PAROXYSMAL DYSPHASIA AND THE PROBLEM OF CEREBRAL DOMINANCE

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The problem of the functional specialization of the two cerebral hemispheres has its origin in studies of aphasia. In a report which received little attention at the time, Marc Dax maintained (1836) that aphasia was associated with lesions of the left hemisphere. Broca admitted in his earlier writings that both hemispheres might be involved in language functions but in his later observations he insisted that the left hemisphere was dominant for this function. This suggestion was soon confirmed by the observations of Bastian, Hughlings Jackson, and Wernicke.

However, exceptions to this rule were soon noticed. In fact, as from 1865, Broca conceded that there might be a small number of individuals "who were exceptions and would speak with the right hemisphere", and he approached present-day explanations when he wrote:

" Just as there are left-handers in whom the inherent predominance of the motor activity of the right hemisphere confers a natural and irreversible predominance 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 convolutions of the right hemisphere will reverse the order of the phenomena which I have just described."

Broca thus emphasized the correlation between cerebral dominance and contralateral hand preference; left hemisphere lesions in right-handers and right hemisphere lesions in left-handers were supposed to result in aphasia.

But gradually, as evidence in contradiction to this principle arose, either in relation to aphasia (crossed aphasia) or in relation to agnosia, there developed a tendency to abandon the concept of absolute dominance and to substitute the idea of lateral dominance which was not necessarily the same for different functions, or else the idea of a simple functional predominance of one of the hemispheres. Thus Nielsen (1946) speaks of a major and minor hemisphere for a particular function, suggesting thereby a simple quantitative difference in functional potential between two symmetrical zones.

On the other hand, the question has been tackled from the point of view of the handedness of the subject. The classical rule (aphasia with left-sided lesions in right-handed patients; aphasia with rightsided lesions in left-handed patients) rapidly encountered exceptions which were designated " crossed aphasia". Most of these anomalous cases were left-handers with right hemiplegia and it has been suggested that these were actually either ambidexters or left-handed people who had learned to write with the right hand. Crossed aphasia in fully right-handed people appears, however, to be much more rare.

Thus Ettlinger, Jackson, and Zangwill (1955) were able to trace only 15 published cases of dysphasia occurring in association with a right hemisphere lesion in ostensibly right-handed patients. Three of these were ambidextrous and in nine there was familial evidence of left-handedness. They state in fact that they could find only two well authenticated cases which were not ambidextrous and which had no familial left-handedness. The personal case reported by Ettlinger, Jackson, and Zangwill regarded himself as right-handed but appeared to have greater facility with the left hand than is usual in right-handed people.

Recent studies (Conrad, 1949; Humphrey and Zangwill, 1952) suggest that cortical organization for language is different in left- and right-handers. In fact, the study of aphasic left-handers shows that cerebral dominance is far from being as definite in left-handers as in right-handers, since in the former the lesion may sometimes be in the right hemisphere and sometimes in the left. Of the 18 aphasic lefthanders of Conrad, 10 had a left-sided lesion, seven a right-sided lesion, and in one case the lesion was bilateral. In the 10 left-handed cases reported by Humphrey and Zangwill with unilateral cerebral damage to the supposed language area, dysphasia in a permanent form was present in five subjects, with left-sided lesions and in only one of the five subjects with a right-sided lesion. Moreover, the

severity of the disturbance was less in the second group.

Wepman (1951), on the basis of his personal cases and a critical review of cases in the literature, reached the radical conclusion that permanent aphasia depends solely on left hemisphere lesions irrespective of manual preference.

In a recent paper on aphasia in left-handers, Goodglass and Quadfasel (1954) on the basis of 13 personal cases and 110 cases in the literature made a more refined analysis and drew more cautious conclusions. They found that in left-handers 50 out of 62 cases with right hemisphere lesions and 53 out of 61 cases with left hemisphere lesions suffered a dysphasia.

