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

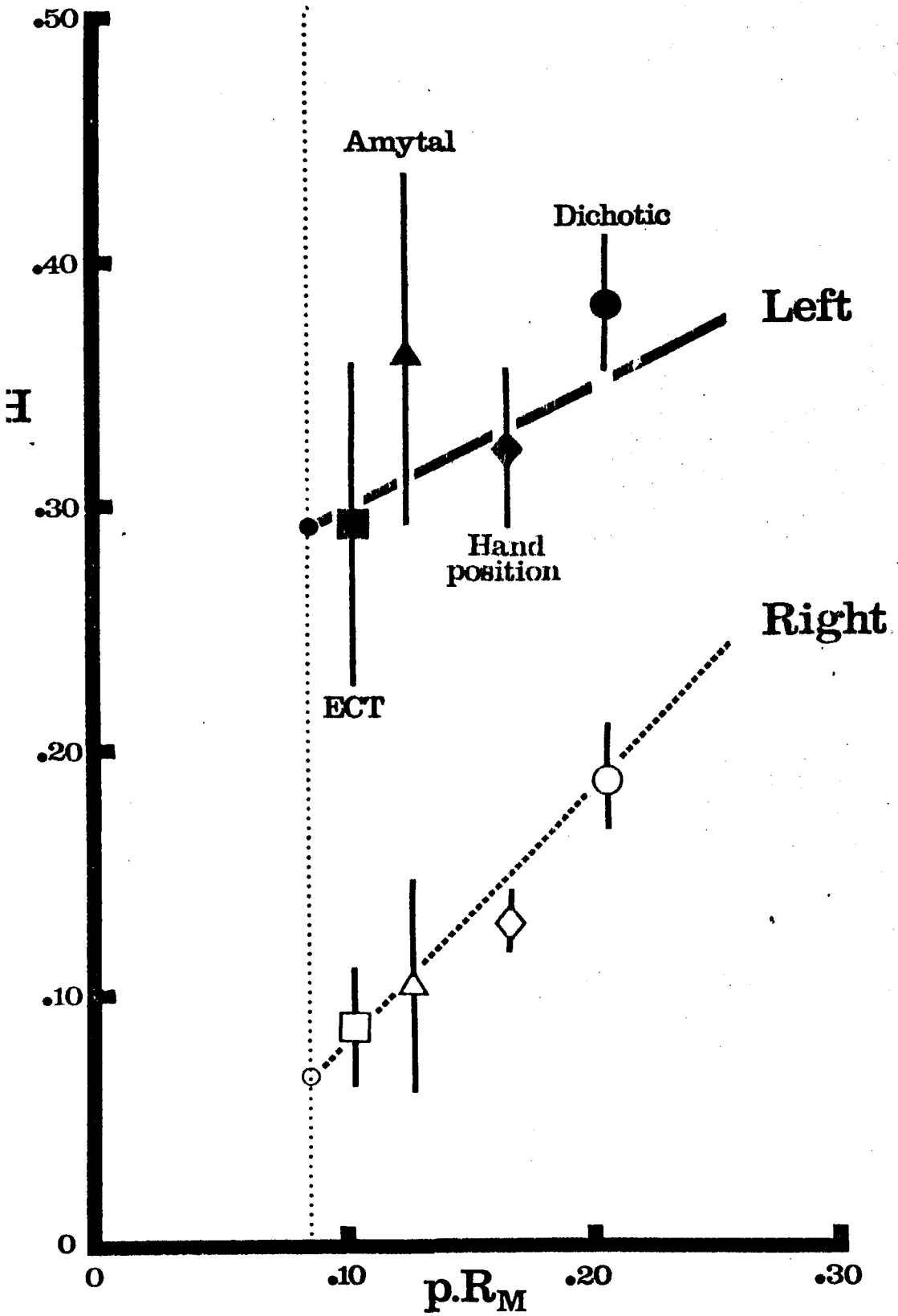
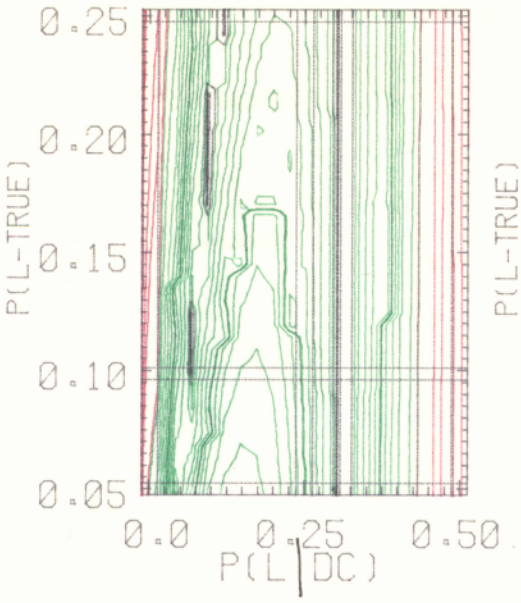
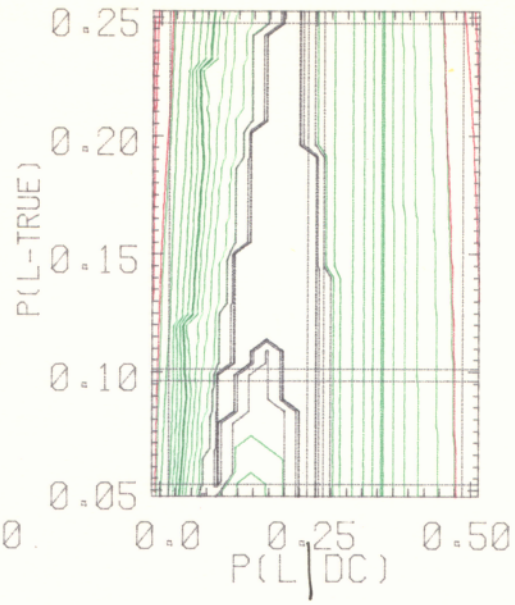


Figure 8.2 Shows the goodness of fit of my own model to the data of Figure 8.1 The contour maps are for the overall fit of the model, and are essentially similar to those of the previous paper (see caption to Figure 7.2), except that a 'liberal' number of degrees of freedom has been used throughout, 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.

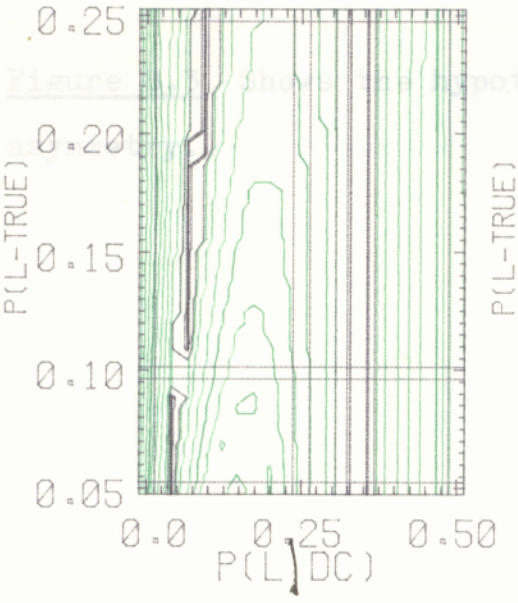
DICHOTIC LISTENING



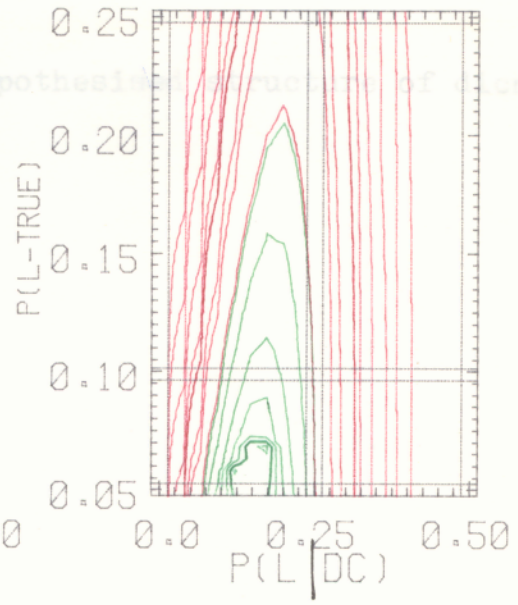
UNILATERAL ECT



INTRA-CAROTID AMYTAL



HAND-WRITING POSITION



ICM B1

Figure 8.3 Shows the hypothesised structure of dichotic asymmetry.

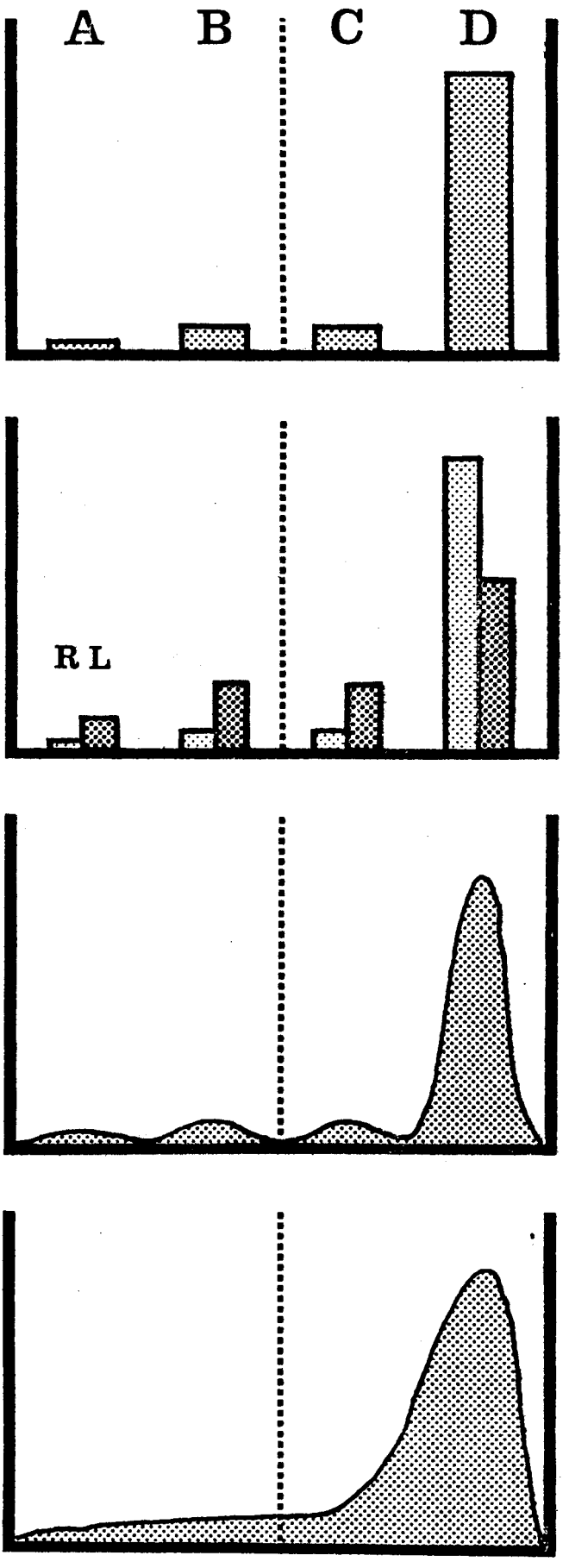

A
B
C
D
R L
LEA
REA

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.

