LANGUAGE LATERALITY IN LEFT-HANDED APHASICS

BY

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DEFINITION, HISTORY AND PROBLEM

Laterality refers to the relationship of a cerebral function, or the symptom of its disturbance, to one cerebral hemisphere. Although the term is used mostly in connexion with handedness, aphasias, and agnosias, it has much wider implications and, in its broader sense, it constitutes the basis for localizing the lesion in cerebral hemiplegia in the opposite hemisphere.

The association of right hemiplegia and speech disturbance can be found in the description of aphasia by Goethe (1796) and others. The idea of localization of speech in the left hemisphere was first conceived by Marc Dax in 1811 and presented at a lecture in 1836, but was not published until 1865 (Broca, 1865). It was reiterated by his son, Gustave Dax, in 1863 (Souques, 1928). When Broca, too, concluded that the posterior part of the third frontal convolution, especially on the left, was essential for intactness of the faculty of speech (1861–1864); he offered no explanation for this lateralization, nor suggested any significance in terms of handedness or cerebral dominance (Broca, 1863a, b).

The idea of the linkage of handedness and speech was first formulated by Bouillaud in April 1865, when he spoke of “gaucherie cerebrale,” again by Baillarger in May 1865, and Broca in June 1865 (See Broca, 1865; Souques, 1928; Riese, 1947). In 1874 Jackson spoke of the “leading hemisphere” and of the localization of both propositional and emotional speech in the left hemisphere, and of emotional speech alone in the right hemisphere. Jackson (1880), and Ogle (1871) contributed some cases of left-handed individuals who had been rendered aphasic by right cerebral lesions. In 1877 Kussmaul stated that innervation centres are bilateral for all manual work but most people are right-handed and train the left hemisphere for most skills. “On this fact and the preponderance of aphasic disturbances in lesions of the left hemisphere rests the hypothesis that most people are ‘left-brained speakers’.”
From such facts and opinions the inference was made that there is cerebral dominance as a property of one hemisphere. The dominant hemisphere now was assumed to control the preferred hand, often the preferred eye and foot, and, if damaged, to produce aphasia. Handedness was now used as evidence of dominance of the hemisphere opposite to the preferred hand (e.g. Weisenburg and McBride, 1935, p. 435).

While Kussmaul does not speak of the influence of handedness on speech or its use for treatment, others like Brown-Séquard, (1874), suggested that by development of the movements of the two sides of the body the two sides of the brain might be developed as regards the mental functions. Buzzard (1882) more specifically believed that the use of the left hand would develop the corresponding convolution in the right cerebral hemisphere. “The idea is, as I have said, in the failure from diseases of Broca’s convolution, to develop by afferent impulses the corresponding convolution in the right cerebral hemisphere. With the same view, I make the patient go through gymnastic exercises with the left arm and delicate movements with the fingers of the left hand, with the aim of producing that dexterity (using the term in its etymological sense) with which the faculty of expression by speech and signs is so intimately associated” (p. 439).

Similar ideas have continued to limit the thinking of neurologists to the present day. They are implicit in the following two explanations offered for “crossed aphasia,” or aphasia caused by a cerebral lesion on the same side as the preferred hand. When a left-handed person becomes aphasic as a result of a left cerebral lesion, the circumstances of his having learned to write with his right hand is generally credited with conferring dominance to the left hemisphere (cases of Bramwell, 1899, Chesher, 1936). When a right-handed person becomes aphasic as a result of a right cerebral lesion, one explanation offered is that he was congenitally left-handed and right-brained (from left-handed stock) but learned to prefer the right hand as a result of environmental pressure.

These two theories contradict each other. The first holds that the training of a hand for writing can transfer the dominance for speech to the hemisphere opposite the hand used. The second theory holds that dominance for language persists in spite of the pre-eminence of the ipsilateral hand in all activities.

The occurrence of aphasia with left hemisphere lesions in the great majority of patients, most of whom are right-handed, is undisputed. However, the assumption that this constitutes a dominance of the left hemisphere, and that conversely left-handedness is necessarily linked with a dominance of the right hemisphere in language, requires further study.
Evidence contradicting this traditional view has accumulated rapidly in the last five years. It has even led some investigators (Wepman, 1951, and Milner, 1952), to hold that right hemisphere language representation is extremely rare or non-existent. It has led Girard (1952) to define handedness in terms of language laterality: "Le gaucher n’est pas celui qui écrit avec la main gauche mais celui qui parle avec le cerveau droit."

**DATA ON LATERALITY IN LEFT-HANDED PATIENTS**

One method of investigating this question would be to examine a large number of cases of left-handed individuals with lesions of the language area. Individual series of such character are so small as to have little statistical significance. In our own two consecutive series totalling 320 cases examined for aphasia, 13 cases of left-handedness with unilateral lesions of the anatomical language area were found. The first series consisted of 76 aphasics found during a post-traumatic epilepsy study at Cushing General Hospital, in which all (246) cases were also examined for aphasia and handedness. Out of these, 7 were found to be left-handed and have a unilateral lesion only. In the second series of 244 consecutive cases examined in the Aphasia Unit at Cushing Veterans’ Administration Hospital, and now at the Boston Veterans’ Administration Hospital, 6 such cases were found. In addition, we have searched the neurological literature for cases of this type and have been able to compile a series of 110 additional cases.

The criterion of left-handedness used in this paper is that the individual performs most skilled acts (eating, cutting, handling tools) with the left hand and considers himself left-handed. Writing with the right hand by an otherwise left-handed person does not constitute ambidexterity. No cases were included in which left-handedness was known to be the result of an early left cerebral injury. It is recognized that this criterion lumps together individuals of every degree of left-handedness. It would be desirable to investigate the strength of left-handed preference in relation to language laterality if it were possible to obtain a satisfactory inventory of the manual skills of each aphasis patient. Bethe (1933), however, believes that even such determination in adults would be inadequate as an indication of native tendency to handedness. Chesher (1936) suggests that writing with the preferred hand is necessary for pure left- or right-handedness, and that “pure” handedness is always correlated with exclusively contralateral language laterality. However, the cases of Mendel (1912), of Weisenburg and McBride (1935), and of Ballantine and White (1953), contradict this rule. Moreover, the few case reports which mention the writing hand indicate that the vast majority
of left-handers were taught to write with the right hand. Thus, left-handed writing cannot be used as a definite criterion for predicting language laterality.

Another possible basis for classifying left-handers (Bramwell, 1899; Kennedy, 1916) is the presence of left-handedness in the family background. There is little or no evidence that the strength of the individual tendency to left-handed preference is determined by the amount of left-handedness in the family, and it is impossible to evaluate the strength of familial influence unless all of the patient's antecedents are left- or right-handed.

