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Neuropsychology and the Localization of Cognitive Function

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The brain being brain must try to establish laws.

Iain Crichton Smith

That the brain, and especially the cerebral cortex, is not anatomically homogeneous is immediately obvious on inspection; the primary concern of this chapter will be whether the brain, and particularly the cortex is *functionally* homogeneous, and if not, how we may ascribe particular functions to particular areas, and how such knowledge may be of use in psychology and psychiatry. A more extensive introduction to the problems of neuropsychology in general will be found in Kolb and Whishaw (1989).

Methodological Problems

Clearly the first priority if we are to have an adequate theory of cerebral *localization* is that we must be able to localize damage in the brain substance. In the absence of lesions of brain tissue (or alternatively of abnormal brain stimulation) we can conclude nothing about localization (and this was probably one of the principal errors of the phrenologists). Until recently, knowledge was based almost entirely on the traditional methods of observing the brain at post-mortem or at operation. The limitations of both are obvious. In the first case one may have to wait a number of years, there may be subsequent or progressive pathology, and there is no question of going back to make further observations in the light of unusual or surprising findings, while the only positive advantage is the ability to make a complete and, if necessary, microscopic analysis of the entire brain. In the case of operative lesions we cannot be assured of the completeness of a lesion, or of its extent, or of the non-existence of other pathology in unexplored areas, and of course we do not have the freedom to make lesions as would interest us or inform us; we are completely dominated by the therapeutic needs of the patient. Recent advances, which are already revolutionizing neuropsychology, allow non-invasive visualization of the living brain. The first major advance was the development of computerized tomography (CT scanning) which allowed one to observe areas of damage, down to a resolution of a millimetre or so. The procedure could be repeated to assess change, although the problem of radiation dosage limited its usage. More importantly from a

diagnostic point of view, the technique suffers from being unable to distinguish white and grey matter. Nuclear magnetic resonance imaging (MRI; see Pykett, 1982) circumvents both problems: no radiation is involved, and because of their differing proportions of water, white and grey matter are readily distinguishable. However both CT and MRI only allow the demonstration of structure (that is, of anatomy); more recent techniques such as PET (positron emission tomography; see Ter-Pogossian *et al.*, 1980; Phelps and Mazziotta, 1985) and SPECT (single proton emission computed tomography) not only show structure but also show function (i.e. physiology), by observing changes in local metabolism (generally or of specific substrates) or local cortical blood flow (Lassen, 1982). In each case, by observing changes from a resting baseline whilst carrying out particular psychological tasks, such as speaking aloud or carrying out mental arithmetic, one can obtain detailed information about localization (see e.g. Petersen *et al.*, 1988).

Electrical recordings from the brain are less helpful than might be expected in localizing function due to their being dominated both by the gross electrical activity of the brain (the α rhythm, etc.) and because of confusion with other, simultaneous but unrelated activity. These problems can be circumvented by the method of evoked potentials in which repeated presentation of a carefully timed stimulus (perhaps 256, 1024 or even 4096 times) allows one to average out background and extraneous noise and thereby extract a specific signal (see Chiappa and Ropper, 1982). The method is useful for observing the early stages of sensation and perception (up to 200 or 300 ms) but is otherwise limited, in part by the fact that many psychological events cannot be repeated precisely many times. In recent years the advent of powerful computer imaging methods to EEG activity has resulted in the methods of *neurometrics* which produces sophisticated maps of cortical activity, albeit at fairly low resolution (e.g. John *et al.*, 1988); at present it is still unclear whether such techniques will contribute substantially to neuropsychology.

Localization as such is not the end but is only the beginning in the cerebral localization of *function*. To go from the existence of a lesion in a particular place to the function of the lesioned tissue is a giant step. An adequate theory of neuropsychology can only be found in a sound psychological theory of the normal processes, and no reasonable theory is likely to emerge solely as a result of making clinical observations of lesioned individuals.

A further problem of interpretation concerns the necessary and sufficient lesion to produce an effect. In neuropsychology this is a peculiarly difficult problem. A large brain lesion will destroy various abilities, but many of these deficits may be secondary to some other function which has been damaged. To avoid these problems, Teuber introduced the principle of *double dissociation*: if lesion A damages function P and not function Q and lesion B damages function Q and not function P, then we are entitled to conclude either that the area of lesion A is involved in function P, or that area B is involved in function Q. Clearly, the lesions must be relatively small and not overlapping for such logic to work, and as a general principle small lesions tell us more about the function of the underlying tissue than do large lesions. Despite being the keystone of most neuropsychology, the theoretical implications of the method are still not fully

clear (for discussion and developments of the method, see Weiskrantz, 1968; Dunn and Kirsner, 1988).

An important assumption underlies the principle of double dissociation. In general it is highly unlikely that a single individual will have lesion A and then lesion B and it is almost impossible that lesion A will be 'cured' after the subsequent occurrence of lesion B. Hence the principle necessarily deals with lesions in different individuals, and thus assumes that the organization of different brains is similar. Whether this assumption is reasonable is unclear. Obvious counter-examples may be found in the case of language lateralization where, for one reason or another, the majority of the population have their language function in the left hemisphere but a minority (perhaps 8–10%) have right-sided language. More worrying still, there is evidence that left-handers perhaps have a more 'diffuse', and hence qualitatively different, brain organization from right-handers; similar suggestions have been made for differences between the sexes. Such factors complicate the application of the double dissociation principle.