Their material suggests that aphasia following a lesion in the hemisphere ipsilateral to the preferred hand is much more frequent in left-handers than in right-handers, since aphasia occurred following a left cerebral lesion in 80% of the left-handers studied. For these authors, there is no direct and necessary relationship between manual preference and cerebral lateralization of language functions; they maintain that left cerebral dominance is more general than right-hand preference and that right cerebral dominance is much less frequent than left-handedness.

It might be added that, to the extent that their data is a fair sample of left-handed patients with appropriately situated unilateral lesions, it can be concluded that many left-handed people have bilateral representation of language. For both the left-sided and the right-sided lesions the incidence of dysphasia significantly exceeds 50% (80% and 86% respectively). Such a degree of overlap is not consistent with unilateral representation of language in most left-handed people.

All of these studies have been concerned with dysphasia occurring in association with a permanent cerebral lesion. We have undertaken a study of paroxysmal dysphasia in the hope that the theoretical position as to cerebral dominance for language functions might be advanced a little further by an analysis of the incidence of dysphasia which occurs in association with an abrupt and short-lived disturbance of cerebral function. The present communication reports evidence concerning the incidence of such paroxysmal dysphasia occurring as an aura to an epileptic fit or as an epileptic equivalent. This has been studied in relation to the laterality of the focus of cerebral disturbance and in relation to the handedness of the patient.

Incidence of Paroxysmal Dysphasia

The examination of the case records of patients suffering from epileptic auras or equivalents was carried out with the aid of a punch-card system. Punch-card records had been kept of all patients seen in the service since 1947. All these patients had been examined by one of the authors (H. H.) and all the cards had been punched by the examiner. All cards punched for "epileptic aura or equivalent" were selected and from these were selected all cards punched for unilateral focus of cerebral dysfunction involving the cerebral cortex or the subcortical white matter. The detailed case notes of these patients were then examined to verify the evidence for epilepsy and for the localization of the cerebral disturbance. Only cases which conformed to at least two of the following criteria were accepted as having unilateral cerebral dysfunction: (1) Jacksonian type seizures; (2) cerebral pathology verified at operation or necropsy; (3) unilateral sensory or motor signs on clinical examination; (4) unequivocal lateralized E.E.G. focus; (5) unilateral lesion demonstrated by air-encephalogram or arteriogram.

Over 3,000 cases were checked with the punch-card system and of these 126 were found, on reference to the detailed case notes, to have had epileptic auras or equivalents and to conform to the criterion defined above as to the localization of the focus of cerebral dysfunction; 97 of these were righthanded and 29 were either left-handed or ambidextrous. Table I shows the incidence of paroxysmal

TABLE I INCIDENCE OF PAROXYSMAL DYSPHASIA

-	Side of Epileptic Focus	Le	ft	Right		
Right-handers	Type of Dysphasia	Expressive	Receptive	Expressive	Receptive	
	Aura with dys- phasia	31	14	4	1	
	Aura without dysphasia	3	1	30		
	Total aura	63		34		
Left-handers	Aura with dys- phasia	17	1	9	1	
	Aura without dysphasia	1		2		
	Total aura	1	8	11		

dysphasia in these cases, occurring during an epileptic aura or equivalent. Expressive dysphasia in the table refers to a severe interference with speech and in most cases amounted to a complete aphemia. Receptive dysphasia refers to either a severe defect of auditory comprehension (temporary deafness excluded) or an alexia. Only two cases had such a paroxysmal alexia. With the exception of one right-handed patient with a left temporal lesion, all cases that presented a receptive defect also presented an expressive defect.