Out of the total of 123 left-handed cases, 50 patients showed aphasia in connection with right hemisphere lesions, and 8 showed absence of aphasia in spite of seemingly adequate left hemisphere lesions. All 58 were apparently right-brained for speech. Sixty-five patients who were apparently left-brained for speech were made up of 53 who showed aphasia with left hemisphere lesions (crossed aphasia), and 12 who showed absence of aphasia with right hemisphere lesions. This proportion is not necessarily an accurate representation of the entire population of left-handed aphasics because on one hand the cases were published as examples of crossed aphasia, and on the other hand as illustrations of the classical theory of dominance. If anything, the prejudice is in favour of the latter. It is certainly to be expected that a history of left-handedness has gone unnoticed in many apparently ordinary cases of right hemiplegia and aphasia, and which are therefore unreported. If we take only those sources which claim to offer an impartial tabulation of consecutive left-handed cases with aphasia from their own experience (Weisenburg and McBride, 1935; Chesher, 1936; Conrad, 1949; Humphrey and Zangwill, 1952a; Roberts, 1951; Wepman, 1951, and the present report), the proportion is 43 to 23 in favour of left-handedness and left cerebral lesions. The total figures, as summarized in Table 1, indicate that 53 per cent of the patients fail to show the expected relationship of left-handedness with right-brainedness for language.\textsuperscript{1} The addition of the negative cases (those in which laterality is inferred from the absence of aphasia in a lesion of the supposed language area), does not change the proportion significantly.

\textsuperscript{1} The foregoing figures do not include 27 cases of speech disturbances in left-handers and ambidexters with right-sided brain injuries reported by Ludwig, who, in 1939, in 979 German World War I soldiers with right brain lesions, found that 27 of 99 left-handers and ambidexters in this group had aphasia. As Ludwig was primarily concerned with the right-handers, he combined his figures for left-handers and ambidexters, did not mention his criterion of ambidexterity, the types of lesions involved or any other individual case data.
TABLE I.—INCIDENCE OF APHASIA IN LEFT-HANDERS FOLLOWING UNILATERAL LESIONS OF THE ANATOMICAL LANGUAGE AREAS

<table>
<thead>
<tr>
<th>Language area apparently on left</th>
<th>Language area apparently on right</th>
<th>Total:</th>
</tr>
</thead>
<tbody>
<tr>
<td>63 cases (53%)</td>
<td>58 cases (47%)</td>
<td>123 (100%)</td>
</tr>
</tbody>
</table>

All left-handers seen or reported

<table>
<thead>
<tr>
<th>Writing hand:</th>
<th>Lesion on left with aphasia</th>
<th>Lesion on right without aphasia</th>
<th>Lesion on right with left without aphasia</th>
<th>Lesion on left with aphasia</th>
<th>Lesion on right without aphasia</th>
<th>Lesion on right with left without aphasia</th>
<th>Lesion on right with left with aphasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>17</td>
<td>3</td>
<td>14</td>
<td>1</td>
<td>35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>4</td>
<td>0</td>
<td>9</td>
<td>1</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not given</td>
<td>32</td>
<td>8</td>
<td>27</td>
<td>6</td>
<td>73</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All cases</td>
<td>53</td>
<td>12</td>
<td>50</td>
<td>8</td>
<td>123</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

From these data two conclusions are inevitable:

First: The tendency for aphasia to follow brain lesions contralateral to the preferred hand is much greater in right-handed than it is in left-handed individuals.

Second: The frequency of left hemisphere lesions with aphasia is greater than the frequency of right-handedness, in fact, about one-half of left-handed individuals with aphasia have a left hemisphere lesion.

In the authors' opinion, these data are most consistent with the hypothesis that the various functions, e.g., language, handedness, eyedness, establish themselves independently more on one side or the other, but most often on the left. The authors agree with Jasper and Raney (1937) that factors responsible for the development of one highly integrated functional region might well favour a similar development of other such regions on the same side. If aphasia following right-sided lesions were as infrequent in left-handers as in right-handers this would tend to indicate the complete independence of handedness and language laterality.

DIFFERENCES BETWEEN LANGUAGE LATERALITY AND HANDEDNESS

A further examination of the accumulated data from this and other studies leads to the identification of properties of language laterality which are distinct from those of handedness.

As far as we know, there is no way of identifying language laterality by observing normal language behaviour. Its characteristics have been inferred only from the performance of patients with damaged brains, where the side of the lesion was known. Focal epileptic discharges accompanied by aphasia have given additional clues. Electrical stimulation of the cortex has been shown by Penfield and his associates (1954) to produce temporary
aphasia similar to that which follows the removal of the brain tissue at the site of stimulation.

Language laterality also differs from handedness in that it is not defined in terms of discrete acts, some mediated by the left and some by the right hemisphere. Rather, each language performance is usually conceived of as arousing associative activity in both hemispheres, though to an unequal degree. This imbalance in the participation of the two hemispheres may vary from one modality of communication to another (speaking, reading, writing) and, as Jackson believed, between propositional speech on the one hand, and automatic or emotional speech on the other.

**CONSTITUTIONAL AND CULTURAL FACTORS IN LATERALITY**

Blau (1946) cites anthropological evidence which indicates that handedness is less strongly developed among primitive people who do not use many implements. In his own investigation he found that the more highly skilled an act was, the more reliable it was as an indicator of an individual's over-all handedness. The emergence of hand preference is a function of the opportunity for activities in which one hand must be used alone, chiefly in the manipulation of small objects. It is therefore based on the consensus of many separate manual habits, all originally subject to voluntary control and to influence by environmental pressure.

Neither social pressure to prefer the right hand, nor the great susceptibility of either hand to training, prevents a certain number of children from using the left hand for most skilled manipulations. Inspection of the statistics on the distribution of left-handed individuals leaves little room for doubt that heredity plays a role in hand preference. Brain (1945) cites the following data of Chamberlain’s on the incidence of left-handedness:

- Both parents right-handed .................. 2.1%
- One or both parents left-handed ............ 17.3%
- Out of a group of 33 families having both parents left-handed .................. 46.0%

The manner of inheritance is not known. Gates (1946) cites Rife as considering "handedness as essentially a quantitative character due to more than one pair of factors."

While genealogical studies of handedness are possible, it is hardly possible to trace familial tendencies in the cerebral lateralization of language function. This can be inferred only in those individuals who develop or fail to develop aphasia with a unilateral lesion. In his paper, "Stockbrainedness." Kennedy (1916) shows that right-handed individuals...
with crossed aphasia may come from families where left-handedness was predominant and vice versa. In these cases, it is the subject's own hand preference which has supposedly deviated from the expected trend. However, Mendel's (1912) description of a right cerebral lesion causing aphasia in a completely right-handed setting, indicates that the cerebral language laterality is as apt to deviate from the tendency of the stock as is the hand preference. In 3 of our cases of crossed aphasia in left-handed individuals, the family background is known, and in all 3 cases there is sufficient evidence of left-handedness to make Kennedy's theory inapplicable. This is also true of Tilney's (1936) own crossed aphasia and right hemiplegia. He and a grandmother, mother, brother, and son, were left-handed.

**Flexibility of Language Laterality**

Evidence from patients with congenital defects of the left cerebral hemisphere indicates that cerebral laterality does not depend on a predetermined localization of function on one side. Injury to the area which is originally more efficient can reverse the balance and, by causing the other side to become the relatively more efficient, lead to establishment of laterality on the side which would naturally have been the minor one. Thus, of the 10 hemispherectomized right hemiplegics reported by Krynauw (1950) (from 2 years, 9 months, to 21 years of age) none suffered any language disturbance after operation, and several improved markedly in speech as a result of it — even to the point of better than average achievement. In these cases of congenital left brain injury, as Gooddy and McKissock point out (1951), "The familial handedness changes sides. The naturally dominant eye and limbs take second place, though not necessarily far behind. The child continues to grow, to learn, and to act."

