16 315-344.

JOURNAL OF COMMUNICATION DISORDERS 00 (1983), 0–00

PATHOLOGIC LEFT-HANDEDNESS: DOES IT EXIST?

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

> . . . the study of the thing caused must precede the study of the cause of the thing. . .[1]

Introduction

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

Definition and a Challenge

Before commencing this review I feel obliged to state what I understand by the concept of pathologic left-handedness. Pathologic left-handedness could be agreed to occur in a patient *previously of known right-handedness* who, as a result of some injury to the brain or similar cause, changes from a *preferential* use of the right hand to a *preferential* use of the left hand. The concept of hand *dominance* inevitably implies a relative preference between two otherwise similar organs or systems. One cannot have dominance if there is only one organ, hence, it is clearly

[1]From Hughlings Jackson, quoted in Wilson, 1908, p. 211.

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0021-9924/83/$03.00

trivial or absurd to claim that a person is pathologically left-handed if, for some reason, the right hand has been severed from the body. Similarly, it is trivial to describe as pathologic left-handedness a case in which a person has suffered damage to one-half of the brain; the shift to left-hand usage must be preferential, not inevitable, and must occur in the absence of signs of a hemiplegia, hemiparesis, lower-motor-neuron weakness or other defect of the limb, or lack of control due to tremor, athetosis, ataxia, etc. The shift in hand preference in pathologic left-handedness also implies that the *previous* preference had been ascertained; one cannot claim that a change has occurred unless one knows the nature of the earlier state. This might seem to impose unacceptable limits in the case of very early brain damage, for it would be difficult if not impossible to assess the handedness of an unborn fetus. This problem may be readily and satisfactorily surmounted by the use of a control group; the incidence of left-handedness must be shown to be significantly higher in a pathologic group than in an appropriate control group (both groups being assessed blindly, etc.). While one might not be able to know with certainty the handedness that an individual would have shown if a particular brain insult had not occurred, speculation on the various probabilities are possible.

Defining pathologic left-handedness as mentioned, it is suggested that there is not a single case in the literature that can be regarded as acceptable proof of the existence of the phenomenon.

Aphasia and Left-Handedness

To understand the origin of the concept of pathologic left-handedness we must return to the period of the first realization of the asymmetry of the brain. In 1861, Broca described a patient in whom a lesion of the left hemisphere had resulted in aphasia; the lesion was confirmed postmortem (for a translation, see Rottenberg and Hochberg, 1977). At that time, the particular importance of the *left* hemisphere does not seem to have been realized.[2] The first mention of the predominance of lesions of the left side is in Broca (1863), where it is noted in passing. Broca's best description of "la singulière prédilection . . . pour l'hémisphère gauche," was published in 1865. For a good account of the early history of

[2]And this was not particularly unusual. As Benton and Joynt (1960) pointed out:

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

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

aphasia, see Ombredane (1951). Whether or *not* Broca was truly anticipated by M. Dax, in an obscure paper of 1836, is still controversial (see Critchley, 1964, for a discussion).

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

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

A few patients, however, had been described in whom aphasia was associated with a *left* hemiplegia (i.e., right hemisphere damage). One reaction to such cases was to attempt to explain them away and to simultaneously explain the overall predominance of left-sided lesions in aphasia, by suggesting that left-sided brain lesions were more common than right-sided brain lesions (e.g., due to emboli being more likely to enter the left carotid artery than the right artery; Bateman, 1870). Such a possibility was rapidly eliminated by the simple statistical analysis of Ogle (1867); while 90% of aphasics had a right hemiplegia, only 55% of hemiplegias affected the right side (and, hence, were due to defects in the circulation of the left side of the brain.)[3]

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[3]Bateman (1865) put forward a further argument against those physiologists who claimed:

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

Bateman said in reply:

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

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

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

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

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

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

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

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

Further cases were produced that supported Broca's symmetric conception of

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

aphasia and handedness. Wadham (1869) described an 18-year-old youth with an aphasia and a left hemiplegia. This youth:

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

Wadham suggests that this case might be explained:

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

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

Exceptions to this revised rule, of the language center being contralateral to the dominant hand were, however, occurring. Ogle (1871) discussed such cases and introduced a new conept that was to have disastrous consequences, eventually resulting, and inexorably, in nonfalsifiable hypotheses.