Expressive Defects.—The most striking finding which emerges from these results is the fact that,

although only 31 out of the 63 right-handed cases with left hemisphere dysfunction had paroxysmal expressive dysphasia, as many as 17 out of the 18 left-handed cases with similar cerebral pathology had a paroxysmal expressive dysphasia. This difference in the incidence of dysphasia between the left-handers and the right-handers is statistically significant on a χ^2 test ($\chi^2 = 11.1$, P<0.001). In the case of right-sided cerebral dysfunction, the incidence of expressive dysphasia is again more marked in the left-handers (nine out of 11) than in the right-handers (four out of 34). This latter observation is, of course, consistent with the theories which attribute right-sided cerebral dominance for language to left-handers. However, the findings for left-sided dysfunction mentioned above are in apparent contradiction to such theories. Moreover, when the results for the left-handers are examined in isolation it is seen that the side of the responsible focus has no obvious effect on the incidence of paroxysmal dysphasia: 17 out of 18 for left-sided lesions as against nine out of 11 for right-sided lesions. Furthermore, it is noteworthy that in both groups of left-handers (left-sided lesions and rightsided lesions) the incidence of dysphasia is well above 50%. This fact makes it extremely improbable that the results for left-handers could be accounted for in terms of some left-handers being left hemisphere dominant for language and others being right hemisphere dominant. The amount of overlapping is great enough to suggest that most left-handers are liable to have a paroxysmal dysphasia from a focus of dysfunction situated in either hemisphere. This is consistent with Goodglass and Quadfasel's (1954) material on permanent dysphasia in left-handers.

In contrast to this stand the results for righthanders. A left hemisphere focus results in a paroxysmal expressive dysphasia in 31 out of 63 cases, whereas a right hemisphere focus results in paroxysmal dysphasia in only four out of 34 cases. If the left- and right-handers are compared irrespective of the side of the focus, it is seen that, whereas 61 out of the 97 right-handers have no dysphasia during their aura or its equivalent, this can be said of only three out of the 29 left-handers.

Thus it is clear that when an epileptic aura or equivalent occurs as a result of unilateral cerebral dysfunction it is very much more likely to be associated with a paroxysmal dysphasia in lefthanders than it is in right-handers, irrespective of the side of the focus. It would seem also that in right-handers the side of the focus is much more relevant to the incidence of paroxysmal dysphasia than is the case in left-handers. These results must be considered in relation to the special nature of the disturbance which occurs if they are to be reconciled with existing evidence as to cerebral dominance for language based on observations of permanent dysphasia. This is considered more fully below.

It may be noted that in the cases reported here there is an unusually large proportion of left-sided lesions both for left-handed and for right-handed patients. This suggested to us the possibility that epileptic auras and equivalents may be more common with left-sided than with right-sided foci. Accordingly the punch-cards were again checked and all cases with adequately localized unilateral cerebral lesions were selected and these cards were divided into two groups according to the side of the lesion. Each of these two groups was divided into two groups corresponding to the presence or absence of epileptic auras or equivalents. The result is shown in Table II,

TABLE II CONTINGENCY OF INCIDENCE OF AURA UPON SIDE OF LESION

		Lesion	
	Left	Right	Total
Aura present	81	81 45	
Aura absent	142	146	288 414
Total	223	191	

where the contingency of incidence of aura or its equivalent upon the side of the responsible lesion is found to be statistically significant ($\chi^2 = 9 \cdot 2, P < 0 \cdot 01$). There is no significant difference between the total incidence of left- and right-sided lesions ($\chi^2 = 2 \cdot 4, P > 0 \cdot 1$).

It is thus clear that a disturbance of the left hemisphere is particularly apt to result in the sequence of cerebral events involved in an epileptiform aura or its equivalent. Presumably the behaviour and experience which is disturbed during an aura is more profusely represented in the left than in the right hemisphere and so paroxysmal left hemisphere dysfunction is more liable to produce clinically obvious changes.