A shift in laterality for language is also assumed to be possible later in life. The extent to which the other hemisphere approaches the activity of the originally major area apparently depends on individual differences in potential, on the age of the individual, and the suddenness with which the major area fails. Thus, permanent aphasia is rarely seen in children after injuries to the left hemisphere. We do not know the age up to which such recovery may occur easily without a long period of re-learning. We know, however, that there is considerable flexibility in the acquisition and loss of speech automatisms, because before the age of 9, children who become deaf and are not forced to continue using speech will forget it, and will have to be re-trained. This lack of fixation of speech patterns is probably related to the amazing readiness with
which children learn new languages and discard old ones. It may also be related to the observation (Freud and Ric, 1891; Sachs, 1926) that aphasia follows right-sided lesions in children much more frequently (about 25 per cent) than in adults.

A more disputable point is whether cerebral language laterality can be influenced by a forced change in hand preference. Needles' (1942) discussion of this problem permits no definite conclusion. It is virtually impossible to present absolute proof that a change of laterality for language function has taken place in any case as a result of a change in hand preference. Such proof would require evidence of the original language laterality in the brain—evidence which can come with certainty only from the results of a brain injury. Two cases are reported in the literature (Oppenheim, 1899; Lovell, Waggoner and Kahn, 1932) of individuals forced to change handedness by injury to the right hand, at ages 10 and 17, and who were later rendered aphasic by right cerebral lesions at ages 31 and 59 respectively. In both cases only the history of right-handedness supports the assumption of original left cerebral language representation. In view of the comparatively great number of cases in which lifelong hand preference has failed to carry along language dominance in the brain, these two cases are not convincing. They could easily be among the number in which language originally preferred right cerebral activity or in which both hemispheres were active.

In the mature individual, the participation of the right hemisphere in speech is apparent in the partial recovery of the aphasic with a left-sided lesion. A new lesion in the right hemisphere will again make him aphasic, but a repeated lesion in the left hemisphere will also make him aphasic again. Such cases have been observed by Gowers (1893) and Nielsen (1946). Henschen (1922) has pointed out that the performance of the right hemisphere is always inferior, but no attempt will here be made to discuss whether this activity constitutes a true substitution or a partial return of premorbid functioning.

Statistical Distribution of Handedness and Language Laterality

Estimates of left-handedness in the population cluster around 5 per cent. Surveys tend to find decreasing frequency of left-handedness with increasing age. Moutier (1908) estimates left-handedness to be present in at least 10 per cent (about 9 per cent in his own observation) of the population; he also states that he found among 600 old people one ambidexter in 45. Other writers do not give figures on the incidence of ambidexterity, but it would appear less frequent than left-handedness, if we do not consider as ambidextrous those individuals who have been forced
to write with the right hand, and who prefer the left for other skilled acts. The distribution curve for handedness in the population would appear bimodal with the major concentration for right-handedness, a very much smaller concentration for left-handedness, and a dip in the centre for ambidexterity. Expressed in graphic form, this distribution might be presented as in fig. 1.

![Hypothetical distribution of degrees of right- and left-handedness.](image)

The proportion of people who have aphasia as a result of right hemisphere lesions is very small. Wepman (1951) cites from the literature a total of more than one thousand cases of brain injury with and without aphasia, in which no instance of left hemiplegia with aphasia was noted. This is an unusual figure because, if the cases were unselected, about 50 should have been left-handed, and it seems reasonable that some of these should have had a right cerebral lesion. It seems remarkable that none of them should have had an aphasia. A strongly contrary finding on the incidence of crossed aphasia in right-handers was reported by Ludwig (1939) on the basis of 880 right-handed German World War 1 veterans with right brain injuries, but otherwise unselected. One hundred (11 per cent) of these had aphasia, and about 30 of this number (3 per cent of the total) had aphasia lasting more than three months. Ludwig found that the frequency of aphasia following right-sided injuries, in left-handers and ambidexters (27 per cent), was only about two and a half
times as great as for right-handers (11 per cent). He does not report on
the permanence of the uncrossed aphasias in left-handers. Nielsen (1951)
gives 6 per cent as the frequency of crossed aphasia for right-handers and
left-handers.

Humphrey and Zangwill's data (1952a) indicate that 9 of their 10
cases of left-handed individuals developed some aphasia after unilateral
lesions of the language areas of either hemisphere. This is also true in
10 of 13 of our left-handed cases. Conrad (1949) presents still stronger
statistical evidence to the effect that, including all cases with brain lesions,
left-handers are more frequently made aphasic by unilateral lesions than
right-handers. Out of a total of 760 brain-injured right handers, 26 per
cent (198) had a history of aphasia. However, 38 per cent (18) out of a
group of 47 left-handers were aphasic, at least temporarily. Such a
difference in proportion could occur by chance less than six times in one
hundred. This suggests that the situation with most left-handers is one
of significant participation of both hemispheres in language.

Conrad also found that left-handed patients frequently had only a
transitory language loss where the nature of the injury would lead to the
expectation of a more profound loss. He concludes that left-handedness
implies less fully developed specialization of either cerebral hemisphere.
This possibility of a transitory aphasia from either side in a left-hander,
if confirmed, should be of interest to the neurosurgeon. It would also
demonstrate the inter-relationship of the two hemispheres in language
because there should be no aphasia if language were represented bilaterally
and independently. Subirana (1952) believes that when aphasia is absent
or transitory "in all apparently contradictory cases," there is no true or
exclusive cerebral dominance. Statistical analysis of Conrad's figures
reveals no significant difference between left- and right-handed patients in
percentage of transient aphasias. But statistical analysis does not take
into account the severity of the lesion in the individual case, on which
Conrad based his impression. The experience of the present authors in
screening the cases in their own series parallels that of Conrad. Only 5
left-handers were discovered among 244 cases who had speech disturbances
severe enough to cause them to be referred to the Aphasia Unit. On the
other hand, 7 left-handers (Appendix cases 7 to 13) were discovered in the
group of 76 epileptics with unilateral lesions of the anatomical language
areas. Of the 5 who had an aphasia, only 1 had detectable aphasic residuals
one year after injury, the remaining 4 having had only partial and
transient language disturbances.

Roberts' (1951) figures on left-handers who have undergone cortical
excisions indicate a much smaller proportion of right hemisphere language
participation than data collected from other sources. He reports only 3 aphasics out of 23 operated upon in the right hemisphere, against 13 out of 18 operated upon in the left hemisphere. Roberts does not state whether all of his cases involved excisions of the language areas. However, he also reports a similar low proportion of left-handers who demonstrated speech disturbance on electrical stimulation of the right hemisphere. This reminds one of Riese's (1950) remark that "the occurrence or absence of aphasia or apraxia after unilateral lesions does not admit of any conclusions as to dominant processes in the intact hemisphere."

The speech disturbances resulting from both electrical stimulation and cortical ablations differ in important respects from the effects of vascular lesions, tumours or injury. Thus, Roberts reports no systematic difference in the kinds of aphasia produced by excision or stimulation of different areas of the brain. Penfield and Jasper (1954), and Roberts (1951) report that cortical areas previously found responsible for aphasia when they were damaged (e.g. the insula) have no such effect when removed operatively. Thus the picture of language laterality obtained from cases of vascular or traumatic aphasia may be much different from that contributed by controlled stimulation or excision.

