christ, and thus showing the left cheek more frequently. Table A13:3.1 shows, perhaps surprisingly, that the saints are placed randomly to left or right of centre, and show no tendency for the more important ones to show the left cheek. Table A13:3.2 shows the fifteen most commonly portrayed saints and the number of occasions they are placed to right or left, and the cheek shown. None of the differences are greater than chance expectations.

APPENDIX A13:4 THE ICONOGRAPHY OF THE CRUCIFIXION

This subject is complex: nevertheless, as Réau emphasises, iconographical hypotheses must be open to testing in the same way as any others, for theories of symbolism are easy to beget but difficult to destroy.

The Crucifixion is central to the present study for it is the clearest example of a left/right asymmetry. Its central figure, Christ, represents one end of a spectrum, the other end of which is the artist himself: any individual may then be placed along this spectrum. The main question therefore is, Why does Christ show the left cheek in almost every example of the Crucifixion? There are, inevitably, several subsidiary questions regarding the portrayal of asymmetries in the Crucifixion. Réau (1957) describes how the left side of the picture (i.e. on Christ's right hand) represents the Church, whilst the right is the Synagogue. As a result Mary stands to the right of Christ and John to the left. The spear which made the wound in Christ's side is held in the left half of the picture, for the spear signifies the solider, St. Longinus, who inflicted the wound and later became converted to Christianity, whilst the vinegar-soaked sponge represents the mocking synagogue and is thus held in the right half of the picture. As a corollary churches are built on an east-west axis with the altar at the east

end (footnote 1); hence on entering a church and looking along the nave, the north (equated with darkness, cold, the devil and the synagogue) is to the left, whilst the south (and light, warmth and God) is to the right. The Old Testament frescoes are placed on the north side of the aisle and the New Testament frescoes on the south. This north-south symbolism extended so that in some French churches the men sat on the right side of the nave and the women on the left (or north). In mediaeval crucifixions we also find the sun shown to Christ's right and the moon to his left. In Dante's Divine Comedy the synagogue is placed in the Northern Hemisphere, whilst the church is placed in the Southern Hemisphere (Purgatory, IV, 76-84). In Paradise Lost there are several references to the Devil coming from the North (V, 689; V: 726; VI: 79); Fowler (1971) has suggested that all of these associations derive from a patristic interpretation of the text of Isaiah, xiv, 13-14.

The two thieves, who are often portrayed alongside Christ, have traditionally been divided into good and bad. In about one third of such pictures examined (7 out of 18) there is definite pictorial evidence that the thief on Christ's right hand is the good thief whilst in the others there is no evidence, or only dubious evidence, for such a differentiation. In the gospel accounts there is no mention of the good thief being on the right hand (Matt., 27, 38; Mark, 15, 27; Luke, 23, 39; John, 19, 18). From evidence

of cheeks shown by the two thieves (sections 13:4 and 13:6) it would appear that the differentiation into good and bad is a Renaissance phenomenon.

The spear wound in Christ's side, if present, is invariably present in Christ's right side (Footnote 2). The evidence for placing the wound in the right side is not clear. The biblical account (John, 19, 34; 30, 24-7) makes no mention of the side of the wound, we are merely informed that "forthwith there came out blood and water", an event for which various theories have been proposed; these include pericardial effusions and a haemohydrothorax, but none is totally convincing, and certainly do not unequivocally locate the wound on the right side. It may be that the spear-wound is placed on the right side because 'spear' in Roman military usage was a synonym for right (and 'shield' for 'left', cf. Paradise Lost, iv, 785; "Half wheeling to the shield, half to the spear", Fowler, 1971).

The 'Doctrine of the Concordance of the Testaments', by which it is claimed that for each event in the New Testament there is an equivalent one in the Old Testament, is of some relevance to the positioning of the spear wound. The wound may be related, by analogy, to Moses striking the river with his stick and turning it to blood (Exodus 7, 20), to the removal of the rib from Adam's side to fashion Eve (Genesis, 2, 21), or even to the killing of

Asabel, Abner, Ish-bosheth, and Amasa, in each case under the fifth rib (II Sam., 2, 23; 3, 27; 4, 6; and 20, 10). However none of these events unequivocally describe the right or the left. Indeed the use of Adam's right or left rib seems to be confused. Thus in Lucas Cranach the Elder's Das Paradies of 1530, in the Kunsthistorisch-esmuseum, in Vienna, Eve is clearly being created from Adam's right side. In 1608, Andrew Willett, in his Hexapla wrote "It is a superficial question out of what side of Adam, Eva was taken ..... It is resolved by most, out of the left because Adam's heart lay there; but these are frivolous and needelesse matters". Nevertheless, Milton thought the matter sufficiently important to refer to it twice in Paradise Lost (IV, 484; VIII, 465). To confuse the symbolism even further, Milton also states that during the battle between Michael and Satan, the former's sword enters the devil's right side (PL, VI, 327).

The biblical evidence has so far given us no reason for the particular portrayal of left and right in the crucifixion. The most difficult, but by far the most challenging and interesting explanation of the asymmetries of the Crucifixion is that they find their origins in the actual events of Christ's Crucifixion. Their universal occurrence, through all ages, suggests, even if tradition is a secondary factor, that some primary orienting event of great moment must have occurred to account for these asymmetries. Two separate lines of evidence may be

followed, one more useful than the other.

The positioning of the Cross on Golgotha, and the direction faced by Christ, are not told us in the Gospels. However, Molanus in 1547, in a volume produced by decree of the Council of Trent, in which were set down rules for the painting of all religious pictures, tells us:-

> "He was crucified (and therefore should be portrayed) with his back to Jerusalem and the East, looking towards the West, with his right to the north and his left to the south ..... In this position also he ascended, turning his eyes towards the West, and over the Roman Church set in the West, to which he was about to send his chief apostles Peter and Paul, and where he was to keep his pact with Paul".
>
> (Molanus, 1547).

The evidence for these statements is not given, but in conjunction with the recent discovery and excavation of Golgotha, it may well provide useful information.

The most important evidence of all is the Shroud of Turin (Plate A13:4.1). This remarkable piece of cloth is kept in the Cathedral at Turin, and is claimed to be the actual Shroud in which Joseph of Arimathea wrapped the body of Christ. As a result of an ill-understood chemical reaction between the embalming fluids and blood and sweat upon the body (Lancet, 1902) we are left with a sort of 'natural photograph' in which many of the details of the body may be seen. The shroud itself has had a chequered

history, being partially destroyed by fire on one occasion. Its first definite historical identification is at Chambery in the mid-15th century, although Green (1969) has provided good evidence, on iconographical and historical grounds, for its existence at least back to the sixth century, and by implication back to the time of Christ himself. There is no reason to doubt at present that the Shroud is anything but entirely authentic, although further credence will be given to this point of view by the carbon-14 tests being undertaken at present.

For the purposes of this study the Shroud provides several important items of evidence. Firstly there is a large wound between the fifth and sixth ribs on the right side, which is compatible with a spear-wound, and, according to Bucklin (1970), "There is a sign of another fluid which has been mingled with blood". Secondly, on the face, corresponding to the right cheek, there is a swelling of the malar region which has resulted in partial closure of the right eye (Plate A13:4.2). This may perhaps be more convinving in the reconstructed three dimensional image of Plate A13:4.3.

In view of the evidence described above, I would propose that the most reasonable theory of the iconography of the crucifixion is as follows. Christ is portrayed with a spear wound in his right side because the wound actually was in his right side. His left cheek was portrayed because

the right cheek actually was badly bruised (a process for which a classical precedent has been set in Appelles' portrait of the one-eyed King Antigonus; Pliny, Nat. Hist., XXXV, xxxvi, 90; Quintillian, Inst. Orat., II, xiii, 12). From these two primary asymmetries all others follow as secondary processes. The spear which produced the wound is placed in the left half of the picture, the good thief, to whom Christ would be preferred to be looking, is to his right, and the light of the afternoon sun, to which Christ would alsobe looking, comes from the left side of the picture. Evidence for the subsequent, and contingent, development of the secondary asymmetries lies in the facts that light only begins to come from the left side toward the beginning of the Renaissance (Figure A13:1.3); the thieves are portrayed in only 8% of mediaeval crucifixions and in many cases there is little evidence of any differentiation between the two; the spear, ultimately held on the left side only is initially, in a few early pictures, present on both sides and even in late mediaeval crucifixions is present in only 30% of Cases (Figure A13:.4.1). The secondary asymmetries probably represent a result of the Mediaeval and Renaissance obsessions with the symbolic meaning of objects. Such an approach also allows for the apparent inconsistencies in symbolism; the left half of the Crucifixion represents both West (the light and the setting sun) and also South (the Church, warmth, light, and the 'good' thief), and yet in Churches, Christ in an altar Crucifixion inevitably looks away from the New

Testament (the south) and towards the Old Testament (the north).

The transmission of such iconographical knowledge presents an epistemological problem that is most parsimoniously solved by taking the hypothesis of Wilson (1978) that the Shroud of Turin is identical with the Image of Edessa, for which there is good historical evidence from 544AD, and which left Constantinople in the thirteenth century. This holiest of relics, the 'True likeness', was probably shown to only a handful of artists in Constantinople in the sixth century. This image we may then speculate was transmitted down through generations of artists, the accuracy of this process being assisted by the strict canons of Byzantine art. By the Renaissance these details would have become tradition, a tradition which for most artists there was no rational explanation.

Appendix 13:4 Notes

1. It is worth pointing out, en passant, that the well-known deviation between the axis of the nave end of the apse in many churches, which was once considered to be portraying the inclination of Christ's head on the Cross (Lasteyne, 1905; Anthyme, 1906), is almost certainly not iconographical in origin. Reau (1957) considered the deviations to be probably random, and the data of Cave (1950) support this suggestion. Wordsworth, quoted by Benson (1975), felt that deviations from true east were due to churches being aligned "by the point in the horizon at which the sun rose upon the day of the saint to whom the church was dedicated". Benson attributed internal deviations within a church to the misalignment of the Julian calendar and the true year. An alternative, and possibly more convincing,hypothesis has recently been proposed by Searle (1974), who has shown that the deviations are probably a function of the shifting of the magnetic north pole and hence the use of different east-west axes whilst building different portions of the church.

2. An interesting exception to this is in Carpaccio's 'I diecimila crocifisi del Monte Ararat' in the Accademia, Venice, where there are a score or more crucified figures, in some of whom the spear wound

is on the left side: of course there figures are not portraying Christ himself. Other occasional exceptions do occur. The Rubens Descent from the Cross, in the Courtauld Institute Gallery clearly shows a left-sided spear wound. But this is within Western Europe, unique, as far as I am aware. Whilst in Cochin, in Kerala, Southern India, a while ago, I noticed a locally carved wooden crucifix with a left-sided wound. Further investigation in the local Cathedral of St. Thomas showed that of 8 crucifixions, two had left-sided wounds and seven had right-sided wounds. The two left-sided wounds were painted on to walls, and were clearly produced locally, whilst the right-sided wounds were all carved crucifixes and looked as if they had probably been imported from Europe. The Church of Southern India was founded circa 60 AD, possibly by St. Thomas (the doubter) himself, and thus the local tradition of a left-sided spear wound might perhaps represent a tradition founded before knowledge of the Shroud became extant. It may of course also be argued that Thomas was perhaps in a better position than most to know the side of the spear-wound, having placed his fingers into it.

APPENDIX A13:5 THE DISCRIMINATION OF ORIGINAL AND MIRROR-IMAGE REPRODUCTIONS OF WORKS OF ART BY ART STUDENTS

In a previous paper (Blount et al, 1975) we have described the results of an experiment in which subjects were unable to discriminate the original from a mirror-image reproduction of a painting unless they had seen the picture before. We have since repeated the experiment using art students and including pictures with known asymmetries.

Fifty subjects, all of whom were studying fine arts at University or were at Art School, were each shown sixty pictures by means of a pair of slide projectors. The original version and a mirror-image were displayed side-by-side, the side of the original being randomly ordered. The subjects were asked to indicate on a 6-point rating scale (Strong, Medium, Weak for each version) which picture they thought was the original, and on a four-point scale (Never, Possibly, Probably, Definitely) whether they had seen the picture before. In other respects the conditions were the same as in the previous paper.

One picture was excluded after the experiment was completed as it inadvertently contained writing. Some subjects omitted to check all responses.

Of the 2947 choices of original (out of a possible 2950),1512 (51.30%) were correct: this is not significantly better than chance (X^2 = 2.03, 1 df). Of the 1495 cases in which the picture had never been seen before, 752 (50.30%) were correct (X^2 = 0.054, 1 df, NS). Analysis of responses by individual pictures and individual subjects, as in the previous experiment, showed no evidence of individual differences beyond the expectations of chance. Of particular interest is the analysis of the different types of picture. Twenty-three of the pictures were a miscellaneous collection of portraits, male and female, right and left cheek. Figure A13:5.1 shows that there is no evidence for subjects being able to detect the original by any simple rule of thumb; in the previous experiment there was a slight, although not significant, tendency for the right cheek to be chosen rather than the left.

Eight of the pictures were Rembrandt portraits, chosen for the direction of light, four having the light from the left and four from the right. These were chosen in view of Coles' (1974) analysis of the direction of light in Rembrandt portraits. Figure A13:5.2 shows that subjects made no use of knowledge of the direction of light in choosing the original version, although there is a non-significant tendency to choose pictures in which light comes from the right.

Seven of the pictures were Italian Renaissance Annun-

ciations, all of the usual type with the angel entering from the left. Four of the pictures were Italian Renaissance Crucifixions, all showing the usual portrayal of Christ's left cheek. Figure A13:5.3 shows that there is no evidence for detection of original version of these pictures beyond chance expectations.

At the end of the experiment subjects were asked to record the features they were using to choose the original version. Fourteen of the fifty subjects mentioned some aspect of light or shadowing, eleven mentioned memory of the pictures, ten mentioned 'Instinct', 'Intuition', or a feeling that the picture was 'just right', and seven admitted that they were guessing. One subject stated, "Crucifixions - Christ just had to face right", and analysis of his results showed that "right" meant right cheek in the same sense as in this paper. Another subject commented "Direction of looking - portraits usually to left", whilst a third commented, "People very rarely pick up babies in L.H.", by which it is presumed is meant Left Hand.

In general we may conclude that whilst some students are willing to admit that simple rules might exist for carrying out this task, and some have formulated such rules (which are wrong in some cases), in the majority of cases there is no evidence for knowledge of even the very simple rules governing pictures like the Annunciation and the Crucifixion.

(The experimental work described in this Appendix A13:5 was carried out by Ms. P. Blount, then a final year student at the University of Reading, under the joint supervision of myself and Professor M. Colthart. The original design of the experiment was entirely my own, as was most of the statistical anslysis: hence the inclusion of the work here).

Table A13:1.1 Cheeks shown in Rembrandt's known paired portraits.

		Left Cheek	Right Cheek
Sex of Subject	Male	3	26
	Female	24	2

All linked portraits are of non-kin individuals. The difference between males and females is highly significant. Numbers of males and females are not the same due to some members of pairs being lost.

Table A13:1.2

Analysis of cheeks shown in Rembrandt portraits after exclusion of all cases known to be members of linked pairs. Compare results with those of Figure 13:6. Differences between kin and non-kin are not quite significant (X^2 = 5.49, 2 df, $0.10 > p > 0.05$).

	Left Cheek	Right Cheek	% left cheek
Self-Portraits	9	48	15.7%
Male Kin	3	14	17.6%
Male Non-Kin	58	69	45.7%
Female Kin	22	17	56.4%
Female Non-Kin	28	12	70.0%

Appendix 2: Tables

Table A13:2.1 The cheek shown by the direction of light: 'English Icon' portraits from Strong (1969).

Direction of Light	Cheek Shown: Left	Cheek Shown: Right	N	%L	X^2
From left side	141	67	208	68%	6.25 $p < 0.01$
Straight on or indeterminate	99	24	123	80.2%	
From right side	4	5	9	44.4%	

Table A13:2.2 Cheek shown by direction of light and sex of subject. Data from Berenson (1957, 1963, 1967, 1968).

Direction of Light	Cheek Shown: Males Left	Males Right	Females Left	Females Right
From left side	157	62	55	16
From right side	18	27	5	16

$X^2 = 16.7$, 1 df, $p < 0.001$ $X^2 = 20.56$, 1 df, $p < 0.001$

Table A13:2.3 Data of Table A13:2.2 re-arranged.

Direction of Light	Direction subject facing with respect to source of light: Males — Towards light	Males — Away from light	Females — Towards light	Females — Away from light
From left side	157	62	55	16
From right side	27	18	16	5
	X^2 = 2.41, 1 df, NS		X^2 = 0.014, 1 df, NS	

Table A13:2.4 Data of Coles (1974) combined with data from Tables A13:2.1 and A13:2.2.

Direction of Light	Cheek Shown: Left	Right
From left side	598 (48.03%)	463 (37.18%)
From right side	120 (9.63%)	64 (5.14%)

X^2 = 4.68, 1 df, $p < 0.05$

Table A13:3.1 Position of saints to left or right of centre and cheek shown, by number of portrayals of saint (data from Berenson, 1968). Note: totals are not all consistent since, for various reasons, it was not possible to classify all saints on all categories.

Number of portrayals	Number of different saints	Position with respect to mid-line				Cheek Shown		
		Left	Centre	Right	$\frac{L}{L+R}$ %	Left	Right	% L
1	56	21	1	26	44.6%	29	23	55.7%
2	14	12	0	12	50%	13	15	46.4%
3	8	10	0	11	47.6%	15	9	62.5%
4	5	4	0	12	25.0%	12	8	60.0%
5-9	8	14	1	32	30.4%	31	23	57.4%
10-19	12	76	3	61	55.7%	69	92	42.8%
20+	7	110	7	103	51.6%	119	105	53.1%
Difference between groups in proportion of right and left		$X^2 = 4.001$, 6 df, NS				$X^2 = 6.77$, 6 df, NS		

Table A13:3.2 Portrayal of the fifteen most commonly occurring saints in Italian Renaissance Polyptychs (Berenson, 1968).

Total number of polyptychs = 229. Note: tables are not all consistent in their totals due to some pictures not being classifiable on some dimensions.

Saint	Number of images	% of polyptychs	Position with respect to mid-line			Cheek Shown	
			Left	Centre	Right	Left	Right
John the Baptist	54	23.58%	30	0	19	19	30
Francis	43	18.77%	17	3	16	23	17
Catherine of Siena	33	14.41%	10	0	18	22	11
John Evangelist	28	12.22%	9	0	12	19	8
Anthony Abbott	26	11.35%	14	0	12	14	11
Jerome	26	11.35%	13	1	10	11	15
Peter	25	10.91%	17	3	16	11	13
Michael	17	7.42%	5	1	6	5	11
Augustine	16	6.98%	8	0	2	5	10
Mary Magdalene	16	6.98%	4	1	6	7	8
Paul	16	6.98%	5	0	10	11	4
Sebastian	15	6.55%	8	1	4	6	8
Nicholas	15	6.55%	5	0	9	9	5
Bartholomew	14	6.11%	9	0	4	3	10
Lucy	14	6.11%	8	0	3	5	9

Difference between groups in proportion of left and right $X^2 = 8.34$, 14 df, NS $X^2=13.12$,14df,

Figure 13:1. Shows the proportions of portraits in which the left or the right cheek is shown. Data from McManus and Humphrey (1973) and Humphrey and McManus (1973). All groups are significantly different from a chance distribution of 50% left and 50% right cheeks (Females $X^2 = 72.5$, 1 df, $p < 0.001$; Males $X^2 = 14.2$, 1 df, $p < 0.001$; Self-portraits $X^2 = 13.14$, 1 df, $p < 0.001$). The difference between males and females is also highly significant ($X^2 = 20.44$, 1 df, $p < 0.001$).

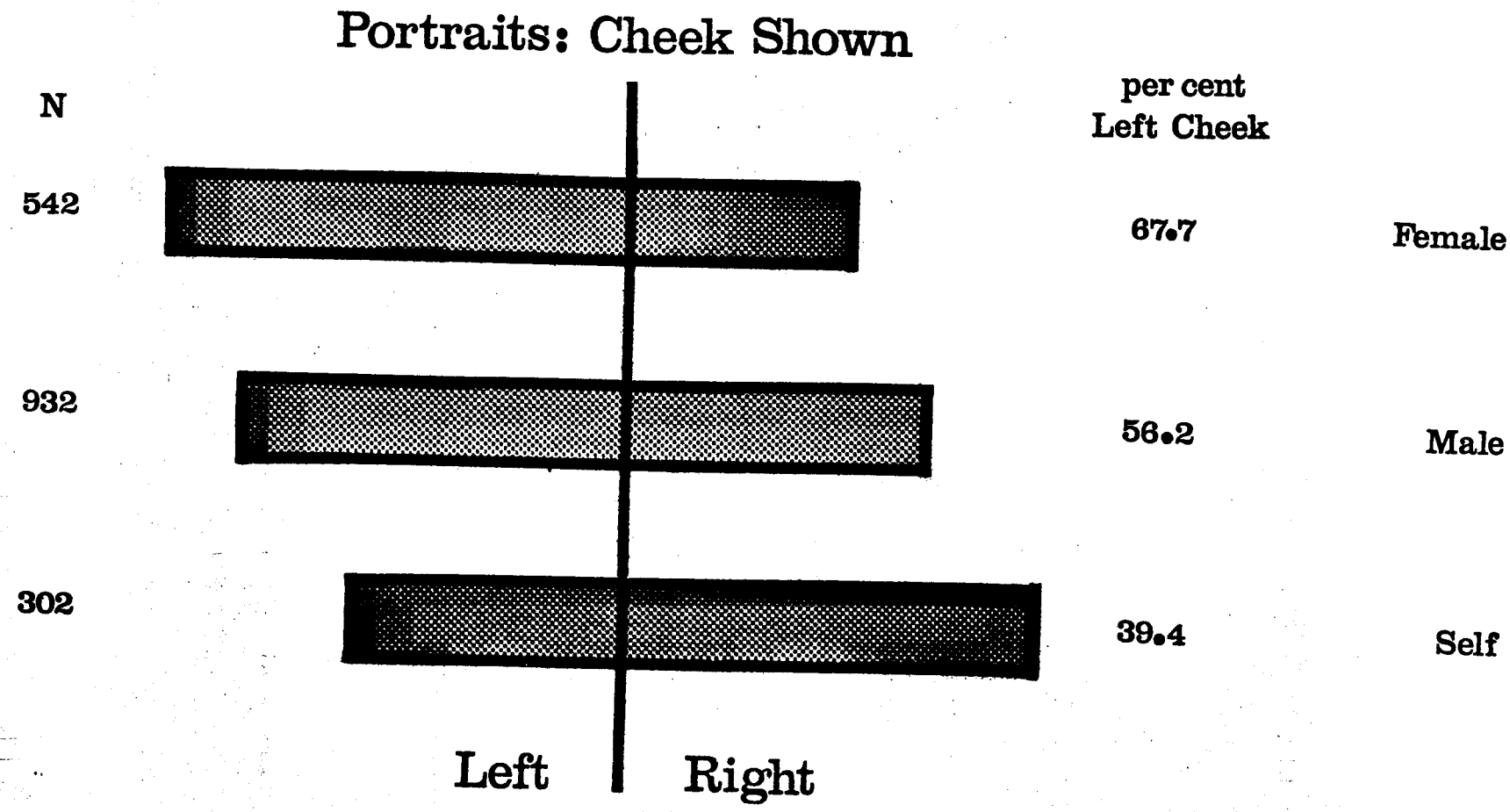

Portraits: Cheek Shown
N
per cent
Left Cheek
542
67.7
Female
932
56.2
Male
302
39.4
Self
Left
Right

Figure 13:2. Shows the percentage of left cheeks shown in Western portraits (excluding self-portraits) by the degree of turn of the head and the amount of the body of the subject shown. 'Full-face' portraits are those in which both eyes are completely visible, whilst 'profiles' are those in which one eye is partially or completely obscured. 'Head and shoulders' (HS) portraits usually cut off at about the level of the mid-sternum, whilst 'others' display more of the body than this. The ordinate shows the percentage showing the left cheek. Points which are shown as filled are significantly different from a value of 50% at at least a significance level of 0.05 by a X^2 test; unfilled points are not significantly different from chance. Head and shoulders portraits are represented by circles, and 'others' by squares. Female portraits are joined by a dashed line, male portraits by a solid line. The figures above the points (in order as shown) give the sample size for each point. Taken overall there is no significant difference in proportion of left cheeks between HS portraits and 'other' portraits ($X^2 = 0.98$, 4 df, NS). The difference between full-face and profile portraits overall is significant ($X^2 = 10.35$, 4 df, $p < 0.05$).

Portraits: Cheek Shown

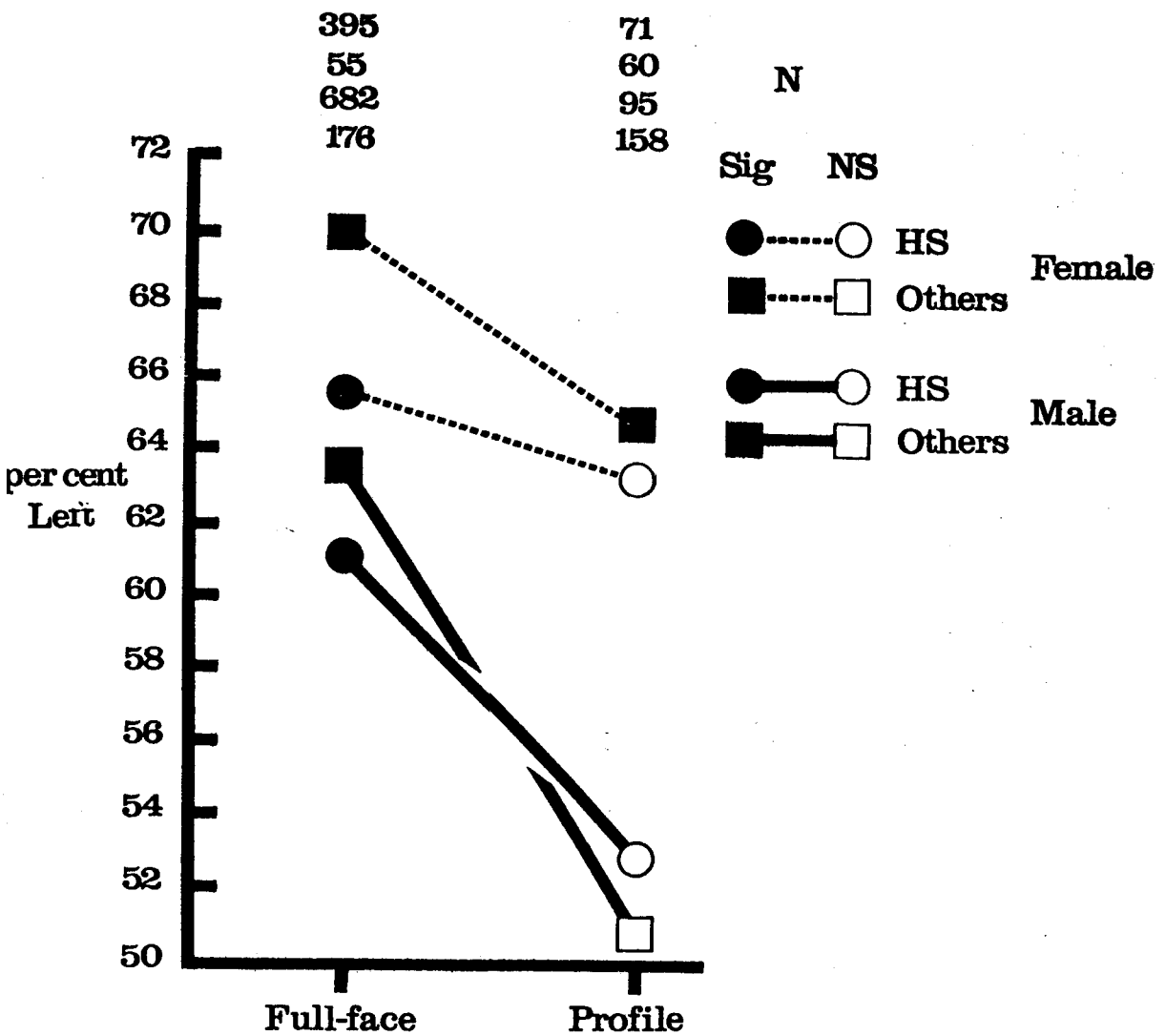


Figure 13:3. Shows the percentage of male portraits showing the left cheek in the National Portrait Gallery, London, by date of production. The sample size for each point is shown above it. The vertical bars from each point represent ± 2 standard errors. Standard errors have been omitted from all other figures to avoid undue complexity and confusion; if required they may be estimated from the sample size which is given for all data points.

Male Portraits: Cheek Shown

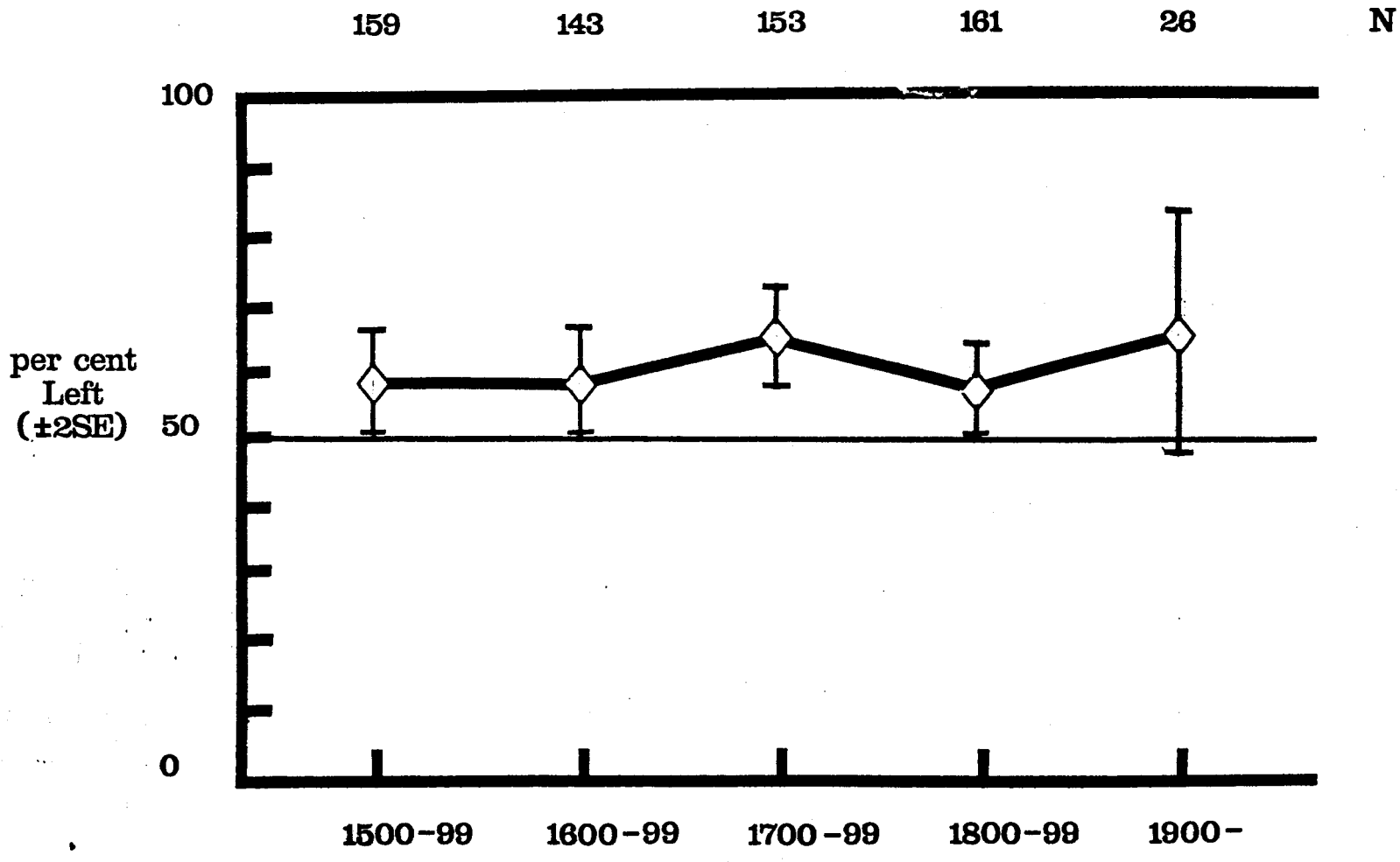


Figure 13:4. Shows the percentage of left cheeks shown in Italian Renaissance portraits (data from Berenson, 1957, 1963, 1967, 1968, and Pope-Hennessy, 1966). The proportions of both male and female portraits showing the left cheeks are significantly different from a chance hypothesis of 50% right and 50% left cheeks (Males, $X^2 = 26.5$, 1 df, $p < 0.001$; Females $X^2 = 10.6$, 1 df, $p < 0.005$). The difference between the male and female portraits is not significant. The self-portraits show a significantly lower proportion of left cheeks than do the male portraits (Fisher's exact test, $p = 0.025$, one-tailed test).

Italian Renaissance Portraits: Cheek Shown

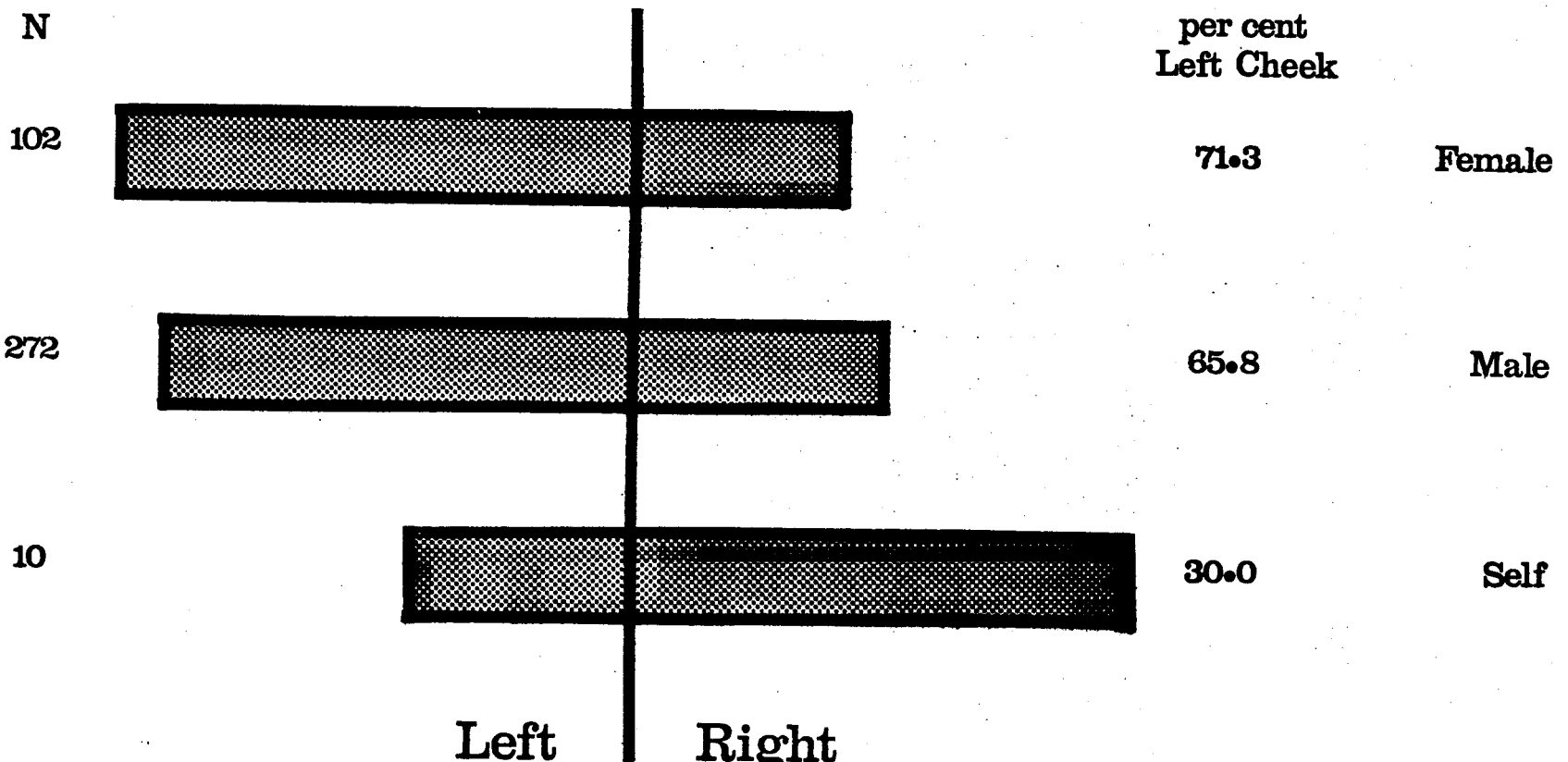


Figure 13:5. Shows the percentage of Italian Renaissance portraits showing the left cheek, by date of production. The chronological trends are not significant for either male or female portraits (X^2 = 5.26, 3 df, NS; X^2 = 4.33, 3 df, NS respectively).