Receptive Defects.—It is immediately apparent from Table I that a paroxysmal receptive dysphasia during an aura or its equivalent is a less frequent occurrence than is a paroxysmal expressive dysphasia. Receptive paroxysmal dysphasia would also appear to be particularly infrequent in left-handed patients (1/18 with left-sided lesions; 1/11 with right-sided lesions). In the case of right-handed patients the frequency of receptive dysphasia is related to the side of the lesion (14/63 with left-sided lesions as against 1/34 with right-sided lesions). Thus the results concerning receptive defects resemble those concerning expressive defects in that the incidence of the defect is related to the side of the lesion in right-handers but not in left-handers, but differ from them in that receptive defects are less frequent in left-handers than in right-handers whereas expressive defects are more frequent in left-handers than in right-handers.

Exceptional Cases

It is of some interest to consider in more detail the cases which depart from two of the general principles suggested above: (1) that a paroxysmal receptive dysphasia is very rare in left-handed patients; (2) that any kind of paroxysmal dysphasia is very rare in association with right-sided lesions in right-handed patients.

Left-handed Cases with Comprehension Defect.— Two of our left-handed patients showed a definite comprehension defect. One other patient was unable to understand what was said to him during the aura but questioning made it clear that this was a complete paroxysmal deafness and was not specific to language comprehension. This case is not reported here.

In the first of these two cases with comprehension defect there was a tumour of the posterior part of the left temporal lobe (oligodendroglioma). The epuivalents consisted of an abrupt inability to speak, understand, or read. Pre-operatively the only permanent language difficulty was a very mild amnesic dysphasia. However, two years after the operation there was a considerable degree of amnesic dysphasia, a literal alexia without agraphia, paralexia in reading words, and an agnosia for colours. Verbal comprehension was consistently intact, even for complicated orders. The patient was also able to imitate correctly the movements of the examiner in Head's hand-eye-ear test.

The other case was a patient with a right temporal glioma who had generalized convulsions and loss of consciousness. After a few weeks these changed in character. The seizure started with numbness of the left hand leading to numbness of the whole of the left side of the body. At the same time his speech became indistinct and he was unable to understand what was said to him. Except for difficulties occurring during seizures, the only disturbance of language was mild word-finding difficulties on naming tests and a dysgraphia which was marked by both literal and verbal errors. There was a mild constructional apraxia. At no time were there any difficulties of language reception apart from during seizures. There was reduced muscular tone on the left side (hemihyperextensibilité of André Thomas) but no disturbance of sensation.

These last two cases would appear to be of special interest in so far as there is a complete absence of permanent comprehension difficulty although the lesions were extensive, progressive, and involved the temporal lobe. Comprehension defects were, however, present in a paroxysmal form during epileptic equivalents and these are our only cases of left-handed patients showing a paroxysmal receptive defect of language.

Right-handed Cases with Right-sided Lesions.— Four right-handed patients showed a disturbance of language functions during an aura or equivalent resulting from a right hemisphere disturbance. In one of these cases (right temporal trauma) there was no true comprehension defect but a total deafness followed by tinnitus. The patient was, however, unable to speak. Ventriculography, E.E.G., and neurological examination all confirmed the rightsided focus of the lesion.

A second case (right temporal angioma) showed an unusual disturbance of language functions. In addition to a defect of articulation, which on occasions amounted to a complete aphemia, the patient, although able to understand what was said to him, reported that he could no longer " mentally deal with the rhythm of a sentence " and could not " imagine the sounds and pronunciation of words". The patient's description of his difficulty (quoted above) raised the possibility that this was largely a feeling of unreality involving the words and sentences which he read or imagined. This suggestion is supported by the fact that the patient also reported feelings of unreality which did not involve language.

The third observation in this group concerns a child of 12 who had had convulsive seizures for nine years following an anti-typhoid inoculation. These seizures were preceded by paraesthesia starting in the left thumb and spreading to the left side of the face. Clonus of the left upper limb followed and the fit then rapidly generalized. After a series of fits the child remained mute for two days but without any receptive difficulty. After each isolated fit she also showed some dysarthria of brief duration. The E.E.G. showed diffuse changes and the air-encephalogram was normal.

