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.