The best evidence for strong right hemisphere language laterality is the destruction of the left hemisphere language areas without consequent aphasia. There are in all our collected material only 8 such cases out of 61 left-handers with left-sided lesions. This may indicate that strong right hemisphere language laterality is infrequent even in left-handers. This is in contrast to the specialization of the left hemisphere for language in right-handers, as evidenced by the rare incidence of aphasia with right-sided lesions and left hemiplegia.

A theory which comes close to fitting these facts was offered by Bramwell (1899), who is better known for taking the position that crossed aphasia in left-handers was an "exceptional" condition which had to be explained by their training in right-handed writing, or by familial right-handedness. He suggests that there is a universal tendency to revert to left-brained speech as the natural or "original" condition of the species—a view which makes his other explanations unnecessary. Bramwell does not, however, go on to point out that regression of left-handers to the "original" left-brainedness for speech does not make them right-handers in spite of enforced training in writing with the right hand.

The addition of this observation transforms Bramwell's theory into one which is entirely congruent with the position of the present authors; i.e. the tendency for language to centre predominantly in the left hemisphere is in large measure independent of handedness: it is more nearly universal
than the tendency to right-handedness; it therefore operates to make many left-handers left-brained speakers, while the reverse condition is very rare.

From the evidence presented it appears that the distribution curve of language laterality differs from that of handedness by being more one-sided, unimodal, and tapering off, with more people showing cerebral ambilaterality for speech than there are having a clear-cut right-sided language dominance (see fig. 2). This independence of distribution from that of handedness means that laterality for language is not identical with laterality for handedness. Handedness does not determine brainedness.

**LATERALITY, SPEECH AND LANGUAGE DISORDERS**

There is no room here for an exhaustive treatment of the question of the relation of laterality to developmental disorders of speech or reading, but a review of the evidence is relevant to the topic of this paper. The observation has been made repeatedly by Orton (1937), Dearborn (1940), Harris (1947), and many others that stammerers and poor readers show a greater-than-average incidence of left-handedness, ambidexterity, and
mixed hand and eye preferences. This relationship must now be accepted as a fact to be explained.

An increased incidence of mixed or left-sided preference is a repeatedly verified finding for stutterers who have no known brain damage and whose verbal intelligence may be above average. Here, however, there is a question of interference with innervation at a level which is better considered one of speaking, in a narrow sense, than an interference with language on a symbolic level.

Speaking of unrecognized minimal birth injuries to the brain, Geseil and Amatruda (1941) state: “The type which expresses itself in speech difficulties, poorly defined unilateral dominance, and in delayed integration, may later result in a serious difficulty in the acquisition of reading.” Some minimal brain injury at or after birth, or mal-development may affect one or all phases of language while leaving the rest of the intellect intact. This is in our opinion the explanation for some specific disabilities, such as circumscribed reading and spelling defect, partial amusia (poor ability to recognize, appreciate and perform music), poor spatial orientation, poor sense of rhythm, and others. There is little justification for regarding mixed or left-sided hand-eye preference as the primary cause of a predisposing factor towards a deficiency in acquiring language. This coincides with Brain’s (1945) opinion, seconded by Subirana, that “anomaly of handedness is a symptom and not the cause of the disorder underlying the congenital aphasias.”

An example of flexibility in the laterality of agnosic symptoms generally attributed to one hemisphere is the localization of lesions producing visual-spatial disorientation. McFie, Piercey, and Zangwill (1950) present 8 cases, and Hécaen, Ajuriaguerra, and Massonnet (1951) present six more in which disturbed visual-spatial performances occurred in right hemisphere lesions in right-handed individuals without aphasia. Brain (1941), Hermann and Pötzl (1926) and Humphrey and Zangwill (1952b), present cases of originally left-handed persons who had marked visual-spatial disorientation with right cerebral lesions and aphasia. Milner (1952) reports visual-spatial defects consistently more severe in right temporal lobe excisions than left. She suggests that this is a function which is usually lateralized on the right. This disorder is also known to occur with left-sided lesions in right-handed individuals, or with bilateral lesions, as Critchley (1953) points out. These samples show that there is no fixed laterality for these disturbances, nor is localization determined by laterality for handedness or laterality for language.

**On a Theory of Lateral Specialization**

We know, from cases with congenital right hemiplegia and severe left
hemisphere damage, that the right hemisphere alone can mediate the normal use of language. Why then should this function develop asymmetrically, preferring the left hemisphere in the intact brain? To say that language laterality results from the use of a preferred hand would require universal correspondence of hand and language laterality, which is not the case. The external and voluntary elements in the choice of hand preference are so obvious that some, like Blau (1946) have chosen to lay the entire phenomenon of handedness at the door of cultural determination. However, the hypothesis of cultural determination of handedness is heuristically valueless for understanding the asymmetry of language laterality in the brain, since this is beyond the reach of direct cultural influence. Thus, whatever weakness there may be in the case for constitutional determination of hand preference, no other plausible explanation has yet been forthcoming for language laterality.

No theory of lateral specialization has yet been offered to account for these facts: (1) Either hemisphere can function alone for language if the other is severely damaged early in life. (2) In children, aphasia from right-sided lesions occurs more frequently than in adults. (3) In children aphasia is more often transitory. (4) Left-handed people are possibly more susceptible to aphasia from lesions of either hemisphere than are right-handed. (5) The specialization of the left hemisphere for language in most right-handers is much more extreme than specialization of either hemisphere in left-handers. (6) The period of consolidation of language laterality in childhood is one of increasing learning ability in both verbal and nonverbal areas; thus the consolidation does not constitute a cessation of learning ability.

The ideal theory of cerebral laterality would permit us to start with two potentially trainable language association systems, differing slightly in their readiness to learn. The theory would have to allow for individual differences in the amount of imbalance between the two hemispheric systems, with inheritance tending to favour that on the left, especially in right-handers. Finally, and most important, the ideal theory should show how, after beginning with almost equal bilateral participation, a self-reinforcing process causes the slight lead in language performance to snowball into a nearly complete unilateral representation.

**ORIGINAL CASE DESCRIPTIONS**

**Case 1.**—Right hemiplegia with aphasia: R. S., aged 19, received a left frontal penetrating missile wound May 25, 1951. On operation three missile tracts, all within the left cerebral hemisphere, were found, although X-ray later showed one fragment lodged in the right posterior parietal area. On examination six months later the patient showed a right homonymous hemianopsia with macular sparing, right central facial weakness, right hemiplegia, hyperactive deep tendon reflexes.
and pathological reflexes on the right. Pneumoencephalogram showed the left ventricle dilated in the anterior horn and a normal right lateral ventricle. EEG showed a slow wave focus in the posterior portion of the left hemisphere and smoothing of the wave forms over the left central temporal area. At this time the patient had a severe mixed aphasia, with a marked articulatory component, and he was almost totally alexic and agraphic. He could evoke only a few single words in response to pictures or questions. During attempts to repeat back any but the simplest spoken words, he often perseverated with the same jargon-like utterance. He wrote his name easily and rapidly with his preferred left hand, and he later revealed that he had been taught to write with his right, but had reverted to the left in the fourth or fifth grade. The patient's mother verified that she herself was left-handed, as were the patient and his brother. Six other children and the father were right-handed.