In these three cases of right-handed patients with right cerebral hemisphere disturbances there was definite disturbance of verbal expression. The fourth case, however, is exceptional. A right-handed patient, aged 50, with no left-handedness in the family, since the age of 2 had had fits which started with clonus of the left superior limb. These were succeeded at the age of 34 by equivalents which involved a complete aphasia: " I did not understand the person who was speaking to me for several minutes ", said the patient, " and I myself could not express my thoughts except for 'yes' and 'no'". Neurological examination revealed reduced muscular tone on the left side. The E.E.G. showed anomalies (a form of spike and wave) restricted to the right hemisphere. Skull radiographs showed calcification involving an area the size of a half-crown in the right posterior parietal region. Thus, in the first three cases of this group, the motor disturbances were the only disturbances of language and can possibly be described as "arrest of speech" rather than as genuine language disturbance. However, it must be emphasized, on the basis of the last observation, that it is possible for receptive language functions to be disturbed in a paroxysmal fashion in association with a right cerebral lesion in a righthanded patient.

Permanent Cases

For purposes of comparison we now present the observations made on 12 left-handed patients (all using the right hand for writing before the illness or accident) with a unilateral cerebral lesion involving the language area (as verified at necropsy, operation, or—in one case—on the basis of associated clinical evidence). These 12 cases comprise all left-handed patients with appropriate lesions who were seen in the neurological service during the period covered by the investigation of paroxysmal dysphasia. Two of these 12 cases had a paroxysmal dysphasia during an aura or its equivalent and are included in the results reported above.

Table III shows that, regardless of which hemisphere is involved, comprehension difficulties are very rare. In fact, such difficulties were present in only one case in a permanent form, and in this case were only moderate in severity. In two other cases comprehension difficulty was present in a transitory or paroxysmal form. Word-finding difficulties were more frequent since they occurred in a permanent form in five cases with left-sided lesions and one case with a right-sided lesion, and in a paroxysmal or transitory form in one case with a right-sided lesion. Agraphia occurred in a permanent form in two cases (one right-sided lesion, one leftsided lesion) and was doubtful in two other cases. Dyslexia was observed in one patient with a rightsided lesion and in three with left-sided lesions and was doubtful in a fourth case with a left-sided lesion.

On the other hand, irrespective of the site of the lesion, anterior or posterior, right or left, verbal expressive difficulties were observed in all but one of these cases in a greater or lesser degree, usually only in a paroxysmal or transitory form. These observations thus evince the mildness of permanent aphasic manifestations in left-handed people regardless of which hemisphere is damaged. The conservation of comprehension in all of these patients except one is very striking when compared with the incidence of expressive difficulties. This is in accordance with the evidence obtained from the data on epileptic auras in left-handers. It is worth mentioning also that constructional apraxic difficulties were fairly marked and permanent in these cases, both with left and right hemisphere lesions.

Conrad (1949), Humphrey and Zangwill (1952), and Goodglass and Quadfasel (1954) suggest that in left-handers cerebral lesions involving the so-called language area involve aphasic difficulties which in general are more transitory and less severe than in right-handers, and this irrespective of the hemisphere

Pe = Permanent

Case	Expression	Comprehension	Word-finding	Writing	Reading	Localization of Lesion
Left-sided lesion P (No. 1) L (No. 2) D (No. 3) S (No. 4) F (No. 5) B (No. 6) H (No. 7)	$ \begin{array}{c} s \\ + \\ + \\ + \\ + \\ + \\ + \\ + \\ + \\ + \\$	0 + 0 0 +(Pa) 0	+ (Pa, Pe) ++ 0 0 0 +++ ++ ++	0 0 ± ++++ ±	0 0 0 ± + + 0	Temporal Temporal Unilateral Temporo-parietal border Parietal Postero temporal Temporal (lobectomy anterior 2/13)
He (No. 8)	+ (Tr)	0	+	0	+++	Unilateral
Right-sided lesio N (No. 9) B (No. 10) P (No. 11)	ns + (Tr) + (Pa) + (Pa)	+ (Tr) 0 0	+ (Tr) + 0	0 +++ 0	0 0 0	Postero parietal Temporo-parietal Parietal (1st and 2nd con-
G (No. 12)	++ (Tr, Pa)	0	0	0	++	Parieto-temporo-occipital border

TABLE III* LANGUAGE DEFECTS IN LEFT-HANDED PATIENTS

Tr = Transitory Pa = ParoxysmalDefinite defects are marked with + and doubtful defects with +.