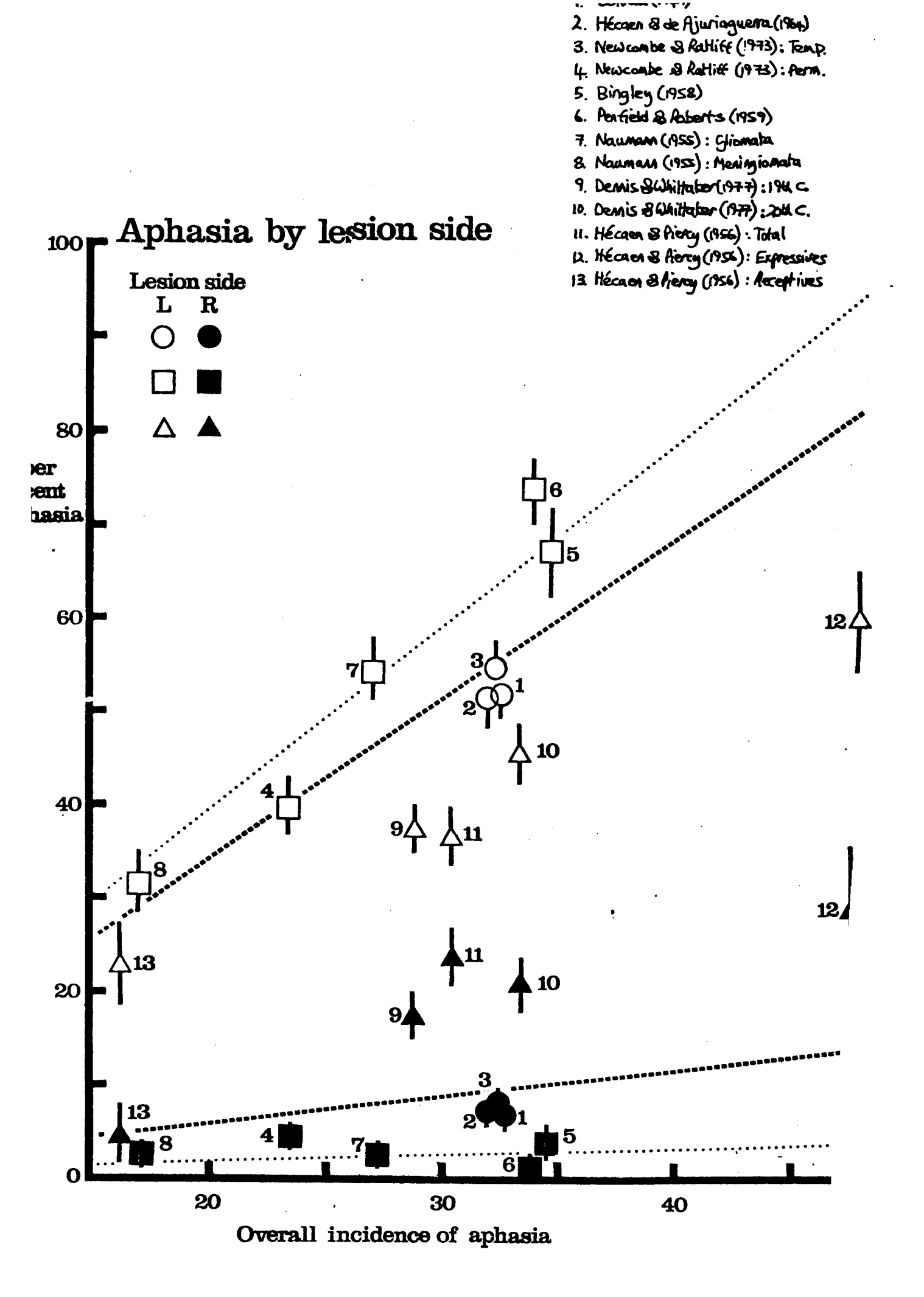
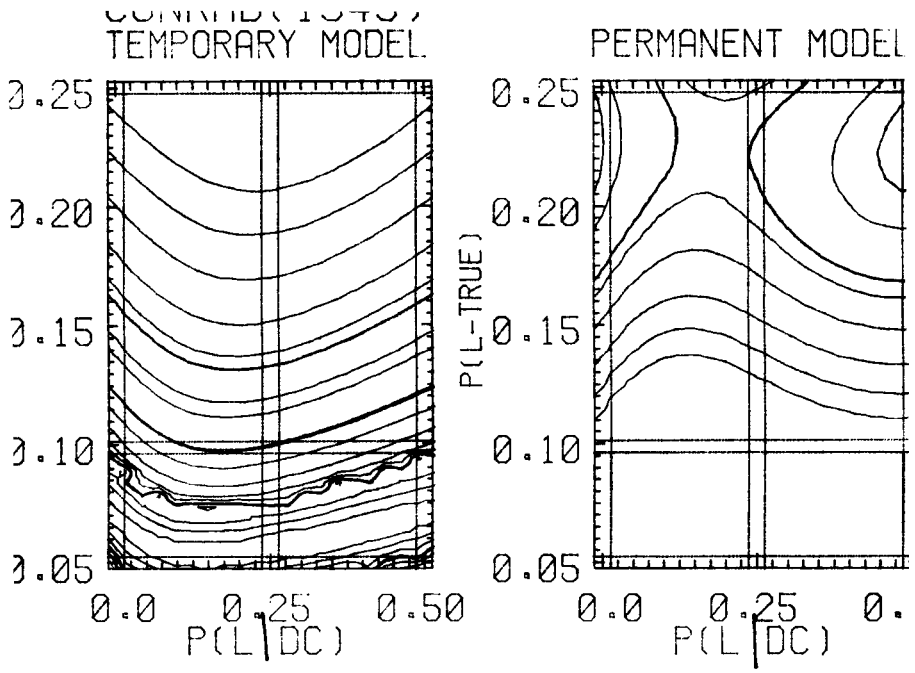
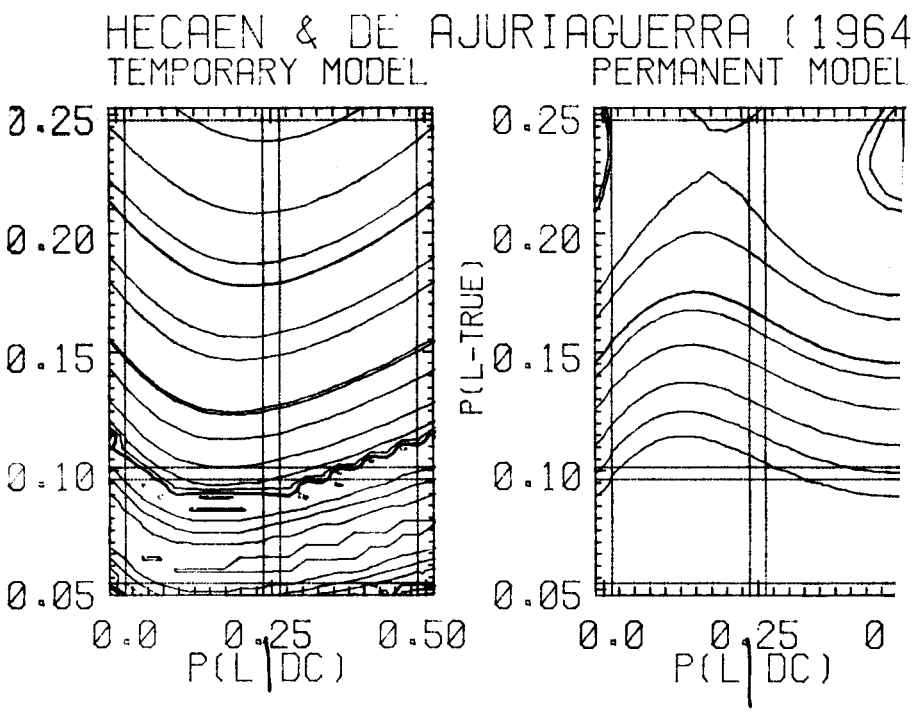

2. Hécaen & de Ajuriaguerra (1964)
3. Newcombe & Ratliff (1973): Temp.
4. Newcombe & Ratliff (1973): Perm.
5. Bingley (1958)
6. Penfield & Roberts (1959)
7. Naumann (1955): Gliomata
8. Naumann (1955): Meningiomata
9. Dennis & Whitaker (1977): 19th C.
10. Dennis & Whitaker (1977): 20th C.
11. Hécaen & 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'.


TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)


HECAEN & DE AJURIAGUERRA (1964
TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

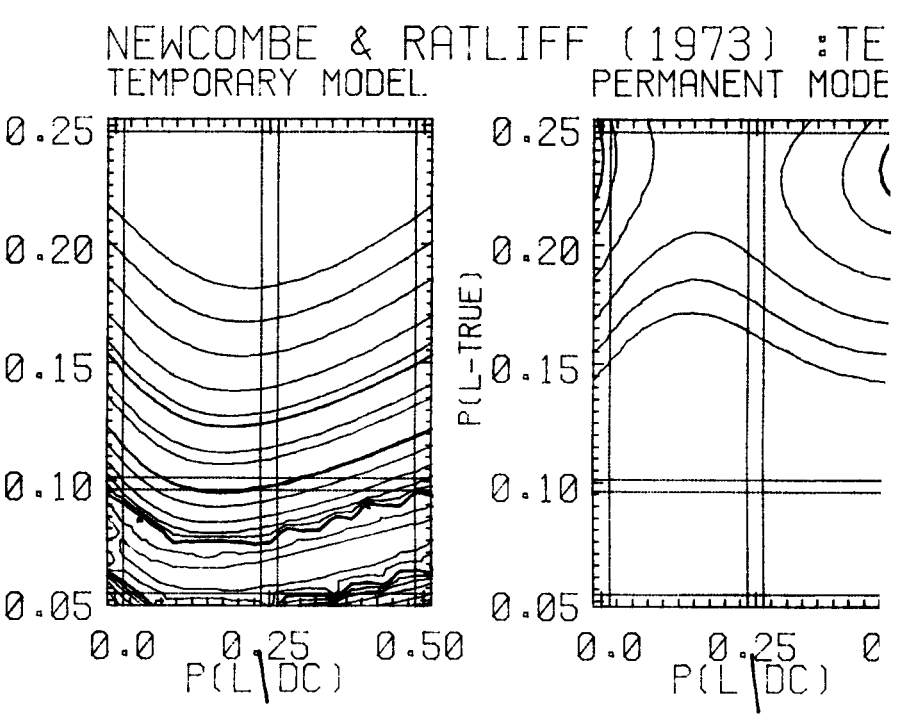

NEWCOMBE & RATLIFF (1973) :TE
TEMPORARY MODEL
PERMANENT MODE
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

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.

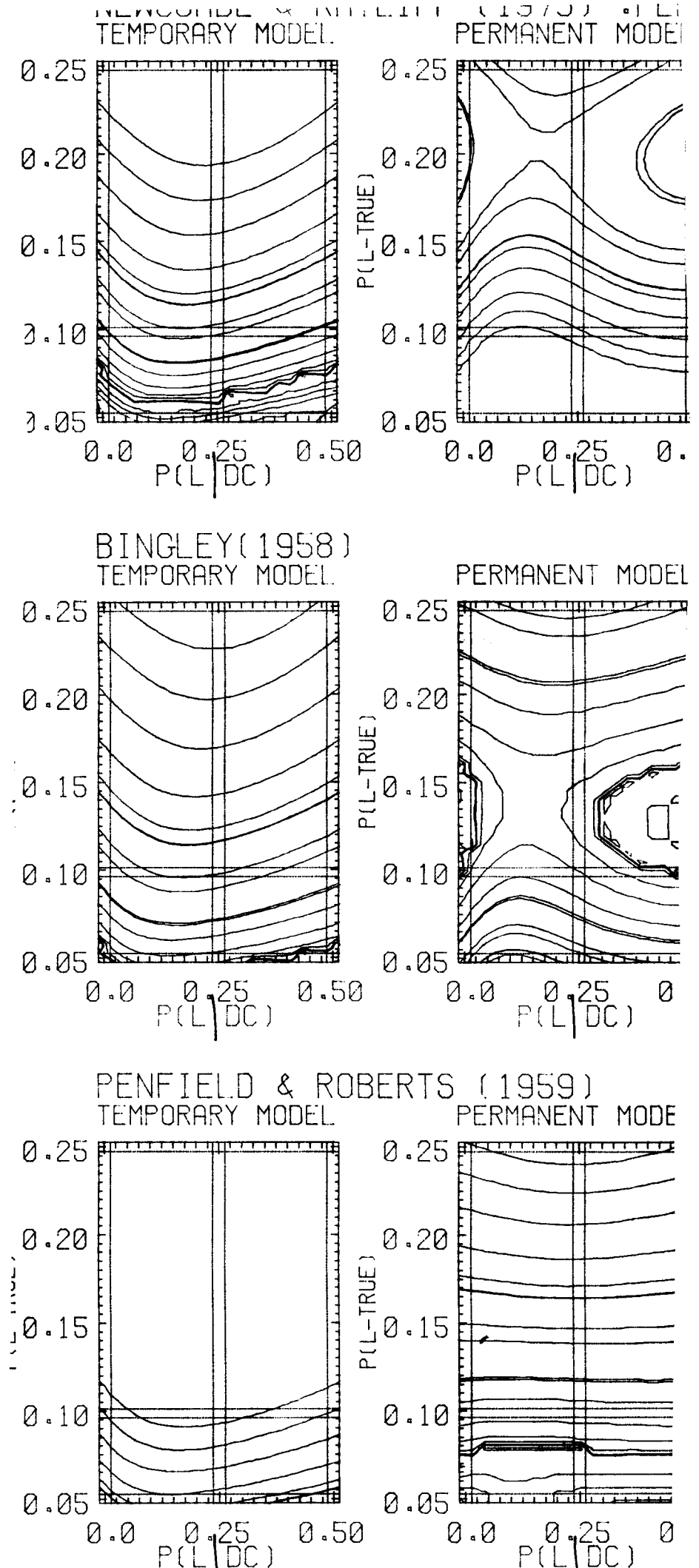
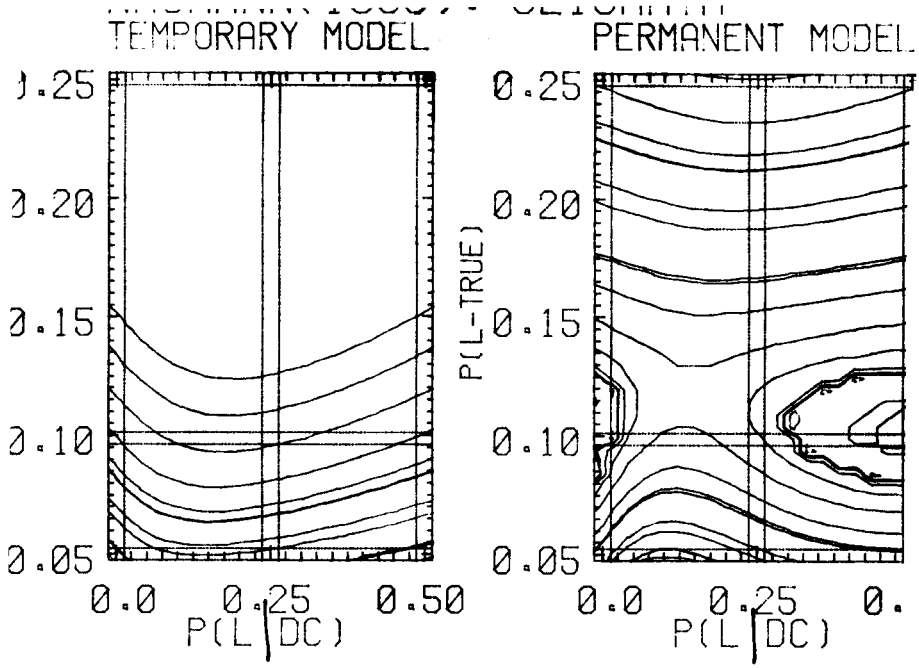

TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)
BINGLEY(1958)
TEMPORARY MODEL
PERMANENT MODEL
PENFIELD & ROBERTS (1959)
TEMPORARY MODEL
PERMANENT MODE

Figure 8.7 As for figure 8.5, except for the data of Naumann (1955).


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)

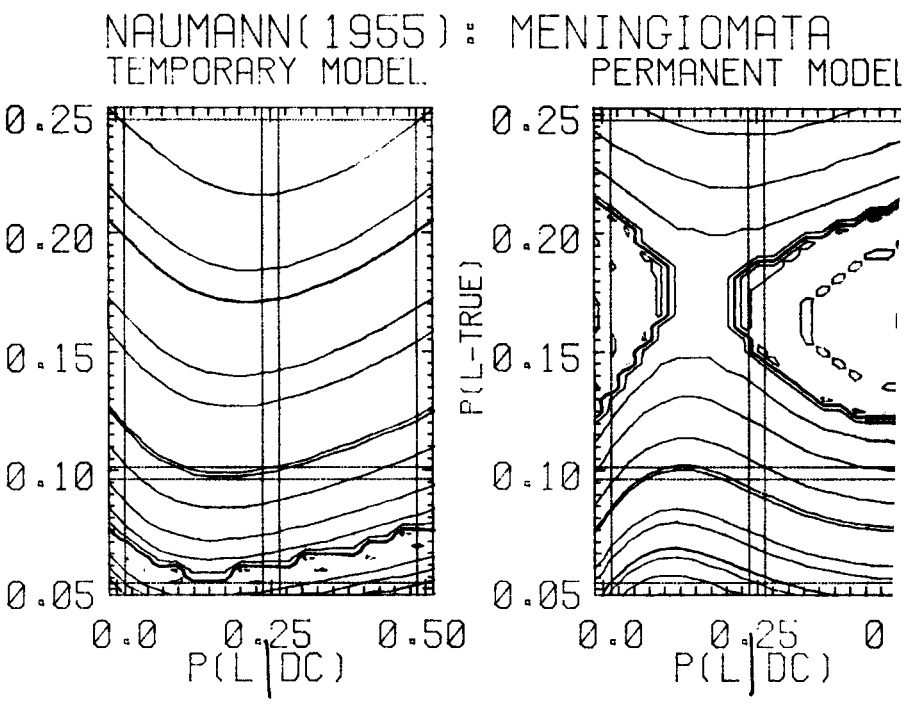
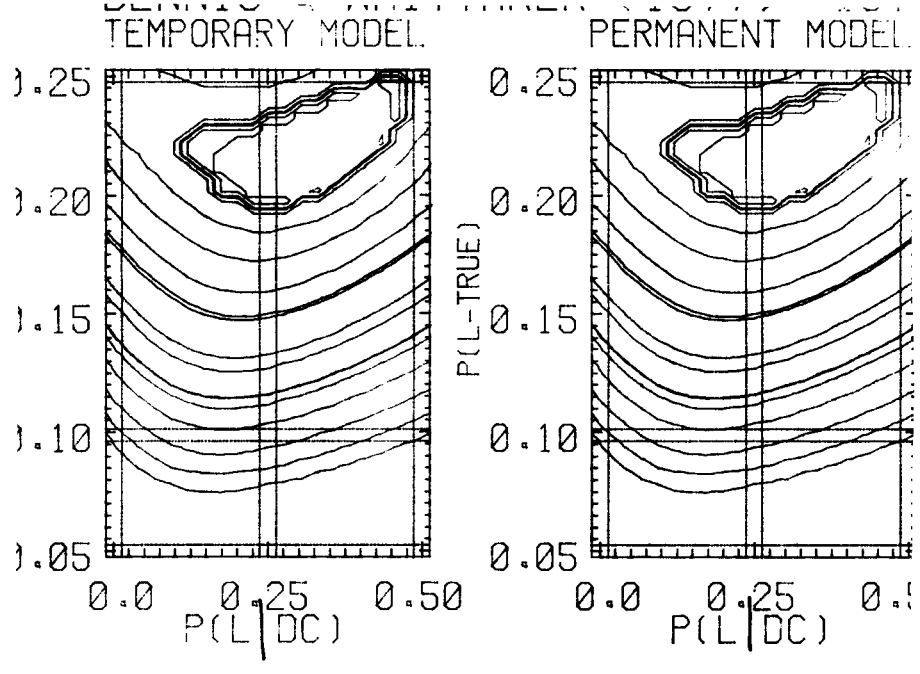

NAUMANN(1955): MENINGIOMATA
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.25
0.50
P(L|DC)

Figure 8.8 As for Figure 8.5, except for the data of Dennis and Whittaker (1977).


TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

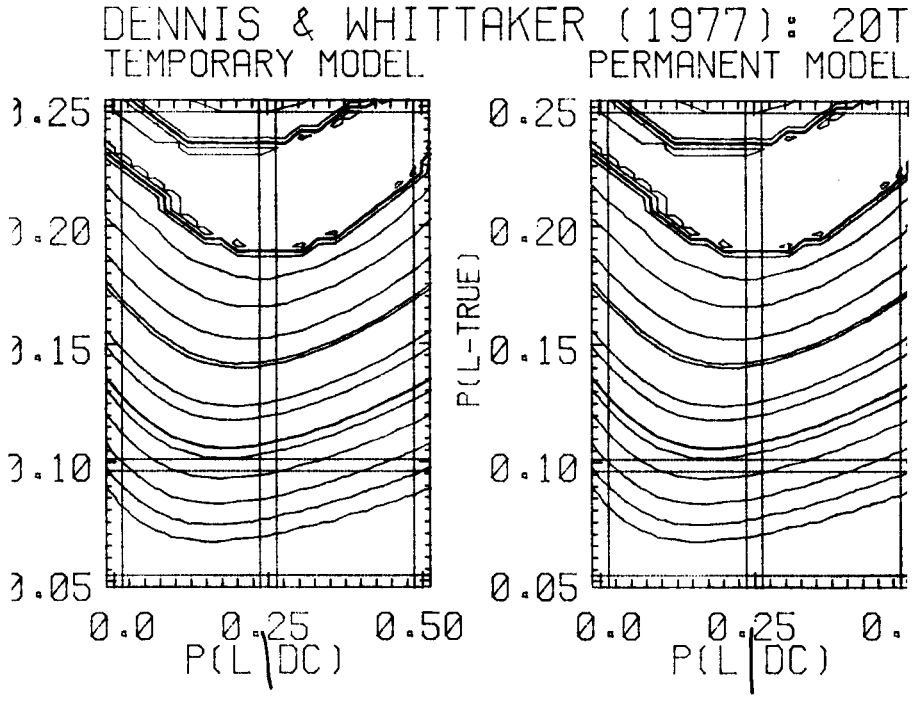
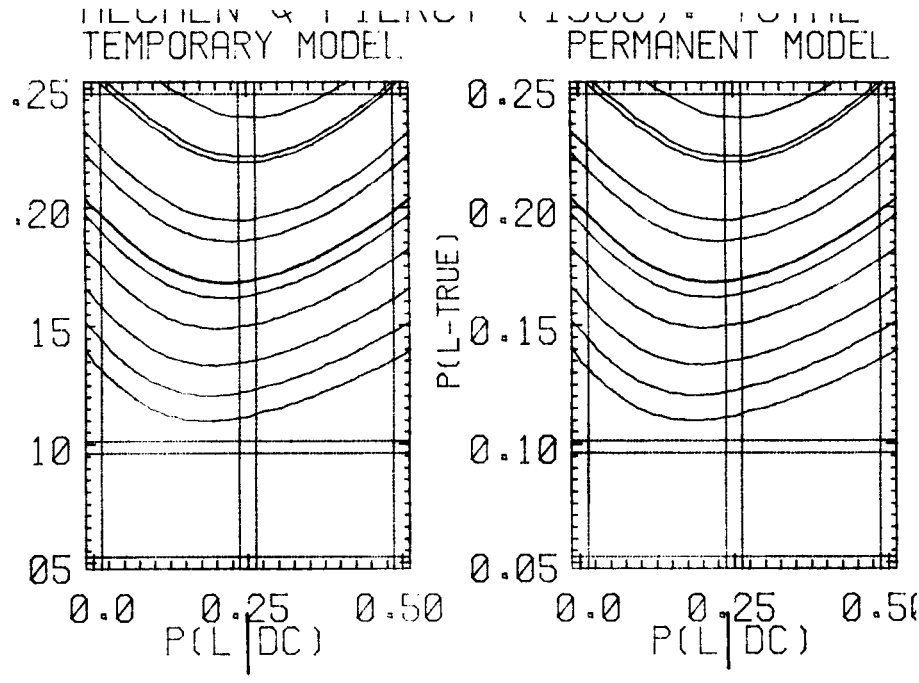
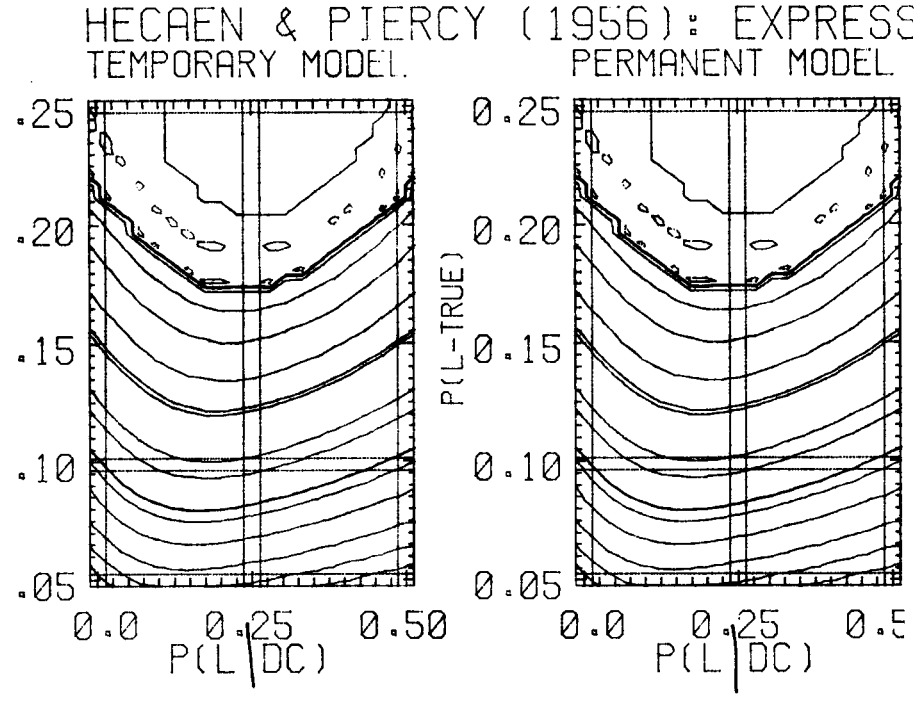

DENNIS & WHITTAKER (1977): 20T
TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

Figure 8.9 As for Figure 8.5, except for the data of Hecaen and Piercy (1956).