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

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

> Even should such a case occur it would not be incompatible with the views now expressed. For, as I have already stated, there are probably persons with a natural left-handed tendency in whom the bias is so feeble that its external manifestations become completely masked by education. In such a person aphasia might occur with left hemiplegia, and such a case would thus appear erroneously to stand in contradiction with Dax's Law.
>
> Ogle, 1871[4]

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

Despite Ogle's stricture ("*even* should such a case occur"), cases of crossed aphasia (i.e., dominant hemisphere for speech ipsilateral to dominant hand; the

[4]Critchley (1964) suggested that the term "Dax's Law" was first used by Grasset, in 1873; the present usage clearly predates that essay. In the present paper, term will be used in the sense of *all* right-handers having left hemisphere speech, and *all* left-handers having right hemisphere speech.

term apparently was first used by Bramwell, 1899) continued to appear, and presented theoretical problems. In his Gulstonian Lecture of 1878, Ferrier could only point out that:

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

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

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

> It is admitted that there are cases of left hemiplegia with aphasia in persons who are not right-handed. Besides granting fully the significance of the fact that in the vastly greater number of cases loss of speech is caused by disease in the left half of the brain, the thing of infinitely greater significance is that damage in but *one* half can produce speechlessness.
>
> Jackson, 1880

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

> The explanation of the anomaly may, I think be found, in the fact that, though left-handed for other actions [the patient] wrote with his right hand, and was in the habit of writing much . . . The habit of writing much seems to be the only assignable cause for this reversal.
>
> Paget, 1877

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

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

In the last 2 years of the 19th century, two works appeared, which introduced a series of ideas that would modify approaches to cerebral dominance. Both authors assumed at least partial veracity of Dax's Law[5]

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[5]At this point, I cannot resist quoting from the writings of at least one man who believed in the strict truth of Dax's Law. Collins (1898), in an essay awarded the "Alvarenga Prize of the College of Physicians of Philadelphia, 1897," writes thus:

> Every one knows that the faculty of speech is maintained by the left hemisphere of the brain, and by the right hemisphere in those who are left-handed. And this for the same genetic reason that the organism is right-handed in the majority of people and left-handed in the few:

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

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

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

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

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

The Concept of Hand Usage Modifying Speech Laterality

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

> It would thus seem that the predominant use of the right hand or the left hand carries with it, as one of its associated effects, the leading activity in the production

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when the organism is left-handed, then the right hemisphere contains the zone of language. It would seem that this is one of the facts of physiology which is indisputable; nevertheless, every now and then some one brings forward evidence purporting to deny it. One of the most recent purveyors of such evidence is Moltshanow, of Moscow, . . . [he describes a case reported by Moltschanow]. The writer says that this case speaks unequivocally against what he is pleased to call Broca's dictum concerning the location of the speech area. He says further that other authors have probably had similar cases, for in looking over the literature of aphasia he had noted that it is often times impossible to determine from the reported cases whether the patient was right-handed or left. This point the writer need have no difficulty in determining in the future. If the patient whose history he reads has a left hemiplegia with aphasia, that patient is a right-handed man, and *vice versa*. It is almost incredible that physicians will attempt to convince themselves that evidence of this kind can have the slightest effect in overthrowing such an invariable rule as the one relating to the location of the speech area, just cited. An attempt to discredit a law so firmly established as this by the citation of testimony of a woman concerning the right-handedness or left-handedness of her husband [the source of the handedness information in Moltschanow's case] is like trying to trip up Atlas by putting a microbe to obstruct his path.

several decades, admittedly under pressure. Thompson (1921) felt that b and c were either each zero or each very small. Even in 1936 Chesher felt that the normal form was for a and d to equal 100% together. Although Bramwell had begun to realize that c was not equal to 0%, he still suspected that c < d. Conrad (1949) realized that for left-handers the true relation might actually be that c>d; this was supported by Zangwill (1960) and by Brain (1961). Espir and Rose (1970), however, felt that c = d.

The zero nature of b continued for much longer, being clearly held by Elliott, Hughes, and Turner (1952), and still held by Zangwill (1960), although doubt was beginning to develop. Russell and Espir (1961) concluded that:

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

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

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

Milner, Branch, and Rasmussen (1964) presented empirical evidence for b not being zero and accepted that "the overwhelming predominance of left-sided speech representation in right-handed persons of right-handed ancestry rests unchallenged" (i.e., a > b and b = 0). Clarke and Zangwill (1965) stated:

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

Weinstein (1978) claimed that b might be as high as 10% of a + b (although he also claimed that for left-handers, c was only 30% of c + d.) Zangwill (1979) estimated that b was between 1.5% and 2% of a + b.

If we now return to our four explanatory concepts, we find that, certainly at the time of their introduction, they were not supported by adequate empirical evidence; they were *post hoc* postulates to maintain Dax's Law, or variants upon it.[10] It should be clear by now that the non-zero nature of b and c cannot support the

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

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

existence of all four of the original speculations; further evidence is required or else some, if not all, of them must flounder.

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

We may find this process of "truth by definition" occurring in the large series of patients described by Hécaen and de Ajuriaguerra (1964), and analzyed in some detail by Annett (1975), who somewhat discretely suggests that:

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

To put it less discreetly, Hécaen and de Ajuriaguerra were classifying a patient's handedness *after* they had known whether he was dysphasic and whether he had a right- or left-sided lesion. This is the error exemplified by Girard (1952).