Italian Renaissance Portraits: Cheek Shown

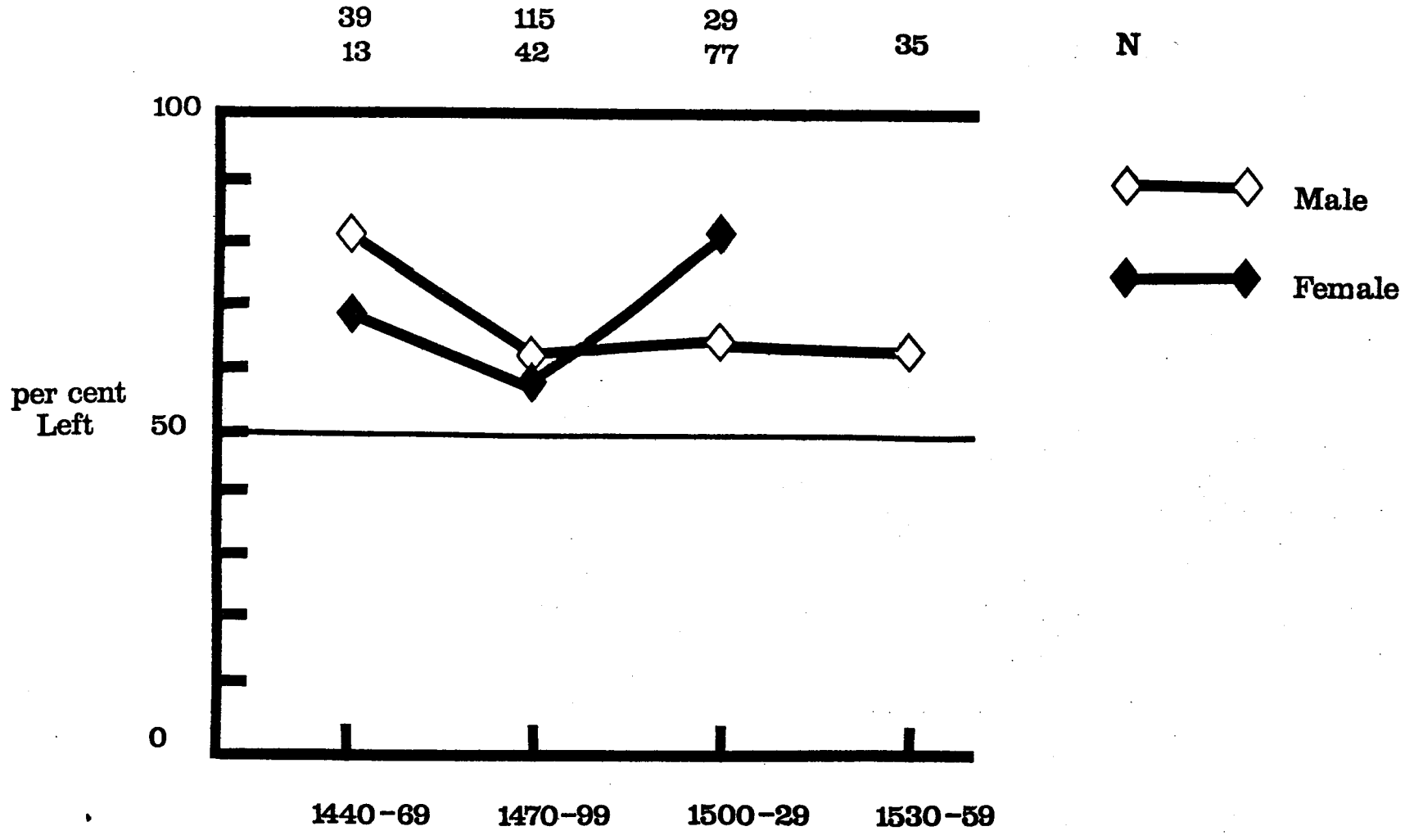


Figure 13:6. (from Humphrey and McManus, 1973). Shows the proportions of Rembrandt portraits showing the left or right cheek, by sex and kinship. The self-portraits Male Kin portraits, Male-Non-Kin portraits, and Female Non-Kin portraits are all significantly different from chance expectations (X^2 = 25.3, 1 df, $p < 0.001$; X^2 = 5.88, 1 df, $p < 0.05$; X^2 = 6.98, 1 df, $p < 0.001$; X^2 = 20.74, 1 df, $p < 0.001$ respectively). The Female Kin group is not significantly different from chance expectations (X^2 = 0.41, 1 df, NS). Taken overall there is a significant difference between kin and non-kin portraits (X^2 = 8.01, 2 df, $p < 0.02$).

Rembrandt: Oil Paintings: Cheek Shown

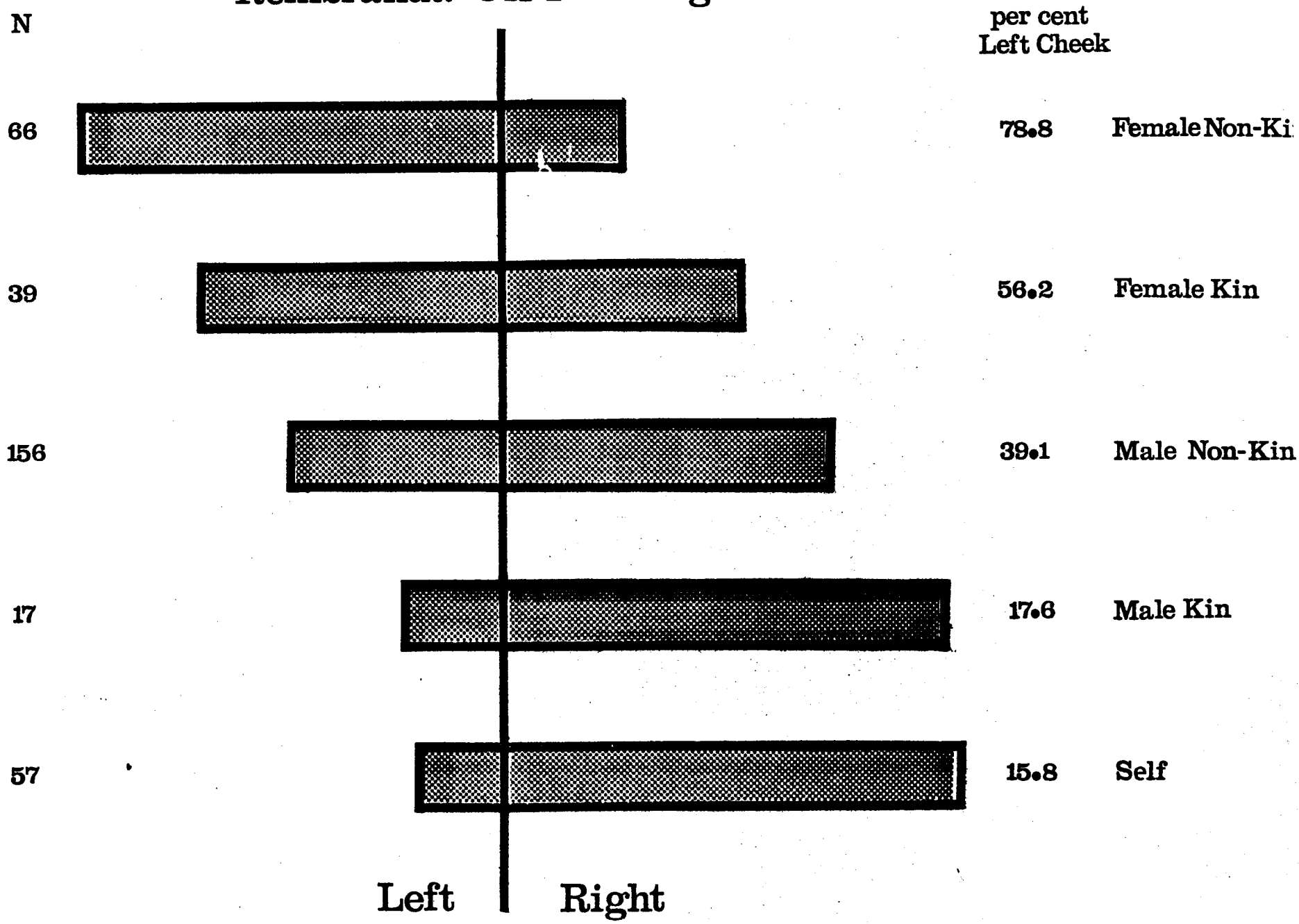
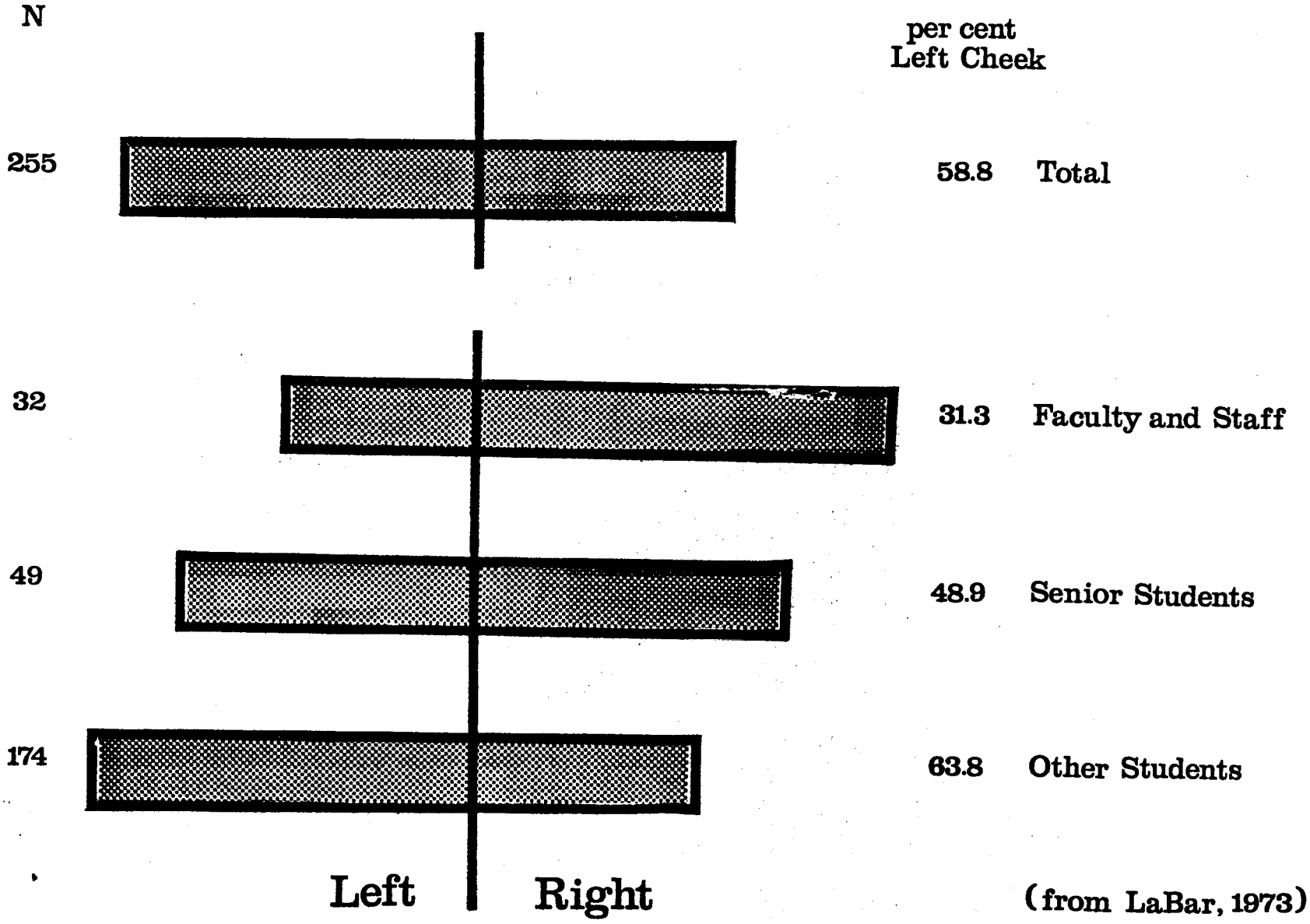


Figure 13:7. Shows data derived from LaBar (1973) concerning cheeks shown by students and staff in a school year book. The proportion of left cheeks overall is significantly greater than would be expected by chance ($X^2 = 4.53$, 1 df, $p < 0.05$). The bottom part of the figure shows LaBar's data for the Central Wesleyan College, the groupings being those of LaBar himself. Overall the differences between Faculty and Staff, Senior students and Other students are significant ($X^2 = 13.2$, 2 df, $p < 0.01$). The difference between Senior students and Other students is almost significant ($X^2 = 2.92$, $p < 0.10$), the difference between Seniors and Faculty and Staff is not significant ($X^2 = 1.82$ 1 df, NS), whilst that between Faculty and Staff and 'Other Students' is significant ($X^2 = 10.51$, 1 df, $p < 0.01$).

Modern Photographs: Cheek Shown



(from LaBar, 1973)

Figure 13:8. Shows data regarding Dante Gabriel Rosetti's portraits, the data being derived from Surtees (1971). The male portraits are significantly different from a chance distribution (X^2 = 7.22, 1 df, $p < 0.01$), whilst the female portraits are not different from chance (X^2 = 0.591, 1 df, NS). The difference between the male and female portraits is significant (X^2 = 4.2, 1 df, $p < 0.05$).

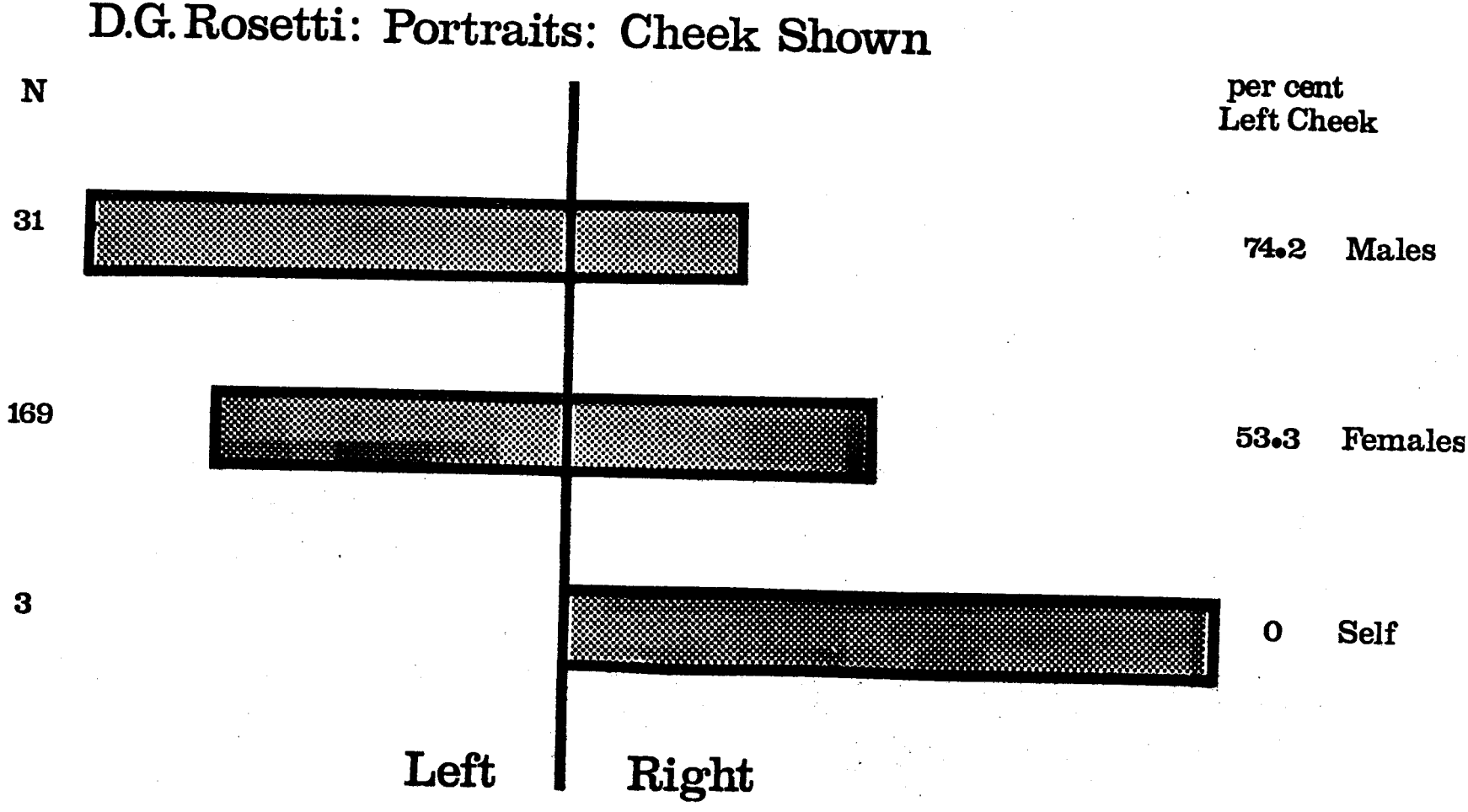

D.G. Rosetti: Portraits: Cheek Shown
N
per cent
Left Cheek
31
74.2
Males
169
53.3
Females
3
0
Self
Left
Right

Figure 13:9. Shows data on the paintings and sketches of van Gogh, from de la Faille (1970). Only male subjects are considered. For the Heads and Half-Lengths the differences between groups are significant ($X^2 = 8.79$, 2 df, $p < 0.02$). For the Whole-lengths the lower-class portraits show a distribution significantly different from chance ($X^2 = 4.69$, 1 df, $p < 0.05$). There were no middle-class full-lengths.

van Gogh: Male Portraits and Drawings

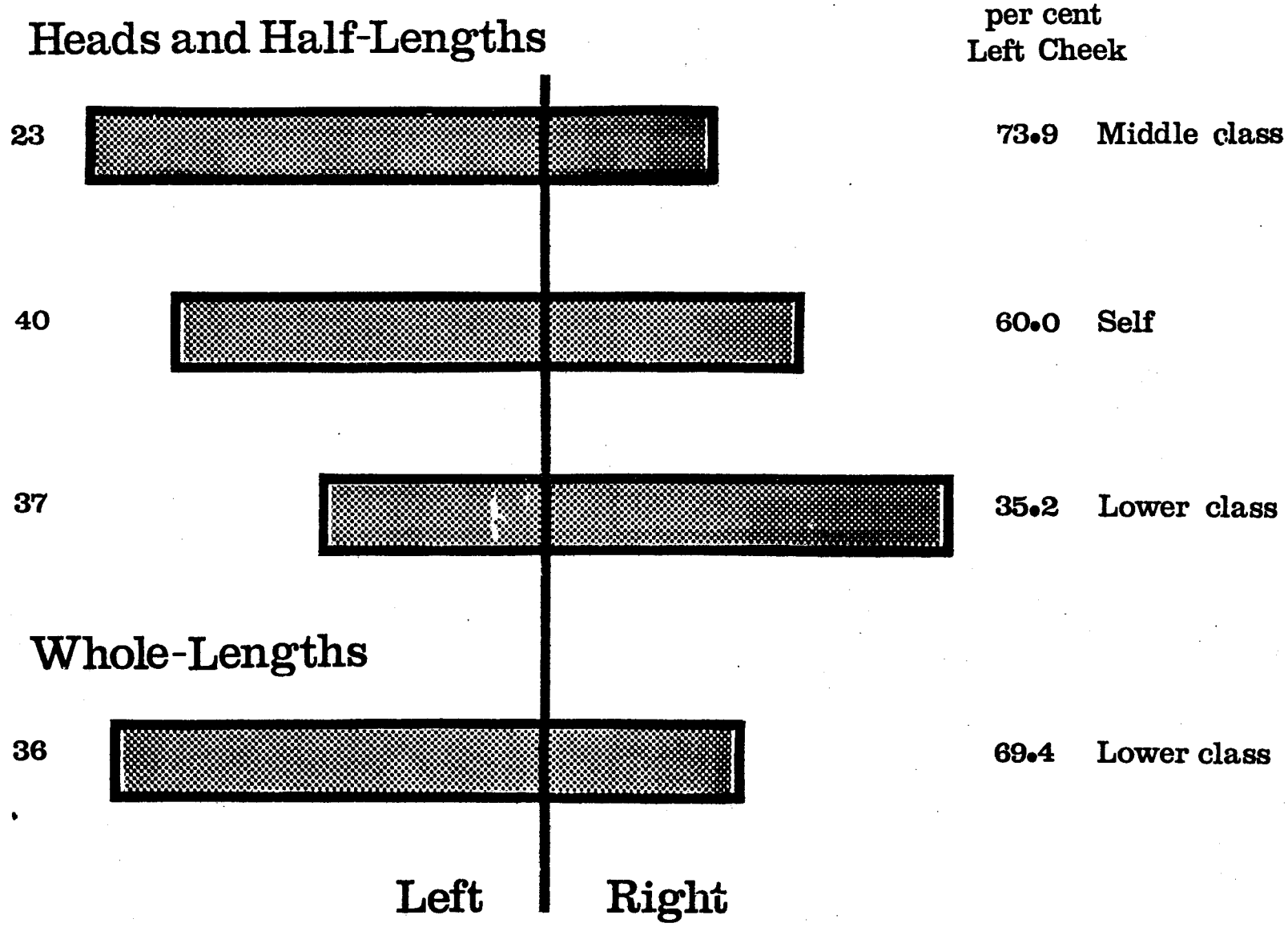


Figure 13:10. Shows the percentage of pictures of the Madonna and Child in which the child is held on the left side. Data from Berenson. The shift by date is highly significant (X^2 = 151.8, 5 df, $p < 0.001$). Individual data points are shown as open points if they are not significantly different from 50% on a X^2 test with 1 degree of freedom at the 0.05 level. The sample size for each datapoint is shown above the top of the figure.

Madonna and Child : Side of Child

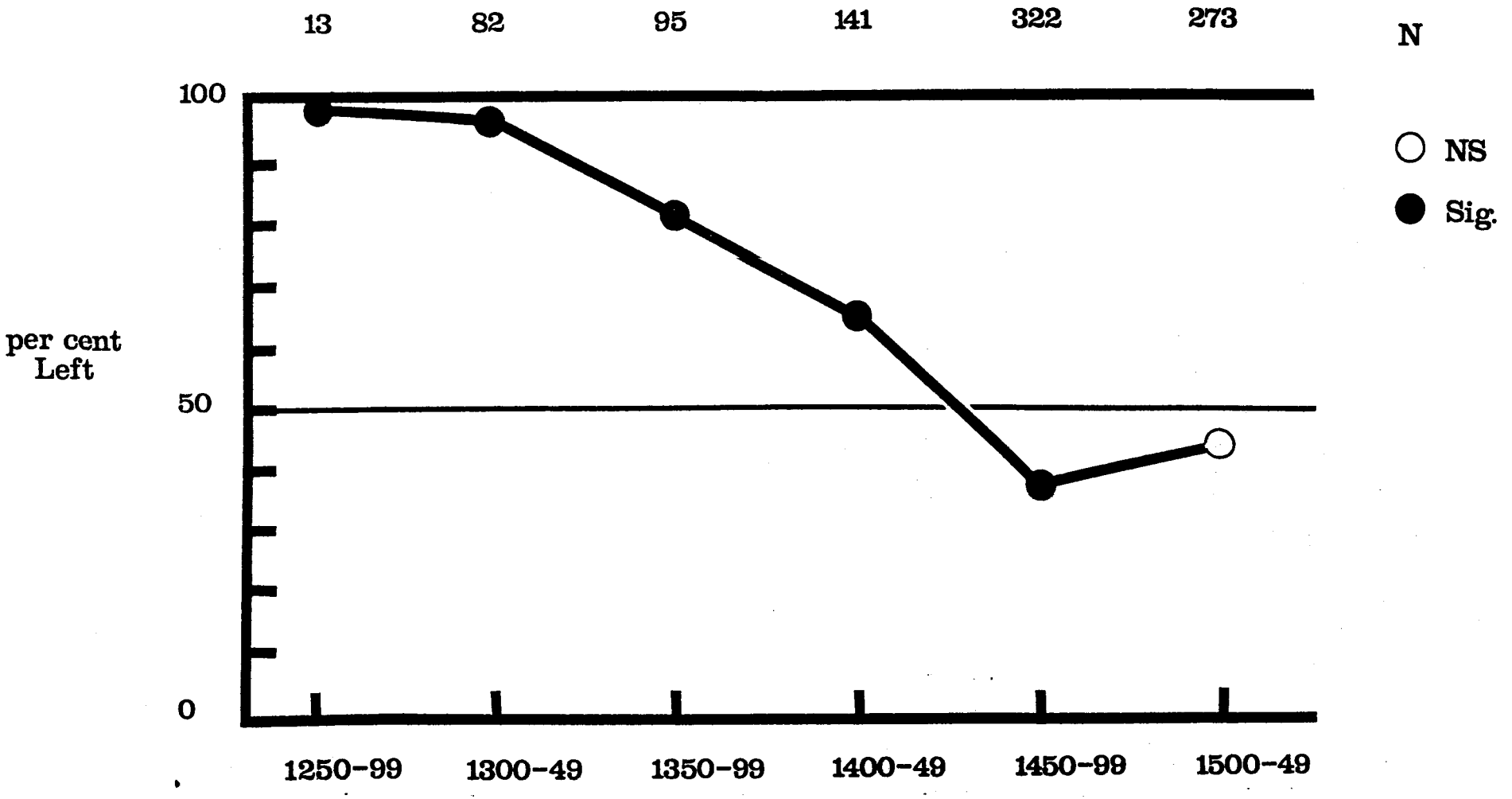


Figure 13:11. Shows the side of holding the child by date and by the school of painting. Insufficient Venetian and Northern pictures are available for the period 1300-1449. Differences between schools are not statistically significant (1350-99 X^2 = 0.27, 1 df, NS; 1400-49 X^2 = 3.65, 1 df, NS; 1450-99 X^2 = 5.75, 3 df, NS; 1500-49 X^2 = 5.51, 3 df, NS). The 1300-49 group is not testable with a X^2 statistic.

Madonna and Child: Side of Child

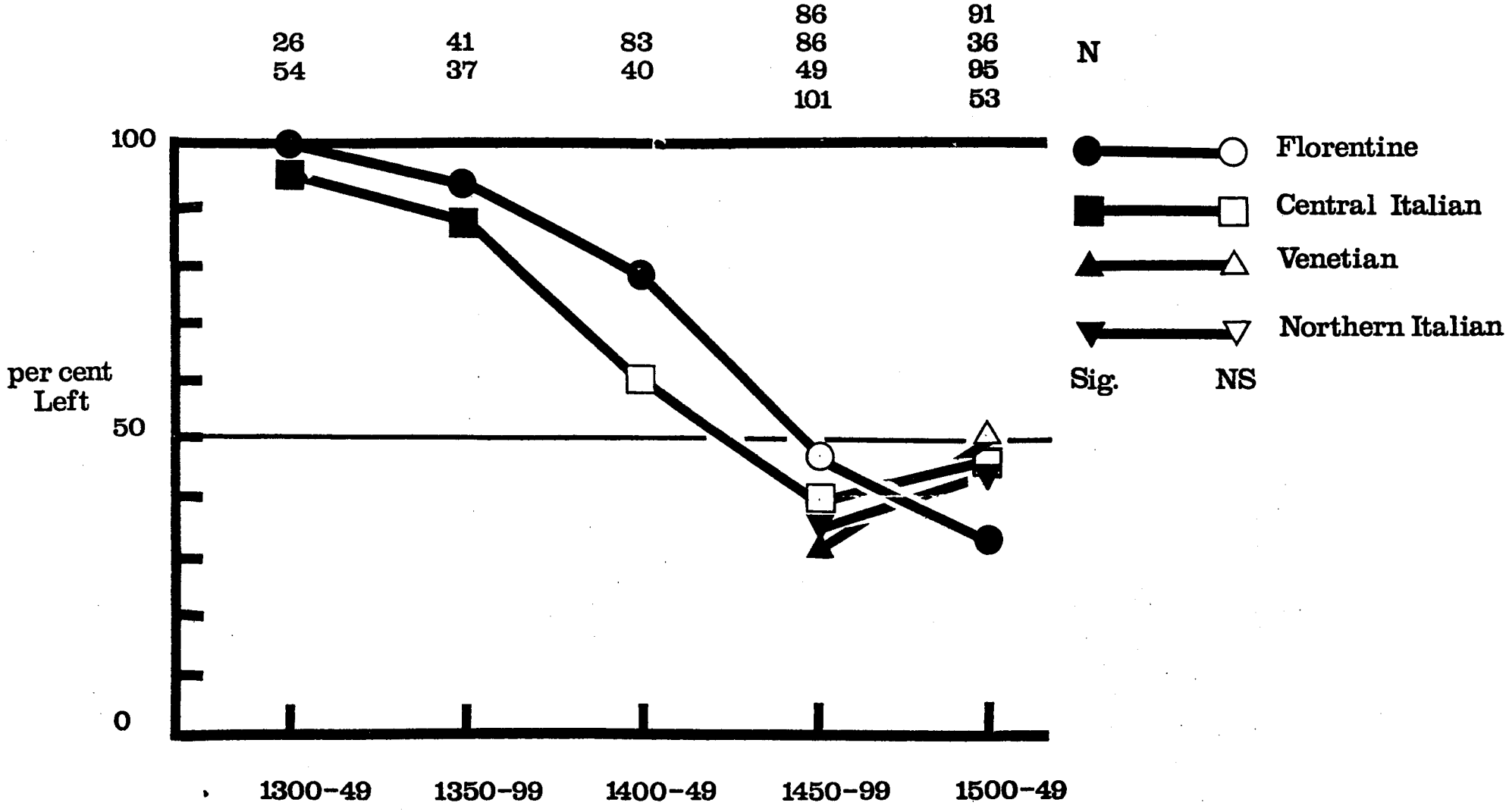


Figure 13:12. Shows the side of portrayal of the child by date and by the importance of the artist. Differences are not significant except for the 1350-99 group which gives a X^2 value of 5.10, p = 0.024; this value would not be significant after correction for repeated X^2 testing. For other groups:- 1400-49 X^2 = 4.28, 2 df, NS; 1450-99 X^2 = 5.89, 2 df, NS; 1500-49 X^2 = 3.29, 2 df, NS. The data for the 1300-49 group are not suitable for statistical testing.

Madonna and Child: Side of Child

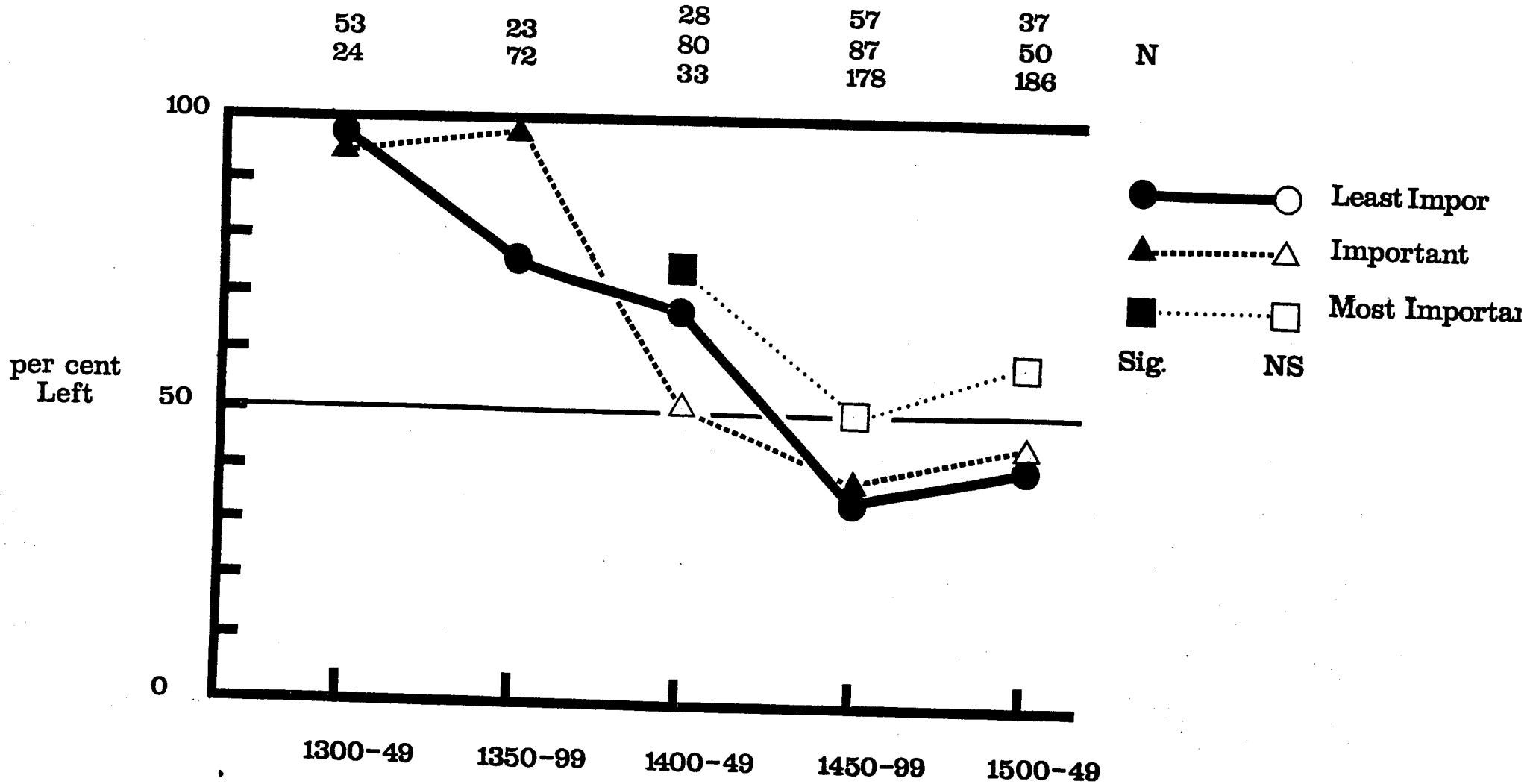


Figure 13:13. Shows the percentage of paintings in which the Madonna, the Child, and the Madonna and Child combined, show the left cheek, by date of production. Differences between the date groups are highly significant (Madonna $X^2 = 141.52$, 4 df, $p < 0.001$; Child $X^2 = 29.50$, 4 df, $p < 0.001$; Madonna and Child combined $X^2 = 38.94$, 4 df, $p < 0.001$).

