GRAPPLING WITH THE HYDRA

Review of *Handedness and Brain Asymmetry: The Right Shift Theory* by Marian Annett, ISBN 1-84169-104-6, Hove: Psychology Press, 2002. 416 pages, Price: U.S.$ 80.00, U.K. £49.95.

There are times when I seem to have spent my entire academic life grappling with Annett's Right Shift theory. Rather like Heracles fighting the Hydra, just as one head is chopped off, so another appears, so that every year or two the theory seems to mutate and a half dozen new heads to spring forth. Although mutation might imply evolution, evolution would probably be the wrong word, for the theory is essentially creationist, change resulting only from some insight on the part of Dr Annett. And like any fundamentalist theory, there is no adaptation to the comments and criticisms of others (and least of all, to my own criticisms, which throughout this book seem to be described as not just wrong, but totally wrong, and invariably misconceived or due to a complete misunderstanding). There is thus an almost Mosaic certainty, further truths being revealed in the patterns of new data, which only become apparent to the theory's only begetter. Of course none of that is unusual or even undesirable for scientific theories – as Imre Lakatos said long ago, theories need those who fight for them, guard them, explore them and defend them when they are young and vulnerable to premature rejection. Having said that, arguing for a model and ignoring the prior work of other academics is another matter. In an academic monograph it is conventional to acknowledge prior workers in the field – and I confess to feeling somewhat miffed that neither the Alexander and Annett model of aphasia (Annett and Alexander, 1996), nor the description in chapter 4, makes any mention of my own earlier model of multiple cerebral dominances, which accounts in a formally equivalent way for the different patterns of acute and chronic aphasia (McManus, 1979; McManus, 1985).

For those who have read *Left, Right, Hand and Brain: the Right Shift Theory* (1985) the present book will provide a strong sense of *déjà vu.* Once again every significant step in the development of the theory is unveiled in the order it happened, including not only the correct steps but also the incorrect ones (and it is difficult to see why, for instance, an appendix should be devoted to the calculations for the dominant version of the model for handedness when even the author has long accepted that the additive version is better). Throughout there is a rather blinkered view which sees only Annett's view of handedness to be the correct one, and to believe that the world wishes to know every detail of its creation. It is a shame. Annett has been a creative researcher, who has measured in much detail many aspects of handedness and its relationship to other aspects of psychological functioning, and on many occasions she has been right in her approach and has had important insights.

None of that of course means that I think the Right Shift theory is correct, at least in many of its details. It does though have a large and important truth lurking at its centre – that randomness is a key part of the biological basis of the genetics of lateralisation. Around that though is a mass of difficulties which in the end make it difficult for me to accept the theory in its totality. Some of the problem lies in the continual temptation to conflate data, modelling and prediction, as for instance in the relatively new aspect of the model conerning the relationship between handedness and footedness.

Although Annett goes to great lengths in chapter 16 to criticise the genetic model which I first presented in 1979, the similarities of her model and my own are far greater than the differences. Essentially my model, like Annett's, proposes that there are two alleles (D and C in mine, RS+ and RS– in hers), and that these result in different proportions of left-handers in the three genotypes (0%, 25% and 50% in my own model for DD, DC and CC), and not dissimilar proportions in the RS shift model (1.4%, 13.6% and 50% for RS +/+, RS+/– and RS –/– when the threshold is at zero). Annett's model and the original version of my own model both argued that there is a fixed gene frequency (43% for RS- in the case of Annett, and 15.5% for C in my own case). However since data sets invariably differ in their actual proportion of left-handers (for a host of possible reasons), both models also assumed that some phenotypic right-handers manifest as left-handers or vice-versa. My own model was explicit about this process (and it used the same approach as is conventional for similar corrections in signal detection theory in psychometrics). Annett is scathing about such corrections (pp. 205-206), but I think that reflection will show that her 'threshold', which is adjusted anew for every fitting of a new data set, has an equivalent effect.