TEMPORARY MODEL
PERMANENT MODEL
.25
.20
15
10
05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)


HECAEN & PIERCY (1956): EXPRESS
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

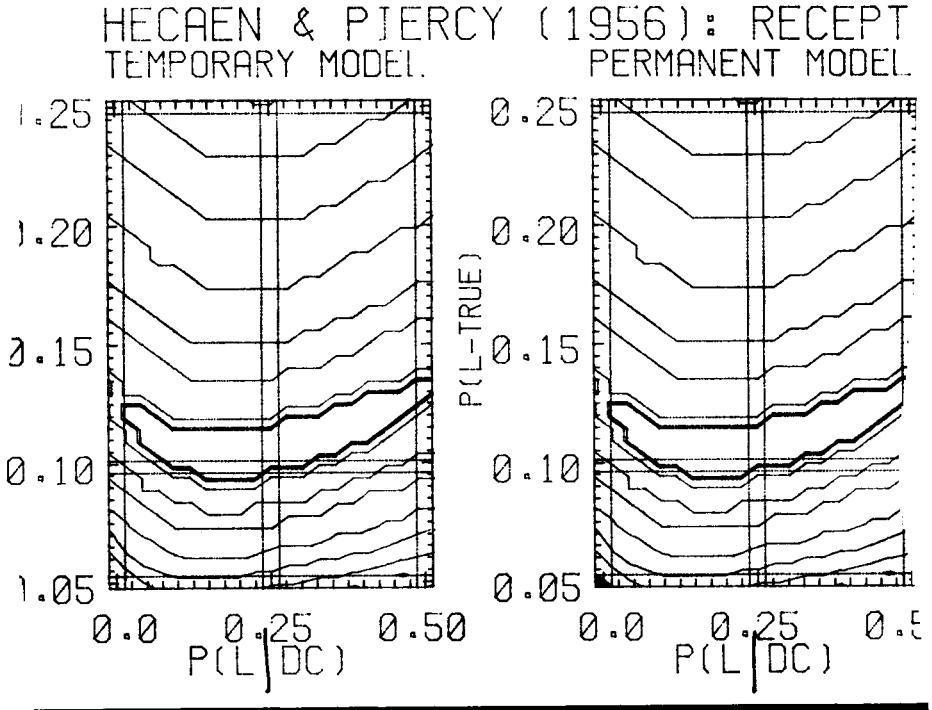
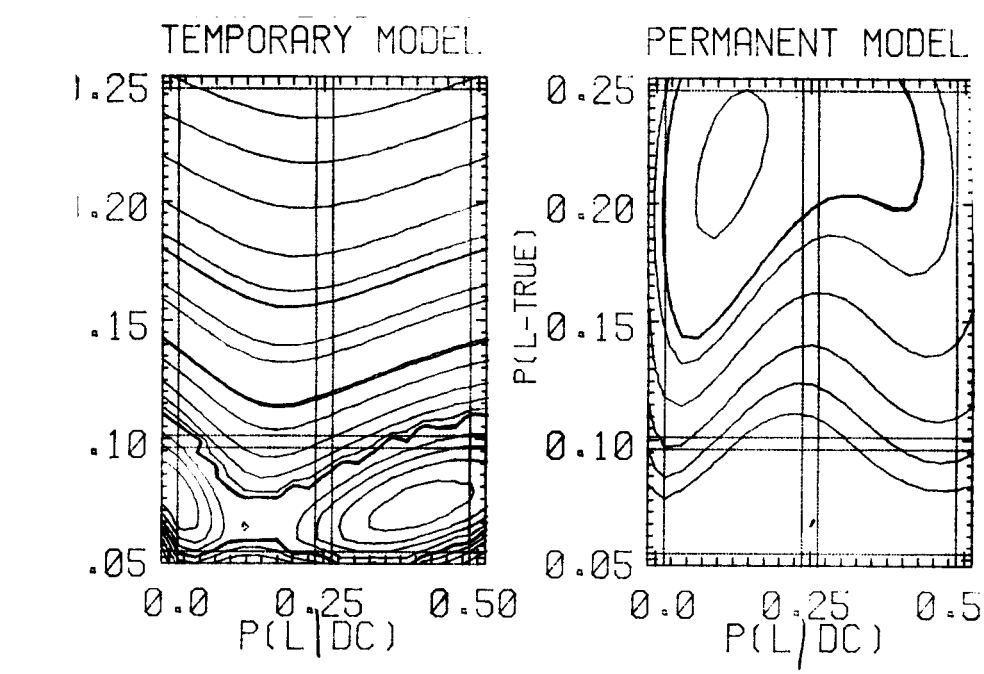
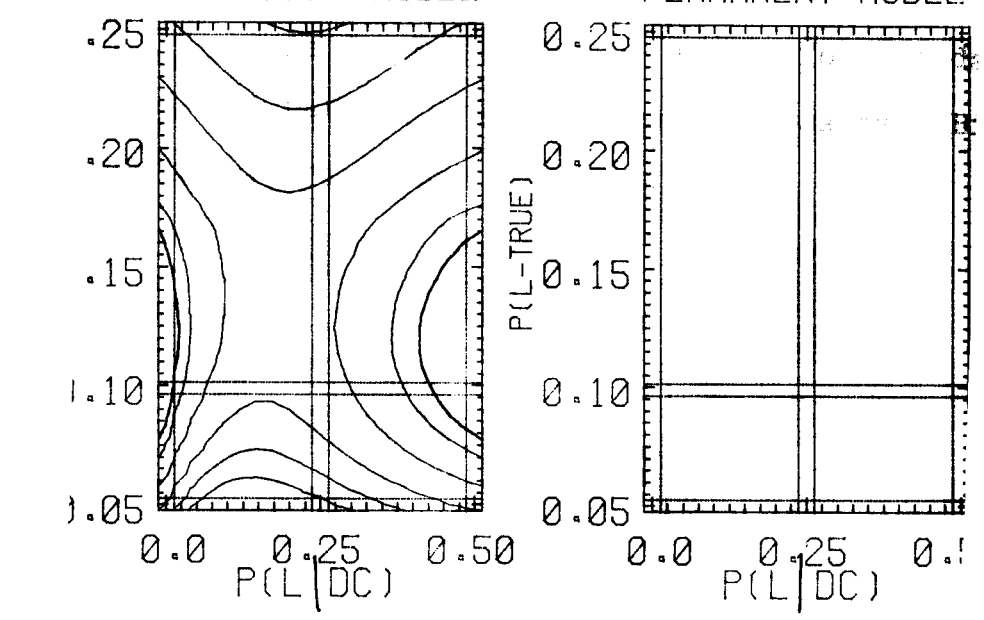

HECAEN & PIERCY (1956): RECEPT
TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.25
0.50
P(L|DC)

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.



HECAEN & DE AJURIAGUERRA (1964)



NEWCOMBE & RATLIFF (1973) :TEM

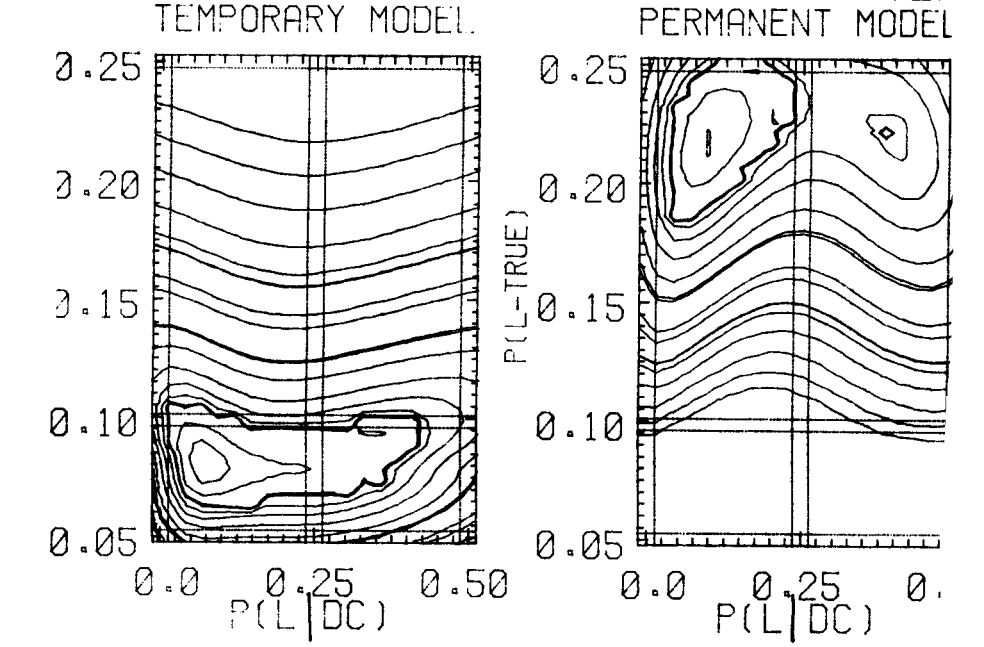
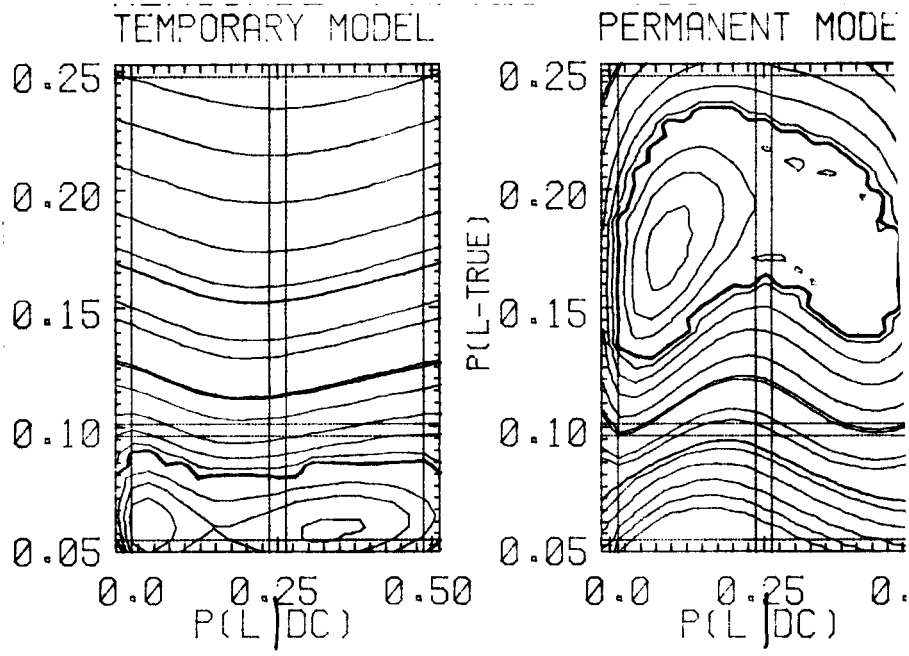
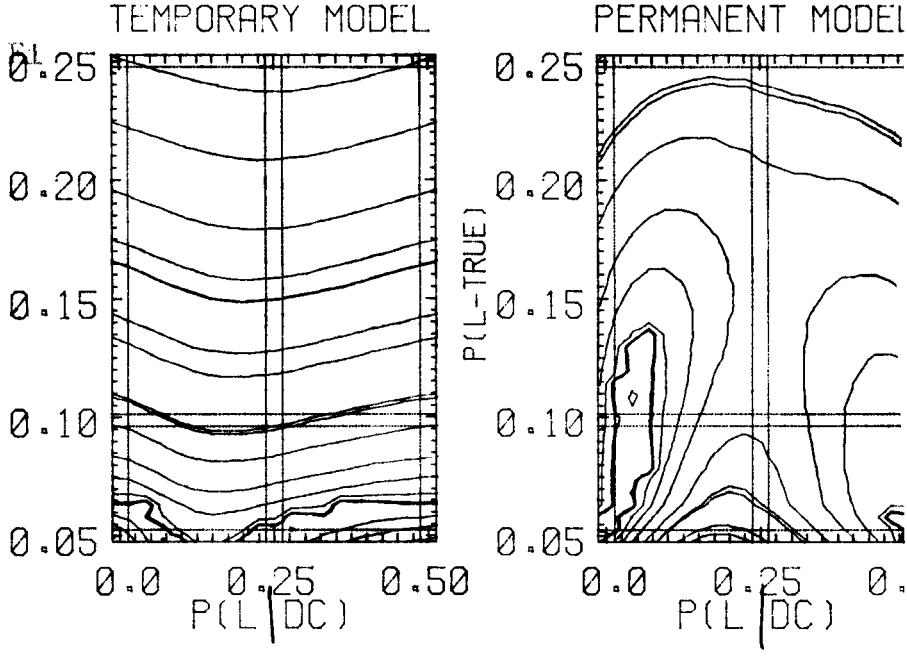


Figure 8.11 As for Figure 8.10, except for the data of Newcombe and Ratliff (1973-permanent aphasics), Bingley (1958) and Penfield and Roberts (1959).