In May 1952, after six months of re-training, the patient had a great many words available for spontaneous speech, but was still unable to use sentences, and had moderate articulatory difficulty. Comprehension of speech was now almost normal, but only slight gains in written word-recognition and spelling were measured on re-testing.

Case 2.—Right hemiplegia and aphasia: C. M., aged 47, developed a right-hemiplegia and severe aphasia in the course of treatment for osteomyelitis of the left tibia. A diagnosis of "encephalomalacia of the left internal capsule and thalamus due to vasospasm and thrombosis," was made at that time. When admitted for language re-training in January 1948, a pneumoencephalogram showed dilatation of the left ventricle in an otherwise normal ventricular system. EEG showed abnormal focal activity in the left temporo-occipital area. His right hemiplegia had receded sufficiently for the patient to resume writing with his right hand. He gave a history of left-handedness from birth, and stated that he had been forced to learn to write with his right hand. He readily taken to the use of his left hand for writing during the period of severe hemiplegia.

His aphasia had improved greatly up to the time of his admission and was principally sensory in nature. He had fairly fluent speech, with normal articulation and inflection. However, there was still groping for words, and circumlocution in his speech. He understood spoken commands poorly, had a severe word-deafness for words spoken out of context. and had considerable difficulty in following conversation. He also had a great deal of spelling difficulty, and when words were dictated he would repeat them aloud, over and over, without appearing to understand what he was saying. Even with this device he would forget the word or wander to another within the same sphere of meaning. After a year and a half of treatment, his understanding of conversation and writing ability had improved, but he still had trouble understanding isolated words.

Case 3.—Right hemiparesis and aphasia: F. F., aged 48, was admitted to the medical service of the hospital on April 23, 1951, because of the onset, three days before, of right hemiparesis and aphasia. The patient had had seven previous admissions for kidney disease, and for a skin ailment. At the time of his sixth admission, in April 1949, signs of an old vascular accident in the right hemisphere were discovered. These included dragging of the left leg, sustained patellar clonus and Hoffman sign on the left. The left arm was slightly clumsy. The patient reported that he had noticed the onset of numbness and weakness in his left arm a year before—at the same time that his left leg felt funny and started to drag. An EEG performed on April 26, 1949, was within normal limits. No speech disturbance was noted, and none was reported by the patient.

At the time of his most recent admission he was reported to have fallen to the floor without losing consciousness, having weakness of the right side, with the right
corner of his mouth drooping. He showed a predominantly sensory aphasia, speaking in phrases with normal rate, inflection, and articulation, but with considerable paraphasia and word hunting. He had difficulty in following spoken commands, and could not read or write. Neurological examination in August 1951 showed that the old signs on the left side of the body were most prominent, while strength on the right had been regained. However, an EEG performed in May 1951 revealed a slow wave focus in the left anterior frontal area.

The patient reported that he was left-handed in all respects except writing. He claimed that he used to prefer his left hand for writing, but was forced to use his right in the early grades. His mother was also allegedly left-handed, except for writing.

In December 1951, language examination revealed that the patient's speech was superficially normal, but there was moderate anomia and a great deal of circumlocution because of it. His own sentences often trailed off in confusion, but he now understood commands and conversation well. Reading comprehension was recovered to the third-grade level of efficiency, and writing had returned to a greater degree during six months of re-training.

Case 4.—Left hemiplegia and aphasia. V. R., a 29-year-old female patient, with severe left hemiplegia and aphasia, was admitted for speech re-training in September 1951. She had suffered severe cerebral contusions in an automobile accident in November 1950, which had been followed within forty-eight hours by left facial paralysis and left hemiplegia. Two days after injury, while still in coma, bilateral trephination revealed brain edema, but no sign of hemorrhage. After this procedure, a right facial paralysis and paresis of the right arm developed and then receded, while the patient remained in coma for two weeks.

Examination in September 1951 revealed a spastic left hemiplegia and a left facial weakness of the central type, hyperactive deep reflexes, Babinski, and Oppenheim sign on the left. There was diminished sensation to touch, pain, and vibration on the left side. The right pupil was smaller than the left, and there was a ptosis of the right eyelid. Pneumoencephalogram done on May 12, 1952, revealed marked dilatation of the lateral and third ventricles without a shift in the ventricular system. Increased amounts of air were seen in the subarachnoid channels, and the interpretation of extensive brain atrophy was made. EEG with metrazol activation, performed in May 1952, showed a low metrazol threshold without a clear focus, while a sleep record later that month showed a lack of sleep spindles and of arousal responses on the right, indicating extensive damage in that hemisphere. Later that month the patient reported her first focal seizures, involving the left arm.

The patient reported that she had been left-handed before her injury, and her family advised that she and one of her brothers were completely left-handed, while the remaining three siblings were right-handed.

Upon admission to the aphasia service, the patient had a moderate mixed aphasia, revealing a varied vocabulary of single words and short phrases, which she used in answering questions, but she almost never initiated speech. She had word-finding difficulty for all categories of words, and made frequent paraphasic word substitutions in tests for evoking single words in response to pictures, printed or spoken cues. Her understanding of spoken language in tests was defective to the point of mismatching some spoken words and pictures, and misinterpreting simple commands. Her understanding of conversation was less obviously impaired. With regard to written language, the patient recognized the meanings of many individual words, but had no success with sentences. The patient could write nothing beyond her name from dictation, but was able to write "cat" and "man" on presentation of a picture stimulus. She was completely acalculic. Aphasia, in this case, can clearly be attributed to the right cerebral lesion. If these facts are considered:
There was no speech before patient came out of coma two weeks after the injury. There was bilateral dilatation of the ventricles, but it seems not likely that the aphasia would have been so severe if it were part of the transient signs observed on the right (right central facial weakness, and weakness of right arm) after trephination. All later examinations, neurological and electroencephalographic, reveal a lesion in the right hemisphere, and fail to reveal evidence for a lesion on the left.

After eight months of therapy, re-examination revealed that her command of spoken vocabulary for spontaneous speech had improved measurably, although her sentence structure was still telegraphic for any but the shortest sentences. Her understanding, while slightly improved, was still defective. Intensive practice with written language had made little inroad into her alexia and agraphia. Although she acquired good right-handed writing, and would copy and memorize sentences with key words, her retention was barely adequate for one day, and she still failed to score on standardized reading or spelling tests.

Case 5.—Right hemiplegia and aphasia. H. C., a 45-year-old male, was hospitalized in December 1953 for control of epileptic seizures. On neurological examination he had a mild, right, residual hemiplegia, and a moderate mixed aphasia, with dyscalculia. The history revealed that he had become aphasic in 1942, during his hospitalization for an appendectomy. At that time he had developed thrombophlebitis, followed by cerebral thrombosis, and acute arterial occlusion of his right leg, which led to a low thigh amputation. During the next eight years he received intensive speech re-training.

While the patient currently referred to himself as "ambidextrous," his handedness history revealed that writing was the only activity in which he had preferred the right hand. He had been converted in school from his original inclination to use the left hand in writing. There were no other known left-handers in his family.