Madonna and Child: Cheeks Shown

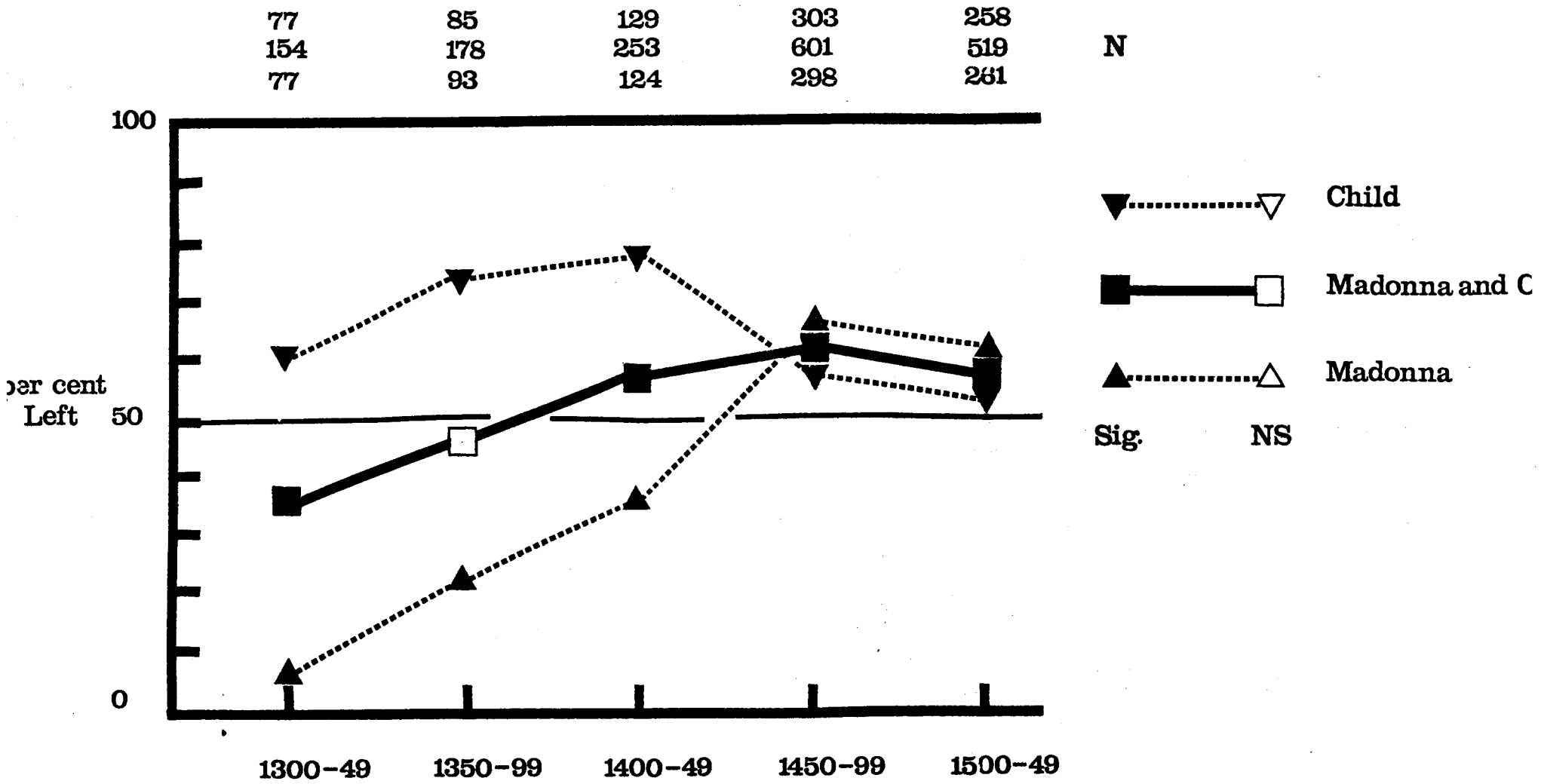


Figure 13:14. Shows the cheek shown by the Madonna and the Child by date and by school of artist. Differences between schools are not significant. Insufficient data was present for the Venetian and Northern schools in the period 1300-1449. (Madonna's Cheek 1350-99, X^2 = 0.005, 1 df, NS; 1400-49 X^2 = 1.59, 1 df, NS; 1450-99, X^2 = 1.00, 3 df, NS; 1500-49 X^2 = 4.37, 3 df, NS; Child's Cheek 1300-49 X^2 = 0.59, 1 df, NS; 1350-99 X^2 = 0.14, 1 df, NS; 1400-49 X^2 = 0.13, 1 df, NS; 1450-99 X^2 = 3.52, 3 df, NS; 1500-49 X^2 = 5.72, NS).

The data for the Madonna's cheek for the period 1300-49 are not suitable for statistical analysis.

Madonna and Child: Cheek Shown

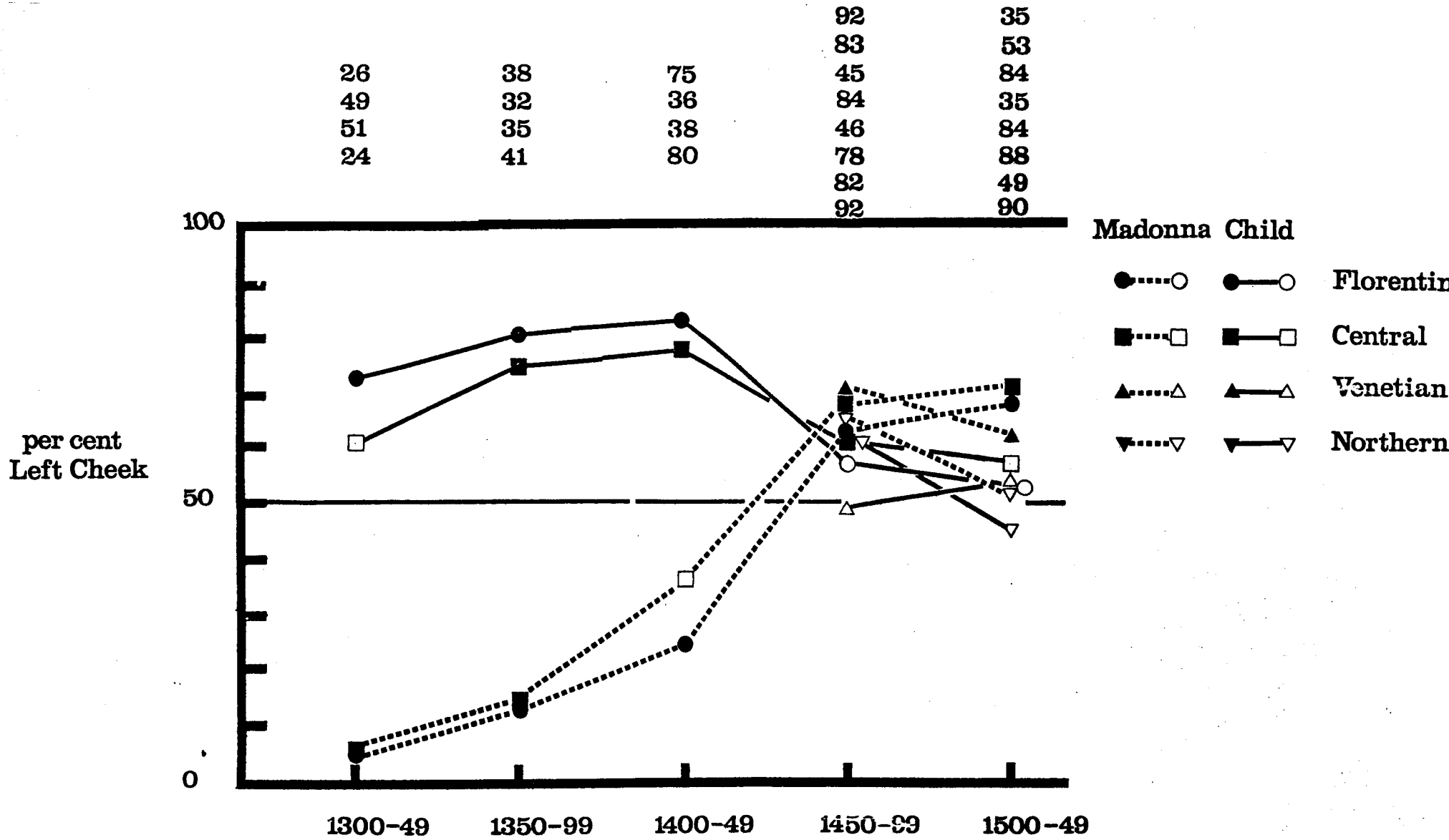


Figure 13:15. Shows the cheek shown by the Madonna and by the Child by date and by the importance of the artist. No statistically significant differences related to the importance of the artist are found. (Madonna's cheek 1350-99, X^2 = 2.04, 1 df, NS; 1400-49, X^2 2.26, 2 df, NS; 1450-99, X^2 = 1.25, 2 df, NS; 1500-49, X^2 = 1.09, 2 df, NS; Child's Cheek 1300-49, X^2 = 0.10, 1 df, NS; 1350-99, X^2 = 2.84, 1 df, NS; 1400-49, X^2 = 4.73, 2 df, NS; 1450-99, X^2 = 5.12, 2 df, NS; 1500-49, X^2 = 1.16, 2 df, NS.

Data for the Madonna's cheek for the period 1300-49 are not suitable for statistical testing.

Madonna and Child: Cheek Shown

		24	172	36
24	21	76	51	176
48	64	29	78	34
22	70	22	53	179
50	23	74	78	46
		28	169	48

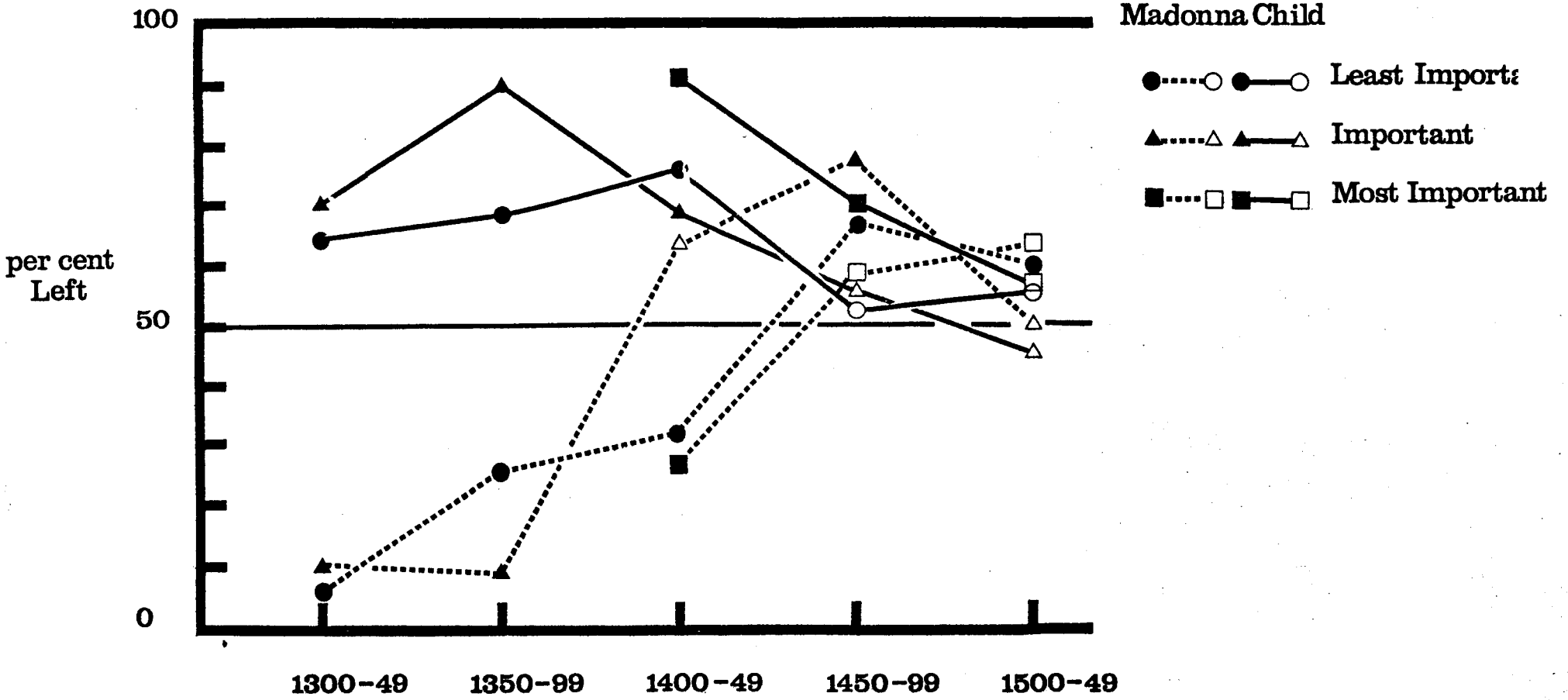


Figure 13:16. Shows the relative proportions of each of the eight possible types of Madonna and Child painting (LRL, LRR, etc.). Each particular type is described by the three letter code described in the text, and is also shown by a 'pin-man'. The large pin-figure represents the Madonna, the small pin-figure the Child, and the small bars in the faces the direction of facing.

Madonna and Child

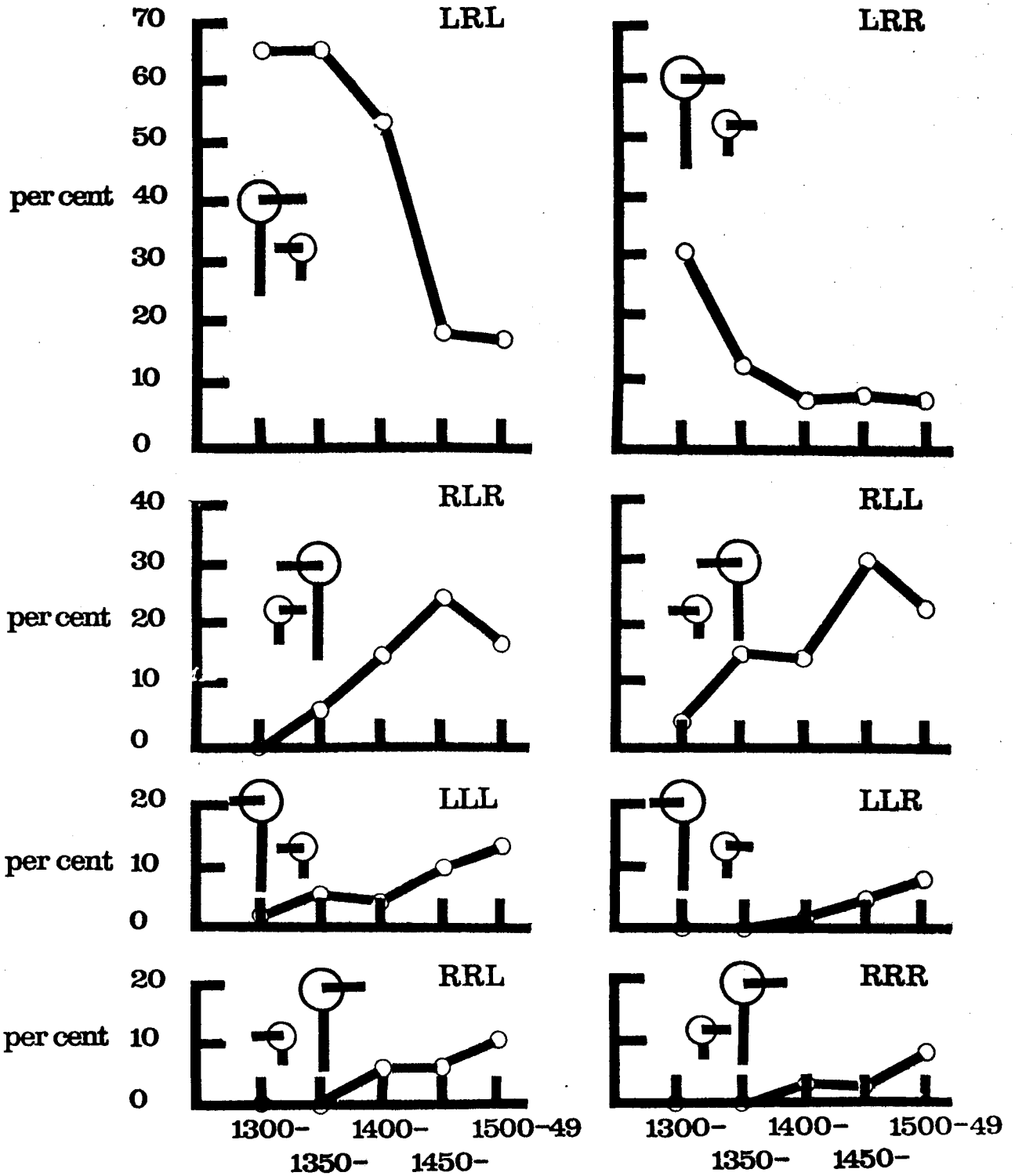


Figure 13:17. Shows for the period 500-1549, the relative proportions of works portraying the Crucifixion or the Madonna and Child. The ordinate is on a logarithmic scale and shows the number of Madonnas divided by the number of Crucifixions. The N values across the top are of the total number of pictures analysed. Data from Berenson, Beckwith (1970), Lasko (1972), Rickert (1954) and the collections of the British Museum and Victoria and Albert Museum, London.

The Cult of the Virgin Mary

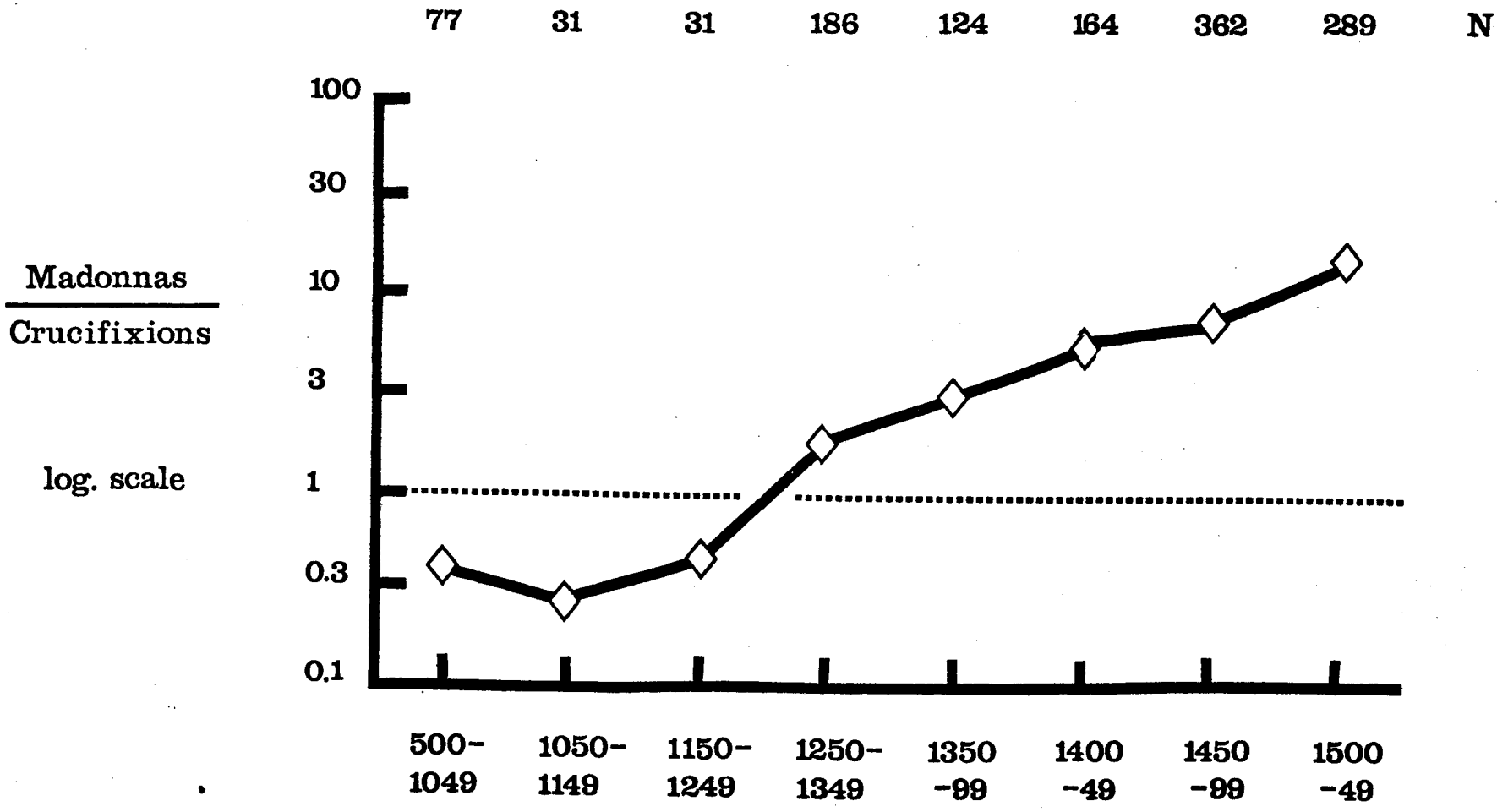


Figure 13:18. Shows the percentage of Annunciations in which the Angel is entering from the left side. As in previous figures, solid datapoints are significantly different from chance, open points are not. Differences between groups are not significant using Fisher's exact test.

Annunciation: Side of Entry of Angel

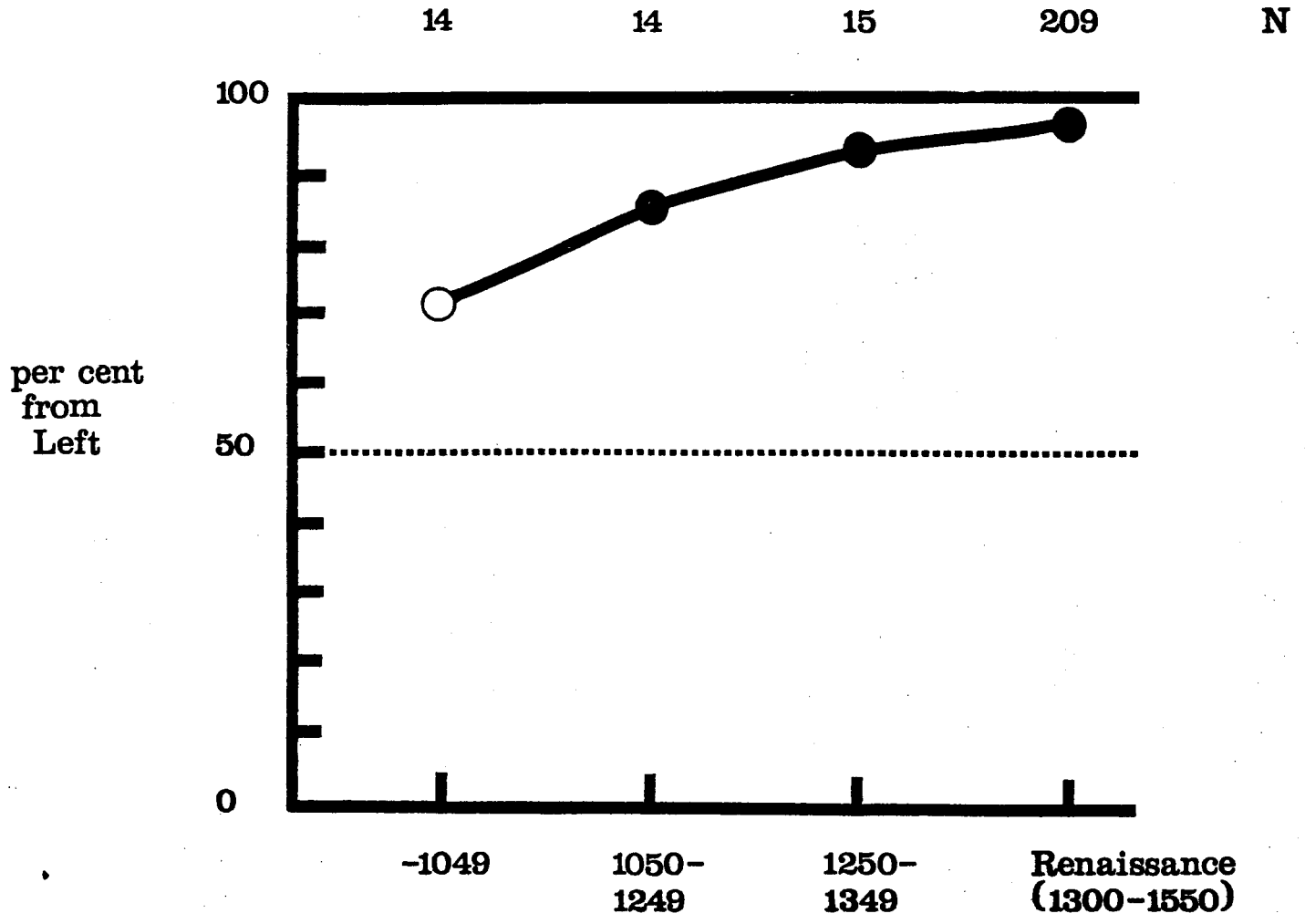


Figure 13:19 Shows for the mediaeval Madonna and Child, the proportions of portraits showing the left cheek, and holding the child on the left side. All graphs are for the same data set, for which sample sizes are given across the top. For the side of the Child there is a significant decrease in the proportion of portraits in which the child is held in the centre (pre-649 vs 1250-1349, Fisher's exact test, $p = 0.0012$). The proportion held on the left side versus the right side in those in which the child is not held centrally is not significant (Fisher's exact test). No other trends are significant.

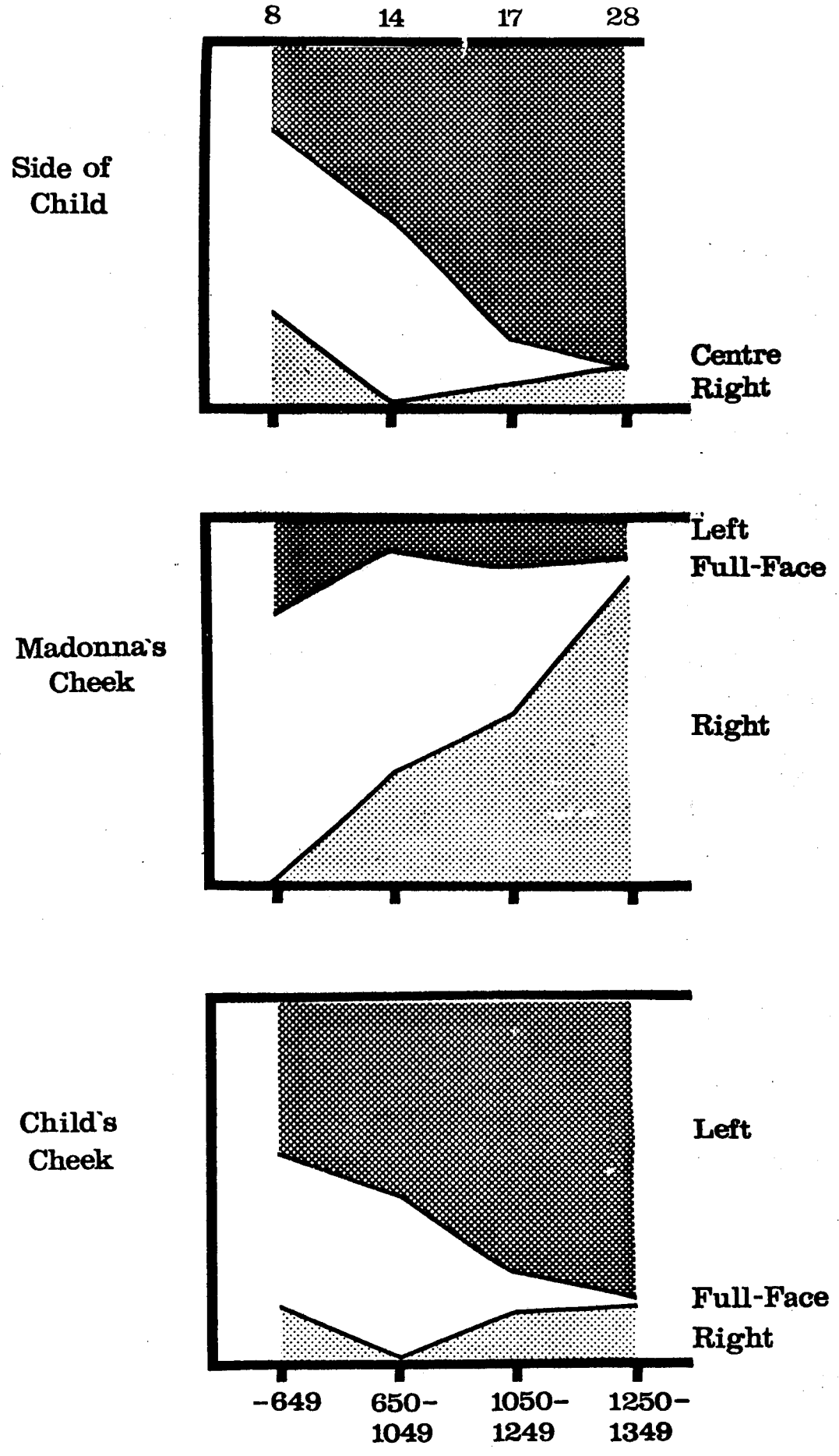

8
14
17
28
Side of Child
Centre
Right
Madonna's Cheek
Left
Full-Face
Right
Child's Cheek
Left
Full-Face
Right
-649
650-1049
1050-1249
1250-1349

Figure A13:2.1 Shows for four different painting types (Portrait, Crucifixions, Annunciations and Madonna and Child) the proportions showing light coming from the left side as a function of the number of pictures showing directional light (i.e. after exclusion of those with light straight on, indeterminate, diffuse, etc.), as a function of the school. The sample size for each data point is shown directly above the column of data points, in the same order as the data points. Considering just the 'Total' data points, there is no statistical difference between the Venetian and Northern schools (X^2 less than 0.01, 1 df), whilst the difference between the Florentine and Central Italian schools is just significant ($X^2 = 4.61$, 1 df, $p < 0.05$). The difference between the combined Northern and Venetian schools, and the combined Florentine and Central Italian schools is highly significant ($X^2 = 48.06$, 1 df, $p < 0.001$).

Renaissance Paintings: Side of Light

8	49	323	19	498
69	47	271	42	370
171	18	287	59	326
62	17	219	28	395

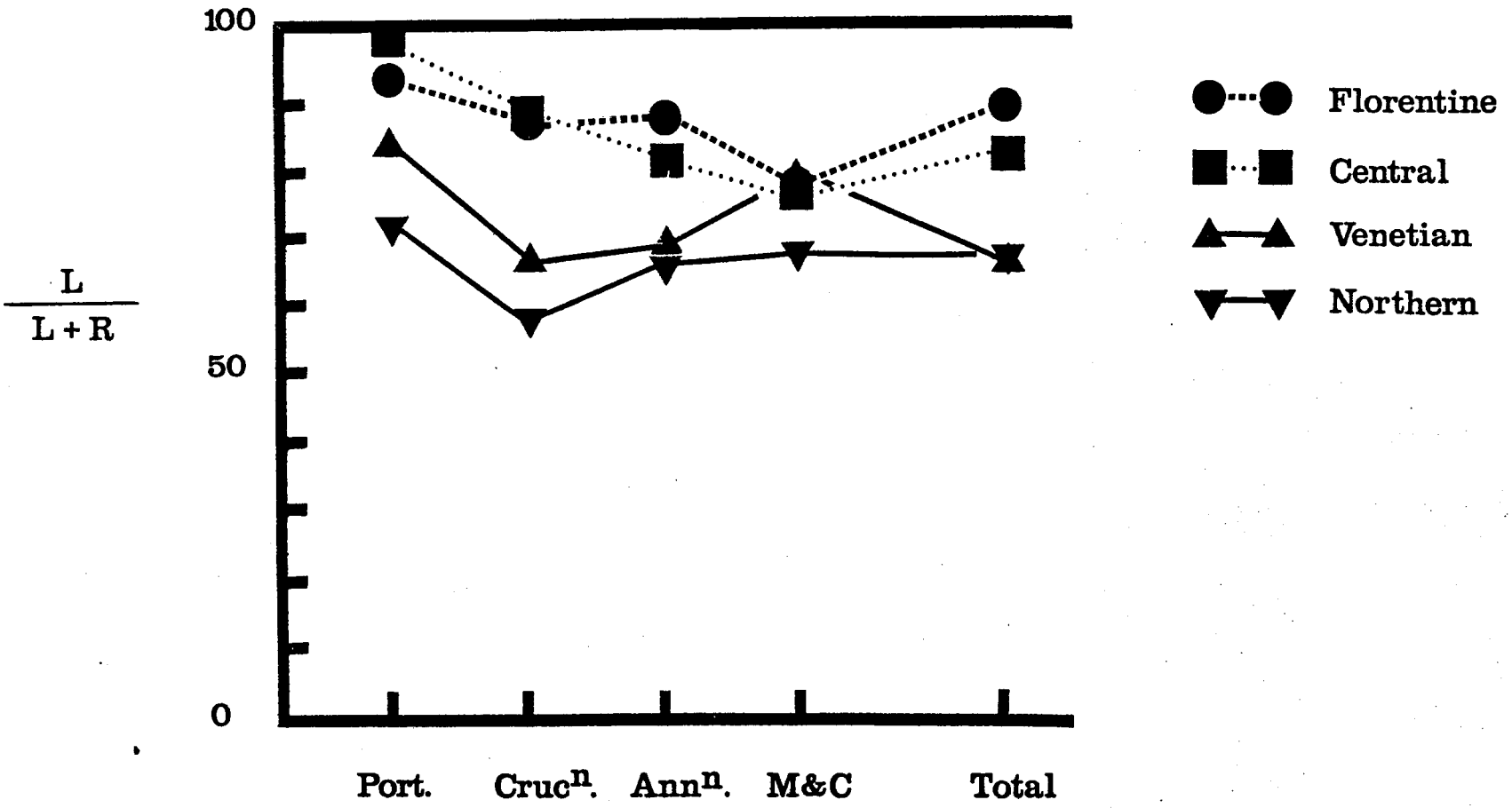


Figure A13:2.2 shows the proportion of Madonna and Child paintings with light from the left side as a proportion of Madonna and Child paintings with directional light. The differences between schools are significantly different from chance distributions for the years 1400-49, 1540)99 and 1500-49 (X^2 = 6.25, 2 df, $p<0.05$; X^2 = 13.08, 3 df, $p<0.005$; and X^2 = 24.66, 3 df, $p<0.005$ respectively).