BINGLEY(1958)



PENFIELD & ROBERTS (1959)

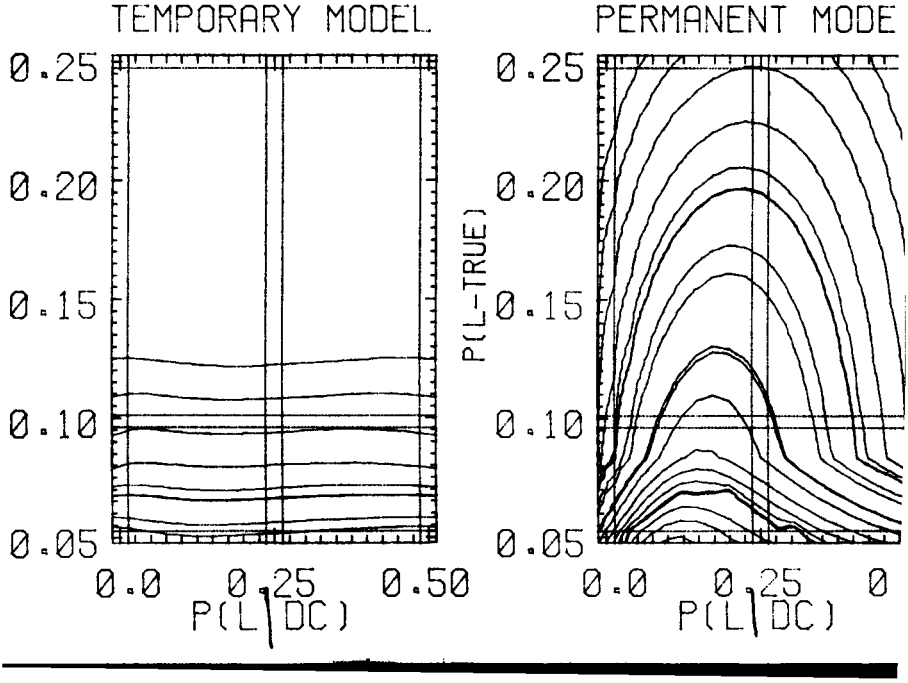
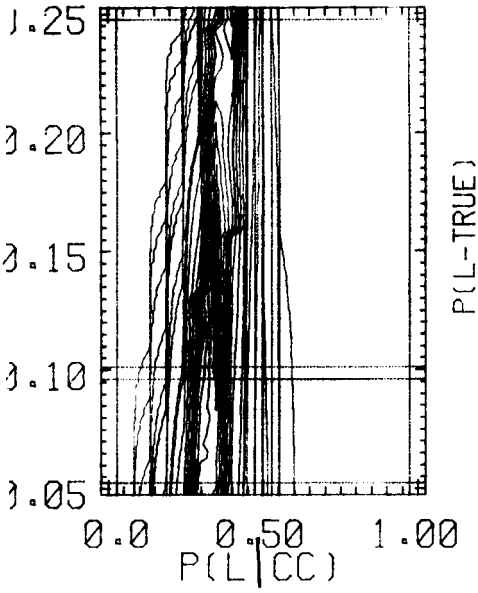
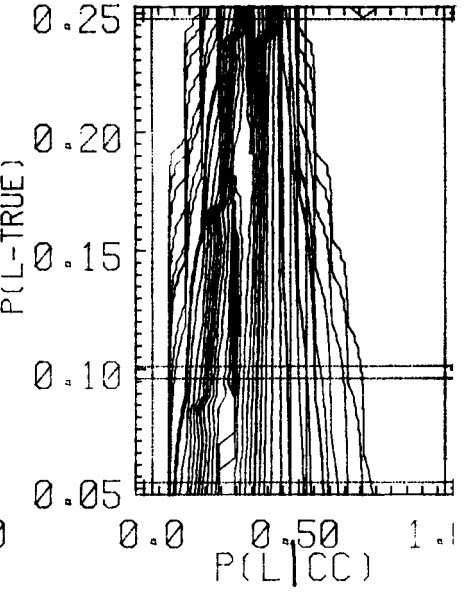


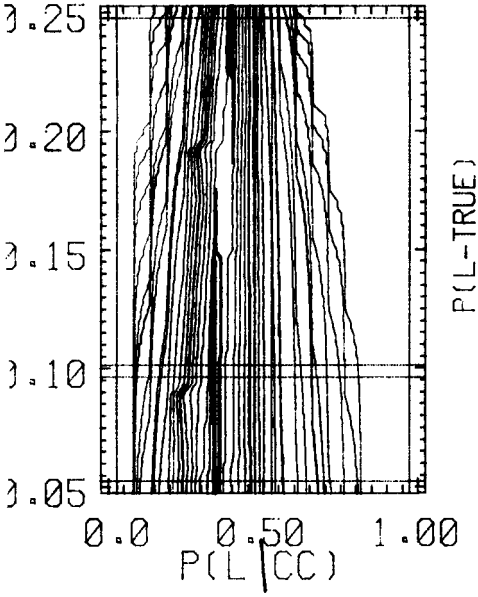
Figure 8.12 As for Figure 8.2 except that the model fitted is the Trankell model. The abscissa is the proportion of left-handers hypothesised in the SS genotype (and on this and succeeding figures should read p(L|SS), and not p(L|CC).





INTRA-CAROTID AMYTAL

HAND-WRITIN



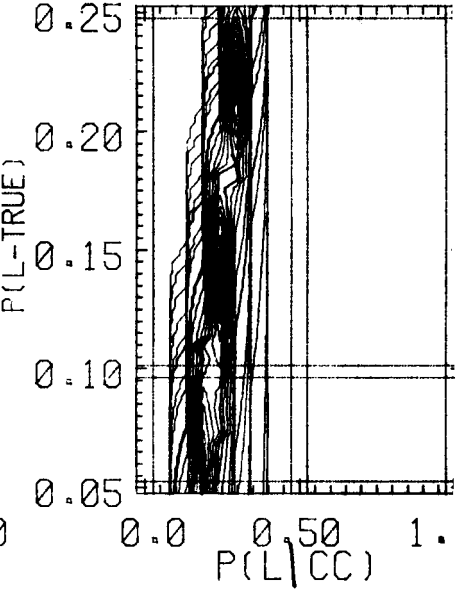
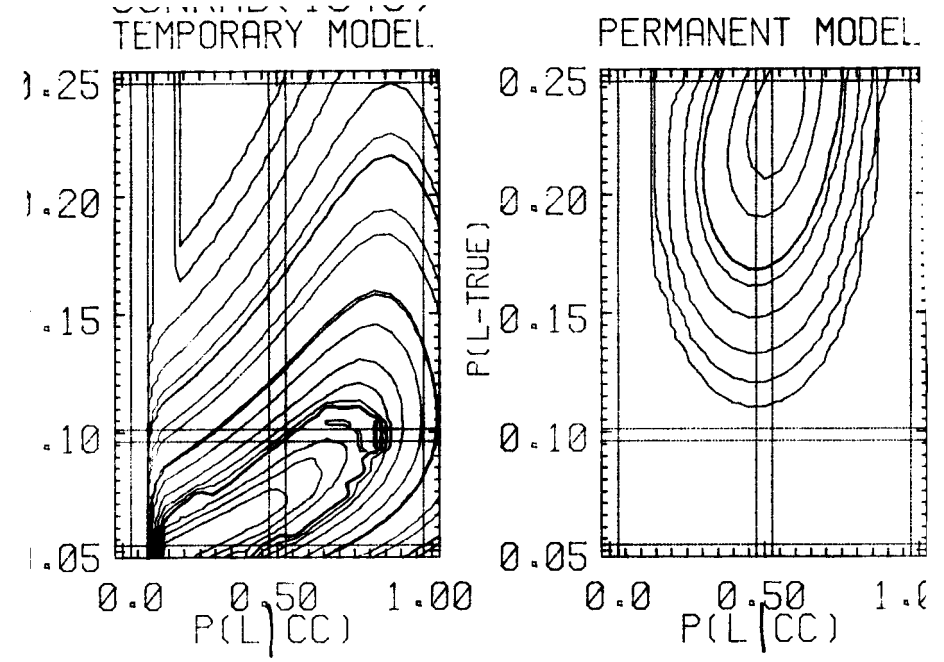
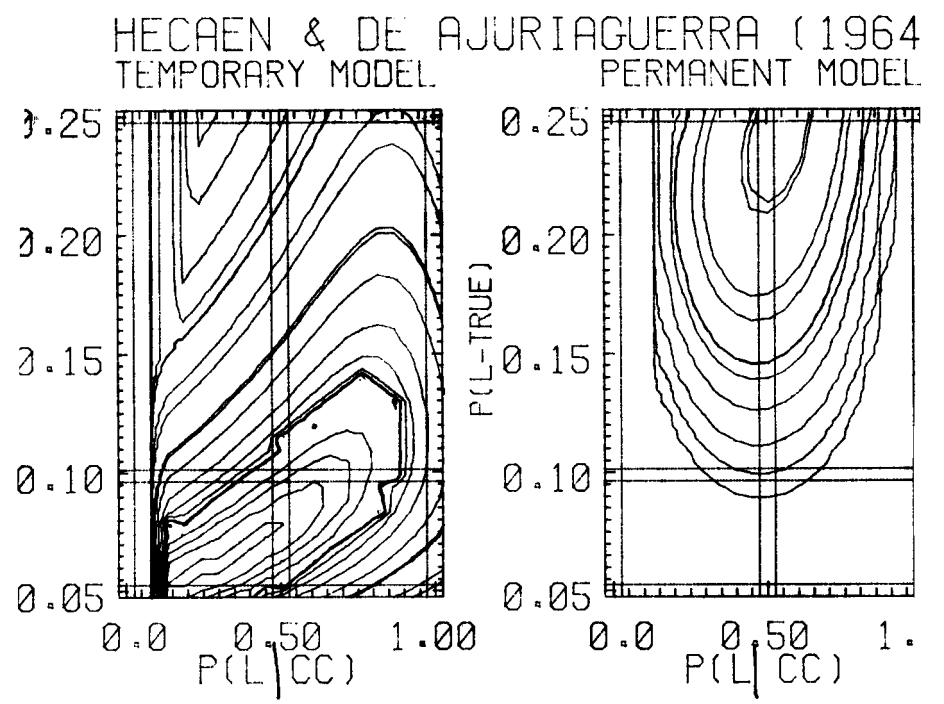


Figure 8.13 As for Figure 8.5, except for the Trankell model (see also note to Figure 8.12).