Case 6—Right temporo-parietal lesion without aphasia. C. P., a 27-year-old machinist was hospitalized on March 24, 1954, with complaints of headache of ten days' duration, and a momentary lapse of consciousness during which he had driven his automobile into a tree just prior to hospitalization. Neurological examination revealed bilateral papilledema, left homonymous hemianopsia with macular sparing, and left supranuclear facial paresis. There was no weakness. Tendon reflexes were increased on the left, but there were no pathological reflexes. There was no disturbance of co-ordination, but the left arm tended to pronate and move up and out. There were no sensory disturbances. Electroencephalogram revealed a slow-wave focus in the posterior portion of the right hemisphere. A right carotid arteriogram revealed tumour stain in the temporo-occipital area. Psychological examination revealed no aphasic defect in either spoken or written language at this time. A disturbance in reproduction of distances in vertical or forward direction was the only perceptual-motor deficit found. Higher mental processes were intact.

In view of the present findings and the history of excision of a melanoma of the chest two years ago, a right temporal craniotomy was performed on April 8, 1954. There was an extensive excision of tissue from the posterior temporal and parietal lobes with partial removal of a metastatic melanoma. No sign of language impairment was found on the day following operation. Five days later, speech and reading were again found to be intact.

The patient reported that he had always been left-handed for all activities, including writing. His mother and one sister are also left-handed, but his father, his three brothers, and his other sister are right-handed. After the radical removal of left chest muscles, with severe scarring near the left shoulder, the patient tended to develop his right hand in the past eighteen months. He was now using this hand predominantly in his work as a machinist. However, he continued to write with his left hand, and was extremely clumsy in attempts to write with the right hand.
Case 7.—Monoplegia, left arm, and transient aphasia following right frontal brain injury. H. C., a 22-year-old officer was admitted to Cushing General Hospital, January 17, 1946, ten months after receiving shell fragment wounds of the brain, eyes, and left arm. At operation, the day after injury, there was found a compound comminuted fracture of the right frontal region with extrusion of the frontal lobe. Patient was unconscious for twelve days following the injury, and then was found to have paresis of the left arm and aphasia, which cleared in six weeks. No details of the speech disturbance available. He suffered the first of a series of lapses of consciousness without convulsions in November 1945.

On examination in January 1946, he was found to be blind in the left eye, with metallic foreign bodies in the orbit. There was mild atrophy and weakness of the left arm and, in addition, a complete left ulnar nerve paralysis, the latter due to the wound in the arm itself. Neurological examination revealed no reflex changes, motor or sensory abnormalities, except those of the left arm and hand. Electroencephalogram revealed a slow wave focus in the right fronto-temporal area. A pneumoencephalogram made in August 1945 had revealed marked dilatation of the ventricular system and especially marked dilatation upwards of the body of the right lateral ventricle. No residuals of aphasia could be found on detailed examination in June 1946.

The patient had been predominantly left-handed, and had always written with the left hand, although he preferred to bat a ball in right-handed fashion. In the fifteen months since injury he had learned to write easily with the right hand.

Case 8.—Left hemiparesis and aphasia after right-sided injury. O. L., a 24-year-old soldier was transferred to Cushing General Hospital in January 1946 with a history of a right temporo-parietal penetrating injury received in March 1945. The shell fragment had traversed the right lateral ventricle and lodged in the medial wall of the ventricle. Patient remained unconscious for thirty-six hours, and was operated on ten hours after the injury. On examination, left hemiplegia, left homonymous hemianopsia, and motor aphasia were found. The patient reported he remained severely aphasic for two months. He also suffered grand-mal seizures from the time of injury.

On examination in January 1946, slight articulatory difficulty was found, without definite aphasic residuals. Neurological examination revealed left hemiparesis with hyperactive reflexes and positive Hoffmann, Rossolimo and Oppenheim signs on the left. Sensation was reduced in all modalities on the left side. No neurological abnormalities on the right. Electroencephalogram showed slight slow wave focus in the lower right parietal area with build-up on hyperventilation. Pneumoencephalogram of April 25, 1945, showed generalized dilatation of the ventricular system, most marked in the region of the right posterior horn. More air accumulated in the region of the right Sylvian fissure than the left.

The patient was completely left-handed before his injury. Familial handedness is not reported.

Case 9.—Right temporo-parietal wound without aphasia. P. M., a 32-year-old officer was admitted to Cushing General Hospital, February 21, 1946, with a history of a penetrating wound received in August 1944. At debridement, one hour after injury, depressed bone fragments and lacerated brain tissue in the right temporo-parietal region were removed. The patient was not unconscious at the time of the injury, and had no paralysis or sensory loss. However, he had no memory for events in the two-day period after injury, and he showed some confusion for four or five days. In January 1945 he had a first grand-mal seizure.

On examination in February 1946 there were no neurological abnormalities apart from deafness of the right ear. There was a skull defect measuring 3 by 3½ cm., in the right parietal region. Pneumoencephalogram of December 11, 1945, revealed very slight dilatation and possible displacement to the right of the posterior horn of
the right lateral ventricle. Electroencephalogram was abnormal with a slow wave focus in the right temporal area. No evidence of aphasia could be detected, although patient recalled a period of stuttering after his injury. His Verbal I.Q. was 133, Performance I.Q., 130.

The patient was left-handed by preference, using the left hand for writing until he was 15, when his father made him change to the right hand in writing. Family hand preference is not known.

Case 10.—Left hemiplegia and mild aphasia following right parietal injury. H. M., a 31-year-old soldier, was admitted to Cushing General Hospital, December 6, 1945, nine months after receiving a penetrating shrapnel wound in the right parietal area. Debridement was carried out twenty-eight hours after injury, and left hemiplegia and mild motor aphasia were found after recovery from unconsciousness lasting three weeks. He started having focal attacks in the left arm in June 1945, and grand-mal seizures with focal onset in the left arm, but without speech disturbances, in December 1945.

On examination in December 1945, patient had a hemiparesis of the left side, with increased tone, increased tendon reflexes, diminished abdominal reflexes, Babinski's and Oppenheim's sign on that side. Position sense in fingers and toes, two-point discrimination and figure-writing were disturbed on the left, but other sensory modalities were normal. Left homonymous hemianopsia was found at this time. Electroencephalogram was normal. Skull film showed no defects apart from metal plate over right parietal bone defect. Verbal I.Q., 133, Performance I.Q., 73.

Language examination revealed a stutter which patient claimed to have had all his life. A mild dysgraphia but no other aphasic residuals were found. The patient had been left-handed in all respects but writing, for which he always used the right hand.

Case 11.—Left hemiparesis and transient aphasia after right parietal injury. J. B., a 21-year-old soldier was admitted to Cushing General Hospital on December 11, 1945. On March 26, 1945 he had received a perforating shrapnel wound with entrance in the right parietal region and exit in the right occipital, and involving the frontal, parietal, temporal and occipital lobes. Craniotomy and debridement were performed the next day. Patient remained unconscious for four days, then was found to be hemiplegic on the left, mildly aphasic, and he had a left homonymous hemianopsia. Grand-mal seizures started in October 1945.

Neurological examination of December 1945 showed left homonymous hemianopsia, left spastic hemiplegia, increased tendon reflexes, positive Hoffman and Oppenheim signs on the left, and general reduction in all sense modalities on the left, including absent position sense. Electroencephalogram showed a slow wave focus in the right temporal area, and pneumoencephalogram in January 1946 showed generalized dilatation of the ventricular system, with marked enlargement and distortion of the right lateral ventricle. Language examination at this time revealed no aphasic residuals, apart from a slight spelling defect. The patient said that his word-finding difficulties cleared up a month and a half after his injury.