Although the McManus and the Annett models both use almost equivalent adjustments to take into account differing incidences of left-handedness between studies, the justifications for that are very different. Annett is compelled to adjust her threshold because, as becomes very clear in this book, the one thing that has not changed since the

RS theory was first published is the assumption that the RS+ gene is primarily for cerebral dominance, and that right-hemisphere language dominant individuals can only be of the RS –/– genotype. (An awkwardness of the theory which never seems to resolve is that while RS+ is dominant for cerebral dominance it is additive for handedness, with the result that although RS +/– heterozygotes, like RS+/+ homozygotes, are *all* left language dominant, a far higher proportion of these heterozygotes will be left-handed than is the case for the RS+/+ homozygotes). Annett presents data from a number of studies of aphasia after unilateral strokes and concludes, quite reasonably, that 9.27% of aphasics have right-sided lesions. It is then a straightforward calculation that 18.54% of the population must be RS –/–, and hence about 18.54% = 43.06% of the gene-pool is RS– and 56.94% is RS+. There is nothing wrong with doing that except that it means the RS– model can only be fitted to handedness data *if data are also available on language dominance.* That will later be seen to be problematic.

The McManus model did not have any grand reasons for assuming the same gene frequency across all studies except that it made life much simpler, particularly as at that time there were few strong reasons for believing the gene frequency may vary between populations, cross-culturally or historically. It was also somewhat reassuring, as I stated in my 1985 paper, that Annett had made precisely the same assumption, albeit for different reasons. I think I can now criticise Annett from stronger grounds because I think we were both wrong in making that assumption of a fixed gene frequency.

It may have simplified things to believe that gene frequencies would be similar at all times and places, but it probably over-simplified and mislead. It was the cross-cultural data of Phil Bryden and Maharaj Singh from Canada and India that first convinced me that the incidence of left-handedness varies in real terms between those two countries (Singh and Bryden, 1994). More crucially, Phil and I also realised that by looking at data within families one can decide if the difference in incidence between the two countries is the result of cultural pressures alone or is due to differences in gene frequency. To put it simply, if the lower incidence of left-handedness in India were due to cultural pressures, then handedness would run *less strongly in families* than in Canada, whereas if it were due to a lower gene frequency then handedness would run *more strongly in families* (Bryden et al., 1997). In fact it runs more strongly in families, as we have also subsequently found to be the case in the United Arab Emirates and Japan, and other workers have found in two African populations. Subsequently we also found that precisely the same pattern applies to secular changes in the rate of handedness in the west; low rates of left-handedness in early twentieth century studies of handedness are also associated with handedness running *more* strongly in families, meaning the difference is due to a lower gene frequency (McManus, 2002). The original McManus model of 1979/1985 is therefore wrong. One does not need to adjust for differences in the incidence of left-handedness, but instead take them at face value – they mean some populations have fewer of the genes that make people left-handed.

Where though does that leave the Annett model? In principle similar arguments apply to the Annett model for explaining how handedness runs in families, and it would be interesting to see how well the Annett model can cope with the data from India and elsewhere. My intuition is that it will only work if Annett is willing to alter the frequency of the RS- gene. That though may be a theoretical bridge too far. There is also the problem, albeit a self-imposed one for Annett, that given the way the model works it is not possible to fit the model to data from India or elsewhere unless one also knows for such populations the proportion of cases of aphasia which are due to unilateral right-sided damage – for only thus in Annett's schema does one know the gene frequency. Without such information the Annett model of handedness has two free parameters which are utterly confounded mathematically – the gene frequency and the threshold. Worse still, if one assumes without evidence that the gene frequency is the same as in the West then the prediction has also to be that cultural pressure explains the lower rate of handedness in India and, as for the McManus model, handedness should run *less strongly* in families, which is not the case.

Annett's right-shift model is first and foremost a model of how handedness runs in families. Whatever the elaborate super-structure, described exhaustively and exhaustingly in this book – fine –tuning parameters to explain small differences in twins, or in males and females, of looking for heterozygote advantage in relation to intellectual ability, of trying to explain autism and schizophrenia, of considering the evolution of the RS+ gene and theorising that it is a gene for cerebral dominance and not for handedness *per se* – all of the hard tests of the model come down to the question of whether the model explains how handedness runs in families. Like my own original model, it does indeed do so to a reasonable approximation in modern Western data. However, just as I know my own original model failed on the cross-cultural and historical data, so the Right Shift model will inevitably encounter problems with the much harder problem of cross-cultural data, because of the extremely interesting possibility that gene frequencies differ geographically and historically. It will be interesting to see what

happens, although I have a sneaking suspicion either that my comments will once again be ignored, or that the Hydra will grow yet another head ...

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