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

The confusion of data and hypothesis was recognized long ago by Moutier (1908), a pupil of that great iconoclast of aphasia, Pierre Marie.

> Chaque fois en effet qu'un fait contradictoire de lésion du centre de Broca sans aphémie a été observé, il s'est toujours trouvé quelque partisan de la localisation classique pour déclarer que le sujet atteint était un gaucheur méconnu, un ambidextre au besoin. Ou bien, on admet que l'aphémie a été fugitive, et que ce caractére transitoire des accidents est dû à une suppléance rapide de l'hémisphère gauche par l'hémisphère droit. Gaucherie, ambidextérité, suppléance, sont des théories que l'on ne saurait appliquer également à l'interprétation de tous les faits. La part de vérité pouvant exister ici est du reste des plus difficiles à déterminer; nous allons nous en rendre compte.
>
> Moutier, 1908, p. 115

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

Weisenberg and McBride (1935) had initiated this search for the true handedness:

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

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

> Thus it is possible to determine *subtle signs of left-handedness* [all emphases are in the original] in cases where gross observation gives no basis for such classification. . . Among the *morphological* signs of latent left handedness are a large left hand, a well developed venous system on the back of the left hand,[7] a wide finger nail on the fifth finger of the left hand, and highly developed expressive musculature on the right side of the face.[8]
>
> The number of *functional signs* of latent left handedness is considerably greater. [These include that] in more primitive functions. . . under conditions of high affect. . . latent left handedness may manifest itself, and the individual may switch to his left hand. Aside from such observation, a detailed interrogation [sic]. . . often reveals irregularities in the dominance of the right hand. . .
>
> Another technique for the discovery of latent left handedness involves *special tests*. Among these are such well known tests as *clasping the hands*[9] . . . *clapping movements*. . . *tonus tests*. . . [and] *sensory dominance*. . .
>
> These types of tests are not limited to functions involving the hand. The procedures include a determination of the *dominant eye*.
>
> These test permit us to detect latent signs of hemispheric dominance. . .
>
> The detection of latent left-handedness is not the only approach to a more precise characterisation of cerebral dominance. [He discusses the genetic literature]. . . We shall [thus] expect that, along with the overt forms of left-handedness, it should

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

[8]This suggestion might originate in the findings of "a friend of Dr Wilbur, of Syracuse, New York" (see Ireland, 1880).

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

> be possible to find *latent forms of left-handedness* which give no external signs of their presence. Such forms could be identified only by reference to the presence of overt left-handedness in other members of the family. . .

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

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

This cataloging of the stigmata of "latent left-handedness" reminds one of nothing more than that notorious index, the *Malleus Maleficarum*, where those diabolic signs ("witchmarks") necessary for the unequivocal detection of witches could be found. Parenthetically, it is ironic to note that after cataloging his signs of latent left-handedness, Luria puts in a footnote stating that, "A new and apparently effective method of detecting hemispheric dominance was introduced by. . . Wada." In many places, Luria's text seems only comprehensible if one infers some confusion and interplay between handedness and dominance, such that the author must have assumed the veracity of Dax's Law.

A Recapitulation

From the works of Bastian (1899) and Bramwell (1898) we may trace four concepts: hand usage modifying speech laterality, early brain damage modifying speech laterality and handedness, stock-brainedness, and latent left-handedness. These four concepts arose in response to a single problem, the presence of crossed aphasics. If the two types of handedness and the two sides of possible aphasia producing brain lesions are considered, a table can be constructed (ignoring the possibility of bilateral representation of language) as follows:

		Side of Aphasia-Producing Lesion	
		Left	Right
Handedness	Right	a %	b %
	Left	c %	d %

Let a, b, c, and d be the percentage occurrences of the four possible combinations. Broca's initial (implicit) assumption was that a + c = 100%. This was modified by cases of aphasia after right-sided lesions, in supposed left-handers; thus, Dax's Law is that a + d = 100%. The concept that both b and c were equal to 0% held for

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

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

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

The twin concepts of pathologic shift of language laterality and pathologic shift of handedness, either capable of occuring in the absence of further evidence, provided a theoretical carte blanche for the neurologist. Now, no case was beyond explanation, or incapable of fitting into a particular theoretical preconception, be it Dax's Law, or some modification of it. Thus, Kinnier Wilson (1921) wrote:

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

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

Stock-Brainedness

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

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

This idea was expanded by Foster Kennedy (1916) in the concept of "stock-brainedness." He cited a crossed aphasic described by Bramwell:

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

Once again, Kinnier Wilson (1926) finds the concept useful in explaining awkard cases:

> In other words, *ectopia* of the speech centres is a possibility in some right-handed members of a sinistral stock, and in some left-handed members of dextral stock.