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)

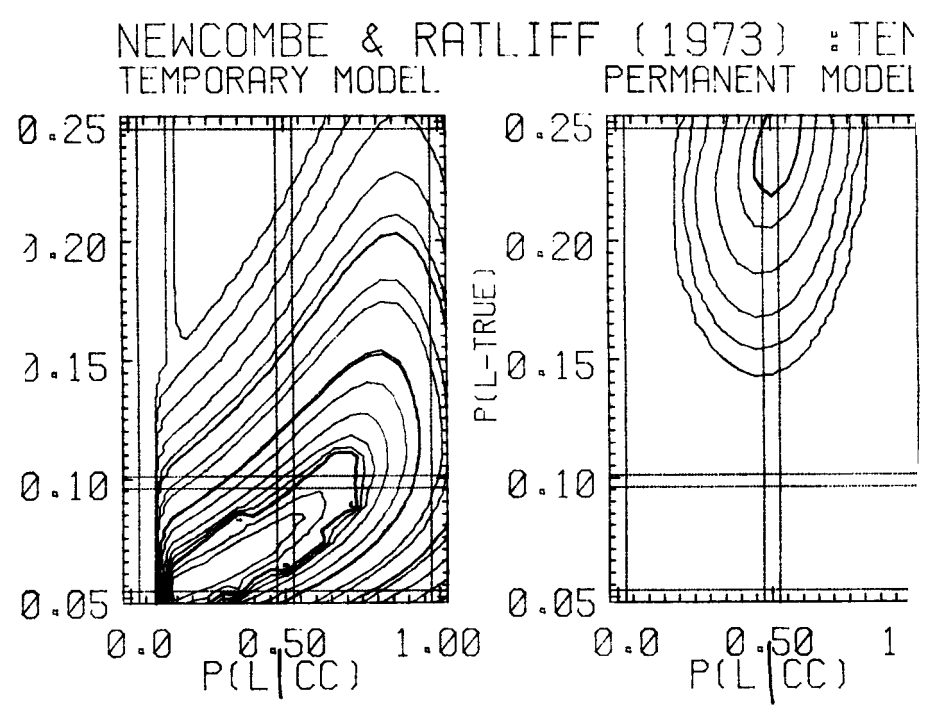
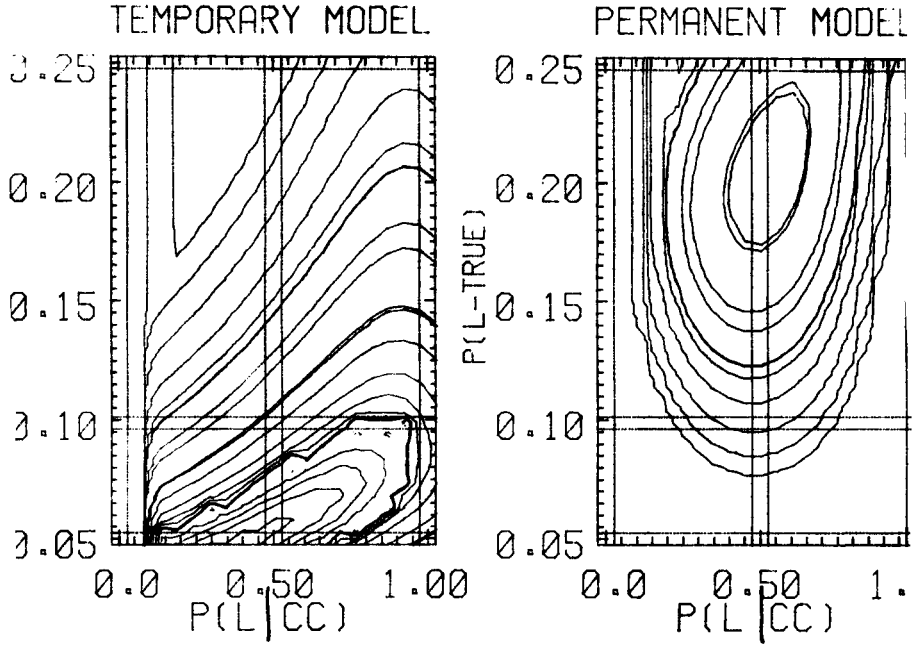
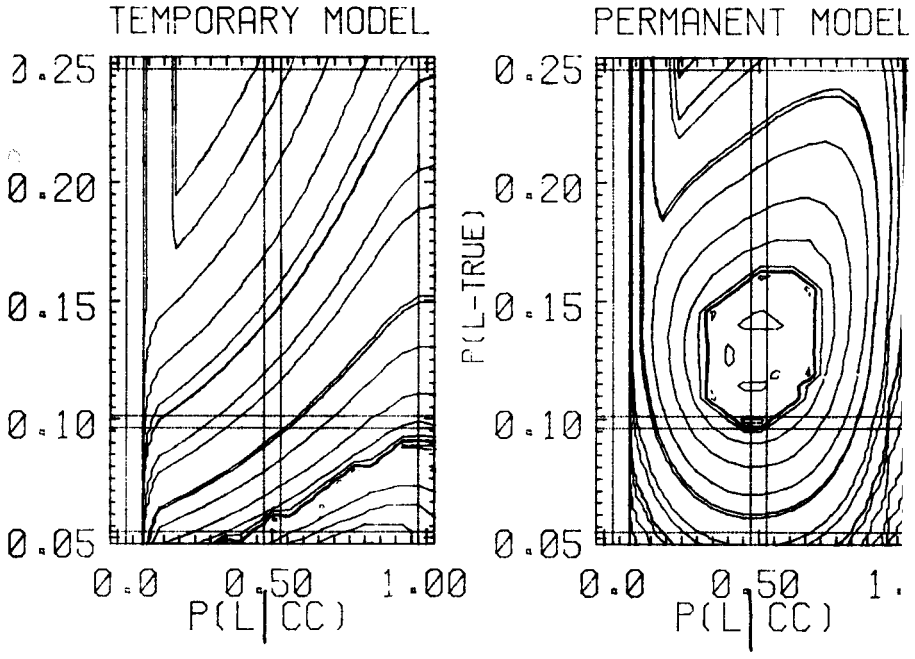

NEWCOMBE & RATLIFF (1973)
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|CC)

Figure 8.14 As for Figure 8.6 except for the Trankell model (see also note to Figure 8.12).



BINGLEY(1958)



PENFIELD & ROBERTS (1959)

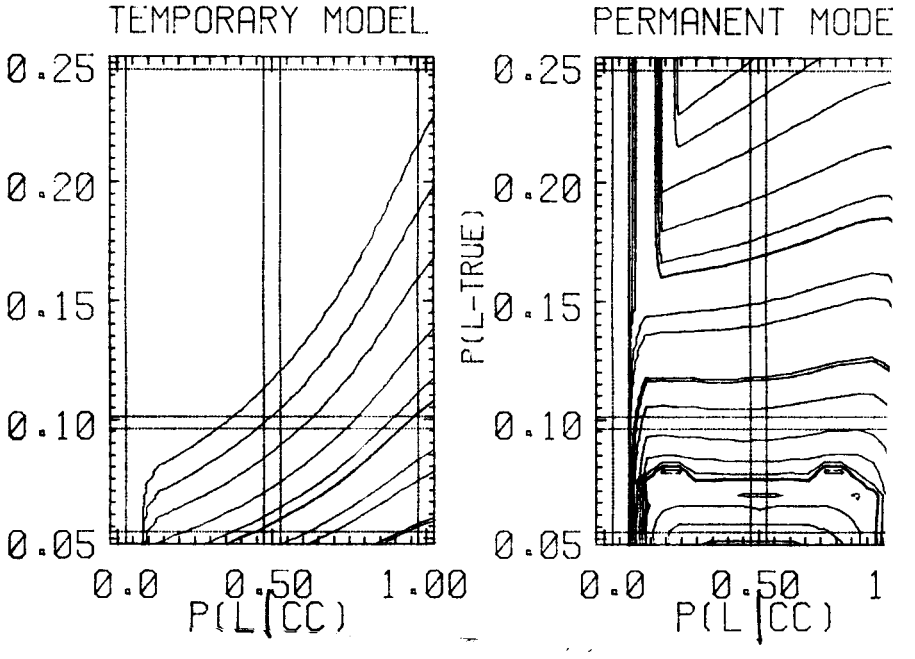
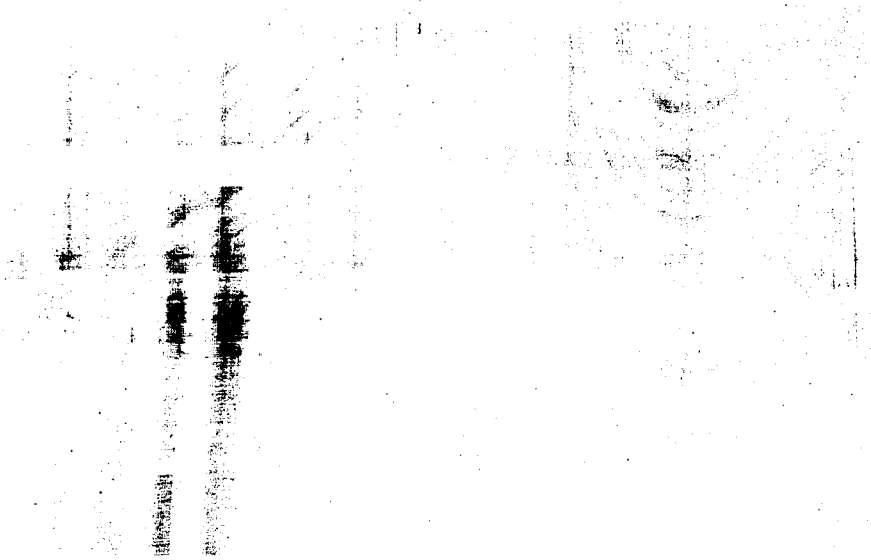
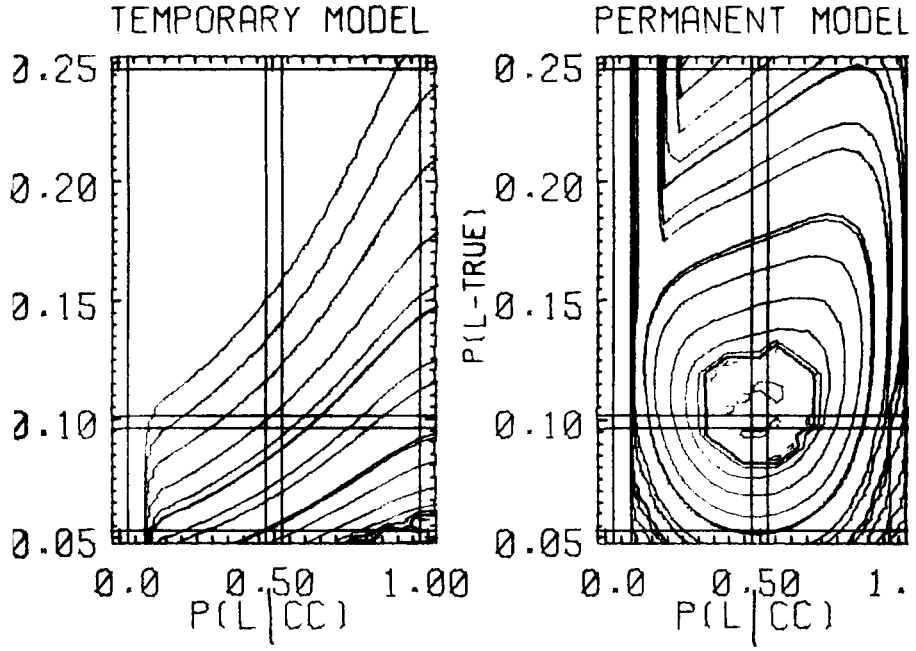
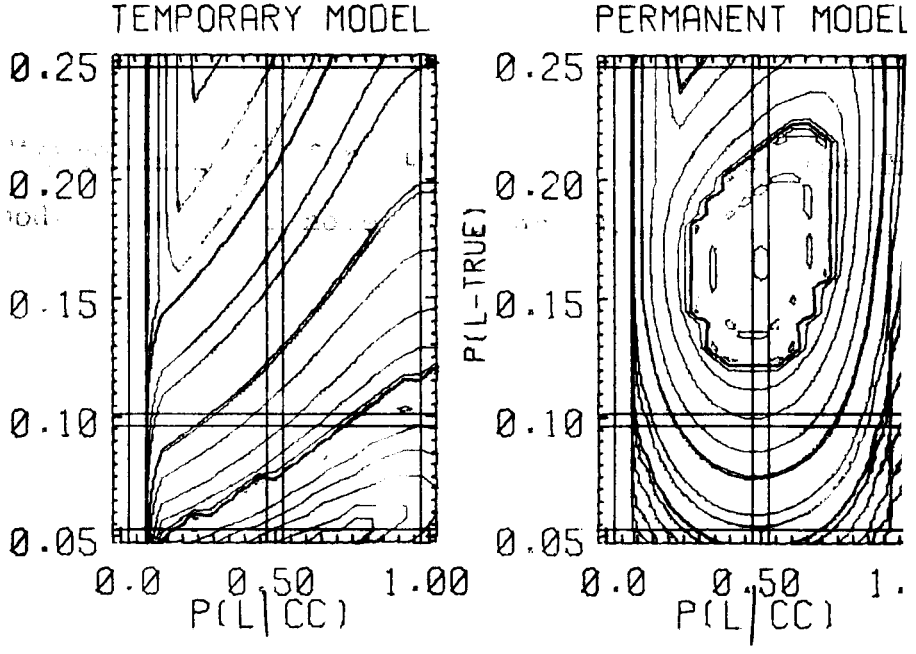


Figure 8.15 As for Figure 8,7, except for the Trankell model (see also note to Figure 8.12).