Patient had been completely left-handed before injury, but family's handedness was not given.

Case 12.—Transient right hemiplegia and mild aphasia following left frontal brain injury. J. M., a 24-year-old officer was admitted to Cushing General Hospital February 5, 1946, seven months after he was struck by a bullet in the left frontal region. The wound was debrided of bone fragments and macerated brain tissue on the date of injury. Patient had a right hemiplegia and mixed aphasia. His first grand-mal seizures occurred eight days later. Paralysis cleared within two months, but a mild word-finding difficulty persisted.

On examination in February 1946, neurological examination was normal throughout. Skull film revealed the metallic plate covering the frontal bone defect.
encephalogram showed a borderline dysrhythmic record without definite focus. Mild aphasic hesitancy in speech was still observed at this time, but there was no defect in written language.

The patient reported he performs most skilled acts with the left hand, but writes with his right. No report of family handedness is available.

Case 13.—Left temporal lesion without aphasia. J. J., a 24-year-old soldier was admitted January 16, 1946, having received a gutter type wound of the left temporal region on June 18, 1944. He was unconscious for three days, and upon awakening he had a complete amnesia for his past life and spoke slowly with a stutter. In October 1944 neurological examination was essentially negative with the exception of a partial peripheral left upper facial nerve paralysis. Pneumoencephalogram revealed a slight dilatation of the left lateral ventricle. The patient was suffering from post-traumatic psychomotor epilepsy, consisting of periods of disorientation and confusion, with amnesia.

Examination January 1946 showed a peripheral paralysis of the left frontalis muscle, and bilaterally reduced auditory acuity, while the rest of the neurological examination was negative. The patient continued to speak slowly, with a severe stutter. Detailed psychological examination led to the conclusion that the stutter was not a residual of any aphasia. Verbal I.Q., 127, Performance I.Q., 119.

Electroencephalogram performed September 5, 1945, following pitressin hydration, showed high voltage abnormal, slow activity directly overlying the skull depression in the left temporal region. Routine electroencephalogram of January 22, 1946, was normal.

The patient reported himself as naturally left-handed, except for writing. He was forced to use the right hand when he started school. No information was given as to handedness in patient's family.

Ballantine's Case (See Bibliography).—This patient and her mother were interviewed by the authors in January 1954, through the courtesy of Dr. Ballantine, and the following supplementary information was obtained. The patient now had a right hemiplegia, spoke and understood normally, but reported some slowness in reading. Her post-operative aphasia had lasted six months, and was chiefly amnesic in character. Her original dysgraphia cleared rapidly.

This patient had always been extremely left-handed in all activities, including writing. She had noticed as a child that she could do manoeuvres in dancing with her left foot, which were awkward with the right. Several trials at sighting with both eyes open revealed that she was right-eyed, however.

The family history was one of left-handed preference on the part of two brothers for most activities. Neither brother is as strongly left-handed as the patient. The patient's mother was predominantly right-handed, but had observed more than usual left-sided motor facility in herself. Her father (patient's grandfather) had been ambidextrous, but all of the eight siblings were right-handed. The patient's father is right-handed.

Short Description of Cases of Left-handers with Lesions of Language Areas

Group 1.—Left-handers with Right Cerebral Lesions and Aphasia, 50 cases.

Jackson (1866). Left hemiplegia and aphasia in a man who had previously suffered right hemiplegia without speech defect. Left-hander, but no mention of writing hand or aetiology.

Jackson (1868). Severe aphasia with left hemiplegia. Patient was left-handed but wrote with right hand. His four children by first wife were left-handed; two by second wife were not. No information on aetiology.
Wadham (1869). 18-year-old boy; left hemiplegia and severe aphasia. Patient wrote with left hand and had four left-handed brothers. Autopsy: Island of Reil on right replaced by fluid-filled cavity. Left hemisphere intact.

Ogle (1871). (Three cases). Three cases of left hemiplegia with aphasia in left-handers out of a series of 100 aphasic hemiplegics.

Russell (1874). 69-year-old man. Sudden onset motor aphasia and left hemiplegia. Patient had learned to use right hand for writing with great difficulty. No information on etiology.


Cuffer (1880). Left hemiplegia and sensory aphasia; rapid recovery. Patient wrote and ate with right hand. Otherwise left-handed. Onset after confinement with third child.

Habershon (1880). 52-year-old man. Progressive motor aphasia for seven weeks, followed by death. Autopsy: Glioma of right insula and posterior part of third frontal convolution. Life-long left-hander. Not known if he used left for writing.

Jackson (1880). Sailor with sudden onset of left hemiplegia and aphasia, presumably of vascular origin. Patient was left-handed, except for writing and sewing sails, in which he was forced to use the right hand.

Dally (1882). Left hemiplegia and aphasia, following apoplectic attack. Wrote with paragraphic errors. After two years, speech and reading recovered but spelling defective.

Fére (1885). Left hemiplegia and aphasia. Patient used right hand for writing. No details regarding etiology or duration of aphasia.

Hale White (1887). 50-year-old, left-handed woman. Left hemiplegia and aphasia. Autopsy: Haemorrhage into left internal capsule and lenticular nucleus.

Bell (1895). Left hemiplegia and aphasia probably due to embolus caused by mitral insufficiency. Died two days after onset. No autopsy.

Runeberg (1898). Case of motor aphasia with left hemiplegia in a left-handed woman consequent to thrombosis of arteria fossæ sylvii.

Left-handed man with aphasia (type unspecified). Jacksonian seizures, crying spells. Autopsy: Diffuse meningo-encephalitis with area of softening in right lower Rolandic region.

Mingazzini (1910). 60-year-old man, life-long left-hander suffers sudden left hemiplegia and complete aphasia, improving to partial sensory aphasia. Autopsy: Destruction posterior portion of insula on right and of dorsal portion of the gyrus temporalis profundis.

Dejerine (1914). Illiterate man becomes aphasic and remains so four years until death. Autopsy: Massive destruction of right hemisphere.

Dejerine (1914). Aphasia with recovery, in a left-handed patient who wrote with right hand. Autopsy: Such severe destruction of right hemisphere that the recovery of speech must be attributed to substitution of left hemisphere.

Head (1926). Traumatic right cerebral lesion with semantic aphasia, no hemiplegia.


Chesher (1936). (Two cases.) Tumours: Discrete lesions in right hemisphere language areas with aphasia, type unspecified. Both cases pure left-handers.

Chesher (1936). (Three cases.) Tumours of right hemisphere language areas with aphasia, type unspecified. Presence of hemiplegia not indicated. All were left-handed except for writing. Classified by Chesher as having mixed handedness.

Brain (1941). Right parieto-occipital abscess. Mixed aphasia. Also has minor hemisphere syndrome with respect to neglect of space on one side.

Kennedy (1947). Right cerebral injury with left hemiplegia, complete motor and partial sensory aphasia for two years, improving with therapy. Pneumoencephalogram and exploratory trephination verify right frontal hematoma and cerebral atrophy.

Nielsen (1948). Right CVA with left hemiplegia and aphasia with good recovery. Later left CVA and right hemiparesis with agraphia for right hand only. Patient had used right hand since childhood for writing.


Conrad (1949). Right parietal lesion with left hemiplegia and transient aphasia.