Madonna and Child: Direction of Light

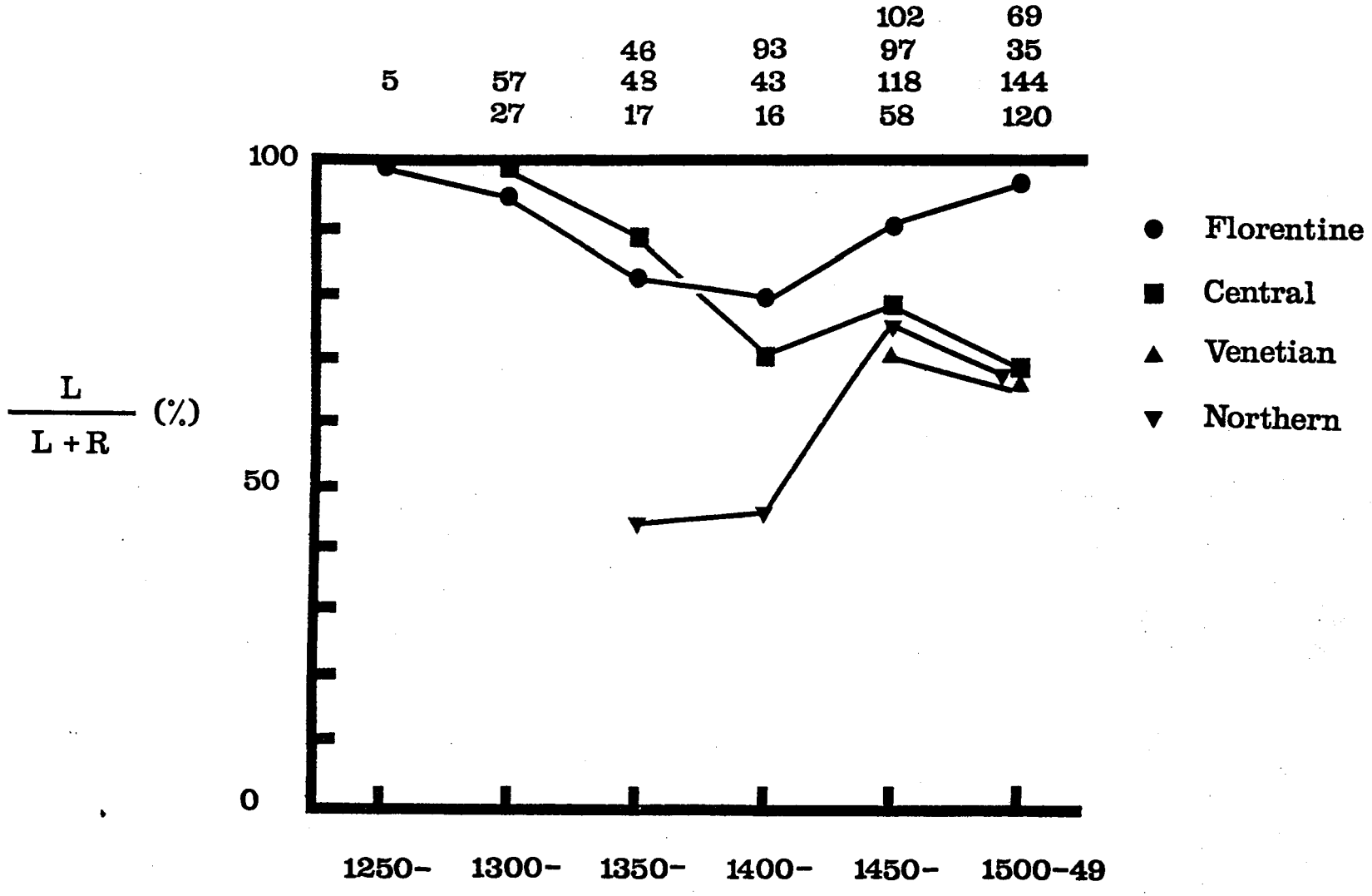


Figure A13:2.3 shows the proportion of Mediaeval and Renaissance Crucifixions having light from the left (dark shading), right (light shading) and indeterminate or straight on (white). During the Renaissance (1250-1550) there is a significant decline in the proportion of pictures with light straight on or indeterminate ($X^2 = 9.02$, 1 df, $p < 0.01$), whilst during the same period there is no significant variation in the proportions of left and right sided light (considering only those pictures with directional light) ($X^2 = 0.803$, 2 df, NS).

Crucifixions: Side of Light

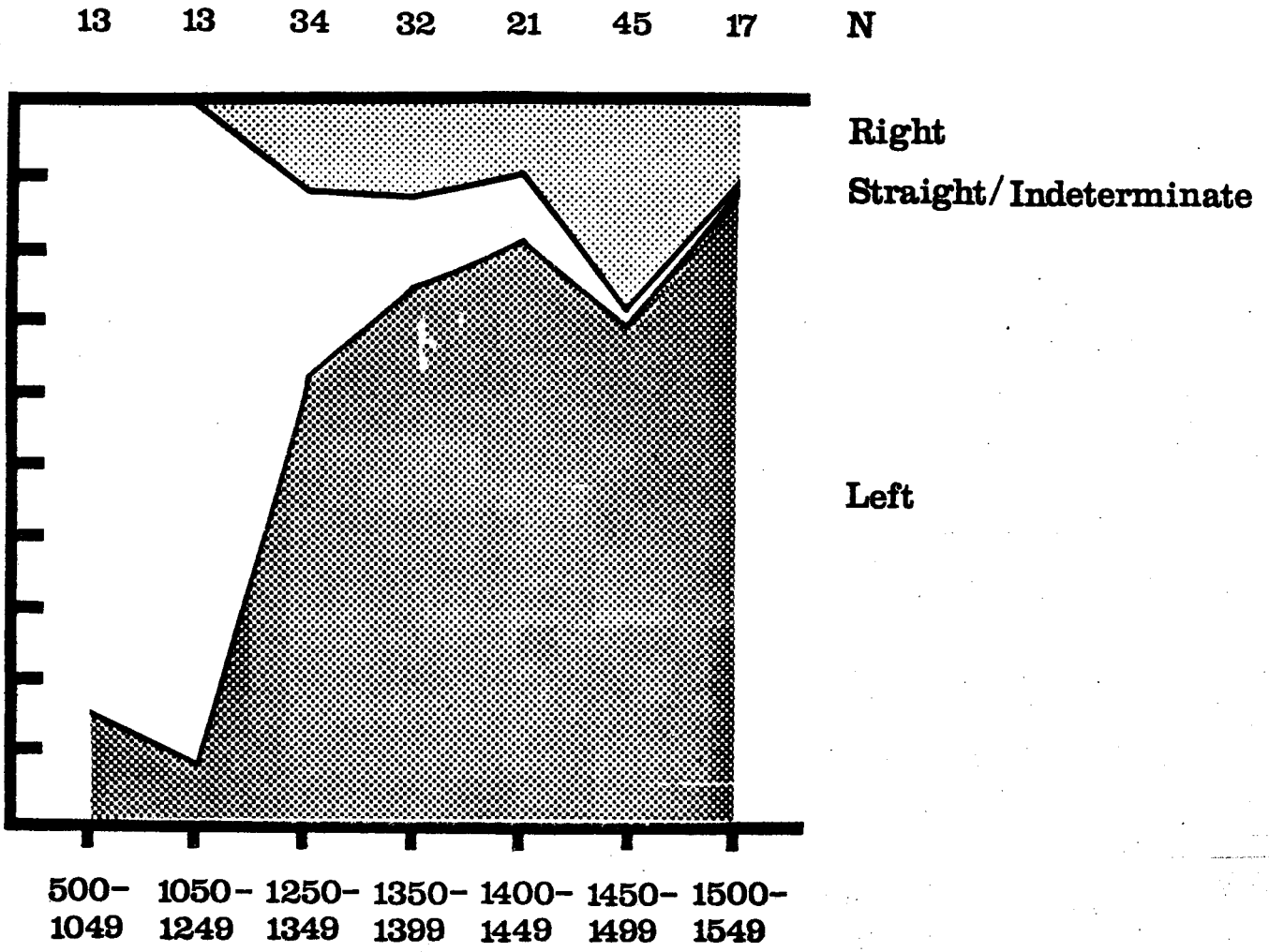


Figure A13:2.4 shows the direction of light in Renaissance Madonna and Child paintings. Light from the right is indicated by light stippling, light from the left by dark stippling, and indeterminate light by white. The proportion of pictures with light of indeterminate origin declines significantly during the period considered (X^2 = 41, 4 df, $p < 0.001$). In pictures with directional light there is a significant increase during the Renaissance of pictures with light from the right (X^2 = 29.28, 4 df, $p < 0.001$).

Madonna and Child: Direction of Light

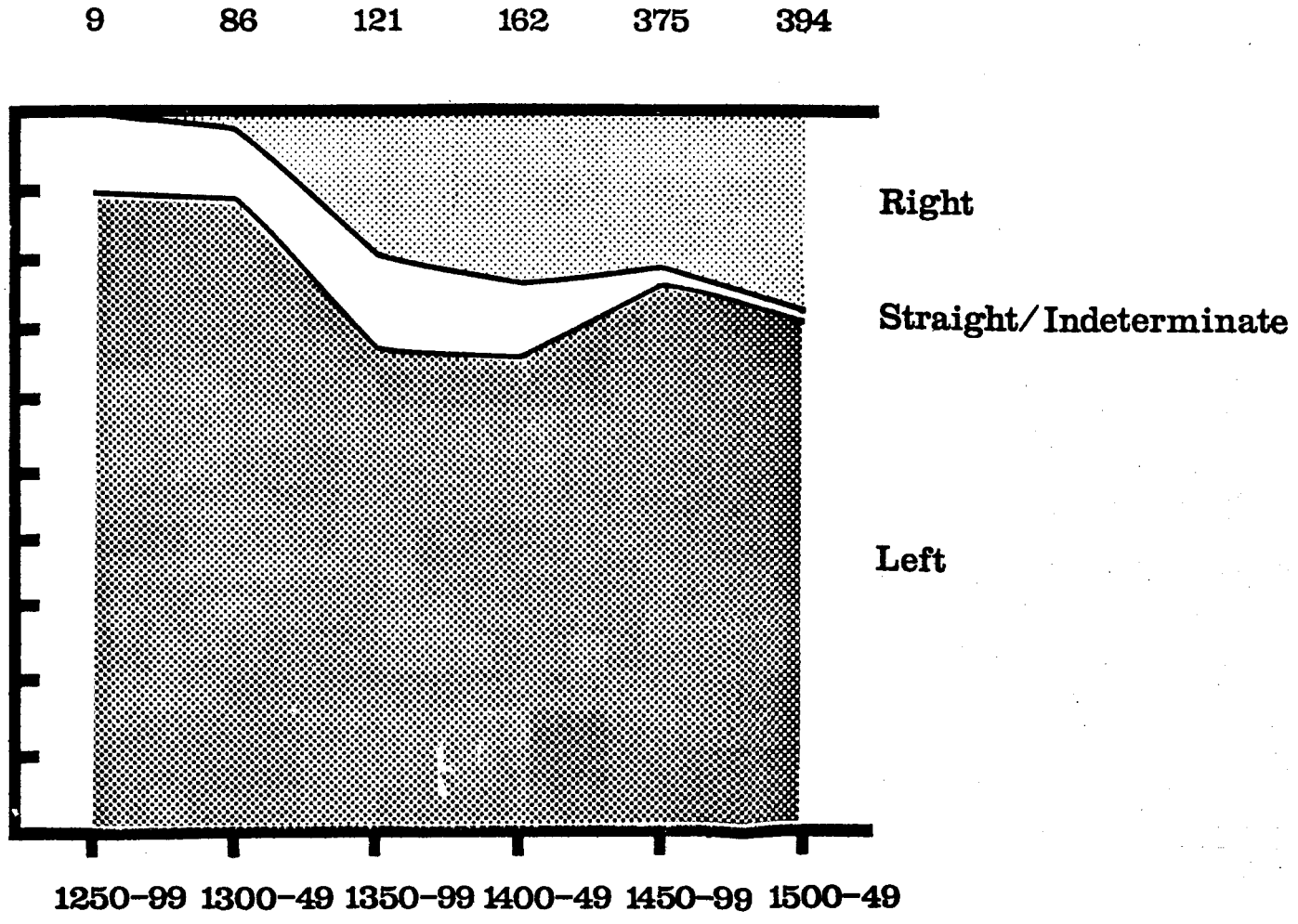


Figure A13:2.5 shows for Madonna and Child portraits with light from either the right or the left side, the proportions of pictures showing the left cheek of the Madonna, the left cheek of the Child, and holding the child on the left side. The changes with time for the group with light from the left side are statistically highly significant (X^2 = 97.6, 3 df, $p < 0.001$; X^2 = 124.6, 3 df, $p < 0.001$; X^2 = 24.9, 3 df, $p < 0.001$ for side of child, Madonna's cheek and child's cheek respectively.) For the group with light from the right side none of the chronological changes are significant by a Chi^2 test (X^2 = 1.17, 3 df, NS; X^2 = 5.83, 3 df, NS; and X^2 = 6.62, d3 df, NS; respectively; however comparing the first data point (i.e. 1350-99) with the last (1500-49) by a Fisher's exact test shows a significant difference in the proportion of left Madonnas cheeks ($p = 0.0208$, one-tailed test) but not in side of child or child's cheeks ($p = 0.232$; $p = 0.80$ respectively).

Statistics are calculated only for the time epochs used on the abscissa; the earlier years produced insufficient pictures with light from the right to merit statistical analysis. For pictures prior to 1350 changes in pictures with light coming from the left are denoted by short dashed lines. The sample sizes for each point on the Madonna's cheek datapoints are shown in the same order as the points themselves. The sample sizes are the same for all three graphs.

Madonna and Child

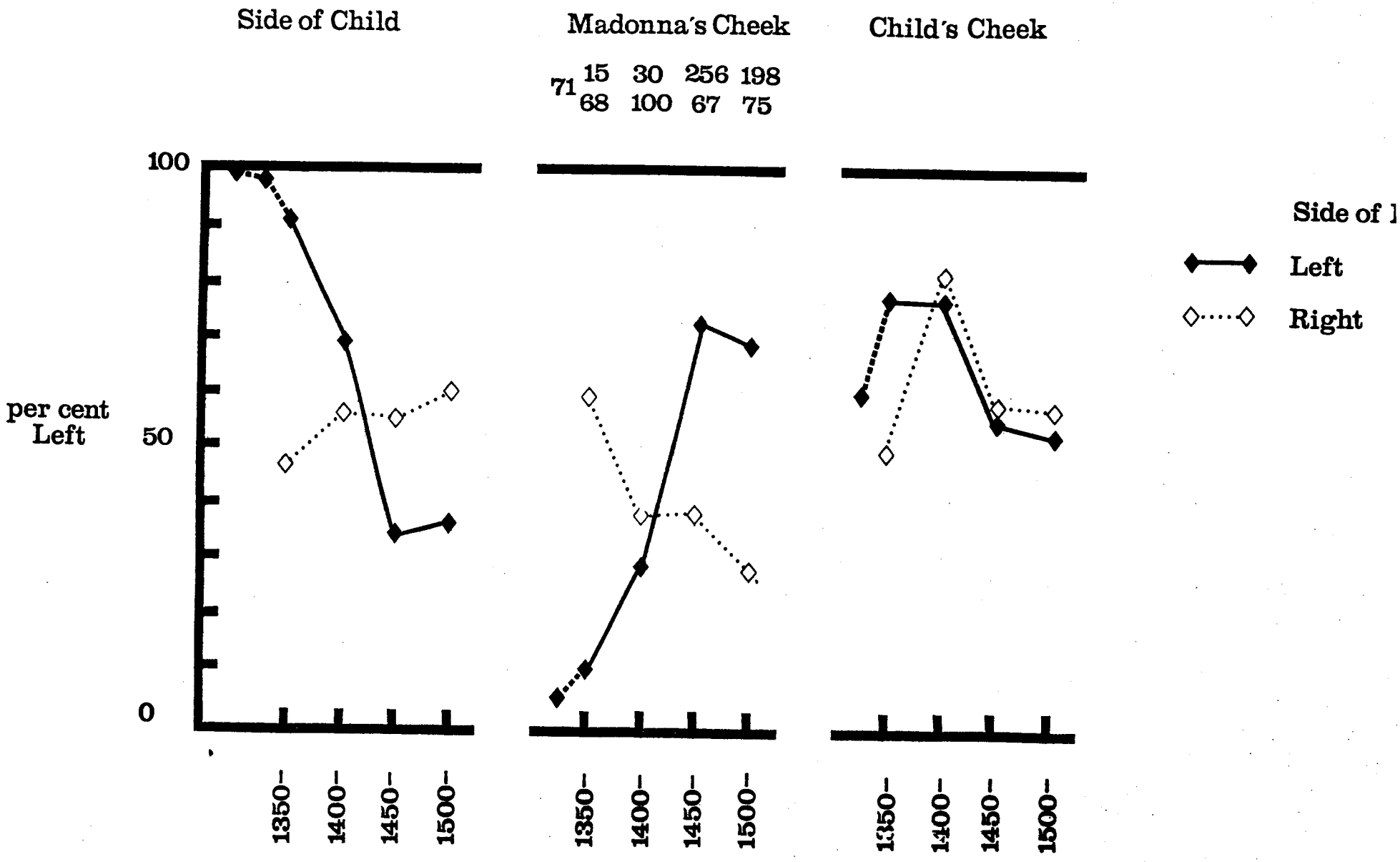


Figure A13:3.1 shows, in the top part, the number of polyptychs in which were shown one, two, three, etc. pairs of saints. n(polyptychs) = 605. Mean =2.09 pairs of saints (4.18 saints); standard deviation = 1.657 (pairs). The lower part shows the proportion of saints portrayed who are in polyptychs with one, two, three, etc. pairs of saints. N = 2532 saints.

Polyptychs

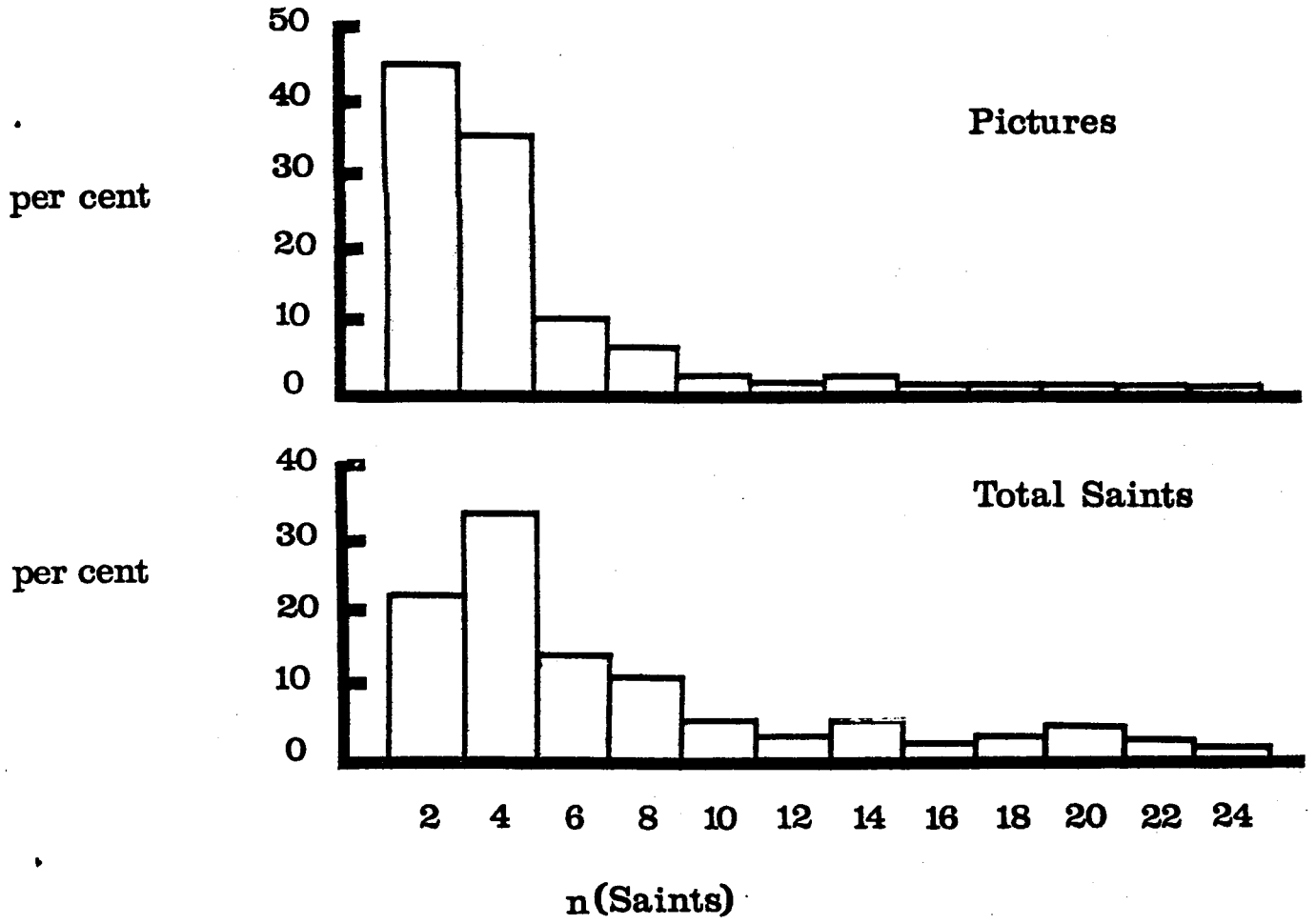

50
40
30
20
10
0
Pictures
per cent
40
30
20
10
0
Total Saints
per cent
2 4 6 8 10 12 14 16 18 20 22 24
n(Saints)

Figure A13:3.2 shows for Italian Renaissance polyptychs, the number of works containing various numbers of symmetry 'errors'. n = 605; mean = 0.27438, standard deviation = 0.52381. The distribution is significantly different from that of a Poisson distribution ($X^2 = 8.65$, 1 df, $p < 0.01$), due to a deficit of polyptychs with no errors and a surplus of those with one error.

Polyptychs: Symmetry Errors

439 153 12 0 1 0 N

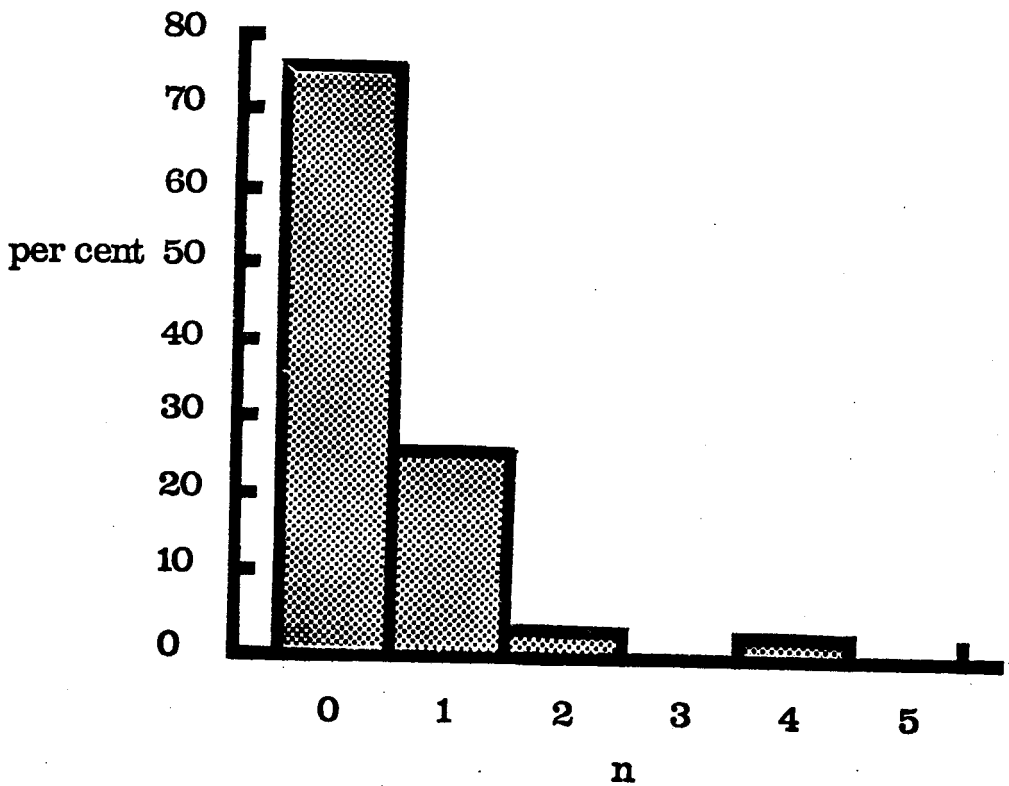
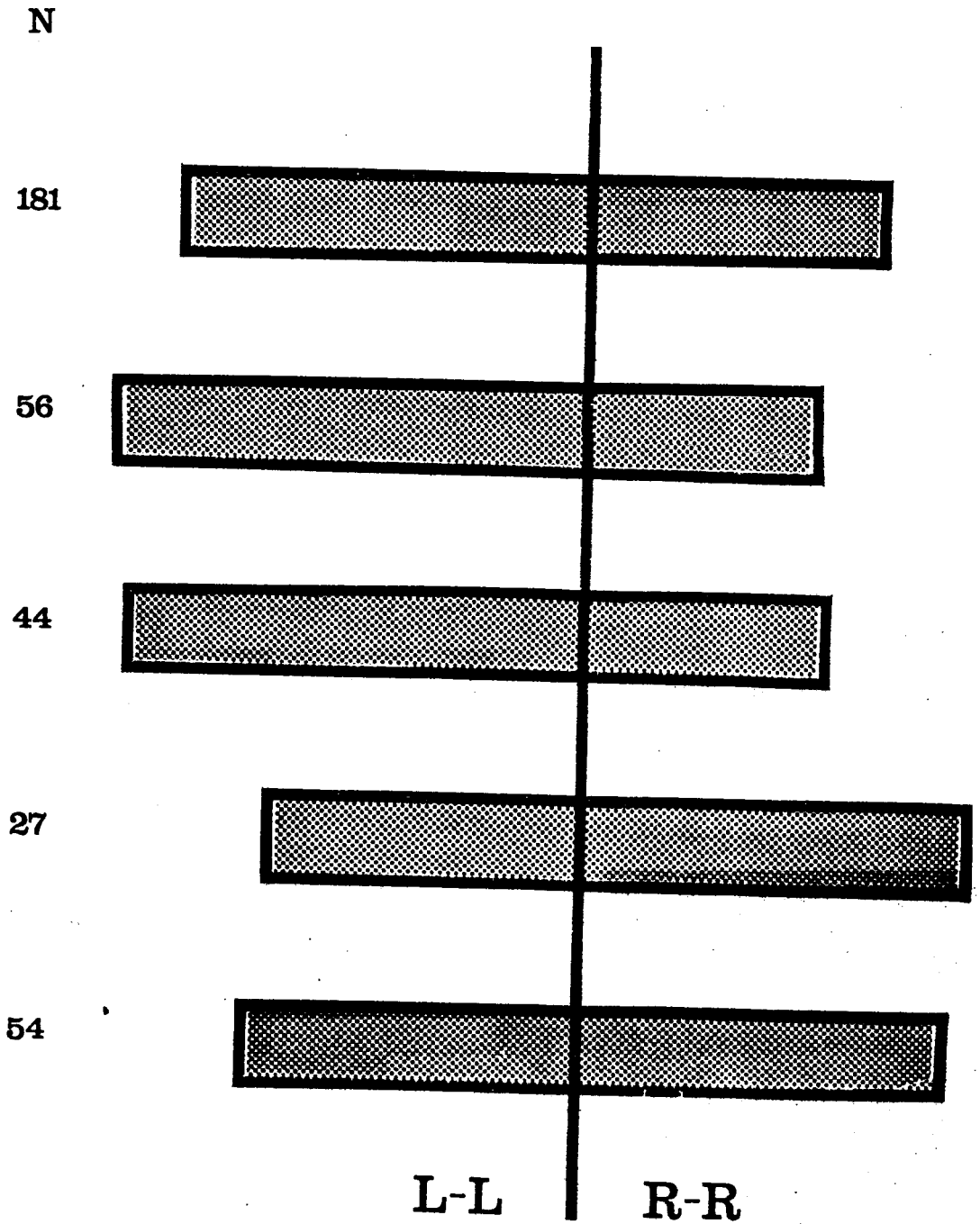


Figure A13:3.3 shows the proportions of symmetry errors in Italian Renaissance polyptychs, by type of error. L-L errors are those in which two left cheeks are actually shown, whilst R-R errors are the converse. The column marked 'per cent errors' is the number of errors overall (see Figure A13.3.1). In the total group the number of L-L errors is significantly greater than the number of R-R errors ($X^2 = 4.34$, 1 df, $p < 0.05$). Further anslysis reveals that this difference is significant only for the Florentine and Central groups ($X^2 = 6.41$, 1 df, $p < 0.02$; and $X^2 = 3.84$, 1 df, $p = 0.05$ respectively), and not for the Venetian and Northern groups ($X^2 = 0.14$, 1 df, NS and $X^2 = 0.02$, 1 df, NS respectively). The difference between the combined Florentine and Central groups, and the combined Venetian and Northern groups is significant ($X^2 = 6.61$, 1 df, $p < 0.02$).



N	per cent L-L		per cent Errors
181	58.0	Total	27.4
56	67.8	Florentine	24.5
44	65.9	Central	27.6
27	44.4	Northern	25.5
54	48.1	Venetian	32.4

Figure A13:4.1 shows the proportion of mediaeval and Renaissance crucifixions in which a spear wound is visible in the right side. Taking the groups pre-1150, 1150-1400 and Renaissance, there is a significant increase in the proportion of Crucifixions with spear wounds ($X^2 = 26.34$, 2 df, $p < 0.001$). The difference between the pre-1150 group and the 1150-1400 group does not achieve statistical significance ($X^2 = 1.61$, 1 df, NS) but the sample size is small.

Crucifixions: Spear Wound

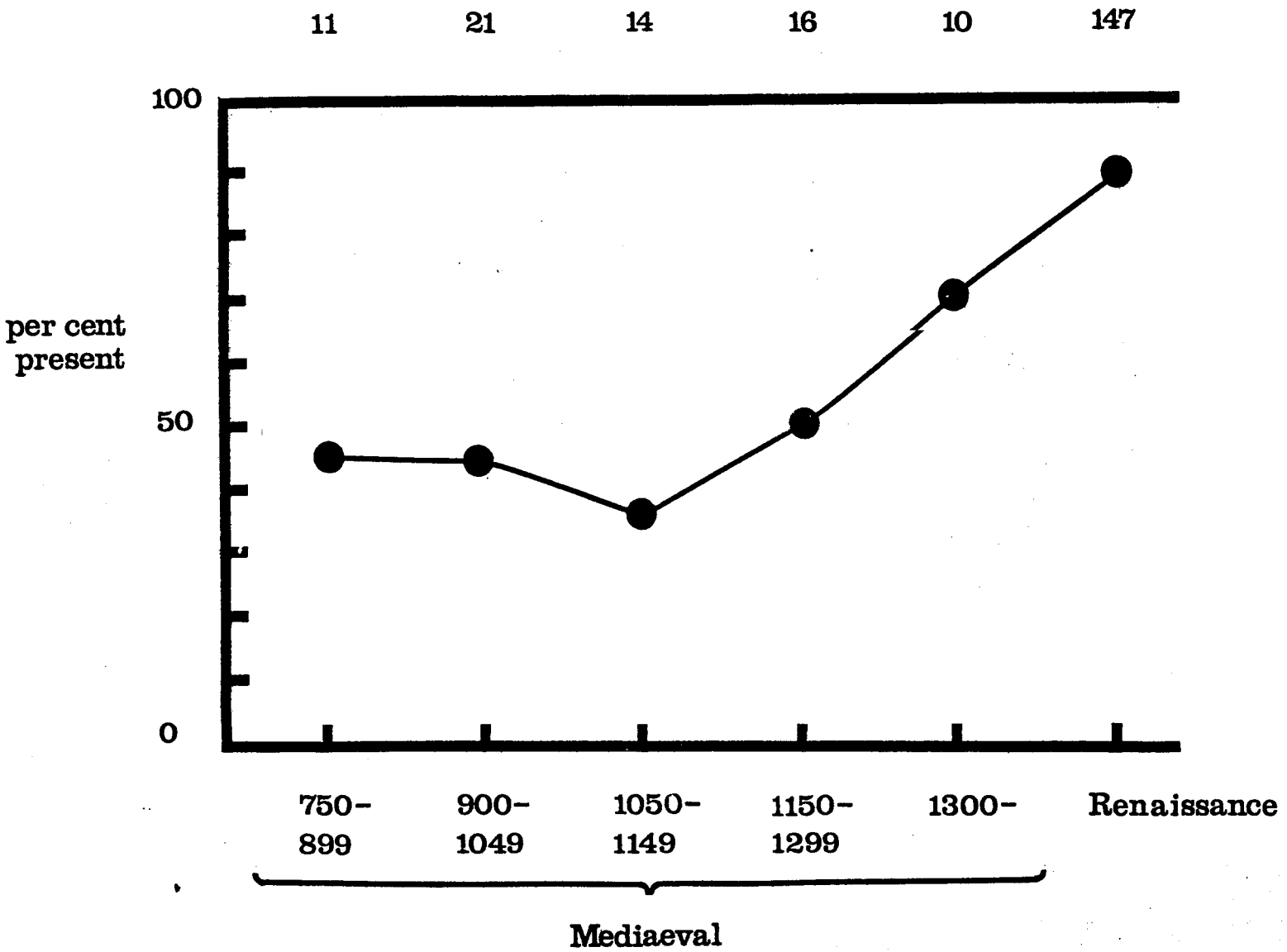
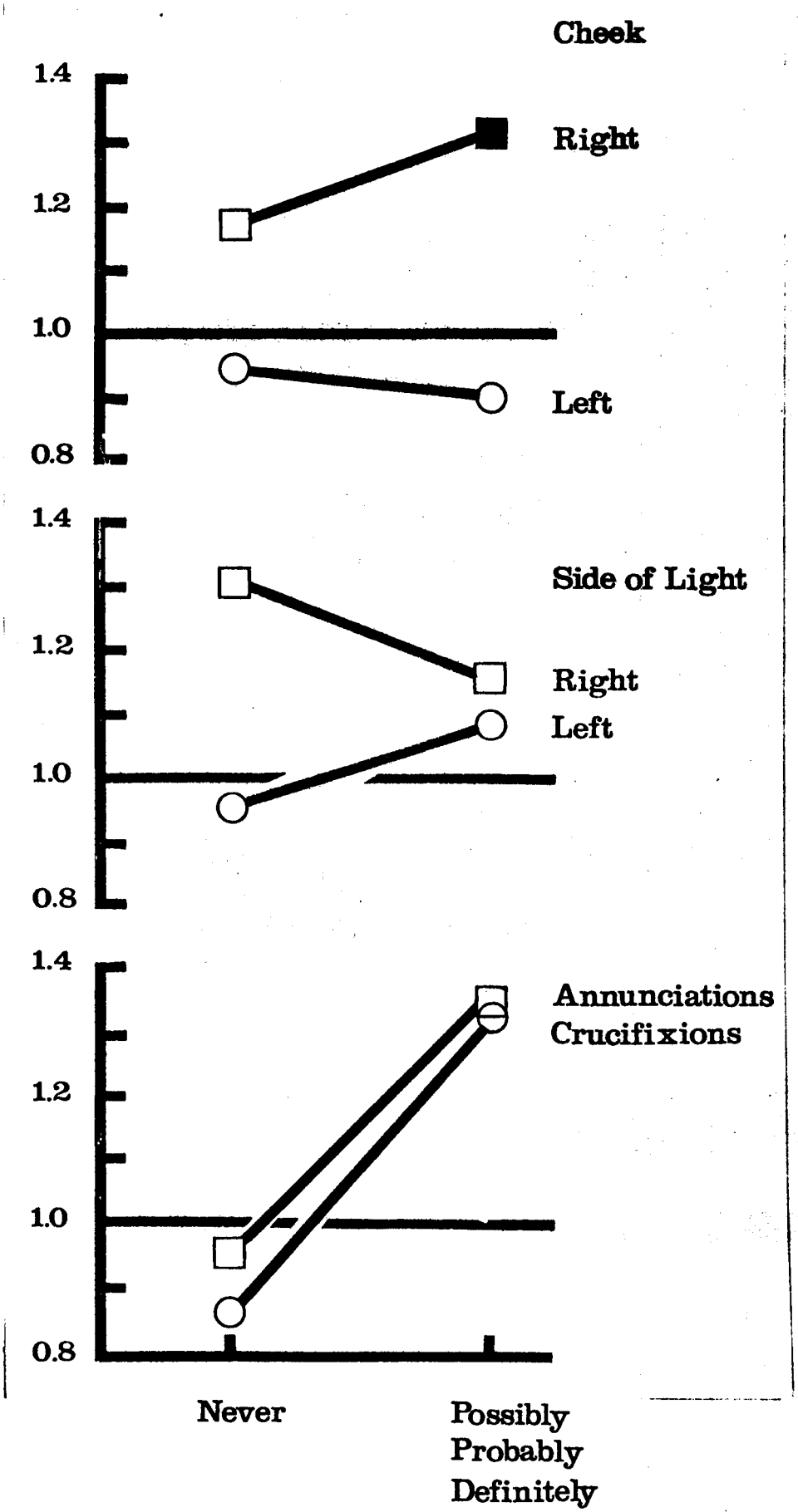


Figure A13:5.1 shows the ability of subjects to distinguish the correct version of a picture from its mirror image. Ordinate represents the ratio of total correct to incorrect choices; chance alone would thus produce a value of 1.0. The abscissa shows the subjects's relative estimates of whether they have seen the picture before; 'possibly', 'probably' and 'definitely' have been lumped together for the purposes of this analysis. Open data points are not significantly different from chance expectations on a X^2 test with 1 df at a probability level of 0.05; the solid point is significantly different from chance. Other details of the experimental groups may be found in the text.