Defects are permanent unless otherwise stated.

* This table has previously been published in a modified form (Hécaen and Ajuriaguerra, 1955).

involved. Our personal experience confirms this point of view, although, in the case of permanent dysphasia, our figures are small and valid comparisons are difficult to make. In this context we may refer to Case No. 4 in the table, who showed, following severe cranial trauma, a paresis of the right arm and an aphemia which had persisted for several months but which remitted almost completely. This patient, who was left-handed but wrote and drew with the right hand before the accident, easily learned to write and draw with the left hand after the accident. He had fits preceded by an aphemic aura, without any difficulty of comprehension. Examination 16 years after the accident revealed a slight right pyramidal deficit associated with a gross sensory deficit of the right upper limb. a doubtful dyslexia and dysgraphia and a slight ideomotor apraxia. An operation carried out for the relief of the concomitant fits revealed an obvious area of cortical atrophy involving the left-parietotemporo-occipital border with an area of corticomeningeal adhesion immediately anterior. The aura preceding the fits was reproduced by stimulation of the post-central gyrus and an area corresponding to the sensory region of the arm was excised. There was no aggravation of language difficulties after the operation.

It is difficult to make a comparison between the duration of expressive aphasia in right- and lefthanders following anterior cortical ablation (precentral gyrus) for epilepsy or abnormal movements since, in both types of case, the disturbance clears up very rapidly. However, it seemed to us that in two such left-handed patients the disappearance of the dysphasic disturbance after the operation was particularly rapid and complete.

Discussion

Previous work on cerebral dominance for language functions has been almost exclusively concerned with the chronic disturbance in language functions which accompanies a permanent cerebral lesion. The present study has been concerned with an acute and short-lived disturbance of normal function, and it is reasonable to seek an explanation of the differences between the results reported here and those obtained by previous workers in terms of the differences in the physiological disturbance that has been studied. Our results are in complete accord with previous work in so far as they reveal a marked left hemisphere dominance for language in righthanded people. Our results concerning expressive dysphasia in left-handed patients are, however, at variance with the usual findings. Not only is paroxysmal expressive dysphasia in left-handers as frequent with left hemisphere disturbance as with

right hemisphere disturbance, but also the incidence of such a dysphasia in left-handers is much higher than in right-handers, irrespective of the hemisphere involved.

It has been suggested that dysphasia in left-handed patients with left-sided lesions is far from an exceptional observation (Humphrey and Zangwill, 1952; Conrad, 1949; Roberts, 1951; Goodglass and Quadfasel, 1954) and some authors have suggested that language may be more equally represented in the two hemispheres in left-handers than is the case with right-handers (Subirana, 1952; Chesher, 1936). Another observation which has been reported by more than one author is that recovery from dysphasia is more complete and more rapid in left-handers than in right-handers. This would, of course, be consistent with a more balanced cerebral representation of language with respect to the two hemispheres. Goodglass and Quadfasel tentatively suggested that left-handed people are more liable to a dysphasia than others, irrespective of the hemisphere damaged. However, the allegedly more transient nature of the dysphasic disturbances in left-handers would seem to point, in one sense, to relative invulnerability.