Conrad (1949). Right parieto-temporal lesion with left hemiplegia and motor aphasia.

Conrad (1949). Right parietal lesion with sensory aphasia and right [sic] hemiplegia.
Conrad (1949). (Two cases.) Right central lesions, one motor and one sensory aphasia. Both have left hemiplegia.

Conrad (1949). Right fronto-temporal penetrating wound with sensory aphasia; no hemiplegia.

Roberts (1951). (Three cases.) Right cerebral operations followed by aphasia in left-handed patients. No details on writing hand or on type or duration of aphasia.

Humphrey and Zangwill (1952a). Right parieto-occipital trauma with mild mixed aphasia and minor hemisphere syndrome for neglect of space and dressing dyspraxia. Good recovery and adoption of right as preferred hand.

Humphrey and Zangwill (1952b). (Three cases.) Right cerebral head wounds with aphasia; recovered. All with left hemiplegia. One wrote with left hand, two with right.

Present Authors. (Five cases.)

Group II.—Left-handers with Left Cerebral Lesions without Aphasia, 8 cases.

Westphal (1884). Left-handed patient with huge gliosarcoma of left temporal lobe and softening of island of Reil, without aphasia.

Chesher (1936). Tumour in language area of left hemisphere without aphasia. Patient is strongly left-handed and from a right-handed family. (Presence of hemiplegia not mentioned.)

Conrad (1949). (Five cases.) Left cerebral wounds of language areas without occurrence of aphasia. Three with paresis of right arm or arm and leg; two without.

Present Authors. (One case.)

Group III.—Left-handers with Left Cerebral Lesions and Aphasia, 53 cases.

Paget (1887). Left-handed pastor who wrote with right hand. Aphasia and right hemiplegia, after three successive vascular accidents.

Wood (1889). 50-year-old man with progressive mixed aphasia and right hemiparesis. Wrote with right hand but was otherwise left-handed for manual work. Autopsy indicates left-sided lesion, but no details given.

Dickinson. 49-year-old marine engineer admitted to hospital after a convulsion, resulting from blow on head. Complete motor aphasia and right hemiplegia, but continued to write fluently with left hand. Recovered completely in a few weeks.
Bramwell (1899). 36-year-old left-hander who wrote with right hand. While straining at the stool was suddenly seized with right hemiplegia and severe motor aphasia with slight impairment of comprehension and written language. Remainder of family right-handed.

Miyake (1909). 4-year-old left-handed child. Developed aphasia with left cerebral lesion. Operation followed by recovery.

Liepmann (1912). Left-handed woman admitted to hospital for hearing voices. Then suffers right hemiplegia, right hemianopsia, and severe disturbance of word-finding, reading and writing. Slight difficulty in repetition. Autopsy: Enormous lesion of third frontal convolution, central convolution, insula, temporal lobe, parts of external capsule. Right hemisphere intact.

Ardin-Delteil, Lévi-Valensi, Derrieu (1923). Severe right hemiplegia, predominantly sensory aphasia, improving markedly over a five-week period, and presumably vascular aetiology. Patient strongly left-handed except for writing. Sister also left-handed.

Long (1913). Right hemiplegia and sensory aphasia without motor aphasia following destruction of Broca's and adjacent areas.

Tilney (1936). Left cerebrovascular accident with aphasia; agraphia most severe for the right hand. Patient had always written with right hand, but otherwise completely left-handed. Half of family also is left-handed. Complete recovery of speech and recovery of writing with left hand, with right hand remaining apraxic for writing.

Weisenburg and McBride (1935). Tumour of left temporal lobe operated. Patient was left-handed except for writing and sewing. (Classified as ambidextrous by Weisenburg and McBride for this reason). Post-operative slight right hemiparesis; predominantly sensory aphasia.

Weisenburg and McBride (1935). Exploratory operation, left temporo-parietal area. Patient (classified by Weisenburg and McBride as ambidextrous) was originally left-handed, but switched to right in school. Post-operatively right hemiparesis and marked, predominantly motor aphasia, which improved rapidly.


Chesher (1936). (Five cases.) Tumours of left hemisphere language areas. Patients are described as claiming to be originally left-handed, but to have acquired a number of right-handed skills. (Classified by Chesher as showing mixed-handedness.) No mention of type of aphasia or presence of paralysis.

Conrad (1949). (Three cases.) Left cerebral lesions (traumatic) followed by transitory aphasia; two cases with right hemiplegia.
Conrad (1949). (Seven cases.) Left cerebral lesions (traumatic) followed by severe aphasia; include four motor aphasics with right hemiparesis, one motor, one amnesic, and one sensory aphasic without paralysis.

Roberts (1951). (Thirteen cases.) Left-handed patients who became aphasic after operation on left hemisphere. No details on writing hand or on type or duration of aphasia.

Wepman (1951). (Two cases.) Left-handed patients with right hemiplegia and aphasia. No other details mentioned.

Humphrey and Zangwill (1952). Penetrating head wound, left hemisphere. Permanent, severe dysphasia. Patient was believed to have written with right hand, but was otherwise strongly left-handed.

Humphrey and Zangwill (1952). (Four cases.) Left cerebral penetrating head wounds with aphasia, recovered. Three hemiplegic, one hemiparetic.

Ballantine (1953) and personal communications. 15-year-old, completely left-handed girl operated for abscess of left parietal lobe. Moderate amnesic aphasia and dysgraphia, recovered after six months, with more persistent dyslexia and dyscalculia. Two brothers partly left-handed.

Present Authors. (Five cases.)

GROUP IV.—Left-handers with Right Cerebral Lesions of Language Areas without Aphasia, 12 cases.

Kennedy (1916). Penetrating, massive right-sided temporo-parietal wound, without an aphasic symptom. Patient was very strongly left-handed, even for eating, but used right in writing. Family was right-handed.

Kennedy (1916). Skull fracture with bone defect in right temporo-occipital area; transient left hemiplegia, but no aphasia. No other left-handers in family.

German and Fox (1934). Resection of right frontal lobe without speech involvement. Patient's family includes five left-handers, six right-handers. Patient was left-handed except for eating and writing.

Conrad (1949). (Five cases.) Injury of language areas of right hemisphere without aphasic symptoms. Two with left hemiparesis and three without paralysis.

Humphrey and Zangwill (1952). Severe mid-parietal wound on right without aphasia, simply a non-aphasic dysarthria. Had left hemiplegia.

Milner (1952). Excision of right temporal lobe because of epileptogenic focus. No speech disturbance either on electrical stimulation of exposed cortex or after operation.

Present Authors. (Two cases.)
Summary

(1) 110 cases from the literature and thirteen new cases of left-handers with unilateral lesions of the language area were collected.

(2) Language was found to be disturbed by lesion of the left hemisphere in 53 per cent of all left-handers.

(3) Cerebral laterality for language and handedness are not directly linked, and one does not determine the other; left cerebral laterality for language is more prevalent than right-handedness, and right cerebral laterality for language much less prevalent than left-handedness.

(4) Hypothetical distribution curves for handedness and language laterality are presented.

(5) The theoretical implications of the problem are discussed.

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1954. Present Authors. This paper. 5 cases.
II.—Left Cerebral Lesions without Aphasia. 8 cases


1954. Present Authors. This paper. 1 case.

III.—Left Cerebral Lesions with Aphasia. 53 cases


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