Cheek
1.4
1.2
1.0
0.8
Right
Left
Side of Light
Right
Left
Annunciations
Crucifixions
Never
Possibly
Probably
Definitely

rect
rrect

Plate 13:1 Jacopo Bassano's The Annuncia

(National Gallery of Art, Washington: al

Plate 13:1 Jacopo Bassano's The Annunciation to the Shepherds (National Gallery of Art, Washington: also in Berenson, 1967).

Plate 13:2 St. John and the Angel, from Apocalypse, by the Canterbury School, third quarter of the thirteenth century (Lambeth Palace Library, London; see Rickert, 1954, plate 113b).

Plate 13:3 A typical Byzantine Madonna and Child with almost total symmetry (except for the hands). Mosaic in the apse of the Euphrasian basilica, Porec (from Beckwith (1970), Figure 97). ca. 550 AD.

Plate A13:3.1. The Madonna and Child enthroned with various saints, by Gregorio Schiavone (National Gallery, London)

Plate A13:3.2. Madonna and Child with Saints, by Carlo Crivelli (National Gallery, London)

Plate A13:4.1 Shows a complete view of the Shroud of Turin, front and rear, in negative.

Plate A13:4.2 shows a detail view of the face of the Shroud of Turin, in negative view.

Plate A13:4.2 shows a computer reconstruction of the three dimensional characteristics of the face of the Shroud of Turin (from Jackson et al., 1977). Note that it is suggested that the small circular objects on the eyes are coins (see Wilson, 1978 for details), and that the irregular object on the forehead might be a Jewish phylactery.

Plate 13:1 Jacopo Bassano's The Annunciation to the Shepherds (National Gallery of Art, Washington: also in Berenson, 1967).

Plate 13:2 St. John and the Angel, from Apocalypse, by the Canterbury School, third quarter of the thirteenth century (Lambeth Palace Library, London; see Rickert, 1954, plate 113b).

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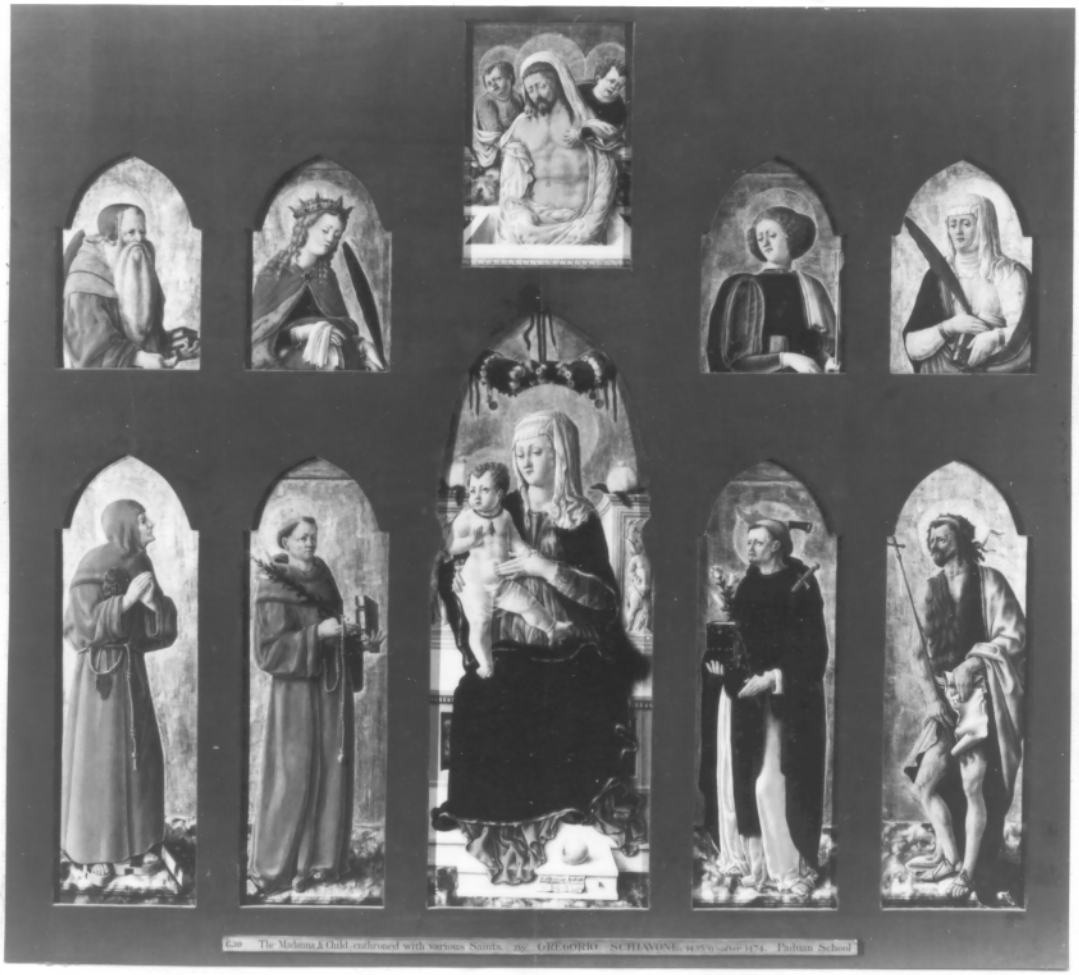


Plate A13:3.2. Madonna and Child with Saints, by Carlo Crivelli (National Gallery, London)

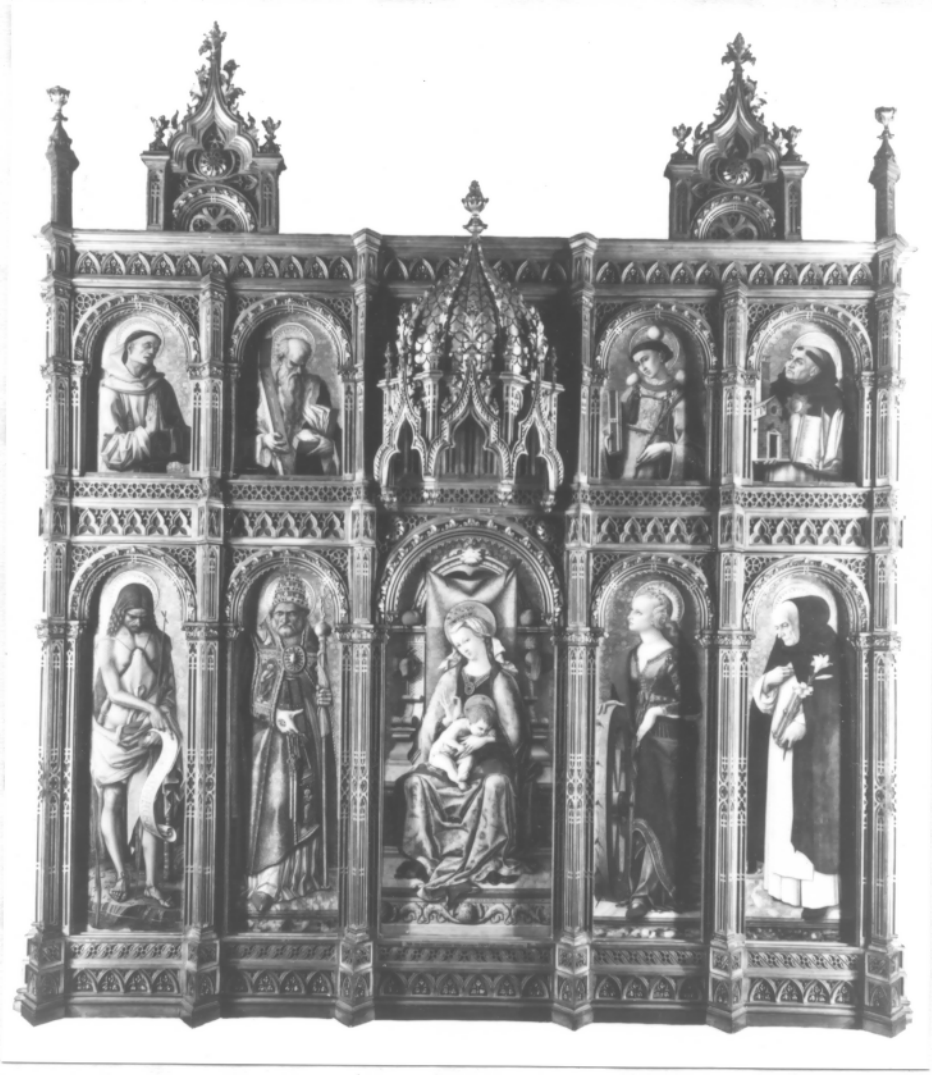


Plate A13:4.1 Shows a complete view of the Shroud of Turin, front and rear, in negative.

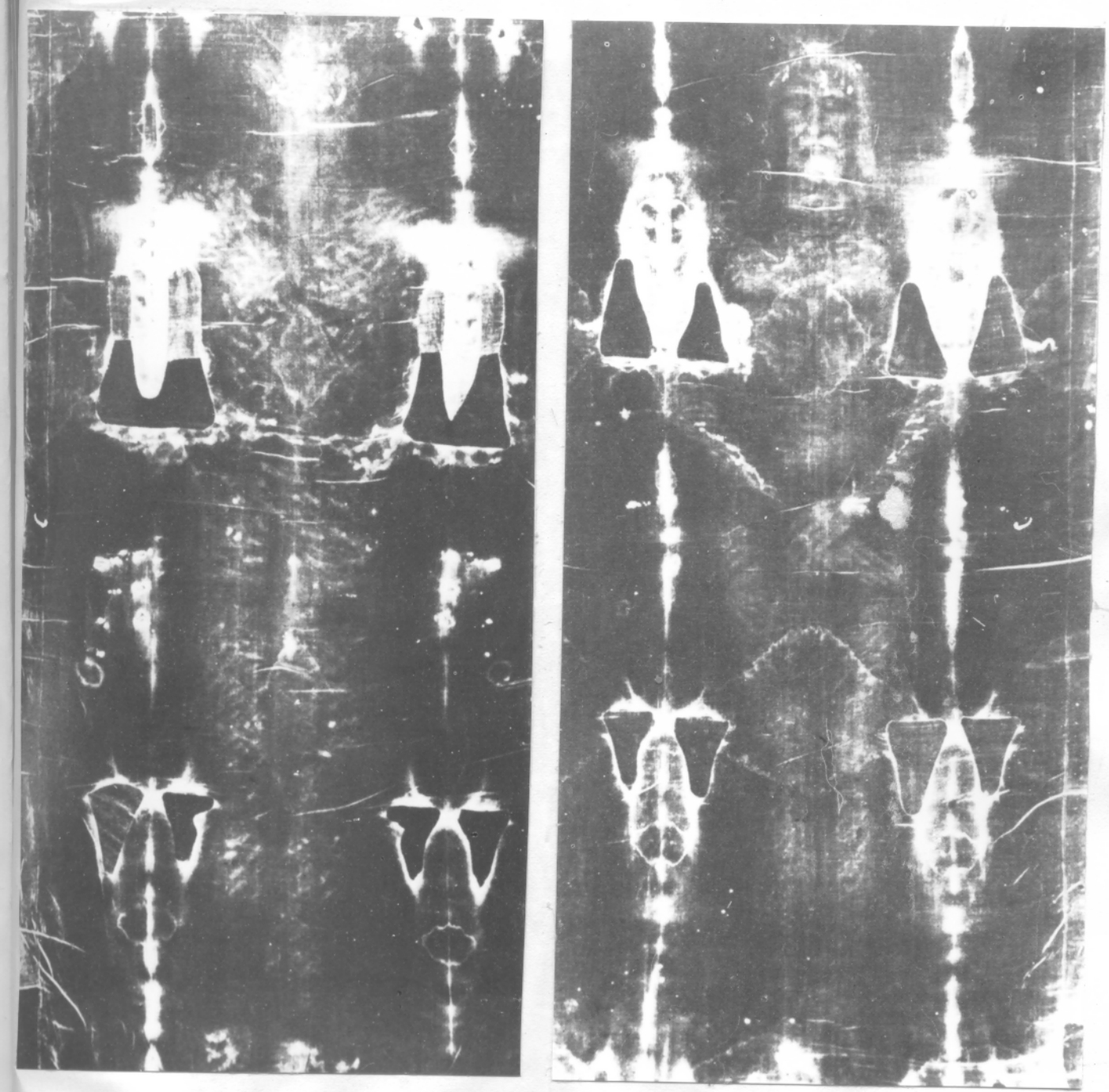
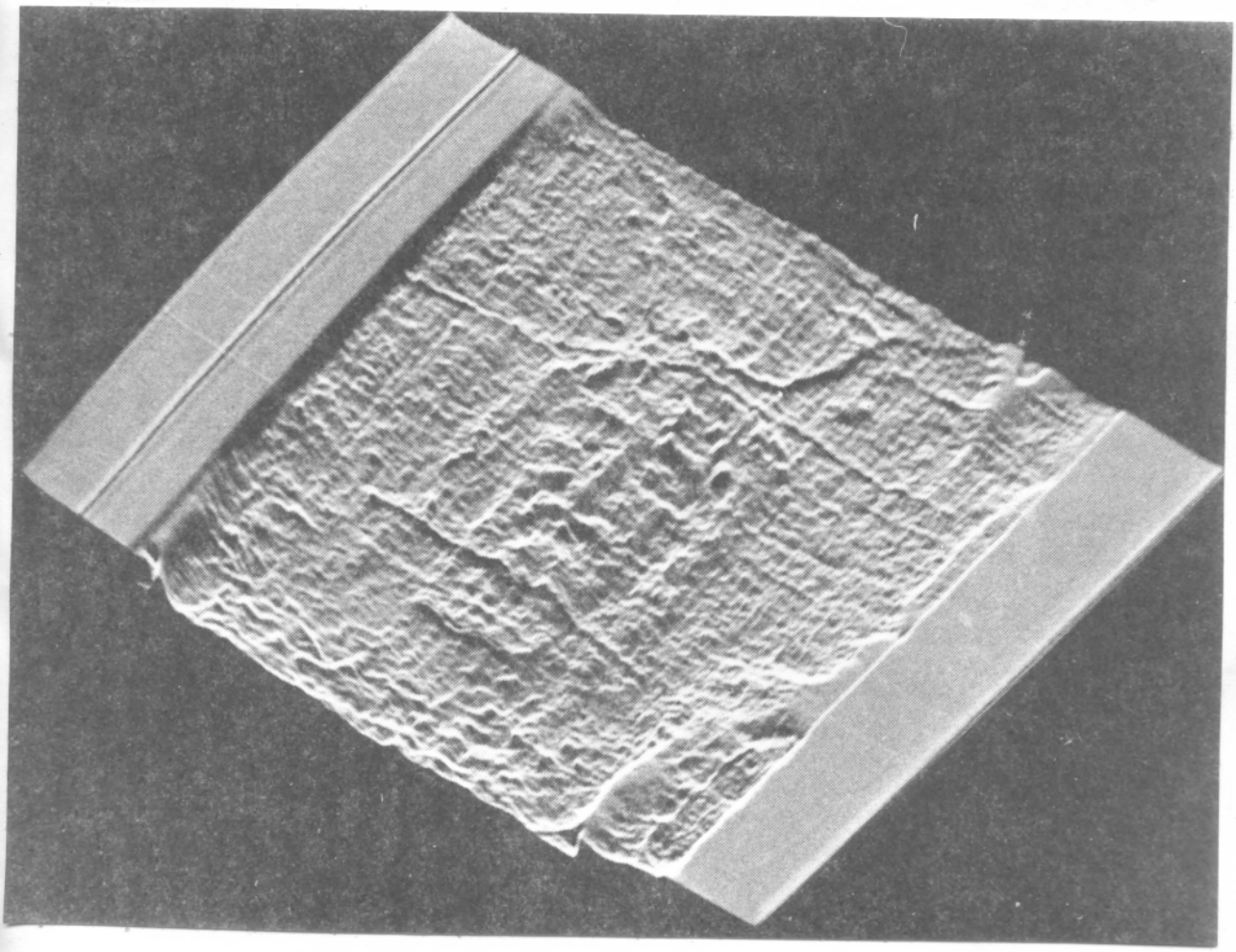


Plate A13:4.2 shows a detail view of the face of the Shroud of Turin, in negative view.

Plate A13:4.2 shows a computer reconstruction of the three dimensional characteristics of the face of the Shroud of Turin (from Jackson et al., 1977). Note that it is suggested that the small circular objects on the eyes are coins (see Wilson, 1978 for details), and that the irregular object on the forehead might be a Jewish phylactery.



CHAPTER 14: THE MEANING OF THE LEFT CHEEK

Summary

The meaning of portraits showing either the left or the right cheek has been studied by means of Semantic Differential technique.

The particular cheek presented to the subject produces little difference in meaning. There are however significant differences in meaning between those portraits originally painted showing the left cheek as opposed to the right cheek.

14:1 Introduction

Portraits tend to show the left cheek more often than the right (see Chapter 13). The difference is more pronounced in portraits of women than of men. For self-portraits however it is the right cheek that is more common. On the basis of the latter finding, and in conjunction with a detailed analysis of Rembrandt's work, Humphrey and McManus (1973) suggested that the meaning of the two cheeks was different, and that when the artist painted an individual showing the right cheek then he was portraying that person as being closer to the artist than if the left cheek was painted; this hypothesis was particularly supported by the relation between the cheek shown in Rembrandt's portraits and the kinship status of the subjects. The hypothesis has since received further support from a detailed analysis of paintings of the Crucifixion, Annunciation and Madonna and Child in Mediaeval and Italian Renaissance art, (Chapter 13), and of the same picture types in Flemish, Dutch and German paintings of the Renaissance and Reformation (Wolfe, 1978).

The question thus arises whether judgements of an ordinary subject, when viewing a portrait, are influenced by the cheek shown. The present study has investigated this question by showing portraits and their mirror-images to a number of subjects and asking them to make semantic differential judgements of the stimuli.

14:2 Experiment 1

Method

Twenty subjects were shown a series of 16 portraits of single individuals, and asked to rate the person in each portrait on 20 semantic differential scales. The 20 scales for each portrait were each placed on an individual sheet in an answer booklet. Each successive sheet in the booklet was the mirror-image of the previous one; thus if sheet 5 had Good-Bad at the top, sheet 6 had Bad-Good, and sheet 7 was the same as sheet 5. 10 of the subjects started with one version of the first sheet, and the other ten with the other version. Each scale had seven divisions on it, and subjects were instructed to use the scales as in the instructions on pages 82-84 of Osgood et al (1957).

Of the 16 portraits seen by each subject exactly half showed the left cheek and half the right cheek. Each portrait was shown to half the subjects showing its left cheek and to the other half of the subjects showing its right cheek (by reversing the slides in the projector). There were thus two series of portraits, the order in one being randomised, and the other being the mirror-image of the first series. Half of the portraits were of males, and half were of females. Each portrait showed only a single person. The stimuli were photographed from post-cards and back-projected on to a ground-glass screen in a dimly

illuminated room. The stimuli were of the order of 40 x 40 cms, and were viewed from a distance of about 1.5 metres. The rate of presentation of the stimuli was controlled by the subjects, by means of a push-botton. No time limits were set; most subjects completed the experiment within 30-45 minutes. All subjects were tested individually. The order of the stimuli, left and right cheek, male and female, was randomised, with the constraint that one of each of the four possible stimulus types occurred in the first four stimuli.

Of the 20 subjects, 8 were left-handed and 12 were right-handed, and 14 were male and 6 were female. Subjects were mostly undergraduates of the University of Cambridge, and none were reading art, history of art or architecture. None were aware of published work on the left cheek phenomenon.

Having carried out the first part of the experiment the subjects were then shown a further four slides, these being repeats of the first four slides shown in the experiment. The subjects were informed that as the first four sets of judgments in an experiment were often unreliable, the purpose of the repetition was merely to allow a more accurate response to the first four stimuli. Six of the subjects did indeed see the first four slides as they had originally been shown, thus allowing a measure of test-retest reliability. The remaining fourteen subjects

saw the first 4 stimuli in the mirror-image of the form in which they had first seen it. Only two subjects noted the reversal, and they noticed it only for a single stimulus each. This part of the experiment thus allowed a within subject analysis.

After the experiment the subjects were asked what they thought the experiment was intended to discover. No-one mentioned any aspects of right or left. The subjects were then informed of the true purpose of the experiment.

Results

The judgements on the 20 scales (shown in Table 14:1) were factor-analysed in order to reduce the number of variables to be considered. This was carried cut individually for each subject (since there was concern that there might be large individual differences in semantic space), and also for the composite data of all subjects. No important differences were found between the two methods, although there were slight inter-subject variations; the population factor-analysis was used for the results in this paper. The judgements on each of the 20 scales were inter-correlated and the resultant 20 x 20 matrix factor analysed by means of the SPSS programs (Nie et al, 1975), using the PA2 option, and then a Varimax rotation. The first three factors had eigen-values of 5.9, 3.8 and 2.1, all highly significant. The next five eigen-values were 0.96, 0.90, 0.83, 0.70 and

0.64, all of which are clearly non-significant, and thus only the first three factors, accounting for 59.6% of the total variance, were rotated. Table 14:1 shows the 20 scales, their communalities, the loading on each of the varimax rotated factors, and the per cent of the common variance attributable to each factor.

To obtain three factors in an experiment such as this one is fairly typical (Osgood et al, 1957). Naming of the factors presented little difficulty. Factor 2 clearly corresponds to Osgood's 'Evaluation', and Factor 1 to Osgood's composite Potency and Activity scale, or 'Dynamism'. Factor 3 does not relate to Osgood's analysis and is clearly a function of the particular scale items chosen in the present experiment, items such as Spiritual and Sacred being included to test the possibility (derived from studies of Italian Renaissance painting), that the left cheek might be more spiritual; Factor 3 has been labelled as 'Spirituality'.

From the factor analysis the judgements of each picture by each subject were recalculated as scores on each of the three factors (these scores each have an overall mean of zero and variance of unity, although this does not apply to the results of individual subjects).

Because of the design of the experiment, by which each subject saw four of the pictures on two separate occasions,

it is possible to produce an error estimate for each subject. However a complete two-way analysis of variance (i.e. subject and picture plus subject picture) is not possible. Considering just main effects (i.e. ANOVA model is subject plus picture) there are highly significant differences in Evaluation, Dynamism, and Spirituality for different pictures ($F(15,365) = 12.50$, $p < 0.001$; $F(15,365) = 2.42$, $p < 0.001$; $F(15,365) = 14.41$, $p < 0.001$ respectively) and also for different subjects ($F(19,365) = 2.415$, $p = 0.0009$; $F(19,365) = 2.375$, $p = 0.0011$; $F(19,365) = 1.613$, $p = 0.0505$ respectively). Since overall differences in means between subjects are not of real interest, these differences have been removed by standardising each subject's scores so that they have a mean of zero, and variance of unity. It is now possible to examine subject-picture interactions by using an ANOVA model of the form (picture + picture*subject). Cheek shown to the subject may also be analysed as a main effect within this model; this produces a model of the form 'picture + picture subject + cheek shown', the three levels having 15, 304 and 1 df respectively, there being a residual with 79 df's.

Picture main effects are still all significant with probability very much less than 0.001. Picture subject interactions for each of Evaluation, Dynamism and Spirituality are all significant ($F(304,79) = 2.408$, $p < 0.0001$; $F(304,79) = 2.380$, $p < 0.001$; $F(304,79) = 2.379$, $p < 0.001$ respectively).

Thus there are differences between the ways in which particular subjects view particular pictures.

The cheek shown does not seem to affect the perception of the picture ($F(1,79) = 0.211$, $p = 0.647$; $F(1,79) = 0.086$, $p = 0.770$; $F(1,79) = 0.289$, $p = 0.592$ respectively).

From experiment 1 we must therefore conclude that it is unlikely that differences exist in the evaluation of pictures according to the cheek shown to the subject.

The hypothesis of McManus and Humphrey (1973), regarding the meaning of the left and right cheek, seems to receive no support from the experiment so far. However the original hypothesis primarily concerned the perception of pictures by artists. It is thus possible that if we carry out an experiment with the original artists, then significant results would be obtained. In the absence of this, an *a posteriori* analysis of the data of experiment 1 was carried out to discover whether the judgement of a portrait related, not to the cheek as perceived by the subject, but to the cheek as originally painted by the artist. Clearly there is no possibility of a within subjects analysis of such an hypothesis since it is not possible to obtain a second, mirror-imaged version of the painting, as painted by the artist. One may therefore carry out only a between subjects analysis.

Table 14.2 shows, for the total results of experiment 1 the difference in mean score on each of the three factorial dimensions between those pictures showing the left cheek in the original (n = 10), and those showing the right cheek (n = 5); (one picture could not be classified in this manner as the original could not be found). For evaluation and dynamism the p levels in a one-way analysis of variance (two-tailed) show a tendency, particularly in the case of dynamism, for the two portrait types to be different. The scale in Table 14.2 is SD (z) units, and hence the left cheek pictures appear to be half of a z unit less dynamic or evaluatively positive. Clearly very little can be made from this result since the analysis is a posteriori, and the sample sizes are small. Hence a second experiment, with more pictures and less subjects was designed to study the question further.

14:3 Experiment 2

In this experiment subjects were shown a series of 106 portraits, and asked to rate the portraits on a semantic differential consisting of a subset of nine of the scales used in experiment 1 (marked with an asterisk in Table 1); these scales were selected for their high loadings on the three main factors. Subjects saw the total of 106 portraits in four separate experimental sessions, the first three sessions consisting of 26 portraits, and the fourth of 28 portraits. Sixty-six of the portraits,

originally showed the left cheek, and 40 the right cheek (approximately the population proportions). In the series of 106 portraits half were presented to the subject showing the left cheek, and half the right cheek, the orientation being determined independently of the original cheek painted by the artist.

Four subjects participated in the experiment, and each took from 25 to 60 minutes on a session. The experimental situation was basically similar to that of experiment 1, except that stimuli were front projected, and were slightly larger. Two of the subjects saw one particular randomised sequence of the 106 portraits, and the remaining two subjects saw the mirror image of this stimulus sequence.

In view of the small number of subjects the data for each subject were factor-analysed individually, using the same method as described in experiment 1. Factor-scores were then calculated for the judgements of each portrait on each of the three dimensions. In each subject the three dimensions of Evaluation, Dynamism and Spirituality were readily identified, although there were minor differences in emphasis and correlation pattern between subjects. For each subject the distribution of factor-scores on each dimension had a mean of zero and a variance of unity.

As with experiment 1, no differences could be found in experiment 2 between pictures according to the cheek

actually shown to the subject, although this was not surprising in view of the between subjects design, and the negative result of experiment 1. Detailed results will not be reported here.

Table 14:2 shows the results of both experiments according to the cheek originally painted by the artist. The a posteriori results of experiment 1 have been taken as indicating the expected direction of any effect in experiment 2, and thus the table shows, separately for each subject, the one-tailed probability of a difference between right and left cheek. Results for the four subjects have been combined using the method of Kendall (1951). For Spirituality and Dynamism there is no evidence of a replication of the effects of experiment 1, ($X^2_8 = 2.96$, and $X^2_8 = 11.63$ respectively, both NS). For Evaluation, three of the subjects show an effect in the same direction as experiment 1, and for one of these subjects the result is highly significant ($p = 0.0007$) and for another the result is almost significant ($p = 0.06$). Combining the four subjects by the method of Kendall (1951) produces a X^2 value of 25.97, which with 8 df is significant ($p < 0.001$). As in experiment 1 this result cannot be put down to the effects of repeated comparisons, as if one combines the three overall values for the four subjects, one still obtains a X^2 value of 40.56, which with 24 df is still significant with $p = 0.0186$.

Lest it be thought that the statistical method of analysing the subjects separately and then combining them is not valid it may be pointed out that if the original scale judgements of each subject are combined and then these composite results factor-analysed and the resultant factor scores then analysed in terms of the original cheek painted by the artist, then the only significant result is for evaluation, with a one-tailed significance of $p = 0.0215$.

Experiment 2 thus provides a replication of the most surprising finding of experiment 1, that the evaluation of a portrait depends not upon the cheek as shown to the subject, but upon the cheek as painted by the artist. The magnitude of the effect is slightly smaller in experiment 2 than in experiment 1, the right cheek being 0.215 SD units more evaluatively positive, as compared with 0.580 SD units in experiment 1.

14.4: Discussion

The results of this experiment are initially surprising; a subject's evaluation of a portrait is not dependent upon the cheek as perceived by him, but instead relates to the portrait as conceived originally by the artist. From judgements made independently of the asymmetry of a portrait we may gain knowledge as to its original asymmetry, a superficially counter-intuitive result.

Two extremes of explanation arise, although intermediate positions are possible. Despite the lack of any significant results in the first part of experiment 1, it is nevertheless still possible that there is a gradient of sensitivity to the meaning of cheeks, ordinary subjects hardly being sensitive at all, and artists being very sensitive.

An alternative explanation is that indeed the ordinary subject has no sensiti ity to the cheek shown, and the artist himself is only sensitive to it in so far as it is related to an iconographic tradition. The ordinary subject would thus have no access to such restricted information, and hence could make no assessment at all on the basis of the perceived cheek.

However in neither case is it unreasonable to postulate that the ordinary subject's judgements will be related to the original cheek, as painted. Since it seems to be typical (or perhaps even necessary) for stimuli to be evaluated in three semantic dimensions only (Osgood et al, 1957), then the artist's sensitivity to the cheek would be linked on to one of the three semantic dimensions. However, there are many other cues as to these dimensions apart from the cheek, and since the subject would have access to this alternative information about the dimensions the subjects judgements would be expected to relate to the cheek as originally painted. If correct this hypothesis would predict that assessments by artists of other artists paintings might depend for moreover the

rather than upon the conceived cheek.

Whether or not the ordinary subject can make any judgement at all of the perceived cheek is of importance in determining the origin of the left cheek phenomenon. If the phenomenon has biological origins, perhaps in the asymmetry of the human brain, we would expect all subjects to show the phenomenon to a greater or lesser extent, although artists would be expected to show the phenomenon far more strongly. If however the phenomenon is primarily iconographical (and hence, in some sense, arbitrary), then ordinary subjects should show no awareness of the phenonenon at all. The present experiment suggests that the iconographical interpretation may well be correct, although a larger replication of the main result of experiment 1 is necessary before this question can be answered definitively.

In summary the meaning of a picture is clearly related to the cheek shown in the original version, but is unlikely to be related to the cheek as perceived.

Table 14:1

Shows the 20 semantic scales of experiment 1, their communalities and loadings on the three Varimax factors. An asterisk next to a scale indicates that it was used in experiment 2.

NOTE: Decimal points have been omitted from all loadings and communalities. Loadings which may be regarded as significant (i.e. ≥ 0.30 or ≤ -0.30) have been underlined.

For each pair of words in a scale the first one is the one referred to in the loadings.

Scale	Dynamism Factor 1	Evaluation Factor 2	Spirituality Factor 3	Communality
*Good-Bad	08	72	33	65
Noble-Ignoble	42	24	39	39
*Wise-Foolish	70	18	27	61
*Kind-Cruel	-18	80	22	74
Beautiful-Ugly	-02	68	10	48
Successful-Unsuccessful	78	-06	-05	62
Important-Unimportant	74	-16	12	60
*Strong-Weak	75	-12	-22	63
*Remote-Intimate	26	-62	28	53
Masculine-Feminine	39	-38	-06	30
Hard-Soft	54	-68	-23	82
*Excitable-Calm	-36	-13	-41	31
Active-Passive	64	-12	-38	58
*Powerful-Powerless	85	24	-15	81
Tragic-Comic	-09	-01	39	16
*Spiritual-Physical	-04	23	70	54
Personal-Impersonal	-24	74	-08	60
*Sacred-Profane	-01	44	55	50
Eerie-This worldly	-05	-11	50	26
Like me-Unlike me	20	53	02	33
% Common Variance	52.9%	32.1%	15.0%	

Table 14:2 Results of Experiment 2, with results of Experiment 1, for comparison.