> This is not mere speculation, but a hypothesis which satisfactorily accounts for not a few recorded instances of "crossed aphasia" and which receives support from the pathological side.

Latent Left-Handedness

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

> The behaviour of left-handed individuals is opposite to that described for dextrals. However there are a few poorly explained cases which, in spite of right-handedness, have right-sided lesions followed by aphasic defects. Lastly a few cases have been reported in which the pertinent foci are partly in one and partly in the other hemisphere. This phenomenon, probably related to ambidexterity, is explained by the conflict of a pre-disposition (e.g. to right-handedness) with individual (latent) left-brainedness.
>
> [Presumably here the pre-disposition is latent, and by latent is intended "manifest."]

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

Brain (1945) emphasized the distinction between natural and pathologic left-handedness:

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

There are now only two causes of true left-handedness mentioned; all others are, by implication, pathologic. Note also that there is actually no adequate evidence for the phenomenon of mirror-imaging, it also being simply a useful concept to explain away apparently awkward data (see McManus, 1980a). Brain continues, "*Natural* handedness is important for neurological diagnosis.") He then cites apparent cases of:

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

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

> of speech by the left or the right hemisphere respectively; and that we must consequently push our question further back, and inquire as to the causes that have led to this predominant use of the right hand.

Notice that now, unlike Broca's conception of the problem, the brain does not have primacy; the use of one particular hand *induces* the development of the contralateral cerebral hemisphere. This hypothesis is implicit in Moxon (1866) ("the brain of educated individuals is manifestly more unsymmetrical than the brains of uneducated individuals"), and possibly also in the case-history described by Jackson (1880b), and almost certainly in Paget (1887). The intellectual origin of the concept probably may be traced back to two cases reported in 1878 and 1880. Gowers (1878) described a case in which the left hand was congenitally absent and the contralateral parietal cortex apparently was atrophied and vestigial. His assistant at the postmortem was the young Victor Horsley, still an undergraduate. Bastian and Horsley (1880) reported an almost identical case, commenting only that, "It seems something more than can be accounted for by mere chance coincidence."

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

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

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

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

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

The importance of utilizing the left hand had been stressed earlier by the Ambidextral Culture Society, which was formed in 1905 by one John Jackson "for the promotion of education reform and two-handed training" (see Barsley, 1966, for an account).[6] By 1926, the esteemed neurologist Kinnier Wilson described the

[6]The utility of ambidexterity was not always accepted. Gould (1905), arguing from the same neurological premises as Coley, Jackson, et al., could only sympathize with the "pitiable victims!" of the Ambidextral Culture Society.

argument of Weber (1904) that it could only have been in the mid-19th century that Broca could make his discovery, since:

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

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

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

Weisenberg and McBride (1935) echoed the same suggestions:

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

Needles (1942) analyzed nine cases from the literature and concluded that in usage of the nonpreferred hand:

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

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

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

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

The Concept of Early Brain Damage Modifying Dominance

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

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

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

> (In effect, each time that a contradictory example is observed, of a lesion of Broca's area without aphasia, there is always found a supporter of classical localisation who declares that the affected patient was an unrecognised left-hander, if need be an ambidextral. Or instead, one suggests that the aphasia was fugitive, and that this transitory nature must be the result of a rapid substitution of the left hemisphere by the right hemisphere. Left-handedness, amibidexterity, plasticity, are theories that one does not manage to apply equally to the interpretation of all the facts. The element of truth which exists here is moreover very difficult to distinguish.

One does not have to look very far for the effects of this confusion of data and theory. Ettlinger, Jackson, and Zangwill's (1955) influential review of crossed aphasia in dextrals concluded that:

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

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

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

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

The effect of the received view upon the interpretation of data can be found in Archibald and Wepman (1968). Although evidence of language in the right hemisphere of eight right-handers was found, they were forced to conclude:

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

Inevitably, observations of patients are made in the presence of prior theories as to the nature of such observations; patients reported in the literature suffer even more from this deficit. Figure 1 shows the cumulative number of reported cases of left-handed patients with crossed and noncrossed aphasia (data from Goodglass and Quadfasel, 1954). Note that we are now fairly sure that crossed aphasia in left-handers is actually more common than noncrossed aphasia; it is only in the late 1940s, when such a hypothesis became more acceptable, that cases were reported. Prior to that, reported cases supported the prevailing theory that left-handers talked via the right-brain.