There is now fairly general agreement that it is not uncommon to find left-handed patients suffering from dysphasia in association with left-sided lesions and it has long been generally recognized that rightsided lesions may produce a dysphasia in lefthanders. Similarly, numerous cases have been cited where there is no dysphasia in left-handers, although an adequate unilateral lesion is present. Such a lesion may be either in the left or in the right hemisphere. It would appear that a unilateral lesion in either hemisphere may or may not produce a dysphasia in a left-handed patient. The negative cases in left-handers do not appear from the literature to be markedly more frequent with respect to one hemisphere than with respect to the other, but these negative cases certainly appear to be more frequent in left-handers with right hemisphere lesions than in right-handed patients with left hemisphere lesions. It is probably not an exaggeration to say that the evidence concerning permanent dysphasia favours the suggestion that a left-handed patient with a unilateral lesion of either hemisphere has a better chance of escaping a dysphasia, or of recovering from it more rapidly, than a right-handed patient with a left hemisphere lesion.

Ettlinger and others (1955) maintain that the negative cases of left-handed patients with unilateral lesions of the language zones imply "strictly unilateral representation of speech... in some sinistrals at least". However, although negative cases are of great importance, these authors'

conclusion is probably not an inescapable one. Bilateral representation of language does not *a priori* imply that a dysphasia must result if there is a unilateral lesion to the language area. It could equally be the case that bilateral language representation involves a certain degree of "equipotentiality" which would afford relative invulnerability to a unilateral lesion. The alleged mildness and transitoriness of dysphasia in left-handed patients is, in fact, consistent with such a possibility.

Our present findings stand in contrast to the evidence from permanent dysphasia discussed above. Ictal expressive dysphasia is more frequent in lefthanded than in right-handed patients irrespective of the side of the unilateral cerebral disturbance. It is, however, possible that differences in the cerebral organization of language in right-handed and lefthanded patients are such as would be consistent both with the evidence as to permanent dysphasia and with the evidence reported here. In the case of our results, the dysfunction was of abrupt onset and almost certainly of too short duration to permit any kind of cerebral adaptation to the interference. In the case of permanent dysphasia, however, the disturbance of language that is observed is the product both of the defect produced by the lesion and of any restitution of function resulting from compensatory activity of undamaged cerebral areas. The first type of observation may be compared to an immediate breakdown and the second to a more or less imperfect repair. Certainly, there is no necessity to assume that the sensitivity of a mechanism to acute interference is positively related to its incapacity for recovery. For example, where radical re-learning of a skilled performance is called for, this is most easily possible when the previous learning has not led to a highly integrated and well established ability. On the other hand, learning which has not achieved the status of a well organized skill is much more liable to break down under temporary stress.

In the case of dysphasia one is concerned less with the level of integration of the skill in using language in left- and right-handers than with the possibility of differences between the two groups in the degree of anatomical focalization of the skill, and it is conceivable that when a function is organized within a comparatively restricted area it may be on the one hand more resistant to acute interference and on the other hand less capable of spontaneous recovery than when such a function is organized more diffusely with respect to cerebral anatomy. We already have some evidence to suggest that language representation is more equally distributed between the two hemispheres in left-handed than in right-handed people. A possibility that should not be overlooked is that the bilateral representation of language in left-handers is only one aspect of the increased diffusion of language organization in these subjects. That is to say, it is conceivable that in left-handers the mechanism of speech involves a greater area of cerebral tissue within a single hemisphere than is the case with right-handers. However, even if this assumption is not made, we are in effect postulating a greater degree of equipotentiality in the cerebral mechanisms for language in left-handed and ambidextrous people than in right-handed people. But in view of our results this supposition involves us in the further assumption that greater equipotentiality is likely to involve greater sensitivity to acute interference of relevant function. So far as we are aware there is no experimental evidence either for or against this suggestion.