	Difference between Cheeks ($\bar{L}$-$\bar{R}$)		
	Evaluation	Dynamism	Spirituality
Experiment 1			
($\bar{L}$-$\bar{R}$)	-0.5801	-0.4991	-0.2749
Two-tailed p (F-test)	p = 0.1537	p = 0.0851	p = 0.2686
Experiment 2			
Subject 1 (WK) ($\bar{L}$-$\bar{R}$)	-.2963	-.0142	-.2990
One-tailed p (F-test)	p = 0.0600	p = 0.471	p = .0545
Subject 2 (AH) ($\bar{L}$-$\bar{R}$)	+.0106	+.2099	+.0790
One-tailed p (F-test)	p = 0.5225	p = 0.858	p = 0.667
Subject 3 (JMF) ($\bar{L}$-$\bar{R}$)	-.5369	+.0456	-.2258
One-tailed p (F-test)	p = 0.0007	p = 0.598	p = 0.1095
Subject 4 (RFD) ($\bar{L}$-$\bar{R}$)	-.0385	+0.2725	+.1198
One-tailed p (F-test)	0.4179	p = 0.9395	p = 0.749
Combined results of Experiment 2			
One-tailed tests			
X^2 (8 d.f.)	25.97	2.96	11.63
P	(p = 0.0011)	(p = 0.936)	(p = 0.167)

Epilogue

"Today's science is built upon yesterday's science; and yesterday's science, in turn, is based on the science of the day before".

Popper, Objective Knowledge, pp 346-7.

The two parts of this thesis bear almost no obvious relationship to one another; at least, not on superficial examination. Some explanation is necessary. But whilst some explanation of the discrepancy between the parts is necessary, one could also argue that an explanation is also necessary for why the topic of laterality is being studied at all. And if why is not possible, at least some attempt should be made to say how one came to be where one is. Thoman Kuhn, in his The Structure of Scientific Revoltuion (p.177-8) discusses the problem of what a scientific community is, and how people begin to work on particular problems, to call themselves, a "school", etc. Firstly he talks about the problem at its lowest level, and then says "It is only at the next lower level that empirical problems emerge. How, to take a contemporary example, would one have isolated the phage group prior to its public acclaim? For this purpose one must have recourse to attendance at special conferences, to the distribution of draft manuscripts or galley proofs prior to publication, and above all to formal and informal communication networks including those discovered in correspondence and in the linkages amongst citations". One might also add that scientists themselves surely have some responsiblity to assist this process by describing the evolution of their ideas (and particularly perhaps the extinct hypotheses buried deep in their past). Scientists should not only say what they have done, but also why they have done it. Such a process will not only assist

the historian or sociologist of science, but will probably also assist most scientists to dissociate themselves from the continued "lie" which Medawar (1963) has argued is perpetuated in most scientific papers. A lie which suggests that scientists actually think in the way they present their papers: Introduction; Discussion of past literature; Hypothesis; Experiment; Conclusion. Science doesn't usually work that way - although quite how it does work is anyone's guess.

In the next part of this Epilogue I propose to be completely autobiographical, in an attempt to explain the rather heterogeneous collection of material in this thesis. To anyone not interested in such inward-looking meanderings, I suggest that they should simply ignore the rest of this chapter.

Like so many Ph.D. theses, this one finds its origins in an undergraduate research project; although the route from there is so convoluted and indirect that if it were presented as an explanatory hypothesis it would probably be rejected as absurd.

The Part II course in Psychology in Cambridge requires one to carry out a research project; and in particular one may, if one wishes, select one's own topic. Since leaving school I had had an interest in art, in particular in the visual arts and music. The question

of quite why man looks for beauty in art strikes me as a profound one to which I had never seen even the slightest hint of a psychological explanation (at that stage I had not read Freud properly). A chance summer encounter, in a Penguin dictionary, with a description of the Golden Section rectangle, and in particular, of Fechner's experiments, interested me, as they seemed to represent a readily testable hypothesis. Over that Long Vacation I carried out some rudimentary statistical analysis of the proportions of several hundred paintings by Turner and Van Gogh. In particular I had found that the proportions of Turner's paintings were dependent upon their size ~~(see Figure 1)~~. On arriving back in Cambridge I had decided that I would like to know if rectangle preference varied with stimulus size. I was particularly fortunate in being directed towards Nick Humphrey, who patiently encouraged me to follow up each of my enthusiasms. During the Michaelmas and Lent Terms I collected together details of portrait paintings, primarily to examine their proportions. Having recently learned to use the University Computer I punched all their details onto cards, and examined a number of features of each one. To my intense surprise there were ~~some~~ more left-facing portraits then right facing ones. Nick Humphrey instantly realised the possibilities of such an obseration, and encouraged to me look further at the result. That encouragement, along with a heavy dose of serendipity, lead to the results presented in Chapter13 (which is also written auto-

biographically). Meanwhile my rectangle experiments continued (also over the next few years); the details (although not the evolution) of these have been published elsewhere (McManus, 1978).

After graduating from Cambridge, I went off to Birmingham to study Clinical Medicine. My heart however remained in research - and particularly in problems of laterality. During my time at Birmingham I continued my research in left and right in paintings, and I also kept an eye out for any other asymmetries, particularly of disease incidence. One asymmetry which particularly intrigued me was that not only did inguinal hernias occur with differing incidences on the two sides, but that the testes themselves were asymmetric. Chang et al (1960) put me on the track of a quotation of Winckelmann (1764; 1968), who had also noticed this asymmetry in Greek sculptures. A holiday in Italy gave me the opportunity to collect some empirical evidence (observing nearly 200 sculptures), and a few months after my return, a short paper, in Nature by Mittwock and Kirk (1975) gave me the necessary excuse to publish my data. This prompted a correspondence with several people. On my return to Cambridge I worked through Aristotle and several other authors, and realised quite how extensive was not only this asymmetry, but ideas about it. Chapter 12 contains a summary of my ideas on the topic.

After graduating from Birmingham, I completed my pre-registration house-job, and then returned to Cambridge to work for a Ph.D. - on the influence of colour upon behaviour, under the supervision of Nick Humphrey. Despite its promising possibilities, this topic never really got off the ground. Several preliminary experiments failed to produce anything interesting, and a review of the literature produced little of worth. After a few months my mind was wandering back to laterality. I finished writing a draft of the chapter on the left cheek (Chapter 13) and continued to scan the literature, hoping to add more items to my catalogue of disease asymmetries. About this time I had re-made an acquaintence with Mike Morgan (who had also taken part in the scrotal asymmetry correspondence in Nature), and he asked me if I had thought of working on the inheritance of left-handedness. I vividly remember saying that I thought the whole area was a mine-field, and that I had every intention of steering well clear of it. About that time I read a draft of his paper (with Mike Corballis) in which he proposed that there was no genetic basis to laterality, in the usual sense of the word 'genetic'. I found this disconcerting, as there seemed to be clear evidence of a familial tendency and whilst 'genetic' and 'familial' are not synonymous, they are often related. One way round this difficulty would be to propose an inherited tendency to, say, pathological left-handedness as a result of brain-damage during borth. To examine this problem (and at that time I was fairly convinced that birth

trauma did cause brain damage and left handedness) I carried out what I have later called survey Ia. This showed no detectable effect of birth stress on handedness, but it did show a strong familial effect. I then began to be interested in the possibility of a genetic model; not knowing any genetics however, I gave myself a week's crash course by reading Cavalli-Sforza and Bodmer's testbook from cover to cover. But I still had no adequate genetic model.

Whilst sitting on an Underground train I suddenly realised that if one homozygote produced only 50% of left handers, then a genetic model would produce relatively low incidences of left-handers in R x L and L x L matings. At this time, incidentally, I had hever heard of the term "fluctuating asymmetry". I constructed three models, dominant, recessive and additive, and tried to fit the available data. The family data fitted the additive model fairly well. But MZ twins presented more of a problem; and DZ twins were never adequately fitted. The problem with MZ twins was that the incidence of sinistrality was mugh higher than in DZ twins. To account for this, and also to fit the data adequately, inspired by the paper of Munsinger ( ), I invoked the concept of the identical twin transfusion syndrome. By now my model fitted, but not well. But there were several major problems.

At about this stage I came to know Nick Mascie-Taylor, and he put further fuel on a fire which had been intriguing me for a while; hand-clasping and arm-folding. After much discussion with him I fitted the data to a three-allele model.

By now it was apparent that several defects in the handedness data needed remedying. The first was the discrepancy between the different studies in the incidence of left-handedness. Initially I had treated all such differences as due to differences in allele frequency; but since most of the differences in incidence were due to differences in criteria this was clearly an absurd thing to do. I therefore derived the method described in Chapter 7, to account for discrepancies between manifest and true handedness. This worked well, and allowed the model to fit all of the family data; and also the previously DZ twins were now brought into the fold. But the MZ twins gave me trouble. I had to retain the identical twin transfusion syndrome in order to explain the increased sinistrality in MZ twins; and yet the model would fit only if I omitted these corrections. At this stage I began to look critically at the twin studies, and I suddenly realised that there was no adequate evidence for twins actually being a higher incidence of sinistrality; this work is described in Chapter 4.

A further problem with the handedness model was that

in the early form of the model I had described the ability of the model to cope with data from dichotic and ECT studies on cerebral speech dominance. However, as Professor Zangwill had pointed out to me, it was just not possible to explain the low incidence of aphasia after right-sided lesions in right-handers using the model. The result of that discussion was Chapter 8, in which I present the next stage of the model in which no less than three asymmetries are independently controlled by one gene.

Perhaps I can summarise briefly. Chance led me towards the problem of handedness, and it also led me to an early model which, by some further happy chance, was wrong on several important counts, but gave me sufficient encouragement to continue making improvements so that a better model could be developed, which now fits most of the available data. No doubt the improved model also has glaring anomalies, and, hopefully, these may perhaps be amenable to correction in the future.

Aas, I.H.M. & Risnes, S. (1979) "The depth of the lingual forsa in permanent incisors of Norwegians II: differences between central and lateral incisors, correlations, side asymmetry and variability", American Journal of Anthropology, 50, 341-347. 11.18

Abler, W.L. (1976) "Asymmetry in the skulls of fossil man: evidence of lateralised brain function?", Brain, Behaviour & Evolution, 13, 111-115. 10.1

Abouna, J.M. & Brown, H. (1968) "The treatment of mallet finger", British Journal of Surgery, 55, 653,-667. 11.19

Acheson, R.M., Chan, Y-K. & Clemett, A.R. (1970) "New Haven Survey of Joint diseases XII: distribution and symptoms of osteo-arthrosis in the hands with reference to handedness", Annals of Rheumatic Disease, 29, 275-286. 11.5,11.19a

Adair, H. & Bartley, S.H. (1958) 'Nearness as a function of lateral orientation in pictures', Percept.Mot.Skills, 8, 135-141. 13.39

Adler, A. (1929) Problems of Neurosis, pp64-65, Kegan, Paul, Trench & Trubner: London 6.22

Afzelius, B.A. (1976) "A Human syndrome caused by immotile cilia", Science, 193, 317-9. 7.8,11.14

Annett, M. (1964) " A model of the inheritance of handedness and cerebral dominance", Nature 204, 59-61. 7.17

Annett, M. (1967) "The binomial distribution of right, mixed and left handedness", Quart. J. Psychol., 19, 327-333. 2.7,2.35, 2.48,2.55

Annett, M. (1970) "A classification of hand preference by association analysis", Brit. J. Psychol., 61, 303-321. 2.28

Annett, M. (1972) "The distribution of manual asymmetry", Brit. J. Psychol., 63, 343-358. 2.7,2.28, 7.17,7.18

Annett, M. (1973) "Handedness in Families", Ann. Hum. Genet., 37, 93-105. 7.9,7.33

Annett, M. (1974) Handedness in the children of two left-handed parents. British Journal of Psychology, 65: 129-131. 9.2

Annett, M. (1975) "Hand preference and the laterality of cerebral speech", Cortex, 11, 303-328. 2.26,2.28, 2.31,6.27 7.18,8.27

Annett, M. (1976) "Handedness and the cerebral representation of speech", Annals of Human Biology, 3, 317-328. 6.27,7.3, 7.18

Annett, M. (1978a) A single gene explanation of right and left handedness and brainedness, Lanchester Polytechnic. 2.28,7.7, 7.9,7.13, 7.14,7.18 7.19,7.20 7.34,7.36 7.48.

Annett, M. (1978b) "Genetic and non-genetic influences on handedness", Behaviour Genetics, 8, 227-249. 7.48

Annett, M. (1979) "Family handedness in three generations predicted by the right shift theory", Annals of Human Genetics, 42, 479-491. 7.20

Annett, M., Hudson, P.T.W. & Turner, A. (1974) "Effects of right and left unilateral ECT in naming and visual discrimination analysed in relation to handedness", Brit.J.Psychiatry, 124, 260-264. 8.36

Annett, M., Hudson, P.T.W. & Turner, A. (1974) "The reliability of differences between the hands in motor skills, Neuropsychologia, 12, 527-531. 2.29

Anon (1963) "Report sur le mémoire de M. Dax, relatif aux fonctions de l'hémisphére gauche du cerveau", Bull. de l'Academic impériale de Médécine, 30, 173. 6.4

Anon (1932) Am. J. Psych., 44, 386. 6.21

Anon (1977) "Preventing Prematurity in Twims", British Medical Journal, I, 1618. 4.10

Anthyme, S.P. (1906) 'Les irregularités de plan dans les églises', Bulletin Monumental, 70. 13.65

Antliff, H.R. & Shampo, D.R. (1959) "Causative factors for the scrotal position of the testis", J. Urology, 81, 462-463. 12.8

Archibald, Y.M. and Wepman, J.M. (1968) "Language disturbance and non-verbal cognitive performance in eight patients following injury to the right hemisphere", Brain, 91, 117-130. 6.30

Arge, E. (1960) "Transposition of the viscera and sterility in men", Lancet, i, 412-414. 11.4

Argyle, M. & Cook, M. (1976) Gaze and Mutual Gaze, Cambridge University Press. 13.41

Ask-Upmark, E. & Bickerstaff, E.R. (1976) "Vertebral artery occlusion and oral contraceptives", British Medical Journal, i, 487-488. 11.19b

Atwell, J.D. (1962) "Inguinal hernia in female infants and children", British Journal of Surgery, 50, 294-297. 11.19

Bakan, P. (1971) "Handedness and Birth Order, Nature, 229, 195-196. 2.24,4.10, 5.4,3.1

Bakan, P. (1975) "Are left-handers brain damaged?" New Scientist, 67, 200-202. 3.2

Bakan, P. (1977) "Left-handedness and birth order revisited", Neuropsychologia, 15, 837-839. 3.1,3.2, 3.4,3.5

Bakan, P. (1978) "Why left-handedness?", The Behavioural and Brain Sciences, 1, 279-280. 3.5,5.1, 6.46,7.3

Bakan, P., Dibb, G. and Reed, P. (1973) "Handedness and birth stress", Neuropsychologia, 11, 363-366. 3.1,3.2, 3.5

Baker-Cohen, K.F. (1961) "Visceral and vascular transposition in fishes, and a comparison with similar anomalies in man", Amer. J. Anatomy, 109, 37-55. 7.8

Bakker, D.J., van der Vlugt, J. & Claushuis, M. (1978) "The reliability of dichotic ear asymmetry in normal children", Neuropsychologia, 16, 753-757. 8.4

Barnes, F. (1975) "Temperament, adaptability and left-handers", New Scientist, 67, 202-203. 3.5

Barsley, M. (1966) The left-handed Book, Souvenir Press: London. 6.15

Barthes, R. (1967) Elements of Semiology, Jonathan Cape, London. 13.24

Barthes, R. (1972) Mythologies, Jonathan cape, London. 13.25

Bartley, S.H. & Dottardt, D.C. (1960) 'A further factor in determining nearness as a function of

lateral orientation in pictures", J. Psychol., 50, 53-57. 13.39

Bastian, H.C. (1898) A Treatise on Aphasia and Other Speech Defects, H.K. Lewis: London 6.12,6.13, 6.23

Bastian, H.C. & Horsley, V. (1880) "Arrest of development in the left upper limb, in association with an extremely small right ascending parietal convolution", Brain, 3, 113-116. 6.14

Basso, A., Faglioni, P. & Vignolo, L.A. (1975), "Étude controlée de la rééducation du langage dans l'aphasie: comparison entre aphasiques traités et non-traités", Revue Neurologique, 131, 607-614. 8.18

Basur, R.L., Shephard, E. & Mouzas, G.L. (1976) "A cooling method in the treatment of ankle sprains Practitioner, 216, 708-711. 11.19a

Bateman, F. (1865) "On aphasia, or loss of the power of speech", Lancet, i, 532-533. 6.6

Bateman, F. (1870) On Aphasia, Churchill: London 6.5,6.7

Beccia, D.J., Krane, R.J. & Olsson, C.A. (1976), "Clinical management of non-testicular intra-scrotal tumors", J. Urology, 116, 476-479. 11.19a,12.14

Beckman, L. and Elston, R. (1962) Data on bilateral variation in man: Handedness, hand-clasping and arm-folding in Swedes. Human Biology, 34: 99-103. 9.23,9.24, 9.25,9.44 9.47

Beckwith, J. (1970) Early Christian and Byzantine Art, Penguin Books, Harmondsworth. 13.19,13.92, 13.107

Beech, H.R. & Fransella, F. (1968) Research and Experiment in Stuttering, Pergamon Press, Oxford.

Beierle, J.W. et al (1971) "Growth promotion by extracts from Wilm's tumour in vitro", Experiemtia, 27, 435-436. 11.7

Beiguelmann, B. (1964) A survey on genetical and anthropological traits among Japanese immigrants in Brazil, Zeitschrift für Morphologie und Anthropologie, 55: 46-59. 9.44,9.47

Bennett, G.C. & Harrold, A.J. (1976) "Prognosis and management of birth injuries to the brachial plexus", British Medical Journal, 1, 1520-1521. 11.19a

Benson, H. (1975) Letter to The Times, March 14th, 1975. 13.65

Benton, A.L. & Joynt, R.J. (1960) "Early descriptions in aphasia", Arch. Neurol., 3, 205-221. 6.3

Berenson, B. (1950) Aesthetics and History, p. 203, Constable, London. 13.1

Berenson, B. (1957) Italian Pictures of the Renaissance: Venetian School, in 2 vols, Phaidon Press, London. 13.72,13.9, 13.79

Berenson, B. (1963) Italian Pictures of the Renaissance: Florentine School, in 2 vols, Phaidon Press, London. 13.9,13.72 13.79

Berenson, B. (1967) The Italian Painters of the Renaissance, Phaidon Press, London. 13.9,13.72, 13.79,13.105

Berenson, B. (1968) Italian Pictures of the Renaissance: Central and North Italian

Schools, in 3 vols, Phaidon Press, London. 13.9,13.72, 13.74,13.75 13.79

Berlin, C.I. & Cullen, J.K. (1977) "Acoustic problems in dichotic listening tasks", in Language Development and Neurological Theory, ed. Segalowitz, S.J. & Gruber, F.A., Academic Press, New York. 8.12

Berman, A. (1971) "The problem of assessing cerebral dominance and its relationship to intelligence", Cortex, 7, 372-386. 2.16

Bernstein, F. (1925) "Beitrage zur mendelistischen Anthropologie ...", Sitzungsberichte Physikal-ische-mathematische Klasse der Preussische Akademie der Wissenschaffen. (Cited by Morgan 1976). 4.11

Bignall, J.R. (1958), editor, Carconoma of the Lung, Livingstone; London. 11.2

Bingley, T. (1958) "Mental symptoms in temporal lobe epilepsy and temporal lobe gliomas", Acta Psych. et Neurol., Supp 120, 33, 1-151. 6.17,6.41, 8.21,8.56, 8.61.

Birkbeck, M.Q. & Beer, T.C. (1975) "Occupation in relation to the carpal tunnel syndrome", Rheumatology & Rehabilitation, 14, 218-221. 11.19

Birkett, P. (1977) "Measures of laterality and theories of hemispheric processes", Neuropsychologia, 15, 693-696. 8.11

Birnbaum, A. (1972) "The random phenotype concept, with applications", Genetics, 72, 739-758. 7.21

Blacklock, (1932) Tubercular disease in children, HMSO, London. 11.2

Blount, P., Holmes, J., Rodger, J., Coltheart, M. & McManus, C. (1975) 'On the ability to discriminate original from mirror-image reproductions of works of art', Perception, 4, 385-389. 13.26,13.38 13.67

Blout, E.R., Doty, P. & Yang, J.T. (1957) "Polypeptides. XII The optical rotation and configurational stability of α-helices", J. Am. Chem. Soc., 79, 749-750. 11.9

Blout, E.R. & Idelson, M. (1956) "Polypeptides. IX. The kinetics of strong-base initiated polymerisations of amino-acid-N-carboxy-anhydrides", J. Am. Chem. Soc., 78, 3857-3858. 11.9

Blumstein, S., Goodglass, H. & Tartler, V. (1975) "The reliability of ear advantage in dichotic listening", Brain and Language, 2, 226-235. 8.4,8.35

Blunt, A. (1973) Artistic Theory in Italy 1450-1600, Oxford University Press, London. 13.16

Bodin, B.J. (1953) "Left handedness and stuttering as signs diagnostic of epileptics", J. Mental Science, 99, 483-488. 5.1

Boklage (1977) – see end of references

Bond, C.J. (1920) "On the left-sided incidence of the super-numerary digit in heterodactylous fowls", J. Genetics, 10, 87-91. 11.17

Bonne, B. (1966) "Genes and Phenotypes in the Samaritan isolate ..." American Journal of Physical Anthropology, 24: 1-20. 9.44,9.47

Bonner, W.A., van Dort, M.A. & Yearian, M.P., (1975) "Asymmetric degradation of DL-leucine

with longitudinally polarised electrons", Nature, 258, 419-421. 11.10.

Bossy, J.G. (1972) "Morphological study of a case of complete, isolated and asymptotic agenesis of the corpus callosum", Arch. d'Anat. d'Hist. d'Embryol, 53, 291-340. 7.32

Botez, M.I. and Westheim, N. (1959) "Expressive aphasia and amusia, following right frontal lesion in a right-handed man". Brain, 82, 186-202. 6.29

Boucher, J. (1977) "Hand preference in autistic children and their parents", J. Aut. Child. Schiz., 7, 177-187. 2.4

Bouterwek, E. (1938) Cited by Zazzo (1960). 4.13,7.63b

Boycott, A.E., Diver, C., Garstang, S.L. & Turner, F.M. (1920), "The inheritance of sinistrality in Limnaea peregra. Philosophical Transactions of the Royal Society of London, B, 219: 51-131. 9.28.

Boyd, W. (1961) Textbook of Pathology, 7th Edition, pp. 656-657, London. 11.2

Brachetto-Brian, D., Grimaldi, F.E., & Costa, A.C.T., (1943) Revta. Asoc. Med., 57, 900; quoted British Medical Journal, (1955) i, 396. 11.8

Bradley, W.G. & Whitty, C.W.M. (1967) "Acute optic neuritis: its clinical features and their relation to prognosis for recovery of vision", J. Neurol. Neurosurg. Psych., 30, 531-538. 11.19a

Bradshaw, J.L. (1978) "Handedness and human cerebral asymmetry: some unanswered questions", The Behavioural and Brain Sciences, 1, 286-287. 5.1

Brain, W.R. (1945) "Speech and handedness", Lancet, ii, 837-841. 6.20,6.21, 6.39.

Brain, W.R. (1961) Speech Disorders: Aphasia, Apraxia, and Agnosia, Butterworths, London. 6.25.

Braitenberg, V. & Kemali, M. (1970) "Exceptions to bilateral symmetry in the epithalamus of lower vertebrates", Journal of Comparative Neurology, 138, 137-146. 11.18

Bramwell, B. (1897) "Lectures on aphasia", Edinburgh Medical Journal, 11, 356-370. 6.19

Bramwell, B. (1898) "A remarkable case of aphasia", Brain, 21, 343-373. 6.18,6.26

Bramwell, B. (1899) "On 'crossed' aphasia", Lancet, i, 1473-1479. 6.10,6.12 6.17,6.24

Brann, A.W. and Myers, R.E. "Central nervous system findings in the newborn monkey following severe in utero partial asphyxia", Neurology, 25, 327-338. 3.5

Braunlich, A.F. (1936) "'To the Right' in Homer and Attic Greek", Am.J.Philology, 57, 245-260. 12.9

Bredius, A. (1969) Rembrandt: the complete edition of his paintings, 3rd edition, revised H. Gerson, Phaidon Press, London. 13.4

Briggs, G.G. & Nebes, R.D. (1975) "Patterns of hand preferences in a student population". Cortex, 11, 230-238. 2.5,2.7, 2.8,2.3

Briggs, G.G. & Nebes, R.D. (1976) "The effects of handedness, family history and sex on the performance of a dichotic listening task", Neuropsychologia, 14, 129-133. 6.38

Briggs, G.G., Nebes, R.D. and Kinsbourne, M. (1976) "Intellectual differences in relation to personal and family handedness", Quart. J. Expt. Psych., 28, 591-601. 2.21

Brittain, H.C. (1978) "Intellectual status in epilepsy", Ph.D. Thesis, University of Cambridge. 5.2

Broca, P. (1861) "Remarques sur le siège de la faculté du langage articulé, suivies d'une observation d'aphémie", Bull, Soc. Anat., 36, 330-357 6.3

Broca, P. (1863) Discussion of a communication by Dr. Parrot, Bull. Soc. Anat., Paris, 8, 372. 6.3

Broca, P. (1865) "Sur le siège de la faculté du langage articulé", Bull. Soc. Anthropol., Paris, 6, 337-393. 6.7

Brock, R.C. (1954) The Anatomy of the Bronchial Tree, Oxford University Press, Oxford. 11.2

Brown-Sequard (n.d.) quoted in Hoadley & Pearson, (1929) 11.2

Brown-Sequard, C.E. (1874) The Toner Lecture, given in Washington, April 22, 1874. "The Dual Character of the Brain". see Riese (1959), p.100-107. 6.6

Bryden, M.P. (1975) "Speech lateralisation in families: A preliminary study using dichotic listening", Brain and Language, 2, 201-211. 8.8

Bryden, M.P. (1977) "Measuring handedness with questionnaires", Neuropsychologia, 15, 617-624. 2.17,2.18

Bucklin, R. (1970) 'The legal and medical aspects of the trial and death of Christ', Medicine, Science and the Law, 10, 14-26. 13.62

Bulmer, M.G. (1970) The Biology of Twinning in Man, Clarendon Press, Oxford. 4.12

Burt, C. (1937) The Backward Child, p. 299, University of London Press, London. 13.13

Burt, C. (1950) The Backward Chuld, 3rd edition, University of London Press: London 6.16

Busk, T. & Clemmeson, J. (1947) "The frequencies of left and right-sided breast cancer", Brit. J. Cancer, 1, 345-351. 12.14

Butler, N.R. and Alberman, E.D. (1969) Perinatal Problems, Livingstone, London & Edinburgh. 3.7,6.2

Butler, N.R. and Bonham, D.G. (1963), Perinatal Mortality, Livingstone, Edinburgh & London. 3.7,3.17 5.2

Calnan, M. and Richardson, K. (1976a) "Speech problems among children in a national survey: associations with hearing, handedness and therapy", Community Health, 8, 101-106. 3.6

Calnan, M. and Richardson, K. (1976b) "Developmental correlates of handedness in a national

sample of 11-year-olds', Annals of Human Biology, 3, 329-342. 3.6,6.46

Campain, R. & Minckler, J. (1976) "A note on the gross configurations of the human auditory cortex", Brain & Language, 3, 318-323. 11.17

Campbell, M.F. (1928) "Varicocoele: a study of five hundred cases", Surg.Gyn.Obs., 47, 558-567 11.5,11.19, 12.14

Carmon, A & Bechtoldt, A.P. (1969) 'Dominance of thr right cerebral hemisphere for stereopsis', Neuropsychologia, 7, 29-39. 13.39

Carter-Saltzmann, L., Scarr-Salatapek, S., Barker, W.B. & Katz, S. (1976) "Left-handedness in twins: incidence and patterns of performance in an adolescent sample", Behaviour Genetics, 6, 189-203. 4.13,7.63b

Cavalli-Sforza, L.L. & Bodmer, W.F. (1971) The Genetics of Human Populations, W.H. Freeman: San Francisco. 7.61,10.2, 10.18

Cave, C.J.P. (1950) 'The Orientation of Churches', Antiquaries Journal, 30, 47-51. 13.65

Chamberlain, H.D. (1928) "The inheritance of left handedness", Journal of Heredity, 19, 557-559. 7.9,7.12, 7.18

Chamberlain, G. and Johnstone, F.D. (1975), "Reliability of the history", Lancet, i, 103. 3.4

Chang, K.S.F., Hsu, F.K., Chan, S.T. & Chan, Y.B. (1960) "Scrotal asymmetry and handedness", J. Anatomy, 94, 543-8. 11.18,12.8, 12.9,E.4

de Château, P. & Andersson, Y. (1976) 'Left side preference for holding and carrying new-born infants II: doll-handling and carrying from 2-16 years", Dev. Med. Child. Neurol., 18, 738-744. 13.13

de Château, P., Holmberg, H. & Winberg, J. (1978) "Left-sided preference in holding and carrying new-born infants", Acta Paediatricia Scandinavica, 67, 169-175. 13.13

Chattopadhyay, P.K. (1968) "Mode du croisement des mains et des bras, dextérie, gaucherie et camptodactylie chez les Bengalis". L'Anthropologie, 72: 317-324. 9.44,9.47

Chesher, E.C. (1936) "Some observations concerning the relation of handedness to the language mechanism", Bull. Neurol. Inst., New York, 4, 556-561. 6.24,6.33

Child, D. (1970) The Essentials of Factor Analysis, Holt, Rinehart and Winston, London. 2.18

Chouké, K.S. (1947) "On the incidence of the foramen of Civini and the Porus Buccinatorius in American whites and Negroes II: Observations on 2745 additional skulls", American Journal of Physical Anthropology, 5, 79-86. 11.17

Ciambellotti, E. (1961) "Localizzazione destra e sinistra delle neoplasie maligne della mammella", Cancro, 14, 375-387. 11.19a

Clark, M.M. (1957) Left-handedness: Laterality characteristics and their educational implications, University of London Press: London. 6.32

Clarke, B. and Zangwill, O.L. "A case of 'crossed aphasia' in a dextral", Neuropsychologia, 3, 81-86. 6.25,6.26.

Clarke, E. & Dewhurst, K. (1972) An Illustrated History of Brain Function, Sandford, Oxford. 13.5

Cobb, S. (1964) "A comparison of the size of an auditory nucleus (n. mesencephalicus lateralis, pars dorsalis) with the size of the optic lobe in twenty seven species of birds", J. Comparative Neurology, 122, 271-279 11.17

Cohn, H.A. & Papez, J.W. (1930) "A comparative study of the visuo-sensory or striate area in the two hemispheres of the human brain", American Journal of Physical Anthrolology, 14, 405-415. 11.18

Colbourn, C.J. (1978) "Can laterality be measured?", Neuropsychologia, 16, 283-289. 2.27,8.12

Colby, K.M. & Parkinson, C. (1977) "Handedness in autistic children", J. Aut. Child. Schiz., 7, 3-7. 6.46

Coles, P.E. (1974) 'Profile orientation and social distance in portrait painting', Perception, 3, 303-308. 13.3,13.33, 13.47,13.50, 13.68,13.73.

Coley, F.C. (1909) "The prophylaxis of aphasia", The Practitioner, 2, 238-240. 6.14,6.15

Collins, J. (1898) The Faculty of Speech: A clinical and Psychological Study of Aphasia, MacMillan: New York. 6.11

Collins, R.L. (1970) "The sound of one paw clasping: An inquiry into the origin of left-handedness". In Contributions to Behaviour-Genetic Analysis - The Mouse as a prototype, ed. Lindzey, G.& Thiessen, D.D., New York: Meredith Corporation. 4.1,4.2, 7.8,7.9

Collins, R.L. (1977) 'Origins of the sense of asymmetry: Mendelian and non-Mendelian models of inheritance", Annals N.Y., Academy of Science, 299, 283-305. 4.12

Collins, R.L., Ward, R. (1970) "Evidence for an asymmetry of cerebral function in mice tested for audiogenie seizures", Nature, 226, 1062-1063. 11.17

Conrad, K. (1949) "Ueber aphasische Sprachstoerungen bei hirnverletzten Linkshaender", Nervenarzt, 20, 148-154. 6.24,8.20, 8.55,8.60

Cooper, A. (1830) Observations of the Structure and Diseases of the Testis, Longmans, London. 12.8

Cooper, A. (1840) On the Anatomy of the Breast, Longmans, London. 12.8

Copeman, S.M. (1919) "Experiments on sex determination", Proc. Zool. Soc., 1919,433-435. 12.12,12.13.

Corballis, M.C. & Beale,I.L. (1976) The Psychology of Left and Right, Hillsdale,

New Jersey: Lawrence Erlbaum. 4.1,4.12, 6.42,7.15, 7.18,13.43

Corballis, M.C. & Morgan, M.J. (1978) "On the biological basis of human laterality: I. Evidence for a maturational left-right gradient, The Behavioural and Brain Sciences, 1, 261-269.

Coren, S. & Porac, C. (1977) "Fifty centuries of right-handedness: the historical record", Science, 198, 631-632. 10.1,13.35

Coren, S. & Porac, C. (1978) "The validity and reliability of self-report items for the measurement of lateral preference", Brit. J. Psychol., 69, 207-211. 2.28

Crew, F.A.E. (1952) in Marchall's Physiology of Repooduction, 3rd Ed'n, Vol II, p. 764. 12.12

Crew, F.A.E. & Munro, S.S. (1937) "Gynandromorphism and lateral asymmetry in birds", Prof. Roy. Soc. Edin., 58, 114-134. 11.8

Crimm, P.D. (1948) "Spontaneous collapse", J. Thoracic Surgeyy, 17, 662-680. 11.19

Critchley, M. (1964) "La controverse de Dax et Broca, Revue Neurologique, 110, 553-7. 6.4,6.9

Critchley, M. (1970) The Dyslexic Child, Heinemann Medical Books, London.

Crovitz, H.F. & Zener, K. (1962) "A group-test for assessing hand and eye-dominance", American J. Psychol., 75, 271-276. 2.8,2.35

de la Cruz, M.V., Espino-Vela, J., Attie, F. & Munoz, C., (1967) "An embryologic theory for ventricular inversions and their classification", American Heart Journal, 73, 777-793. 11.4

de la Cruz, M.V., Anselmi, G., Munos-Castellanos, L., Nadal-Ginard, B., & Munoz-Armas, S. (1971) "Systematisation and embryological and anatomical study of mirror-image dextro-cardias, dextro-versions, and laevo-versions", British Heart Journal, 33, 841-853. 11.4

Curci, J.J. & Horman, M.J. (1976) "Boerhaave's syndrome: the importance of early diagnosis and treatment, Annals of Surgery, 183, 401-408. 11.19a

Curry, F.K.W. (1967) "A comparison of left-handed and right-handed subjects on verbal and non-verbal dichotic listening tasks", Cortex, 3, 343-352. 8.35

Cushing, H. & Eisenhardt, L. (1958), Meningiomas, Thomas: Illinois. 11.19

Dahlberg, G. (1926) Twin births and twins from a hereditary point of view, Tidens: Stockholm, 4.6,4.13, 7.63b,9.44

Damon, A. (1965) "Notes on Anthropometric Technique: II Skinfolds right and left sides; held by one or two hands", American Journal of Physical Anthropology, 23, 305-306. 11.1,11.18

Darley, F.L. (1975) "Treatment of Acquired Aphasia", Advances in Neurology, 7, 111-145. 8.18

David, T.J. & Osborne, C.M. (1976) "Scalp hair patterns in mental sub-normality", J. Med. Genetics, 13, 123-126. 11.18

Davidoff, J. (1976) 'Hemispheric differences in the perception of colour", Quart. J. Exp. Psychol., 28, 387-394. 13.39

Davis, A.E. & Wada, J.A. (1977) "Lateralisation of speech dominance by spectral analysis of evoked potentials", J. Neur. Neurosurg. Psych., 40, 1-4. 8.35

Dawson, R. (1909) The Causation of Sex, London. 12.13

Dax, M. (1865) "Lésions de la moitié gauche de l'encéphale coincidait avec l'oublie des signes de la pensée", Gazette hebdom. de. méd. et de chir., 2nd series, 2, 259-262. 6.4

Dean, A. (1946) Fundamentals of Play Directing, New York. 13.11

Dechaume, M.P. (1957) "Contribution à l'étude de la dominance latérale chez les jumeaux", MD Thesis, Paris. Cited by Zazzo (1960) 4.13,7.63b.