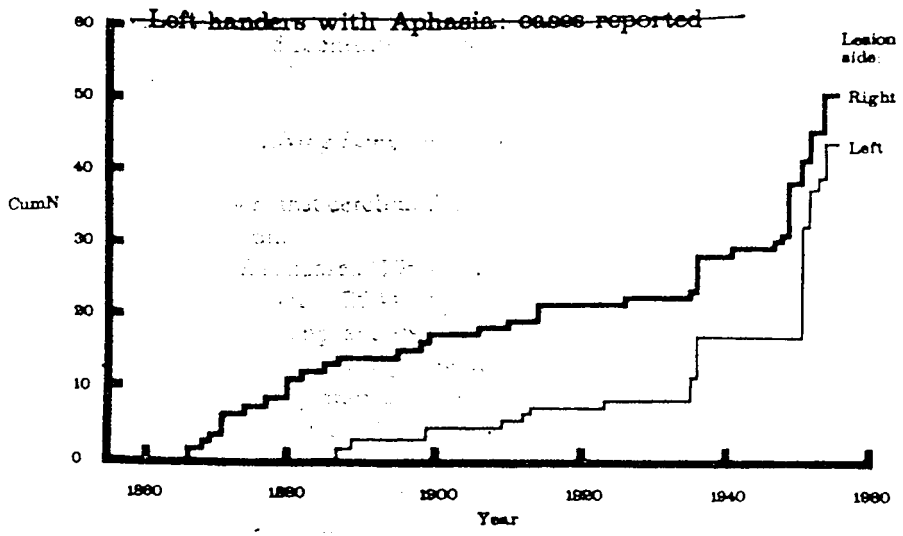

Figure 1: The cumulative number of cases reported of left-handers with either right- or left-sided lesions that produced aphasia, by date of publication. (Data derived from Goodglass and Quadfasel (1954).)

Evidence

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

The Modification of Language Laterality by Hand Usage

Even recent work has assumed that modification of language laterality by hand usage is a potent factor. Humphrey and Zangwill (1952), describing the case of a left-handed man with a right occipitoparietal injury, noted that although he was aphasic, the patient did not suffer from agraphia. They comment:

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

Zangwill (1955) commented that:

> In "congenital" sinistrals. . . the hemisphere dominant for language is typically the right. In these cases, education of the right hand for skilled activity, more

> particularly writing, may lead to some measure of transfer of dominance to the left hemisphere. It is however most doubtful if this transfer is ever complete, except possibly in the case of writing itself. None the less it is to be expected that many "congenital" sinistrals, by virtue of early right-handed training, will come to acquire some measure of bilateral representation of speech.

There is little published evidence on the question. Goodglass and Quadfasel (1954) analyzed 50 cases of cerebrally damaged left-handers, 35 of whom wrote with the right hand, and 15 of whom wrote with the left-hand. The side of lesion is shown in Table 1. While there is a small trend towards a higher incidence of left-sided language laterality in those writing with the right hand, the trend is far from significant ($\chi^2 = 1.52$, NS).

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

Nielsen (1944; see Clark, p. 30) cites a definitive case in which there appears to be prima facie evidence of transfer of the speech center secondary to hand usage. A 10-year-old boy who was right-handed was forced to write with his left hand as a result of the amputation of his right arm. At the age of 31 (21 years after the forced transfer of writing), a localized cyst of the *right* angular gyrus produced a reversible aphasia, thus confirming that speech was in the right hemisphere. However, this patient also had a sinistral family history (one uncle and two cousins on his mother's side) and an alternative explanation to that of Lovell, Waggoner, and Kahn (1932) is that the loss of his hand had not caused transfer of his speech center, but that it had always been in the right hemisphere (see McManus, 1979, for details of the proposed genetic model). Certainly, a single case cannot prove the possibility of transference of dominance. Chesher (1936) also described three

TABLE 1
The Assessed Language Laterality of 50 Left-Handers According to Their Usual Hand of Writing*

	Assessed Language Laterality			
Writing Hand	Left	Right	Total	Percent Left
Right	20	15	35	57.1
Left	5	10	15	33.3
Total	25	25	50	50.0

*Data derived from Goodglass and Quadfasel (1954), with permission.

patients who seemed to disconfirm the possibility of hand usage modifying language laterality.

Early Brain Damage Modifying Language Dominance

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

Milner, Branch, and Rasmussen (1964) determined language dominance by the Wada sodium amytal technique. Of 44 left-handers with no evidence of early brain damage, 63.6% had some language located in the right hemisphere, and 20.4% had language located *only* in the right hemisphere. Of those 27 cases of left-handers with evidence of early brain damage before the age of 2 years, only 22.2% had language exclusively in the left hemisphere, and 66.6% had language exclusively in the right hemisphere (the remaining cases had evidence of bilateral language representation). These differences are highly significant, whether one excludes those with bilateral language ($\chi^2 = 13.17$, 1 df, $p<0.001$), or includes them in the non-left hemisphere group ($\chi^2 = 9.90$, 1 df, $p<0.01$).