NAUMANN(1955): MENINGIOMATA



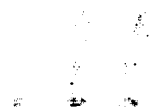
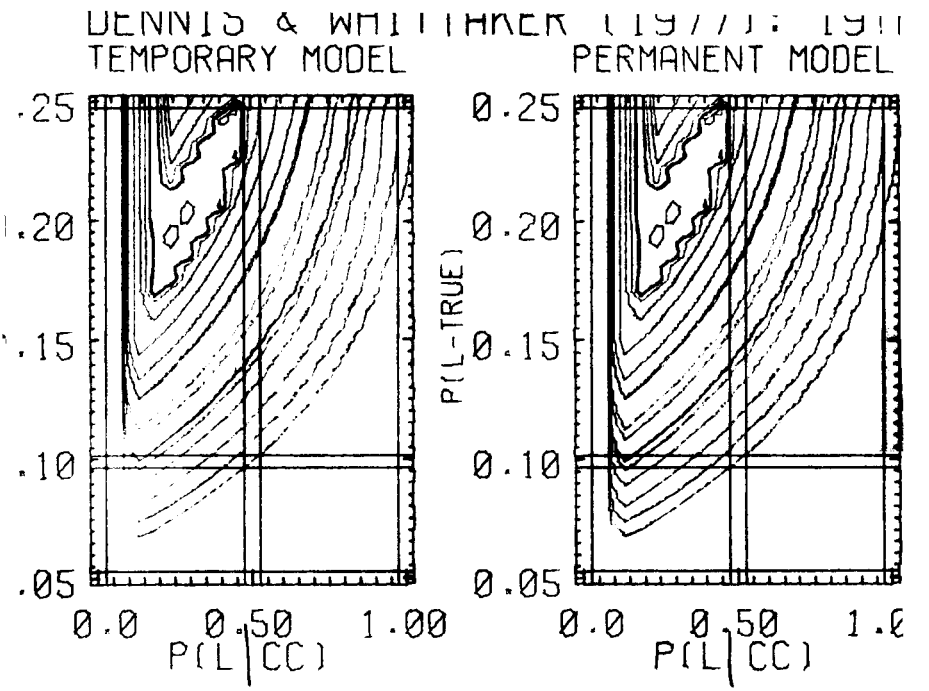


Figure 8.16 As for Figure 8.8 except for the Trankell model (see also note to Figure 8.12).


DENNIS & WHITTAKER (1977): 19
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
P(L|CC)

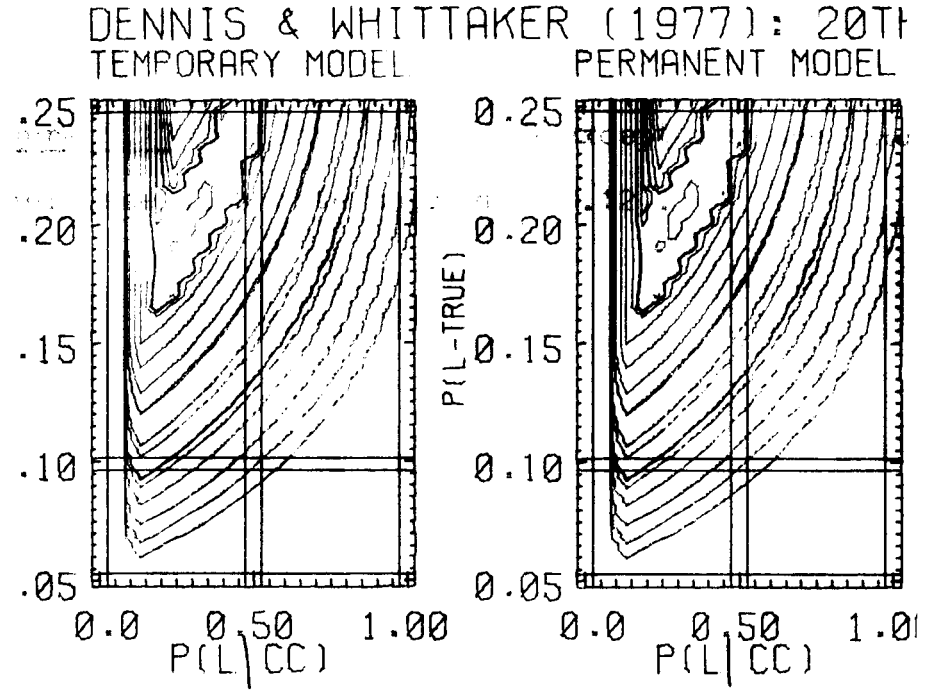
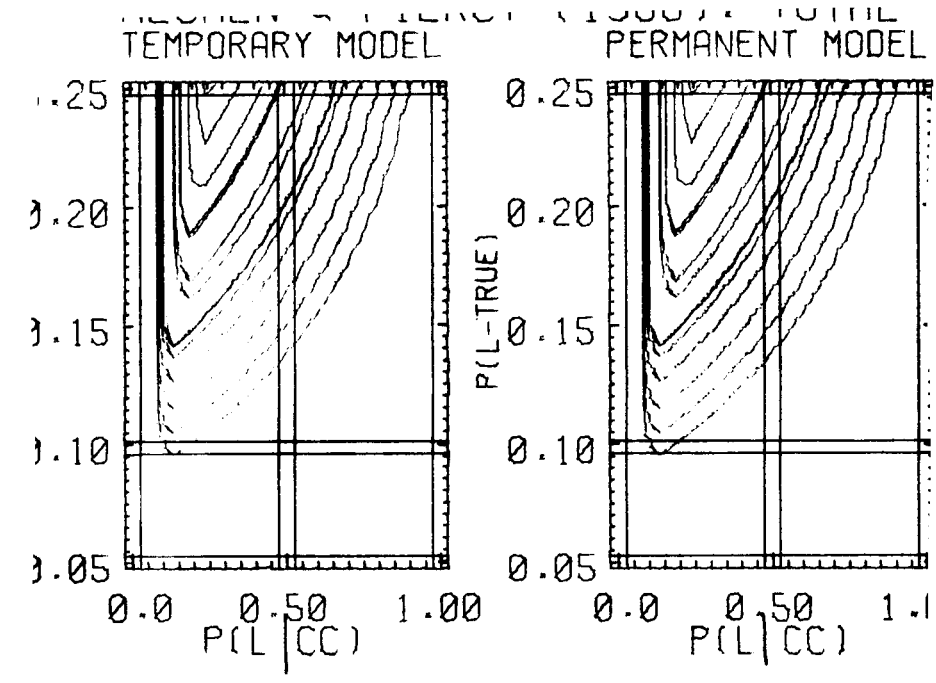
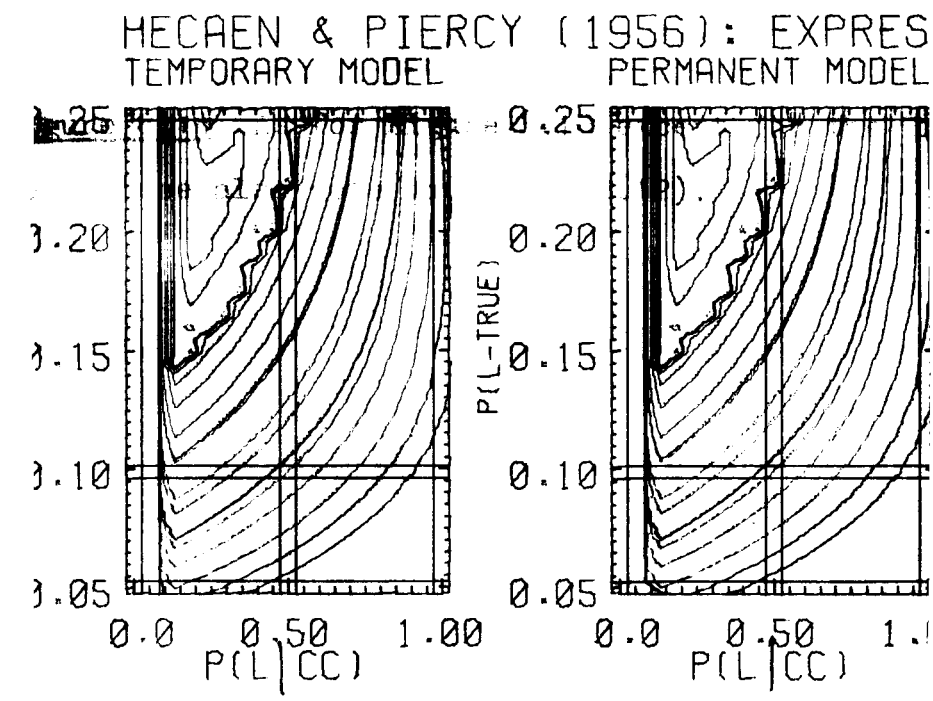

DENNIS & WHITTAKER (1977): 20TH
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
P(L|CC)

Figure 8.17 As for Figure 8.9 except for the Trankell model (see also note to Figure 8.12).


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 & PIERCY (1956): EXPRES
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
P(L|CC)

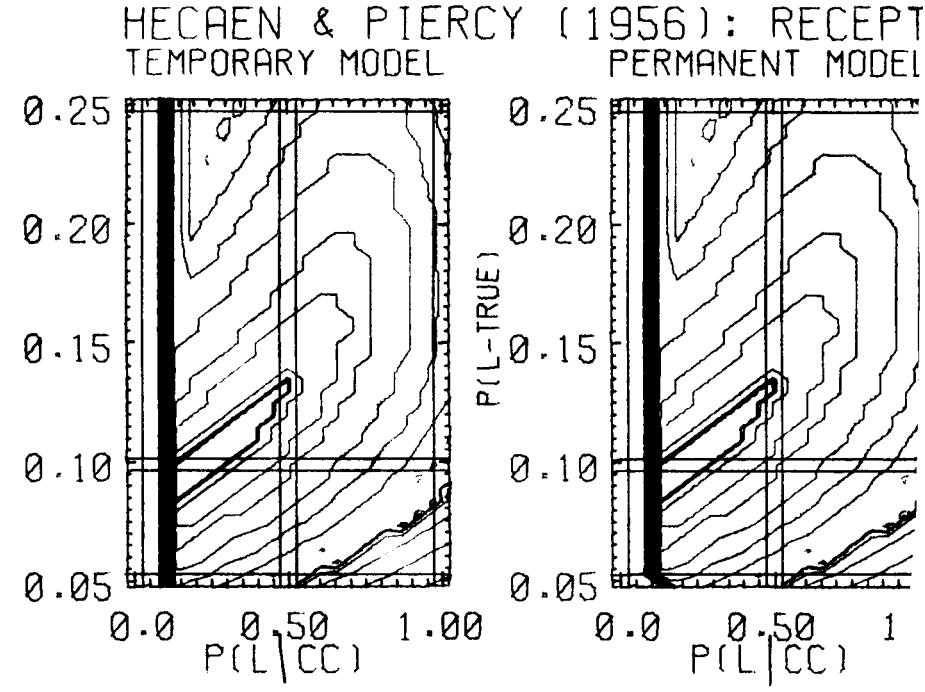
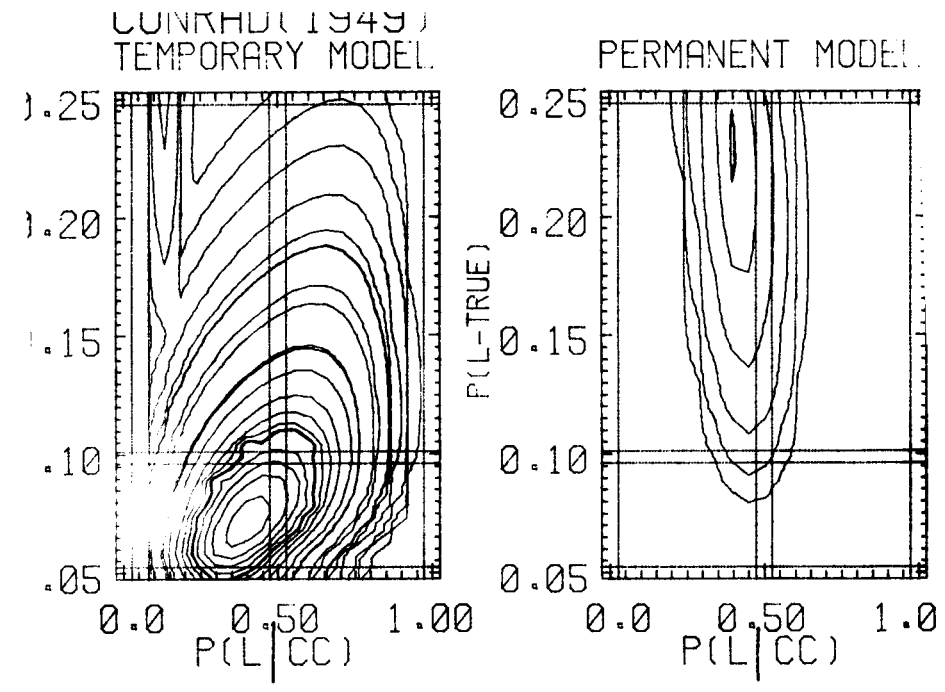
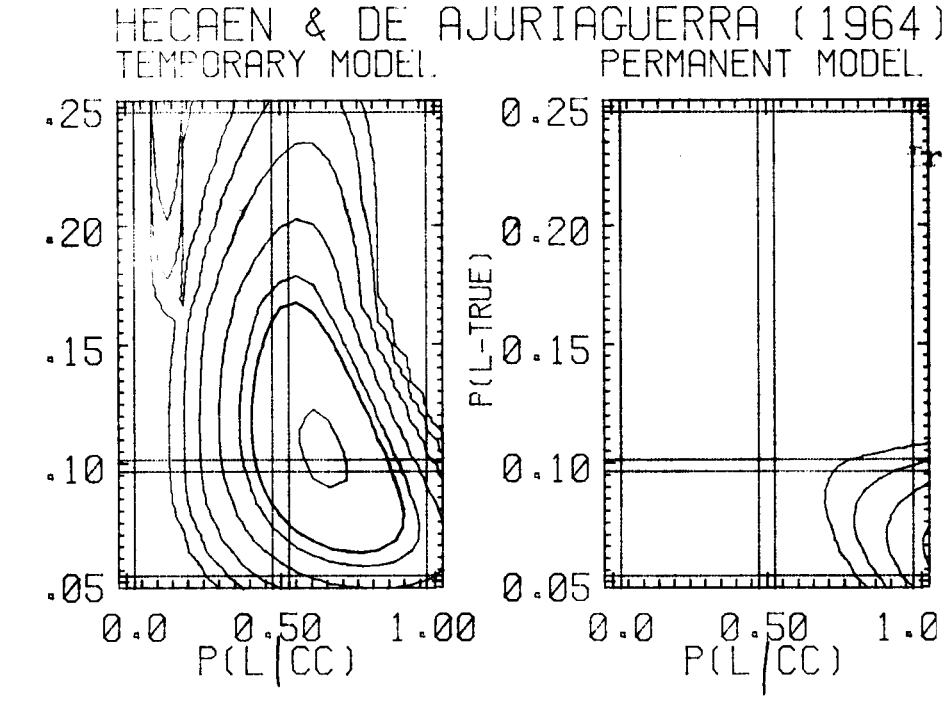