Dee, H.L. (1971) "Auditory asymmetry and strength of manual preference", Cortex, 7, 236-245. 8.35

Denman, J. & Smith, M. (1954) "Intra-cranial Gliomata", Spec. Rep. Ser. Med. Res. Coun., number 284, HMSO. 11.19a

Dennis, M. (1976) "Impaired sensory and motor differentiation with corpus callosum agenesis: a lack of callosal inhibition during ontogeny"", Neuropsychologia, 14, 455-469. 7.32

Dennis, M. & Whitaker, H.A. (1977) "Hemisphere equipotentiality and language acquisition", in Language Development and Neurological Theory, ed., Segalowitz, S.J., & Gruber, F.A., Academic Press, New York. 6.35,8.21, 8.58,11.19, 11.19a

Dennis, W. (1958) 'Early graphic evidence of dexterity in man', Percept. Mot. Skills, 8, 147-149. 10.1,13.32

Dimond, S.J., Farrington, L. and Johnson, P. (1976) 'Differing emotional responses from right and left hemispheres', Nature, 261, 690-692. 13.39

Domhoff, G.W. (1969) "But why did they sit on the king's right in the first place?", Psychoanalytic Review, 56, 586-596. 12.10

Doncaster, L. & Marshall, F.H.A. (1910) "The effects of one-sided ovariotomy on the sex of the offspring", J. Genetics, 1, 70-72. 12.13

Downey, J.E. (1926) "Further observations on the manner of clasping the hands", American Naturalist, 60: 387-391. 9.44

Doyle, A.C. (1902) The Hound of the Baskervilles. 13.2

Duckworth, W.H.L. (1962) Galen on Anatomical Procedures: The later Books, Cambridge Univ.

Press, Cambridge. 12.10

Duke-Elder, S. (1967) System of Ophthalmology Vol, X, Diseases of the Retina, H. Kimpton, London. 11.19

Durost, W.N. (1934) "The development of a battery of objective group tests of manual laterality with the results of their application to 1300 children", Genet. Psychol. Monographs, 16, 225-335. 2.4

Edwards, J.H. (1961) "The recognition and estimation of cyclic trends", Ann. Hum. Genet., Lond., 25, 83-87. 11.15

Elliott, F.A., Hughes, B., & Turner, J.W.A., (1952) Clinical Neurology, Cassell & Co.: London. 6.25

Espir, M.L.E. & Rose, E.C. (1970) The Basic Neurology of Speech, Blackwell, Oxford. 6.25

Ettlinger, G., Blakemore, C.B., Milner, A.D., & Wilson, J. (1972) "Agenesis of the corpus callosum", Brain, 95, 327-346. 7.32.

Ettlinger, G., Jackson, C.V., and Zangwill, O.L., (1955) "Dysphasia following right temporal lobectomy in a right-handed man", J. Neurol. Neurosurg., Psych., 18, 214-217. 6.29,6.36

de la Faille, J-B (1970) The Worls of Vincent van Gogh: his paintings and drawings, Wiedenfeld & Nicholson, London. 13.84

Falek, A. (1959) "Handedness: A family study", Am.J.Hum.Genet., 11, 52-62. 7.10

Falk, C.T. & Ayala, F.J. (1971) Genetic aspects of arm-folding and hand-clasping. Japanese Journal of Human Genetics, 15: 241-247. 9.9,9.21, 9.22,9.25, 9.44,9.47, 9.50,9.63

Farquharson, A.S.L. (1912) De Motu Animalium: De Incessu Animalium, Oxford Univ. Press. Oxford. 12.10

Fergusson, J.D. (1962) "Tumours of the testis", British Journal of Urology, 34, 407-421. 11.19,12.14.

Ferraro, J.A. & Minckler, J. (1777a) "The brachium of the inferior colliculus", Brain and Language, 4, 154-164. 11.17

Ferraro, J.A. and Minckler, J. (1977b) "The human lateral lemniscus and its nuclei", Brain and Language, 4, 277-294. 11.17

Ferrier, D. (1878) The Localisation of Cerebral Diseases, Smith, Elden and co.: London. 6.10.

Ferris, G.J. and Dorsen, M.M. (1975) "Agenesis of the corpus callosum: 1. Neuropsychological studies", Cortex, 11 , 95-122. 7.32

Ferronato, S., Thomas, D. and Sadava, D. (1974), "Preferences for handedness, arm-folding and hand-clasping in families". Human Heredity, 24: 345-351. 9.9,9.21, 9.23,9.24, 9.25,9.44, 9.47, 9.50, 9.63 A.3.

Field, M., Ashton, R. & White, K. (1978) "Agenesis of the corpus callosum: report of two pre-school children and a review of

the literature", Dev. Med. Child Neurol., 20, 47-61. 7.32

Finger, S. (1975) 'Child holding patterns in Western Art', Child Development, 46, 267-271.

Fischer-Dückelmann, A. (1914) La Donna, medico di casa, Turin, 1914. 12.12

Fleminger, J.J., de L. Horne, D.J., & Nott, P.N., (1970) "Unilateral electroconvulsive therapy and cerebral dominance: effect of right and left-sided electrode placement on verbal memory", J. Neurol. Neurosurg. Psychiat., 33, 408-411. 8.36

Fleminger, J.J., Dalton, R. & Standage, K.F. (1977a) "Handedness in psychiatric parients", Brit.J. Psychiat., 131, 448-452. 6.46.

Fleminger, J.J., Dalton, R. & Standage, K.F., (1977b) "Age as a factor in the handedness of adults", Neuropsychologia, 15, 471-473. 2.4

Floris, G. (1975) "Sex and side differences and correlations between quantitative palmar characteristics in a sample Sardinian population", Am.J.Phys.Anthropology, 42, 277-280. 11.18

Forrai, G. and Bankovi, G. (1969) "Relation of hand-clasping and arm-folding to handedness in Hungarian children". Acta Geneticae Medicae et Gemellologiae, 18: 166-174. 9.44,9.47

Fortuyn, A.B.D. (1939) "A polydactylous strain of mice", Genetica, 21, 97-108. 11.17

Fowler, A. (1971) editor Paradise Lost, Longman, London. 13.58,13.59

Fraumeni, J.F., Geiser, C.F. & Manning, M.D., (1967) "Wilm's Tumour and congenitcal hemihypertrophy", Pediatrics, 40, 886-899. 11.7

Freeman, K. (1949) The Pre-Socratic Philasophers, Oxford, Univ. Press, Oxford. 12.10

Freire-Maia, A. & de Almeida, J. (1966) "Hand-clasping and arm-folding among African Negroes". Human Biology, 38: 175-179. 9.19,9.44, 9.47

Freire-Maia, A., Freire-Maia, N. and Quelce Salgado, A. (1960) "Genetic analysis in Russian immigrants". American Journal of Physical Anthropology, 18: 235-240. 9.19,9.44, 9.47

Freire-Maia, N., Quelce-Salgado,A. and Freire-Maia, A. (1958) "Hand clasping in different Ethnic groups". Human Biology, 30: 281-291. 9.44,9.50, 9.63.

Frisancho, A.R., Klayman, J.E., Schessler, T. and Way, A.B. (1977) "Taste sensitivity to phenyl-thiourea (PTC), tongue rolling, and hand-clasping among Peruvian and other Native American populations". Human Biology, 49: 155-163. 9.44

Fritsch, V. (1969) Left and Right in Science and Life, Barrie and Rockliff, London. 1.4,7.0 9.30,11.0

Frohn, M.J.N. (1976) "Left-leg varicose veins and deep-vein thrombosis", Lancet, 2, 1019-1020. 11.19b

Gainotti, G. (1972) 'Emotional behaviour and the hemispheric side of lesion", Cortex, 8, 41-55. 13.39

Garay, A.S., Keszthelyi, L., Demeter, I. & Hrasko, P. "Origin of asymmetry in biomolecules", Nature, 250, 332-333. 11.10

Gardner, E., O'Rahilly, R., Prolo, D. (1975), "The Dandy-Walker and Arnold-Chiari Malformations", Arch. Neurol., 32, 393-407. 7.32

Gardner, M. (1967) The Ambidextrous Universe. London: Allen Lane. 9.28,11.9

Garn, S.M., Mayor, G.H. & Shaw, H.A. (1976), "Paradoxical bilateral asymmetry in bone size and bone mass in the hand", American Journal of Physical Anthropology, 45, 209-210. 11.17

Gause, G.F. & Smaragdova, N.P. (1940) "The decrease in weight and mortality in dextral and sinistral individuals of the snail, Fructicola lantzi", Am. Nat., 74, 568-572. 11.13

Geffen, G., Traub, E. & Stierman, I. (1978) "Language laterality assessed by unilateral ECT and dichotic monitoring", J. Neurol. Neurosurg. Psychiat., 41, 354-360 2.26,8.36.

Geschwind, N. (1975) "Language, Aphasia and Related Disorders" in Textbook of Medicine, ed. Beeson, P.B. & McDermott, W., 14th Edition. 6.41

Geschwind, N. & Levitsky, W. (1968) "Human Brain: Left-right asymmetries in temporal speech

region", Science, 161, 186-187. 10.1,11.17

Gessel,A. & Ames, L.B., (1957) 'The Development of Handedness', J.Genet. Psychol., 70, 155-175. 13.42

Gesell,A. & Ames, L.B. (1950) 'Tonic neck reflex and symmetro-tonic behaviour', J. Pediatrics, 36, 165-176. 13.42

Giannitrapani, D. (1967) "Developing concepts of lateralisation of cerebral functions", Cortex, 3, 353-370. 6.3

Gibiński, K., Giec, L., Zmudziński, J., Waclawczyk, J., & Dosiak, J. (1971) "Body side related asymmetry in sweat gland function", Journal of Investigative Dermatology, 57, 190-192. 11.6,11.18

Gibson, G.A. (1902) "The nervous affections of the heart", Edinburgh Medical Journal, July 1902, 9-24. 6.6

Gibson, J.J. (1950) The Perception of the Visual World, pp. 95-99; Riverside Press, Cambridge, Mass. 13.49

Gilbert, C. & Bakan, P. (1973) 'Visual asymmetry in perception of faces", Neuropsychologia, 11, 355-362. 13.37

Girard, P.F. (1952) "À propos de troubles neurologiques et psychiatriques observés chez les gaucheurs. La notion de diphasie, dipraxie et diphrènie", Semaine hôp., Paris, 28, 750-759. 6.27,6.28

Gloning, I., Gloning, K., Haub, G. & Quatember, R., (1969) "Comparison of verbal behaviour in right-handed and non right-handed patients with anatomically verified lesion of one hemisphere", Cortex, 5, 43-52. 6.32,8.2

Goldscheider, L. (1950) 500 Self-portraits, Phaidon Press, London. 13.3

Goldstein, H. (1969) "The effect of maternal age, parity, social class, height, degree of pre-eclampsia and smoking inpregnancy, on still-birth plus neonatal mortabity rates", pp. 45-46 in Butler and Alberman (1969) 3.14

Gombrich, E.H. (1960) Art and Illusion, Phaidon Press, London. 13.28,13.47, 13.49

Goodglass, H. & Geschwind, N. (1976) "Language disorders (aphasia)" in Handbook of Perception Vol. VII, Language and Speech, ed. Carterette, E.C. & Friedman, M.P., Academic Press: New York. 8.2

Goodglass, H. and Quadfasel, F.A. (1954) "Language laterality in left-handed aphasics", Brain, 77, 521-548. 2.11,6.30, 6.31,6.48, 6.52

Gordon, H. (1921) "Left-handedness and mirror-writing, especially among defective children," Brain, 43, 313-368. 6.17

Gordon, I.E. (1964) 'Left and right in Goya's portraits", Nature, 249, 197-198. 13.3

Gould, G.M. (1905) Biographic Clinics, vol. VIII: Influences of Visual Function upon Health, [illegible] 6.15

Gowers, W.R. (1978) "The brain in congenital absence of one hand", Brain, 1, 388-390. 6.14

Granet, M. (1934) La Pensee Chinoise, Paris. 12.9

Green, M. (1969) 'Enshrouded in Silence", Amplefforth Journal, 74, 321-345. 13.62

Grillo-Lopez, A.J. (1971) "Primary right varicocoele", J. Urology, 105, 540-541. 11.5

Gruneberg, H. (1950a) "Genetical studies on the skeleton of the mouse. I: Minor variations of the vertebral column", Genetics, 50, 112-141. 11.17

Gruneberg, H. (1950b) "Genetical studies on the skeleton of the mouse. II: Undulated and its 'modifiers'", Genetics, 50, 142-173. 11.17

Gur, R.E. (1977) "Motoric laterality imbalance in schizophrenia: a possible concomitant of left-hemisphere dysfunction", Arch. Gen. Psychiat., 34, 33-37. 6.46,8.19

Gur, R. & Gur R. (1977) 'Correlates of conjugate lateral eye-movements in man', in Lateralisation in the Nervous System ed. Harnad, S. et. al, New York, 1977. 13.42

Hamilton, P.V., Nishimoto, R.T. and Halusky, J.G., (1976) "Cheliped laterality in Callinectes sapidus (Crustacea: Portunidae)". Biological Bulletin, 150, 393-401 9.27

Hammer, M. & Turkewitz, G. (1974) "A sensory basis for the lateral difference in the newborn infant's response to somesthetic stimulation", J. Exptal. Child Psych., 18, 304-312. 11.18

Hardyck, C., Petrinovich, L.F. & Goldman, R.D. (1976), "Left-handedness and cognitive deficit", Cortex, 12, 266-279. 10.4

Harnad, S., Doty, R.W., Goldstein, L., Jaynes, J. & Krauthamer, G. (1977) editors, Lateralisation in the Nervous System, Academic Press: New York.

Harrower, G. (1928) "A note on right-handedness", Man, 28, 137-139. 10.1

Hass, J. (1951) Congenital dislocation of the hip, Thomas, Illinois, 11.1,11.19a

Hécaen,H. and de Ajuriaguerra, J. (1964) Left-handedness: Manual superiority and cerebral dominance, Grune and Stratton, New York. 5.1,6.27, 8.29,8.55, 8.60.

Hécaen, H. & Albert, M.L. (1978) Human Neuropsychology, John Wiley & Sons, New York. 8.2

Hécaen, H. & Dubois, J. (1968) "La Naissance de la Neuropsychologie du langage (1825-1865)", Flammarion, Paris, 6.4

Hécaen, H. and Piercy, M. (1956) " Paroxysmal dysphasia and the problem of cerebral dominance", J. Neurol. Neurosurg.Psychiat., 19, 194-201. 6.7,8.21, 8.59

Hécaen, H. and Sauguet, J. (1971) "Cerebral dominance in left-handed subjects", Cortex,

7, 19-48. 2.21,6.36, 6.37

Henry, C.B. & Morant, G.M. (1926) "A preliminary study of the eruption of the mandibular third molar tooth ...", Biometrika, 28, 378-427. 11.19

Hewson, M.A. (1975) Giles of Rome and the mediaeval theory of conception, Athlone Press, London. 12.11,12.15

Hicks, R.E. & Barton, A.K. (1975) "A note on left-handedness and severity of mental retardation", J. Genetic Psychology, 127, 323-324. 5.1

Hicks, R.A., Evans, E.A. & Pellegrini, R. J. (1978) "Correlation betwden handedness and birth order: compilation of five studies", Perceptual and Motor Skills, 46, 53-54. 3.3,4.10

Hicks, R.E. & Kinsbourne, M. (1976) "Human handedness: a partial cross-fostering study", Science, 192, 908-910. 10.5

Hicks, R.E. & Kinsbourne, M. (1978) in Kinsbourne (1978), 5.1,8.2, 8.12

Hicks, R.E. & Pellegrini, R.J. (1978) "Handedness and Locus of Control". Percept. Motor Skills, 46, 369-370. 2.27

Hicks, R.A., Pellegrini, R.J. & Evans, E.A. (1978) "Handedness and birth risk", Neuropsychologia, 16, 243-245. 3.3,3.5, 6.42

Higenbottam, J.A. (1973) "Relationship between sets of lateral and perceptual preference measures", Cortex, 9, 403-410. 6.38

Hildreth, G. (1949) "The Development and Training of Handedness: I. Characteristics of Handedness", J. Genet. Psychol., 75, 197-220. 2.4.

Hilliard, R.D. (1973) 'Hemispheric laterality effects on a facial recognitiion task in normal subjects", Cortex, 9, 246-258. 13.37

Hirsch, N.D.M. (1930) Twins, Heredity and Environment, Cambridge, Mass: Harvard Univ. Press. 4.13,7.63b

Hoadley, M.F. & Pearson, K. (1929) "On measurement of the internal diameters of the skull in relation i) to the prediction of its capacity ii) to the 'pre-eminence' of the left hemisphere", Biometrika, 21, 85-123. 11.17,11.19b

van den Hoeff, A. (1973) New Scientist, 59, 774 (letter) 13.5

Holt, S.B. (1953) "Genetics of dermal ridges: bilateral asymmetry in finger ridge counts", Ann. Eugen. 18, 211-231. 11.18

Hordijk, W. (1952) "Epilepsie en linkshandigheid", Nederl. tijdschr. genesch., 96, 263-269. 5.1

Hubbard, J.I. (1971) "Handedness is not a function of birth order", Nature, Lond., 232, 276-277. 3.2,4.10, 7.9,7.28

Hubbs, C.L. & Hubbs, L.C. (1944) "Bilateral asymmetry and bilateral variation in fishes", Papers of the Michigan Academy of Sciences, 30: 229-310. 9.38

Hubbs, C.L. & Kuronuma, K. (1941) "Hybridisation in nature between two generations of flounders in Japan". Papers of the Michigan Academy of Science, 27: 267-306. 9.37

Hudson, P.T.W. (1975) "The Genetics of Handedness: A reply to Levy & Nagylaki", Neuropsychologia, 13, 331-339. 7.18

Huizinga, J. (1968) "Human biological observations on some African populations of the Thorn Savanna Belt. II". Proceedings Koninklijke Nederlandse Akademie van Wetenschappen, C, 71, 371-390. 9.19,9.44, 9.47.

Hull, C.J. (1936) "Study of laterality test items", J. Exp. Educ., 4, 287: 2.4

Humphrey, M. (1971) "Consistency of hand usage: a preliminary enquiry", Brit. J. Educ. Psychol., 21, 214-225. 2.4

Humphrey, M.E. & Zangwill, O.L. (1952) "Effects of a right-sided occipito-parietal brain injury on a left-handed man", Brain, 75, 312-324. 6.31.

Humphrey, N.K. & McManus, I.C. (1973) 'Status and the left cheek', New Scientist, 59, 437-439. 13.4,13.8, 13.76,13.81, 14.1

Huxley, J. (1942) Evolution: the modern synthesis. London: George Allen & Unwin Ltd. 9.33

Inglessis, (1925) quoted in Hoadley & Pearson (1929) 11.17

Ingram, D. (1975) "Motor asymmetries in young children", Neuropsychologia, 13, 95-102. 2.30

Ireland, W.W. (1880) "Note on left-handedness", Brain, 3, 207-214. 6.6,6.22

Jackson, J.H. (1868a) "Aphasia with hemiplegia of the left side", Lancet, i, 316. 6.7.

Jackson, J.H. (1868b) "Defect of intellectual expression (aphasia) with left hemiplegia", Lancet, i, 457. 6.8

Jackson, J.H. (1880a) "On affections of speech from diseases of the brain", Brain, 2, 323-356. 6.10

Jackson, J.H. (1880b) "On aphasia, with left hemiplegia", Lancet, ii, 637-638. 6.13.

Jackson, J.P., Jumper, E.J., Mottern, B. & Stevenson, K.E. (1977) "The three-dimensional image on Jesus's burial cloth", pp. 74-94 in Proceedings of the 1977 United States Conference of Research on the Shroud of Turin, ed. Stevenson, K.; Holy Shroud Guild, New York. 13.112.

James, W. (1892) Textbook of Psychology, p. 109, MacMillan; London. 6.11

Japper, H.H. (1932) "A laboratory study of diagnostic indices of bilateral neuro-muscular organisation in stutterers and normal speakers", Psychological Monographs, 43, 72-174. 2.4

Jensen, B.T. (1952a)'Left-right orientation in profile drawing', Amer.J. Psychol. 65, 80-81. 13.32,13.36

Jensen, B.T. (1952b) 'Reading habits and left-right orientation in profile drawings by Japanese Children', Amer. J. Psychol., 65, 306-307. 13.32,13.36

Jocelyn, H.D. & Setchell, B.P. (1972) "Regnier de Graaf on the Human Reproductive organs", J. Rep. Fert., Supplement number 17. 12.12

Jones, E. (1955) Sigmund Freud: Life & Work. Vol. II: Years of Maturity, 1901-1918, The Hogarth Press, London, p. 496 1.0

Jones, L.V. & Fiske, D.W. "Models for testing the significance of combined results", Psychological Bulletin, 50, 375-382. 4.6

Jordan, J.E. (1914) "Hereditary left-handedness, with a note on twinning", J. Genetics, 4, 67-82. 7.16

Jost, A., Vigier, B. & Prepin, J. (1972) "Free-martins in cattle: the first steps of organo-genesis", J. Rep. Fert., 29, 349-379. 12.9

Joyce, J. (1922; 1969) Ulysses, Penguin Books, Harmondsworth. 12.0,12.10.

'J.S.' (1870) 'Right-handedness', Nature, 1, 605. 13.36.

Kabakian, H.A., Armenian, H.K., Deeb, Z.L. & Risk, G.K. (1976) 'Asymmetry of the pelvic ureters in normal females", Am. J. Roent., 127, 723-727. 11.18

Kawabe, M. (1949) "A study on the mode of clasping the hands". Transactions of the Sapporo

Natural History Society, 18, 49-52. 9.44,9.50, 9.63

Kawabe, M. (1953) quoted in Fujiki, K. (1960) "On the genetical mechanism of hand-clasping" Japanese Journal of Genetics, 35: 238-246. 9.46,9. 9 9.63.

Kawahara, T. (1974) "Bilateral asymmetry in the transverse processes of the cervical vertebrae in the chicken", Jap. J. Genetics, 49, 1-9. 11.17

Kelsey, J.L. (1975) "An epidemiological study of acute herniated lumbar intervertebral discs", Rheum. & Rehab., 14, 144-159. 11.19b

Kendall, M.G. (1951) The Advanced Theory of Statistics, Volume II, 3rd edition, p. 132; London: C. Griffin & Co. 4.6,3.11, 10.23,14.10

Keller, R. (1942) 'The right-left problem in art', CIBA Symposia, 3, 1139-1142. 13.11

Kember, O. (1971) "Right and left in the sexual theories of Parmenides", J. Hellenic Studies, 91, 70. 12.10

Kennedy, F. (1916) "Stock-Brainedness: the causative factor in the so-called 'crossed aphasias', Am. J. Med. Sci., 152, 849-859. 2.3,6.18

Kimura, D. (1973) 'Manual activity during speaking: I. Right-handers', Neuropsychologia, 11, 45-50. 13.42

Kimura, D. & Durnford, M. (1974) 'Normal studies on the function of the right hemisphere in vision', in Hemisphere Function in the Human

Brain, ed. Dimond, S.J. & Beaumont, g., Elek Science, London. 13.39

Kimura, D.& Vanderwolf, C.H. (1970)"The relation between hand preference and the performance of individual finger movements by left and right hands", Brain, 93, 769-774. 2.30.

King, H.D. (1931) "Studies on the inheritance of structural anomalies in the rate", Am. J. Anatomy, 48, 231-260. 11.18

King, H.E. (1909) "Studies on sex determination in Amphibians II", Biol. Bull., 16, 27-43. 12.13

King, H.E. (1911) "The effects of semi-spaying and of semi-castration on the sex-ratio of the albino rat (Mus norvegicus albinus)", J. Exp. Zool, 10, 381-392. 12.12,12.13

Kinsbourne, M. (1972) 'Eye and Head-turning indicates cerebral lateralisation", Science 176, 539-541. 13.42

Kinsbourne, M. (1978) editor Asymmetrical Function of the Brain, Cambridge University Press: Cambridge.

Klawans, H.L. (1972) "Relation between handedness and side of onset of Parkinsonism", Lancet, i, 850. 11.2

Konstom, N. (1977) 'Rembrandt's use of models and mirrors', Burlington Magazine, 99, 94-98. 13.4

Kovacs, K.L. & Garay, A.S. (1975) "Primordial origins of chirality", Nature, 254, 538. 11.10.

Kreindler, A., Fradis, A. & Sevastopol, N. (1966) "La répartition des dominances hémisphériques", Neuropsychologia, 4, 143-149. 8.20.

LaBar, M. (1973) 'Turning the left cheek re-examined using modern photography', Nature, 245, 338. 13.3,13.5, 13.82

Lagarde, A. (1915) The Latin Church in the Middle Ages, pp. 441-443, International Theology Library,T&T Clark, Edinburgh. 13.17

Lai, L.Y.C. and Walsh, R.J. (1965) "The patterns of hand clasping in different ethnic groups". Human Biology, 37: 312-319. 9.45,9.50, 9.63.

Lake, D.A. & Bryden, M.P. (1976) "Handedness and sex differences in hemispheric asymmetry". Brain and Language, 3, 266-282. 6.38,6.39 8.35

Lancet, (1902) Lancet, i, 1216. 13.61

Lasko, P. (1972) Ars Sacra 800-1200, Penguin Books, Harmondsworth. 13.19,13.92

Lassek, A.M. (1940) "The Human Pyramical Tract: II A numerical investigation of the Betz cells of the motor area", Archives of Neurology and Psychiatry, 44, 718-724. 11.17.

Lasteyne, R. de (1905) 'La déviation de l'axe est-elle symbolique?', Bulletin Monumental, 69, 422-459. 13.65

Latimer, H.B. & Lowrance, E.W. (1965) "Bilateral asymmetry in weight and in length of human

bones", Anatomical Record, 152, 217-224. 11.17

Lauterbach, C.E. (1925) "Studies in twin resemblance", Genetics, 10, 525-568. 4.6,4.13, 7.63b

Lauterbach, C.E. & Knight, J.B. (1927) 'Variation of whorl of the head hair', J. Hered., 18, 107-115. 13.37

Lawson, N.C. (1978) "Inverted writing in right- and left-handers in relation to lateralisation of face recognition", Cortex, 14, 207-211. 2.26,8.5, 8.38

Layton, W.M. (1976) "Random determination of a developmental process: reversal of normal visceral asymmetry in the mouse", Journal of Heredity, 67, 336-338. 7.8

Leguege, A. (1967) "Hand-clasping: Études anthropologique et génétique", Bulletin de la Société Royale Belge d'Anthropologie et de Préhistoire, 78, 81-107. 9.18,9.33, 9.45,9.50, 9.63

Leguebe, A. & Martinez-Fuentes, A. (1971) "Étude génétique du mode de croisement des bras". Acta Geneticae et Medicae Gemellologiae, 20, 267-283. 9.21,9.33, 9.47

Levine, K.N. (1935) "A note on the relation of the dominant thumb in clasping to handedness", Am. J. Psych., 47, 704-705. 6.22

Levita, E. (1978) "Effects of speech therapy on aphasics' responses to functional communication profile", Perceptual & Motor Skills, 47, 151-154. 8.18

Leviton, A. and Kilty, T. (1976) "Birth order and left-handedness", Arch. Neurol., 33, 664. 3.2

Leviton, A. & Kilty, T. (1979) 'Seasonal variation in the birth of left-handed schoolgirls', Arch. Neurol., 36, 115-116. 11.15

Levy, J. (1976) "Cerebral lateralisation and spatial ability", Behaviour Genetics, 6, 171-188. 8.31,8.50.

Levy, J. (1977) "A reply to Hudson regarding the Levy-Nagylaki model for the genetics of handedness", Neuropsychologia, 15, 187-190. 7.9,7.13

Levy, J. & Nagylaki, T. (1972) "A model for the genetics of handedness", Genetics, 72: 117-128. 7.17,7.49.

Levy, J. & Reid, M. (1976) "Variations in writing posture and cerebral organisation", Science, 194, 337-339. 2.25,8.5

Levy, J. & Reid, M. (1978) "Variations in cerebral organisation as a function of handedness, hand posture in writing and sex", J. of Exp. Psychol., Gen., 107, 119-144. 2.25,8.5

Lewis, D. & Burke, C.J. (1949)"The use and misuse of the Chi-square test", Psychological Bulletin, 46, 433-489. 4.2

Lipman, J. (1969) American Primitive Painting, Dover Books, New York. 13.2

Lishman, W.A. and McMeekan, E.R.L. (1976) "Hand preference patterns in psychiatric patients", Brit. J. Psychiat., 129, 158-166. 2.26,6.46, 8.35

Lishman, W.A. & McMeekan, E.R.L. (1977) "Handedness in relation to direction and degree of cerebral dominance for language," Cortex, 13, 30-43. 8.19

Little, C.C. & McDonald, H. (1945) "Abnormalities of the mammae in the house mouse", J. Heredity, 36, 285-288. 11.18

Littre, E. (1840) Oeuvres complètes d'Hippocrate, J-B Bailliere, Paris. 12.9,12.11, 12.13,12.14

Lloyd, G.E.R. (1966) Polarity and Analogy, Cambridge University Press, London, 12.9

Lloyd, G.E.R. (1972) "Parmenides' Sexual Theories: A reply to Mr. Kember", J. Hellenic Studies, 92, 178. 12.10

Lockard, J.S., Daley, P.C. & Gunderson, V.M., (1979), "Maternal and paternal differences in infant carry: U.S. and African data", American Naturalist, 113, 235-246. 13.13

Loehlin, J. & Nichols, R.C. (1976) Heredity, Environment and Personality: A study of 850 sets of twins, Austin: University of Texas Press. 4.9,4.13 7.28,7.63b

Lord, C & Haith, M.M. (1974) 'The perception of eye contact', Perception & Psychophysics, 16, 413-416 13.41

Lourie, J.A. (1972) "Hand-clasping and arm-folding among Middle-Eastern Jews In Israel". Human Biology, 44: 329-334. 9.11,9.45, 9.47

Lovell, H.W., Waggoner, R.W. & Kahn, E.A. (1932),

"Critical study of a case of aphasia", Arch. Neurol. Psychiatry, 28, 1178-1181. 6.33

Lowe, C.R. & McKeown, T. (1953) "An investigation of dextrocardia with and without transposition of abdominal viscera, with a report of a case in one monozygotic twin", Annals of Eugenics, 18, 267-277. 7.8

Lowrance, E.W. & Latimer, H.B. (1957) "Weights and linear measurements of 105 human skeletons from Asia". Am. J. Anatomy, 101,445-459. 11.17

Luria, A.R. (1947; 1960) Traumatic Aphasia, translated from the Russian: Mouton: The Hague, 6.21,6.22, 6.38.

Lutz, F.E. (1908) "The inheritance of the manner of clasping the hands", American Naturalist, 42, 195-196. 9.45,9.50 9.63

Lynn, J.G. & Lynn, D.R. (1943) 'Smile and hand dominance in relation to basic modes of adaptation', J. Abnorm. Soc. Psychol., 38, 250-276. 13.42

Lynn, W.G. (1946) "Situs inversus viscerum in conjoined twins of the brook trout", J. Morphology, 79, 1-29. 7.7

MacCluer, J.W. (1978) "On the probability of demonstrating differential fertility in genetic studies", Annl Hum. Genet., 42, 59-75. 10.5

McCurdy, H.G. (1949) 'Experimental notes on the asymmetry of the human face', J. Abnorm. Psychol., 44, 553-555. 13.37

McGlone, J. & Davidson, W. (1973) "The relationship between cerebral speech laterality and spatial ability with special reference to sex and hand preference", Neuropsychologia, 11, 105-113. 8.31,8.35, 8.50

McGuinness, D. (1976) "Away from a unisex psychology: individual differences in visual sensory and perceptual processes", Perception, 5, 279-294. 12.9

McKeever, W.F. & Van Deventer, A.D. (1977), "Visual and auditory language processing asymmetries: influence of handedness, familial sinistrality and sex", Cortex, 13, 225-241. 8.8.

McManus, I.C. (1976) 'Right-Left and the scrotum in Greek sculpture', Nature, 259, 426. 12.9

McManus, I.C. (1977) "Predominance of left-sided breast tumours", Lancet, ii, 297-298. 11.7,11.19a

McManus, I.C. (1979) "The aesthetics of simple figures, British Journal of Psychology, in press. E.4

McManus, I.C. & Humphrey, N.K. (1973) 'Turning the Left Cheek', Nature, 243, 271-272. 13.2,13.3, 13.76,14.7

Malhotra, K.C. (1968) "Hand-clasping among the eight endogenous groups of Maharashtrian Brahmins". Acta Genetica, Basle, 18: 283-288.

Marshall, J.C., Caplan, D. & Holmes, J.M. (1975),

"The Measure of Laterality", Neuropsychologia, 13, 315-321. 8.11

Martin, L.J. (1906) 'An experimental study of Fechner's principles of aesthetics', Psychol. Rev., 13, 142-219. 13.36

Martin, N.G. (1975) 'No evidence for a genetic basis of tongue rolling or hand-clasping'. Journal of Heredity, 66: 179-180. 4.1,9.17

Mascie-Taylor, C.G.N. (1979a) "Handedness and IQ components". submitted to Annals of Human Biology. 7.10,9.9

Mascie-Taylor, C.G.N. (1979b) "Hand clasping, arm folding and IQ components" submitted to Human Biology. 7.10,9.9

Mascie-Taylor, C.G.N. and Gibson, J.B. (1978) "Social Mobility and IQ Components". Journal of Biosocial Sciences, 5: 17-30. 7.10,9.9

Mascie-Taylor, C.G.N. & Gibson, J.B. (1979) "Assortative Marriage and IQ Components ", Annals of Human Biology, 6: 1-16. 7.10,9.9

Mason-Hohl, E. (1940) The Diseases of Women, Los Angeles. 12.14

May, M.T. (1968) Galen; on the usefulness of the parts of the body, 2 vols, Cornell Univ. Press. 12.10

Mayo, O., Bishop, G.R. & Hancock, T.W. (1978), "The detection of genetical influences on human family size", Human Heredity, 28, 270-279. 10.5

Medawar, P.B. (1963) 'Is the scientific paper a

fraud?', The Listener, 12 Sept. 1963. 13.1,E.2

Melnick, M. & Shields, E.D. (1976) "Allelic restriction: a biological alternative to multifactorial threshold inheritance", Lancet, i, 178-179. 7.33

Milner, B. (1975) "Hemispheric specialisation: scope and limits", in The Neurosciences, Third Study Programme, ed. Schmitt, F.O. & Worden, F.G., MIT Press, Cambridge, Massachusetts. 8.5,8.37

Milner, B., Branch, C. and Rasmussen, T. (1964), "Observations on cerebral dominance", in Disorders of Language, ed. de Rueck, A.V.S. and O'Connor, M., Ciba Foundation Symposium, Churchill, London. 5.2,6.25, 6.33,6.44 8.5,8.37

Mittwoch, U. & Kirk, D. (1975) "Superior growth of the right gonad in human foetuses", Nature, 257, 791-792. 11.18,12.9 E.4

Molanus, Joannes (1570) De pictoribus et imaginibus Sacri, Louvain. 13.52,13.61.