The evidence of Penfield and Roberts (1959) is not so clear. They operated on patients with chronic epilepsy and noted the incidence of postoperative aphasia, according to handedness and side of operation. Their data divided patients according to the presence or absence of early brain damage history. Among left-handers without early brain damage, 72.2% of 18 cases had aphasia after a left hemisphere operation, compared with 6.7% of 15 cases after a right hemisphere operation ($p<0.001$). The corresponding figures for left-handers *with* early brain damage are 12.2% of 49 and 14.1% of 7 (no significant difference). This might seem to indicate a degree of randomness in speech allocation among those with early brain damage, however, such a conclusion is far from certain. A closer examination of the data presented in Table 2, reveals several anomalies. While early brain damage seems to produce a decreased incidence of aphasia after left hemisphere operations, in both right and left-handers ($\chi^2 = 5.78$, 1 df, $p<0.05$; $\chi^2 = 20.47$, 1 df, $p<0.001$), there is no compensatory increase in aphasia in those with early brain damage after right-side lesions. This is a surprising finding. Overall, the incidence of postoperative aphasia is far lower in early brain-damaged patients (12.5 of 136) than in those without brain damage (33.6% of 386) ($\chi^2 = 21.25$, 1 df, $p<0.001$), a finding true for both left-handers and right-handers ($\chi^2 = 8.72$, 1 df, $p<0.01$; $\chi^2 = 12.0$, 1 df, $p<0.001$, respectively). Early brain damage, thus, seems to protect against aphasia secondary to neurosurgical operation; this might imply some degree of bilateral speech representation and, as such, might be consistent with the hypothesis that brain damage modifies language dominance, however, the data is not totally convincing.

Early studies of the effects of early brain damage and of hemispherectomy in

TABLE 2
Incidence of Postoperative Aphasia After Right- or Left-Sided Operations in Right- and Left-Handed Chronic Epileptics*†

		Left-Sided Operations			Right-Sided Operations		
			Aphasics			Aphasics	
Handedness	Early Brain Damage	n	n	(%)	n	n	(%)
Right	−	157	115	(73.2)	196	1	(0.5)
	+	22	10	(45.4)	58	0	(0)
Left	−	18	13	(72.2)	15	1	(6.7)
	+	49	6	(12.2)	7	1	(14.0)

*Patients were divided into those with (+) and those without (−) evidence of early brain damage.
†Data derived from Penfield and Roberts (1959), with permission.

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

Stock-Brainedness

Despite Bramwell's candid admission of a lack of evidence for stock-brainedness, modern work has confirmed the partial validity of the concept, its modern form being that left-handers without a history of left-handedness in the family will be more like right-handers than those with such a history; and the converse for right-handers, a positive history of left-handedness making them more like left-handers, *as a group*.

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

Studies have usually compared persons with a family history of left-handedness (+FHLH) with those without such a history (−FHLH). Criteria vary; Hécaen and