HECAEN & PIERCY (1956): RECEPT
TEMPORARY MODEL
PERMANENT MODEL
P(L-TRUE)
0.25
0.20
0.15
0.10
0.05
0.0
0.50
1.00
P(L|CC)

Figure 8.18 As for Figure 8.10 except for the Trankell model (see also note to Figure 8.12).


CONRAD (1949)
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
1.0
P(L|CC)


HECAEN & DE AJURIAGUERRA (1964)
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
1.0
P(L|CC)

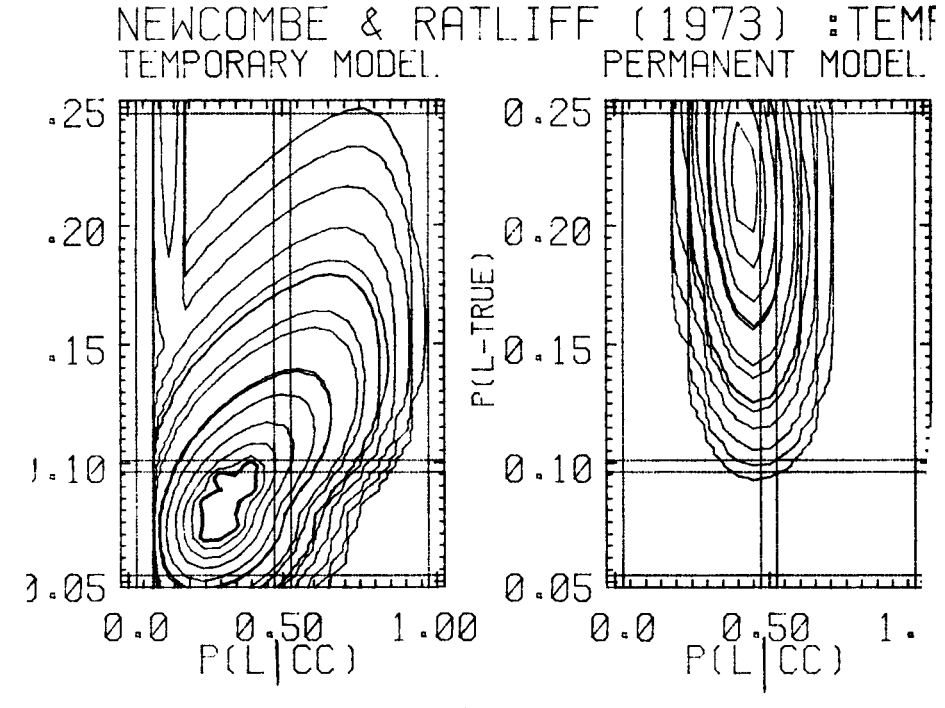
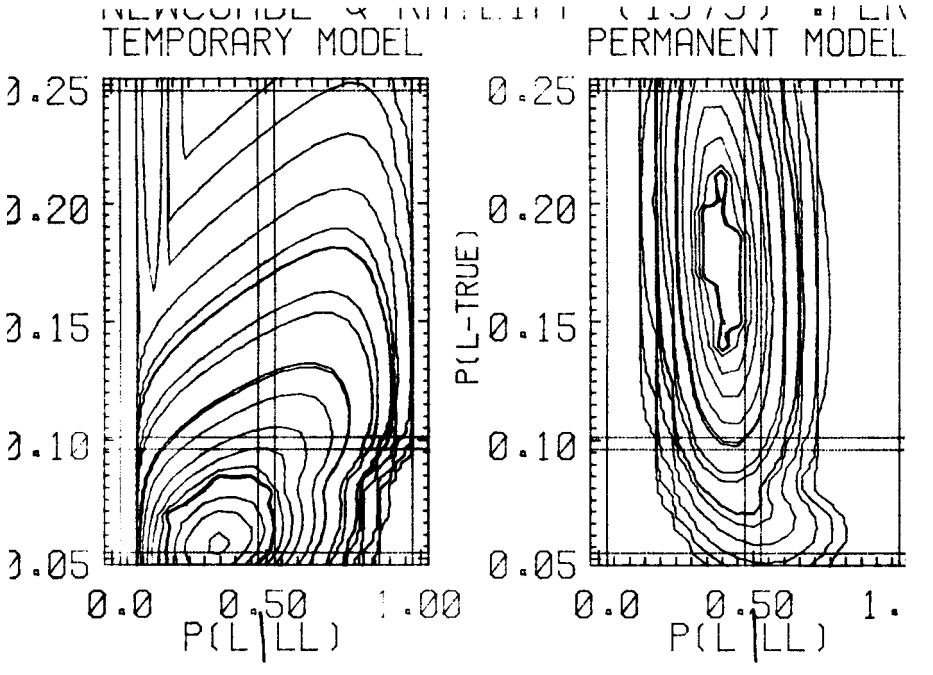
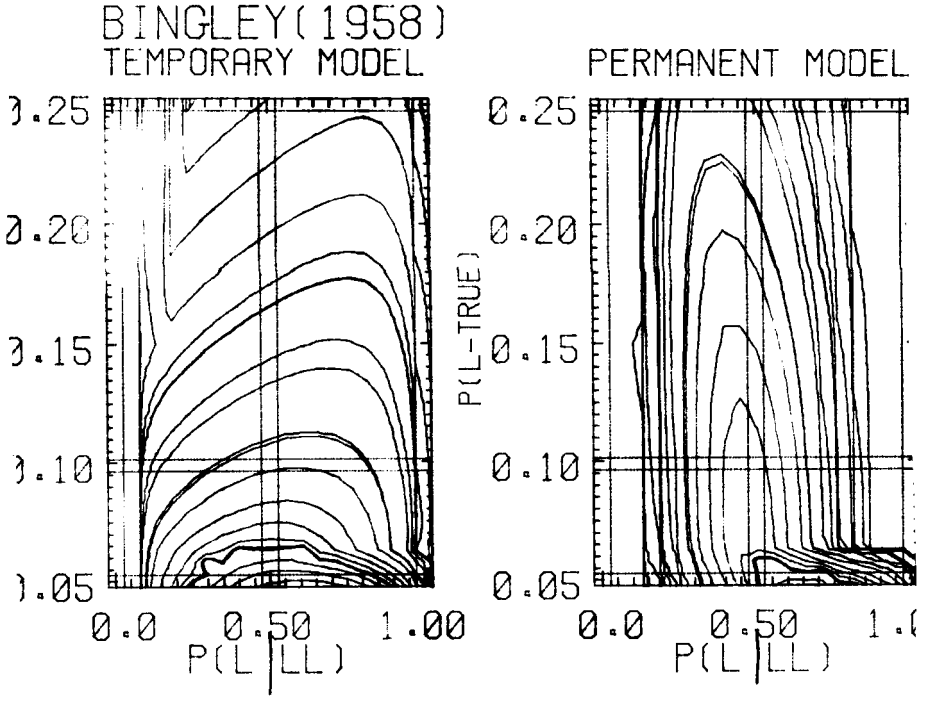
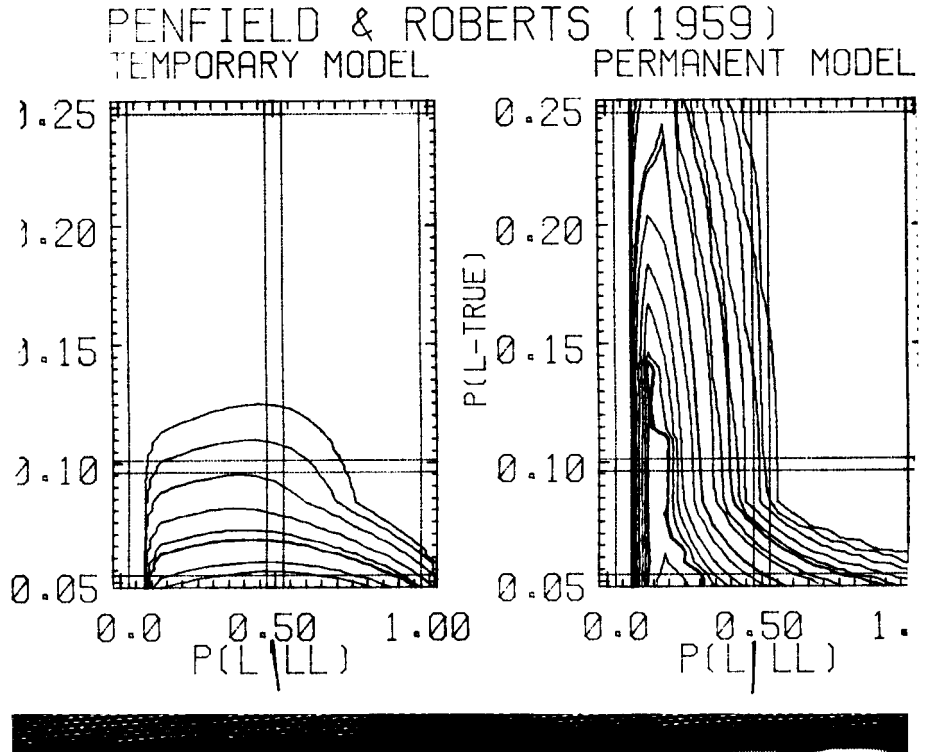

NEWCOMBE & RATLIFF (1973) :TEMP
TEMPORARY MODEL
PERMANENT MODEL
.25
.20
.15
.10
.05
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
1.
P(L|CC)

Figure 8.19 As for Figure 8.11, except for the Trandell model (see also note to Figure 8.12).


TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
P(L|LL)


BINGLEY(1958)
TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
P(L|LL)


PENFIELD & ROBERTS (1959)
TEMPORARY MODEL
PERMANENT MODEL
0.25
0.20
0.15
0.10
0.05
P(L-TRUE)
0.0
0.50
1.00
P(L|LL)