Morant, G., Collett, M. & Adyanthāya, N.K. (1936) "A biometric study of the human mandible", Biometrika, 28, 84-122. 11.17.

Morgan, M.J. (1976) "Embryology and inheritance of asymmetry". In Lateralisation in the Nervous system, ed. Harnad, S., Doty, R.W., Goldstein, L., Jaynes, J. and Krauthamer, G. New York; Academic Press. 4.11,7.2, 7.6,7.30, 9.1,9.4, 9.28,9.39, 11.11

Morgan, M.J. & Corballis, M.C. (1978) "On the biological basis of human laterality: II The mechanisms of inheritance", The Behavioural and Brain Sciences, 1, 270-277. 9.39.

Moutier, F. (1908) L'Aphasie de Broca, G. Stein-Heil, Paris. 6.28

Moxon, W. (1866) "On the connexion between loss of speech and paralysis of the right side", British and Foreign Medico-Chirurgical Record, 37, 481-489. 6.13

Munro, T. (1956) Towards Science in Aesthetics: selected essays. pp. 14-15, Liberal Arts Press, New York. 13.1

Munsinger, H. (1977) "The identical-twin transfusion syndrome : a source of error in estimating IQ resemblance and heritability", Ann. Hum. Genet., 40, 307-321. E.6

Nagylaki, T. & Levy, J. (1972) "A model for the genetics of handedness", Genetics, 72, 117-128. 7.13

Nagylaki, T. & Levy, J. (1973) "The sound of one paw clapping isn't sound", Behaviour Genetics, 3, 298-303. 4.1,4.2, 4.7,4.9, 4.11,.4.12, 7.15

Narang, H.K. (1977) "Right-left asymmetry of myelin development in epi-retinal portion of rabbit optic nerve", Nature, 266, 855-856. 11.18

National Child Development Study (1966) 11,000 Seven-year-olds. Longmans, London. 3.7,5.2

National Child Development Study (1972), From Birth to Seven, Longmans, London. 3.7,5.2

Naumann, B. (1955) "Afasi vid tumor cerebri och dess postoperativa prognos", Nordisk Medicin, 53, 681-684. 8.21,8.57

Needham, R. (1975) Right and Left: Essays in dual symbolic classification, Univ. Chicago Press, Chigago. 11,2,12.9

Needles, W. (1942) "Concerning transfer of cerebral dominance in the function of speech", J. Nerv. Ment. Dis., 95, 270-277. 6.16.

Negus, D. (1970) "Ilio-Femoral thrombosis", Ann. Roy. Coll. Surg., 47, 92-105. 11.2

Nelson, T.M. & MacDonald, G.A.(1971) 'Lateral Organisation, perceived depth, and title preference in pictures', Percept. Mot. Skills, 33, 983-986. 13.40.

Neville, A.C. (1976) Animal Asymmetry. London: Edward Arnold. 9.35

Newcombe, F. & Ratcliff, G. (1973) "Handedness, speech lateralisation and ability", Neuro-psychologia, 11, 399-407. 8.20,8.21, 8.55,8.56, 8.60,8.61

Newman, H.H. (1916) "Heredity and Organic symmetry in Armadillo quadruplets: II. Mode of inheritance of double scutes and a discussion of organic symmetry", Biol. Bull., 30, 173-209. 4.11

Newman, H.H. (1917) The Biology of Twins, University of Chicago Press, Chicago.

Newman, H.H. (1925) "An experimental anslysis of asymmetry in the Starfish, Patiria miniata", Biol. Bull , 49, 111-138. 7.7

Newman, H.H. "Asymmetry reversal or mirror imaging in identical twins", Biol. Bull., 55, 298-315. 4.6,4.13, 7.63b

Newman, H.H. (1940) "Palmar dermatoglyphics of twins", Am. J. Phys. Anthropol., 14, 331-378. 4.12

Newman, H.H. (1940) Multiple Human Births, New York: Doubleday, Doran and Co. 4.6

Newman, H.H. (1940) "The question of mirror-imaging in human one-egg twins", Human Biology, 12, 21-34. 7.7

Newman, H.H., Freeman, F.N. & Holzinger, K.H. (1937), Twins, a study of heredity and environment, Chicago. University of Chicago Press. 4.9,4.13 7.63b.

Nie, N.H., Hull, C., Jenkins, J.G., Steinbrenner, K. & Bent, D.H., (1975) Statistical package for the Social Sciences, Second Edition. McGraw-Hill, New York. 2.6,2.17, 3.8,10.9, 14.4

Nielsen, J.M. (1936) Agnosia, Apraxia, Aphasia: their value in cerebral locatisation, P.B. Hoeber: New York.

Nielsen, J.M. (1946) A Textbook of Clinical Neurology, P.B. Hoeber, New York. 6.32.

Norberg, A. (1968) "Physical factors in directional

hearing in Aegolius funereus (Linné) (Strigiformes) with special reference to the significance of the asymmetry of the external ears", Arkiv for Zoologi (Stockholm), 20, 181-204. 11.18

Norden, B. (1977) "Was photo-resolution of amino-acids the origin of optical activity in life?" Nature, 266, 567-568. 11.10.

Norman, J.R. (1934) A systematic monograph of the flatfishes (heterosomata) Vol. I. London. British Museum 9.36

Notkovich, H. (1955) "Testicular artery arching over renal vein", British Journal of Urology, 27, 267-271. 11.18

Novitski, E. & Kimball, A.W. (1958) "Birth order, parental ages and sex of offspring", Amer. J. Hum. Genet., 10, 268-275. 12.15

Nydick, M., Bustos, J., Dale, J.H. and Rawson, R.W. (1961) "Gynecomastia in adolescent boys", Journal of the American Medical Association, 178, 449-454. 11.19

O'Connor, E. (1967) 'Immaculate Conception" in A Catholic Dictionary of Theology, Thomas Nelson, London. 13.17

Ogle, W. (1867) "Aphasia and agraphia", St. Georges Hosp. Reports, 2, 83-122. 6.5,6.6

Ogle, W. (1871) "On dextral pre-eminance", Medico-

Chirurgical Transactions, 36, 279-301. 6.8,6.9.

Ogle, W. (1912) De Partibus Animalium , Oxford University Oress, Oxford. 12.10.

Oke, A., Keller, R., Meffard, I. & Adams, R.N. (1978) "Lateralisation of norepinephrine in human thalamus", Science, 200, 1411-1413. 11.18

Oldfield, R.C. (1970) "Hand preference in male sexual behaviour", SCU 2/70, MRC Speech & Communication Research Unit, Edinburgh. 10.8

Oldfield, R.C. (1971) "The assessment and analysis of handedness: the Edinburgh inventory", Neuropsychologia, 9, 97-113. 2.7,2.35.

Ombredane, A. (1951) L'Aphasie et l'élaboration de la Pensée explicite, Presses Universitaires de France, Paris. 6.4

Osgood, C.E., Suci, G.J., Tannerbaum, P.H. (1957) The Measurement of Meaning, Urbana: Univ. of Illinois Press. 14.2,14.5, 14.12.

Packard, G.B. & McLauthlin, C.H. (1953) "Treatment of inguinal hernia in infancy and childhood", Surgery,Gynaecology and Obstetrics, 97, 603-607. 11.19

Paget, G.E. (1887) "Notes on an exceptional case of aphasia", British Medical Journal, 2, 1258-1259. 6.10,6.11 6.13

Parker, G.H. (1903) "The optic chiasma in telosts and its bearing on the asymmetry of the hetero-

somata (flatfishes)". Bulletin of the Mueeum of Comparative Zoology, 40, 221-242. 9.35.

Pateria, H.N. (1973) "Ridge counts of plantar dermatoglyphic interdigital areas and the relation of their trends to the anatomical axis", Am. J. Phys. Anthropology, 39, 263-266. 11.18.

Pearsall, R. (1971) The Worm in the Bud, Penguin Books, Harmondsworth. 12.11

Pelecanos, M. (1969) "Some Greek data on handedness, hand-clasping and arm-folding". Human Biology, 41, 275-278. 9.9,9.24, 9.25,9.47

Pelecanos, M., Zacharopolou, A. and Yannopoulos, G. (1974) "Further studies on handedness, hand-clasping and arm-folding in Greece", Biologia Gallo-Helenica, 5, 185-190. 9.10,9.45 9.47.

Penfield, W. and Roberts, L. (1959) Speech and Brain Mechanisms, Princeton Univ. Press: New Jersey. 5.2,6.25, 6.34,6.36 6.44,6.49 8.21,8.55 8.61

Peters, M. & Pedersen, K. (1978) "Incidence of left-handers with inverted writing position in a population of 5910 elementary school children", Neuropsychologica, 16, 743-746. 8.6

Pick, A. (1931: 1973) Aphasia, trans. Brown, J.W., C.C. Thomas: Springfield, Illinois. 6.19,6.20

Pizzamiglio, L., de Pascalis, C. & Vignati, A., (1974) "Stability of Dichotic listening test", Cortex, 10, 203-205. 8.4

Platt, A. (1910) De Generatione Animalium, Oxford University Press, Oxford. 12.10.

Polani, P.E. (1970) "Hormonal and clinical aspects of hermaphroditism and the testicular feminising syndrome in man", Phil, Trans. Roy. Soc. Lond. B., 259, 187-204. 11.19

Pons, J. (1961) "Hand clasping (Spanish data)", Annals of Human Genetics, 25: 141-144. 9.45,9.50, 9.63

Pope-Hennessy, J. (1966) Portrait Painting in the Italian Renaissance, Phaidon Press, London. 13.47,13.79.

Porac, C. & Coren, S. (1976) "The dominant eye", Psych. Bull., 83, 880-897. 12.9

Potter, R.H. & Nance, W.E. (1976) "A twin study of dental dimension: I. Discordance, asymmetry and Mirror-imaging", Am. J. Phys. Anthropol., 44, 391-396. 4.12

Pratt, R.T.C.& Warrington, E.K. (1972) "The assessment of cerebral dominance with 1 unilateral ECT", Brit. J. Psychiatry, 121, 327-328. 8.36

Preece, P.E., Hughes, L.E., Mansel, R.E., Baum, M., Bolton, P.M. and Gravelle, I.H. (1976) "Clinical syndromes of Mastalgia", Lancet, ii, 670-673. 11.2

Pringle, M.L.K. (1971) "The incidence of some supposedly adverse conditions and of left-handedness in schools for mal-adjusted children", Brit. J. of Educational Psychology, 31, 183-193. 10.8

Provins, K.A. & Cunliffe, P. (1972) "The reliability of some motor performance tests of handedness", Nurropsychologia, 10, 199-206. 2.28.

Purnell, D.J. and Thompson, J.N. Jr. (1973) "Selection for asymmetrical bias in a behavioural character od D. melanogaster". Heredity, 31: 401-405. 9.35,9.39, 9.43

Pye-Smith, P.H. (1870) "On left-handedness", Guy's Hospital Reports, 16, 141-146. 1.4,6.8

Raczkowski, D., Kalat, J.W., Nebes, R. (1974) "Reliability and validity of some handedness questionnaire items". Neuropsychologia, 12., 43-47. 2.20,2.28

Ramaley, F. (1913) "Inheritance of left-handedness", American Naturalist, 47, 730-738. 7.9,7.16 10.1

Reau, L. (1957) Iconographie de l'Art Chrètien, Presse Universitaire de France, Paris. 13.57,13.65

Redlich, E. (1908) "Epilepsie und Linkshaendigkeit", Archives Psychiat., 44, 59-83. 5.1,6.17.

Reynolds, L.M. & Locke, S. (1971) "Relation between handedness and side of onset of Parkinsonism", Lancet, ii, 714. 11.19.

Rheingold, H.L. & Keene, G.C. (1965) "Transport of the human young", in Determinants of Infant Behaviour ed. B.M. Foss, Tavistock, London. 13.13

Rhoads, J.G. and Damon, A. (1973) "Some genetic

Traits in Solomon Island populations: II. Hand-clasping, arm-folding and handedness". American Journal of Physical Anthropology, 39: 179-184. 9.21,9.24, 9.25,9.45, 9.47,9.50, 9.63

Riches, E.W., Griffiths, I.H. & Thackray, A.C. (1951) "New growths of the kidney and ureter", British Journal of Urology, 23, 297-356. 11.19

Rickert, M. (1954) Painting in Britain: The Middle Ages, Penguin Books, Harmondsworth. 13.19,13.92, 13.106

Riese, W. (1947) "The early history of aphasia", Bull. Hist. Med., 21, 322-334. 6.3

Riese, W. (1959) "A History of Neurology", M.D. Publications, New York.

Riese, W. (1970) "Cerebral dominance: its origin, its history and its nature", Clio Medica 5, 319-326. 8.16

Riese, W. & Goldstein, K. (1950) "The brain of Ludwig Edinger", J. Comp. Neurol., 92, 133-161. 1.4

Riesenfeld, A.& Simon, M. (1975) "Septal apertures in the humerus of normal and experimental rats", American Journal of Physical Anthropology, 42, 57-62. 11.17.

Rife, D.C. (1940) "Handedness, with special reference to twins", Genetics, 25, 178-186. 4.13,7.9, 7.13,7.63b

Rife, D.C. (1950) "Application of gene frequency analysis to the interpretation of data from twins", Human Biology, 22, 136-145. 4.4,4.13, 7.16,7.53b

Rife, D.C. & Cummins, H. (1943) "Dermatologlyphics and Mirror Imaging", Human Biology, 15, 55-64. 4.12

Ringrose, R.E., Jabbour, J.T. & Keele, D.K. (1965) "Hemihypertrophy", Pediatrics, 36, 434-448. 11.7

Roberts, L. (1969) "Aphasia, apraxia and agnosia in abnormal states of cerebral dominance, in Vinken, P.J. & Bruyn, G.W. (editors) Handbook of Clinical Neurology, vol. 4; North Holland Publishing Company, Amsterdam.

Robin, P.E. & Shortridge, R.T.J. (1979) "Lateralisation of tumours of the nasal cavity and paranasal sinuses and its relationship to aetiology", Lancet, i. 695-696. 11.19b.

Ronn, H.H. (1976), "Fluocinonide compared with beta methasone in the treatment of eczema and psoriasis", Practitioner, 216, 704-706. 11.2

Rosenthal, R. & Bigelow, L.B. (1972) "Quantitative brain measurements in chronic schizophrenia", British Journal of Psychiatry, 121, 259-264. 8.19

Rothman, K.J. and Wepsic, J.G. (1974) "Side of facial pain in trigeminal neuralgia", J. Neurosurgery, 40, 514-516. 11.19

Rotternerg, D.A. & Hochberg, F.H., (1977) Neurological Classics in Modern Translation. 6.3

Roux, C.Z. (1977) "Fecundity differences between mating pairs for a single autosomal locus, sex differences

in viability and non-overlapping generations", Theoretical Population Biology, 12, 1-9. 10.5

Russell, W.R. and Espir, M.L.E. (1961) Traumatic Aphasia, Oxford Univ. Press: London. 6.25

Ruud, G. & Spemann, H. (1923) " Die Entwicklung isolierter dorsaler . . . ", Arch. Entw.Mech. Org . 52., 95-166. 7.7

Sackeim, H.A. & Gur, R.C. (1978) "Lateral Asymmetry in intensity of emotional expression", Neuropsychologia, 16, 473-481.

Sadowsky, C. & Reeves, A.G. (1975) "Agenesis of the corpus collosum with hypothermia", Arch. Neurol., 32, 774-776. 7.32

Saldanha, P.H., Frotapessoa, O., Eveleth, P., Ottensooser, F., Cunha, A.B., Cavalcanti, M.A.A., Abramowicz, M., Beiguelmann, B., Bruno, A. de M., Carvalho, H.C., Casanova, D.M., Castro, R.L.R., Covarrubias, E., Freie-Maia, A., Leme, G., Leon, N., Mello, L.W., Oliveria, A.C.E., Patricio, L.D., Peixoto, L.I.S. (1960), "Estudo genetico e anthropologico de uma colonia de Holandeses be Brasil", Revista de Anthropologia, 8: 1-42. 9.45

Salk, L. (1966) 'Thoughts on the concept of imprinting and its place in early human development, Canad. Psychol. Assoc. J., 11, 295-305. 13.12

Salk, L. (1973) 'The role of the heart-beat in the

relations between mother and infant', Scientific America, 228, May 24-29. 13.12

Sand, P.L. & Taylor, N. (1973) "Handedness: Evaluation of binomial distribution hypothesis in children and adults", Perceptual and Motor Skills, 36, 1343-1346. 2.4

Sanders, J. (1933) "Inheritance of harelip and cleft palate, Genetica, 15, 433-510. 11.19a.

Santschi, D.R., Frahm, C.J., Pascale, L.R. & Dumanian, A.V. (1966) "The sub-clavian steal syndrome", J. Thoracic Cardiovasc. Surg. 51, 103-112. 11.19b

Sarno, M.T., Silverman, M.G. & Sands, E.S. (1970), "Speech therapy and language recovery in severe aphasia", Journal of Speech and Hearing Research, 13, 607-623. 8.18

Satz, P. (1972) "Pathological left-handedness: an explanatory model", Cortex, 8, 121-135. 6.42,6.45, 6.46,8.2

Satz, P. (1979) "A test of some models of hemispheric speech dominance in the left- and right-handed, Science, 203, 1131-1133. 8.20.

Satz, P., Achenbach, K. & Fennell, E. (1967) "Correlations between assessed manual laterality and predicted speech laterality in a normal population," Neuropsychologia, 5, 295-310. 2.4,6.38, 8.35

Schapiro, M. (1969) "On some problems in the semiotics of visual art: field and vehicle in image-signs", Semiotica, 1, 223-242. 13.36

Schnall, B.S. & Smith, D.W. (1974) "Non-random laterality malformations in paired structures", J. Pediatrics, 85, 509-511. 11.19,11.19a

Schultz, A.H. (1926) "Fetal growth of man and other primates", Quart. Rev. Biol., 1, 465-521. 11.17

Schwartz, C.J. & White, T.A. (1964) "Stenosis of Renal Artery: an unselected necropsy study", British Medical Journal, ii, 1415-1421. 11.19b.

Schwartz, M. (1977) "Left-handedness and high-risk pregnancy", Neuropsychologia, 15, 341-344. 3.2,3.3, 4.10

Searle, S. (1974) 'The church points the way', New Scientist, 61, 10-13. 13.65

Seegar, G.E. (1938) "Ovarian dysgerminoma", Archives of Surgery, 37, 697-725. 11.19

Shankweiler, D. & Studdert-Kennedy, M. (1975) "A continuum of lateralisation for speech perception", Brain and Language, 2, 212-225. 2,4,2.29

Shehamfelt, R.E. (1974) "Morphogenesis of malformations in hamsters caused by retinoic acid", Teratology, 5, 103-118. 7.7

Shields, J. (1962) Monozygotic twins: Brought up apart and brought up together, Oxford: Oxford University Press, 1962. 4.5,4.13

Shorr, D.C. (1954) The Christ-Child in devotional images, in Italy during the XIV Century, Glückstadt Press, New York. 13.14

Siegel, M.I. & Doyle, W.J. (1975) "Stress and

fluctuating limb asymmetry in various species of rodents", Growth, 39, 363-369. 7.5

Siegel, M.I., Doyle, W.J. and Kelley, C. (1977), "Heat stress, fluctuating asymmetry and prenatal selection in the laboratory rat", Am.J. Phys. Anth., 46, 121-126. 7.5

Siegel, M.I. & Smookler, J.H. (1973) "Fluctuating dental asymmetry and audiogenic stress", Growth, 37, 35-39. 7.5

Siemens, H.W. (1924) "Die Bedeutungs der Zwillingspathologie ..." Setzungsber.d.Ges. fur Morph. und Physiol. in Munich, Jhg, 35; cited by Nayglaki & Levy (1973) 4.13,7.63b

Simmel, M.L. (1961) "The absense of phantoms for congenitally missing limbs" Amer. J. Psychology, 74, 467-470. 11.19a

Singh, L.and Goel, K.V. (1975) "Mid-phalangeal hair, hand-clasping and handedness among Khatris and Baniyas of Patiola, India", Acta Geneticae Medicae et Gemellologiae, 24: 343-345. 9.45

Snedecor, G.W. & Cochran, W.G. (1967) Statistical Methods, Sixth Edition, Iowa State University Press, Ames. 2.13

Spemann, H. & Falkenberg, H. (1919) "Uber asymmetrische Entwicklung und Situs inversus viscerum bei zwillingen und Doppelbildungen", Wilhelm Roux Arch. EntwMech.Org., 45, 371- 7.7

Spiro, M. & Cotton, L.T. (1970) "Aorto-Iliac Thrombo-endarterectomy", British Journal of Surgery, 57, 161-168. 11.19b

Springer, S.P. & Searleman, A. (1978) "Laterality in twins: the relationship between handedness and hemispheric asymmetry for speech", Behaviour Genetics, 8, 349-357. 8.9,8.35

Srnivasan, S. and Mukherjee, D.P. (1975) "Ear-lobe attachment, hand-clasping and arm-folding among Tamil Brahmins in relation to cultural and ethnic affiliations". Acta Geneticae et Medicae Gemellologiae, 24: 159-160. 9.45,9.47

Staley, R.N. & Green, L.J. (1974) "Types of tooth cusp occurrence asymmetry in human monozygotic and dizygotic twins", Am. J. Phys. Anthropol. 40, 187-196. 4.12

Stannus, H.S. (1914) "Congenital anomalies in a native African race", Biometrika, 10, 1-24 11.19

Steele, K.B. & Javert, C.T. (1942) "The mechanism of labor for transverse positions of the vertex", Surgery, Gynaecology and Obstetrics, 75, 477-484. 11.18,12.13

Stern, C., Schaefer, A. & Ukpe, O,J. (1968) "Hairy Pinnae in Nigeria", Journal of Heredity, 59, 174-178. 11.19a

Stern, D.B. (1977) "Handedness and the lateral distribution of conversion reactions", J. Nerv. Ment. Dis., 164, 122-128. 11.2,11.19b

Stewart, A.F. (1976) "Scrotal asymmetry: an appendix", Nature, 262, 155. 12.9,12.17

Stier, E. (1911) Untersuchungen uber Linkshandigkeit', Jena. 5.1

Stocks, P. (1933) "A biometric investigation of twins and their brothers and sisters", Ann. Eugen., 5, 1-55. 4.13,7.63b

Strong, R. (1969) The English Icon: Elizabethan and Jacobean Portraiture, Routledge, Kegan and Paul, London. 13.2,13.47, 13.72

Subirana, A. (1969) "Handedness and Cerebral Dominance", in Handbook of Clinical Neurology, vol. 4, ed. Vinken, P.J. & Bruyn, G.W., North-Holland: Amsterdaml

Subirana, A. (1958) "The prognosis of aphasia in relation to cerebral dominance and handedness", Brain, 81, 415-425. 8.2

Surtees, V. (1971) The paintings and drawings of Dante Gabriel Rosetti (1928-1882): a catalogue raisonné, Oxford, University Press, London. 13.83.

Sutton, P.R.N. (1963) 'Handedness and facial asymmetry: lateral position of the nose in two racial groups', Nature, 198, 909. 13.37

Tarán, L. (1965) Parmenides: A text with translation, commentary and critical essays, Princeton University Press, New Jersey. 12.10

Taylor, L.W. & Gunns, C.A. (1947) "Diplopodia: a lethal form of polydactyly in chickens", J. Hered. 38, 67-76. 11.17

Thomas, D.G. & Campos, J.J. (1978) "The relationship of handedness to a 'lateralised' task", Neuropsychologia, 16, 511-515. 2.27

Thomas, E.S. (1929) "Right handedness", Man 29, 24. 10.1

Thompson, A.L. & Marsch, J.F. (1976) "Probability sampling of manual asymmetry" Neuropsychologia 14, 217-223. 2.4

Thompson, D'Arcy, W. (1910) Historia Animalium, Oxford University Press, Oxford. 12.10

Thompson, H.C. (1921) Diseases of the Nervous System, 3rd Edition, Revised, Cassell & Co., London. 6.24

Thompson, J.W., McBatts, J. & Danforth, C.H. (1921) "Hereditary and racial variation in the Musculus Palmaris Longus", Am.J. Phys. Anth., 4, 205-218. 11.17

Thyss, J. (1947) "Étude bibliographique et critique du problème des gaucheurs, MD thesis, Paris. Cited by Zazzo (1960) 4.13,7.63b

Tihen, J.A., Charles, D.R. & Sippel, T.D. (1948) "Inherited visceral inversion in mice", J. Heredity, 39, 29-31. 7.8

Tiwari, S.C. and Bhasin, M.K. (1969) "Frequency of colour-blindness and hand-clasping in two endogenous caste groups of Gazrhwal". Human Heredity, 19: 110-112. 9.45

Tomasch, J. & Etemadi, A.A. (1962) "Human Hypoglasal Nucleus (A quantitative study)", J. Comparative Neurology, 119,105-111. 11.18

Torgerson, J. (1950) "Situs inversus, asymmetry and twinning". American Journal of Human Geeetics, 2: 361-370. 9.25,11.3

Torgerson, J. (1951) "Asymmetry and skeletal maturation Acta Radiologica, 40, 521-523. 11.17

Trankell, A. (1955) "Aspects of genetics in psychology", Am.J. Hum. Genet., 7, 264-276. 7.16

Trousseau, A. (1877) "Clinique médicale de l'Hôtel-Dieu de Paris", 5th Edition, Bailliere et fils Paris Reprinted in part in Hécaen & Dubois (1969. 6.6

Tugwell, P. & Williams, A.O. (1977) "Jaundice associated with lobar pneumonia". Q.J. Med. NS., 46, 97-118. 11.2

Turkewitz, G. (1977) 'The development of lateral differences in the human infant', in Lateralisation in the Nervous System, ed. Harnad, S. et al. pp. 251-259, Academic Press, New York. 13.42

Turkewitz, G., Moreau, T., & Birch, H.G. (1967), "Relation between birth condition and neurobehavioural organisation in the neonate". Pediatric Research, 2, 243-249. 3.5

Uhrbrock, R.S. (1973) 'Laterality in Art', J. Aesthet. Art. Crit., 32, 27-35 10.1,13.3, 13.13

van Valen, L. (1962) "A study of fluctuating asymmetry", Evolution, 16: 125-142. 9.2

Vernberg, F.J. & Costlow, J.D. (1966) "Handedness in

Fiddler Crabs (Genus Uca)", Crustaceana, 11, 61-64. 9.27

Verschuer, O. von (1927) "Der Vererbungs_biologische Zwillings_forschung", Ergeb. Inneren. Med. und Kinderheit, 31, 35-120; cited by Nagylaki & Levy (1973) 4.13,7.63b

Victor, D.I. (1976) "The diagnosis of congenital unilateral third-nerve palsy", Brain, 99, 711-718. 11.19

Waddington, C.H. (1957) The Strategy of the Genes, Allen & Unwin, London. 7.4,11.12

Wadham, W. (1869) "Aphasia", St. Georges Hospital Reports, 4, 245-250. 6.8

Wald, G. (1957) "The origin of optical activity", Ann. N.Y. Acadm Sci., 69, 352-368. 11.10

Walker-Smith, G.J., Gale, A.G. & Findlay, J.M., (1977) 'Eye-movement stategies involved in face perception", Perception, 6, 313-326. 13.40

Walsh, G. & Pool, R.M. (1942) "Laterality dominance in the four Gospels", Southern Medicine and Surgery, 104, 317-329. 12.9

Walsh, G. & Pool, R.M. (1943) "Laterality dominance in the Koran", Southern Medicine and Surgery, 105, 112-125. 12.9

Wang, C.C. (1927), "On the post-natal growth of the optic nerve in albino and in Gray Norway rats", J. Comp. Neurol., 43, 201-220. 11.18

Warrington, E.K. and Pratt, R.T.C. (1973) "Language laterality in left-handers assessed by unilateral ECT", Neuropsychologia, 11, 423-428. 6.39,8.36

Warrington, J. (1956) (translator) Metaphysics of Aristotle, Everyman, London. 12.10.

Washburn, S.L. (1946) 'The Effects of facial paralysis on the growth of the skull of rat and rabbit', Anat. Rec., 94, 163-168. 13.37

Watterson, G.A. and Perlow, J. (1978) "Homozygosity in Three Allele Populations". Theoretical Population Biology, 13: 268-275. 9.34

Weinstein, S. (1963) 'The relationship of laterality and cutaneous area to breast sensitivity in sinistrals and dextrals', Amer. J. Psychol., 76, 475-479. 12.10,13.

Weinstein, S. (1978) "Functional cerebral hemispheric asymmetry", in: Asymmetrical Function of the Brain, ed. M. Kinsbourne, Cambridge University Press, Cambridge. 6.26

Weinstein, S., Vetter, R.J. & Sersen. E.A.(1970) "Phantoms following breast amputation", Neuropsychologia, 8, 185-197. 11.19b

Wisenberg, T. and McBride, K.E. (1935) Aphasia: a Clinical and Psychological Study. 6.16,6.4-(

Weitz, W. (1924) "Studien an eineiigen Zwillingen", Zeit für Klin.Med., 101, 115-154. Cited by Nagylaki and Levy (1973). 4.4,4.13, 7.63b

Wethy, H.E. (1971) The Paintings of Titian: 2, the Portraits, Phaidon Press, London. 13.3

Wernicke, C. (1906; 1977) Wernicke's works on aphasia, trans., G.H. Eggett, Mouton: The Hague. 6.14

Weyl, H. (1952) Symmetry, Princeton University Press, New Jersey. 2.0

White, K. & Ashton, R. (1976) "Handedness assessment inventory", Neuropsychologia, 14, 261-264. 2.17,2.18

Wiener, A.S. (1932) "Observations on the manner of clasping the hands and folding the arms", American Naturalist, 66: 365,370. 9.8,9.18, 9.21,9.23, 9.24,9.25, 9.46,9.47, 9.50,9.63

Wilkin,S. (1852) Editor, The Works of Sir Thomas Browne, Henry Bohn, London, 12.12,12.15

Wilson, I. (1978) The Turin Shroud, Gollancz: London. 13.64, 13.112.

Wilson, J.G., Jordan, H.C. & Brent, R.C. (1953), "Effects of irradiation on embryonic development II: X-rays on the ninth day of gestation in the rat", American Journal of Anatomy, 92, 153-177. 7.8

Wilson, S.A.K. (1908) "A contribution to the study of apraxia with a review of the literature", Brain, 31, 164-216. 6.0

Wilson, S.A.K. (1921) "An introduction to thestudy of aphasia", Lancet, ii, 1143-1147. 6.18

Wilson, S.A.K. (1926) Aphasia, Kegan, Paul, Trench, and Trubner: London. 6.15,6.18

Wilson , S.R. (1971a) "The determination of the mode of inheritance from the correlation between relatives" in The Genetics of Mental Disorders.

Slater, E. & Cowie, V. London, Oxford Univ. Press. 9.3

Wilson, S.R. (1971b) "Fitting of models of incomplete penetrance to family data", Annals of Human Genetics, 35: 99-108. 9.3

Wilson, P.T. & Jones, H.E. (1932) "Left-handedness in twins", Genetics, 17, 560-572. 4.8,4.13, 7.63b

Winckelmann, J.J. (1764; 1968) History of Ancient Art, trans. A. Gode, New York. 12.9,E.4.

Witelson, S.F. (1977) "Developmental Dyslexia ; two right hemispheres and none left", Science, 195, 309-311.

Witmer, L. (1902) Analytical Psychology, p. 84 13.11

Wolfe, C.S. (1978) "Theological motivations in artistic portrayal during the Northern European Reformation", BA Thesis (Unpublished) Univ. of Cambridge. 13.23,14.1

Wolff, P.H., Hurwitz, I. & Moss, H. (1977) "Serial organisation of motor skills in left and right-handed adults", Neuropsychologia, 15, 539-546. 2.30

Woo, T.L. (1928) "Dextrality and sinistrality of hand and eye: Second memoir", Biometrika, 22A, 79-148. 2.28

Woo, T.L. (1930a) "On the asymmetry of the human skull", Biometrika, 22, 324-352. 11.17

Woo, T.L. (1930b) "A study of seventy-one Ninth Dynasty Eyyptian skulls from Saqhara", Biometrika, 22, 65-93. 11.17

Woo, T.L. & Pearson, K. (1927) "Dextrality and sinistrality of hand and eye", Biometrika,

19, 165-199. 2.3,2.28

Wood, T.B. (1905) "Note on the inheritance of horns and face-colour in the sheep", J. Agric.Soc. 1, 364- 7.33

Woolf, W. (1933) 'The experimental study of forms of expression", Char. and Pers. 2, 168-176. 13.37

Woolf, C.M. & Gianas, A.D. (1976) "Congenital cleft lip and fluctuating dermatoglyphic asymmetry", Am.J.Hum.Genet., 28, 400-403.

Yamaura, A. (1940) "On some hereditary characters in the Japanese race including the Tyosenese (Coreans)", Japanese Journal of Genetics, 16: 1-9. 9.46,9.50, 9.63

Yarbus, A.L. (1967) Eye-Movements and Vision, Plenum Press, New York, 13.40

Yeni-Komshian, G.H. & Benson, D.A. (1976) "Anatomical study of cerebral asymmetry in the temporal lobe of humans, chimpanzees and rhesus monkeys", Science, 192, 387-389.

Yoshiwara, Y. (1957) "On the inheritance of the manner of clasping hand", Japanese Journal of Human Genetics, 2: 73-74 9.46,9.50, 9.63

Young, A.W. & Ellis, H.D. (1976) 'An experimental investigation of developmental differences in ability to recognise faces presented to the left and right hemispheres', Neuropsychologia, 14, 495-498 13.37

Zangwill, O.L. (1954) "Agraphia due to a left parietal glioma in a left-handed man", Brain, 77, 510-520.

Zangwill, O.L. (1955) "Speech and handedness, Adv. Scil. 12, 55-59. 6.31,6.41

Zangwill, O.L.(1960) Cerebral Dominance and its relation to Psychological Function, Oliver and Boyd: London. 6.25,8.2

Zazzo, R. (1960) Les jumeaux: le couple et la personne, Paris, France, Presses Universitaires de France. 4.8,4.13 7.63b

de Zeeuw, D. Donker, A.J.M., Burema, J. van der Hem, G.K. and Mandema, E. (1977) "Nephroptosis and Hypertension ", Lancet, i, 213-215.

Zurif, E.B. and Bryden, M.p. (1969) "Famililal handedness and left-right differences in auditory and visual perception", Neuropsychologia, 7, 179-187. 6.36,6.38 8.8,8.35

Boklage, C.E. (1977) "Schizophrenia, brain asymmetry, development and twinning: cellular relationships with etiological and possibly prognostic implications", Biol. Psychiat., 12, 19-35. 6.46