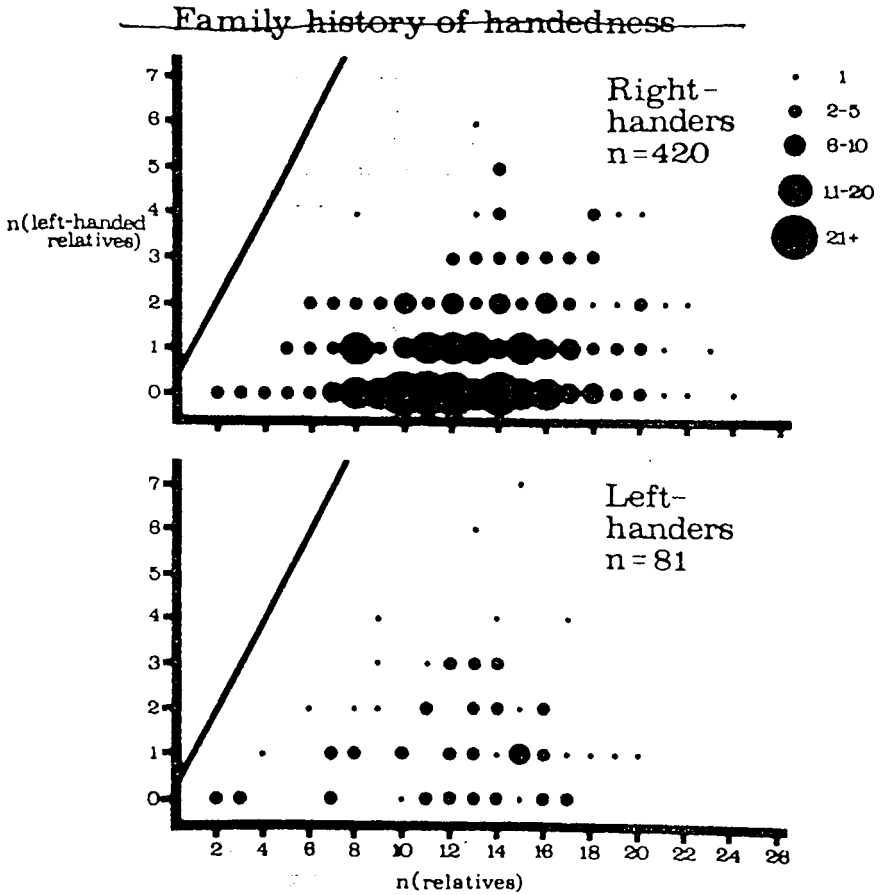

Figure 2: The number of left-handed (relatives reported separately for right- and left-handers) as a function of the total number of relatives reported. [Data are described in more detail elsewhere (McManus, 1979).]

Sauguet (1971) considered a patient to have +FHLH if "there was knowledge of at least one parent, sibling, or direct descendant who was left-handed, or at least two other left-handed relatives e.g. cousins, uncles, or grand-parents." Zurif and Bryden (1969) had a more stringent criterion: "if either a parent or sibling was left-handed; all other left-handers were considered to be non-familial." Clearly, these criteria will produce differing results; neither criterion is perfect, since family size will vary (10 dextral siblings are more convincing evidence of −FHLH than one dextral sibling), and there is a possibility of reporting biases, those with left-handed relations being more likely to report the fact. The problem of dividing a population into −FHLH and +FHLH groups is shown in Figure 2, which presents

data from a survey that was reported in detail elsewhere (McManus, 1979). In that report, a series of 511 students and their parents were asked to report their own handedness and that of their relatives, the questionnaire specifically asking for information on handedness of the aunts, uncles, siblings, grandparents, and great-grandparents of the student. Figure 2 shows (separately for right and left-handers) the number of left-handed relatives as a function of the total number of relatives reported. It would be difficult to argue that there was any clear clustering either of right- or left-handers into two groups, those with and those without a sinistral family history.

Despite the problems of defining +FHLH, useful conclusions probably may be derived from reports in the literature. Two useful studies have examined clinical populations with dysphasia. Hécaen and Sauguet (1971) looked at 73 left-handers. Those with +FHLH showed no difference in verbal symptomatology between right- and left-sided lesions, while for those with −FHLH there was a strong tendency for speech deficits to be nearly absent after right-sided lesions, but to be similar to +FHLH for left-sided lesions. Luria (1970) considered right-handers with +FHLH or −FHLH (his groups A + Ba and Bb + Bc, respectively). Of those with −FHLH, 6.7% had no initial aphasia after injury of the left frontal area, while 30.3% of those with +FHLH had no aphasia ($\chi^2 = 6.77$, 1 df, $p<0.001$). Similarly, no residual aphasia was present in 22.1% of −FHLH cases compared with 71.5% of +FHLH cases ($\chi^2 = 28.21$, 1 df, $p<0.001$). Both sets of data are compatible with the modern concept of stock-brainedness.

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

Briggs and Nebes (1976) found no significant difference between +FHLH and −FHLH on a dichotic task; Lake and Bryden (1976) found a similar result for right-handers, and insignificant trend in the wrong direction for left-handers (i.e., −FHLH in left-handers resulted in *more* right cerebral dominance). Higgenbottam (1973), using a dichotic task, found no evidence of difference between −FHLH and +FHLH in left-handers. Using unilateral ECT as a method of assessment of language laterality, Warrington and Pratt (1973) found an insignificant effect of −FHLH in the opposite direction to that expected (i.e., in the same direction as Lake and Bryden, 1976).

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

Latent Left-Handedness

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

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

Luria's list of signs of latent left-handedness is more extensive. Well-developed veins on the back of the left-hand, a wide finger nail on the left little finger, and right-sided predominance of facial expression are not known to have any empirical evidence in their support. Hand-clasping (and arm-folding) do not correlate at all with handedness (McManus and Mascie-Taylor, 1979), and have never been shown to be related to speech dominance or any other characteristic of right- or left-handers.

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

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

Pathologic Left-Handedness

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

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

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

> [A non-pathologic left-hander]. . . becomes aphasic as a result of [a] lesion in either hemisphere, although left-hemisphere lesions generally produce more lasting disability. . . Pathologic left-handers, who have suffered early childhood injury to the left hemisphere, are usually right-brained for speech. [Note the complete contrast with the view of Zangwill.]