Since the time of the acceptance in general principle of Hughlings Jackson's (1932) concept of a hierarchy of levels of organization within the nervous system, there has been a tendency to regard the "higher levels" of nervous function as the prime example of organization, a view that is encouraged by the complexity and intelligence of the behaviour which these structures make possible. Jackson himself did not view the matter in this way. For him the highest centres are "the ravelled end", and he speaks of "the highest centres, which are the least organized". We find it tempting to echo Jackson's general comment on the highest centres with special reference to the organization of language functions in left-handers: "... the most complexly evolving . . . but the least perfectly evolved ". Such a scheme of organization might be expected to be comparatively unstable in the presence of acute interference, but nevertheless, more capable of reorganization following injury than its "more organized " counterpart in right-handed people.

In this context we would mention that there are certain resemblances between the cerebral organization of language in left-handed people and that in children. Thus, in children, aphasia following a right hemisphere lesion is more common than it is in adults, recovery from aphasia tends to be more rapid, and expressive defects-far more frequently than receptive defects-characterize dysphasia in a child (Guttmann, 1942). These differences between child and adult are strikingly parallel to those between left-hander and right-hander. It is to be expected that cerebral language mechanisms are less differentiated in children than in adults; this may also be the case in left-handed people. A less marked degree of "encephalization of function" for language in left-handers might well be associated with a slighter resistance to acute disturbance but

greater plasticity in respect of spontaneous recovery following brain injury.

Finally, mention should be made of the incidence of receptive defects. These are clearly rarer in a paroxysmal form than are expressive defects and also appear to be rarer in left-handers than in righthanders. We do not feel that the tentative theoretical interpretation which we have advanced in relation to the expressive defects should be extended to explain the incidence of receptive defects. The results are, in fact, somewhat paradoxical. On the one hand, paroxysmal expressive defects are much more frequent in left-handers than in right-handers irrespective of the side of the lesion. On the other hand, both paroxysmal and permanent comprehension defects are rarer in left-handers than in right-handers. It is clear that further evidence is required before interpretation can be extended. However, on the basis of present evidence, we would suspect that the question of the degree of focalization of language function is likely to be crucial. This, in our view, has three aspects. First, there is good evidence to suggest more nearly equal bilateral representation of language in the two hemispheres in left-handed than in right-handed people. Secondly, it is possible that there are differences between the two groups in the diffuseness of language representation within a single hemisphere. Thirdly, there may be important differences in both left-handers and right-handers in the degree of cerebral focalization of language reception on the one hand and of language expression on the other hand. Without attempting to uphold a definite theory, we would suggest that the differences in the incidence of paroxysmal and permanent defects of language expression and reception in right-handed and lefthanded patients may be more fully understood when more is known of the relative importance of the three aspects of focalization of function outlined above. It could be, for example, that language reception is generally more focally organized than language expression, that in left-handers this representation is bilateral, and that in left-handers all language functions are more diffusely organized within any one hemisphere than is the case with right-handers.

Summary

Current views on cerebral dominance for language are briefly reviewed on the basis of evidence from permanent dysphasia.

An analysis of the incidence of paroxysmal dysphasia occurring in association with epileptic auras and equivalents is reported. The data are analysed with special reference to the side of a unilateral focus of cerebral disturbance and to the handedness of the patient in 126 cases.

Paroxysmal expressive dysphasia occurred significantly more frequently in left-handed patients with aura than in right-handed patients with aura, irrespective of the side of the epileptic focus. In right-handed cases the incidence of expressive dysphasia was much greater in patients with leftsided disturbance than in those with right-sided disturbance. No such difference was observed in left-handed cases.

Paroxysmal receptive dysphasia was rare except in right-handed patients with left-sided cerebral foci. In this group the incidence was about half that of expressive dysphasia.

The findings are discussed in relation to evidence from cases of permanent dysphasia and it is suggested that the nature of the physiological disturbance involved may account for the differences between the results reported here and evidence based on cases of permanent dysphasia.

The tentative theoretical position is adopted that differences between left- and right-handed patients in the incidence and nature of dysphasia occurring in association with unilateral lesions or foci is consistent with differences between the two groups in the degree of cerebral specialization for language, both with respect to bilateral representation and with respect to representation within a single hemisphere.

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