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

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

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

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

1. The role of acute anoxia of delivery in inducing left-handedness
2. The increased incidence of left-handedness in twins, particularly monozygotic (MZ) twins
3. Left-handedness in epileptics
4. The increased incidence of left-handedness in the mentally subnormal, the mentally retarded, and the mentally ill

There is also implied evidence that reports of clinical cases support the concept of pathologic left-handedness; earlier in this report, it was argued that, to my knowledge, there is no single satisfactory case in the literature. Certainly, no such cases have been cited by the proponents of pathologic left-handedness. It is now worth considering, briefly, the other evidence for pathologic left-handedness.

The Role of Acute Anoxia of Delivery in Inducing Left-Handedness

I have extensively investigated the role of acute anoxia elsewhere (McManus, 1981) and need only briefly report the conclusions of that study. In two moderately large retrospective studies, and one very large prospective study (n = 12,000), using data derived from the 1958 Perinatal Mortality Survey and the National Child Development Study (NCDS), I was unable to find any evidence for a relation between handedness and birth stress.

The Increased Incidence of Left-Handedness in Twins, Particularly Those of Monozygotic Origin

It is usually claimed that (a) that MZ twins have a higher incidence of left-handedness than do digygotic (DZ) twins and that (b) twins in general have a higher incidence of left-handedness than do singletons. Since neither of these items could conceivably have a genetic basis, it is argued that pathologic factors as well as mirror-imaging in MZ twins are responsible for the increased sinistrality. Elsewhere (McManus, 1980a), I have critically reviewed the evidence for these statements, and concluded that neither is supported by the evidence.

Left-Handedness in Epileptics

Milner, Branch, and Rasmussen (1964) found a higher incidence of left-handedness in chronic epileptics who had an early, as opposed to a late, cerebral lesion. Penfield and Roberts (1959) found a higher incidence of left-handedness in chronic epileptics whose lesion had occurred before the age of 2 years, than in those whose lesion occurred later in life. Neither of these studies contributes anything to the purposes of the present study. The definition of pathologic left-handedness mentioned at the beginning of this paper specifically states that hand preferences *alone* must be altered by a lesion. In the studies described above, we have no idea as to the frequency of right hemiplegia, etc., thus many of these "pathological" left-handers might well fit into Gordon's first category: left-handers of necessity.

Among less severe epileptics, the evidence for increased incidence of left-handedness is poor. Elsewhere (McManus, 1980b), evidence is reported from the NCDS data in which there was no relation between handedness and history of epilepsy (defined in a large number of ways). It is possible that other studies have found an increased incidence of left-handedness in epileptics; these studies are probably all invalidated by the a priori expectations of the researchers causing a criterion shift in the definition of left-handedness in the epileptics. Certainly, future studies in this area as with those concerning twins (McManus, 1980a), should be carried out with the experimenter blind as to the epileptic status of the child being examined.

The Increased Incidence of Left-Handedness in the Mentally Subnormal, Retarded, and Ill

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

Table 3 presents data from analysis of the NCDS data (see McManus 1980 for further details of this dataset); it is clear that mental retardation does, indeed, correlate with left-handedness. Calnan and Richardson (1976), using the same NCDS database, found that left-handedness related significantly to poor speech and to several attainment tests.

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

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

Thus far, all the evidence criticized has been very indirect. To my knowledge, there is no direct evidence of a link between cerebral trauma and left-handedness. Table 4 shows analysis of NCDS data in which mothers of children were asked if their child had ever been knocked unconscious or suffered a fractured skull. While not perfect, these two variables might be expected to correlate with cerebral trauma of the closed head injury type. Despite a large sample size, there is no evidence of a relation between handedness and cerebral trauma. Although very inadequate, these data are unique, and provide~~s~~ yet further evidence for the invalidity of the concept of pathologic left-handedness.

Conclusions

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

Despite the claims of many authorities, a critical review of pathologic left-handedness reveals that there is no adequate evidence to substantiate the concept

TABLE 3
Doctor's Assessment of Child's Degree of Mental Retardation at 7-Years-of-Age

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

$X^2_4 = 35.57, p < 0.001$

TABLE 4
Mother's History of Head Trauma Suffered by the Child

Question	Response	*n*	Percent left	χ^2	Significance
Has the child ever had a fracture of the skull?	No	12010	11.1%	2.69	NS
	Yes	1646	12.5%		
Has the child ever been knocked unconscious in an accident?	No	13096	11.3%	0.004	NS
	Yes	599	11.4%		

*Information was collected at the NCDS second sweep, when the child was 11-years-old.

and, indeed, the idea probably finds its origins in repeated attempts to prove Dax's Law. The present author would accept that pathologic left-handedness might possibly occur in very rare instances, although as yet there is no convincing evidence for such a phenomenon. Certainly pathologic left-handedness would not seem, as many authors would claim, to be a frequent occurrence.

I am grateful to the National Children's Bureau for granting me permission to reanalyze data from the National Child Development Study, and to E. Roughley and the Social Science Research Council Survey Archive for providing that set of data.

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