A further complication concerns the assumption that the anatomical site of a lesion corresponds to the extent of its functional effect. However, to remove any portion of a complex system is to affect many other subsystems. Such neural effects have been termed *diaschisis* (a term introduced by von Monakow in 1914) and involve both a functional interrelation, and perhaps also a more physiological interaction, akin to the phenomenon of spinal shock. In either case the functional extent of a lesion may shrink with time, even though the anatomical lesion may not vary, and thus apparent recovery of function may occur. The anatomical extent of lesions can also vary, due either to secondary processes such as oedema, or to further extension of the lesion. In general, therefore, neuropsychology is the study of chronic patients in whom lesions have largely stabilized, both pathologically and functionally. The theoretical problems of interpreting recovery of function have been well described by Buffery and Burton (1982) (for a general overview, see Finger and Stein, 1982).

The final problem with the principle of double dissociation is that whilst the lesion may indeed be specific to a particular functional defect, it need not necessarily be responsible for that function. Geschwind (1965) published two important papers in which he suggested that many of the characteristic syndromes of neuropsychology were actually *disconnection syndromes*, which arise from disrupted fibres of passage between centres rather than damage to the centres themselves.

Disorders of Language

In 1861 Broca described a patient with the aphasia that now bears his name; the synonyms for the syndrome (motor aphasia, non-fluent aphasia and expressive aphasia) contain the essence of the defect, although they all make theoretical assumptions as well. The patients' problem is that they apparently cannot *produce* spoken language adequately, although *comprehension* of spoken language is normal. Spoken language is not entirely absent; the remaining speech is slow and

deliberate, and is much abbreviated, sometimes having a 'telegraphic' quality, using only the shortest, commonest words. There is however no defect in actually making vocal sounds, since often the patient can exclaim, or even sing (sometimes producing words which they could not speak). In 1875 Wernicke described his eponymous aphasia, which also has a range of synonyms (sensory aphasia, fluent aphasia, receptive aphasia and jargon aphasia), each of which contrasts with the synonyms for Broca's aphasia. The main problem is in the *comprehension* of speech; the *production* of speech apparently being broadly intact. The contrast between the two types of aphasia is heightened by the fact that in the original observations they were described as being due to small, well-circumscribed lesions of the pre-frontal cortex and of the temporoparietal cortex respectively, and these areas have since become known as Broca's area and Wernicke's area. Clinical descriptions of both syndromes will be found in Hecaen and Albert (1978) and Heilman and Valenstein (1979); a more popular and very readable account is given in Gardner (1976). Modern work has shown that whilst there are differences in localization they are not as clear-cut as was originally thought, although undoubtedly patients with Broca's aphasia tend to have more anterior lesions than do patients with Wernicke's aphasia.

It is tempting, on the basis of the preceding descriptions, and many 'diagram-makers' in the late nineteenth century were so tempted, to conclude that the function of Wernicke's area is to comprehend spoken language and that the function of Broca's area is to produce spoken language. That such theorizing is misguided was shown by work in neurolinguistics, which began with the eminent linguists Jakobson and Halle. Jakobson felt that the deficits of these patients were far more widespread than was immediately apparent, and he suggested that the defects corresponded to what linguists know as the syntagmatic and paradigmatic systems, and which we can approximately refer to as syntax and semantics. Careful study of a patient with Broca's aphasia shows that the problem is often not only in the production of speech, but is far broader, in terms of the overall comprehension of syntax. That the patient does understand sentences is in large part due to the redundancy of much of language, so that syntax is essential for comprehension. Thus one may understand sentences such as 'Put your hand in the air' without using syntax since 'Put the air in your hand' does not make sense. However, if syntax is made critically important to comprehension, then a Broca's patient will fail at the task; for example, in distinguishing 'Put the book on your hand' from 'Put your hand on the book'. Since the comprehension of spoken syntax is also very much dependent upon perception of order and timing, it has been asked whether the primary defect is of sequencing, and hence also of production of the closely synchronized vocal tract movements involved in speech.

Closer linguistic analysis also suggests that the defect in Wernicke's aphasia is not just of the reception of the spoken language, but might be more extensive. The speech of Wernicke's aphasics shows several abnormalities; the words which are produced show phonemic confusions (paraphonias: /b/ is confused with /p/ and /l/ with /r/) and there are also confusions of meaning (paraphasias: 'chair' may be said instead of 'table'). Note that in neither case is the error completely random, but rather a close approximation to the correct version is produced. Thus in both Wernicke's

and Broca's aphasia there is evidence of defects of language as a whole, rather than just of the reception and production of speech, although a comprehensive theoretical account of these defects is still in its infancy. The development of systematic assessments of linguistic ability (such as the Porch Index of Communicative Ability (PICA), the Boston diagnostic aphasia test, and the Minnesota test for differential degrees of aphasia) will help both in research and diagnosis.

Of course most patients with aphasia are neither Broca's nor Wernicke's in type; they instead have a 'global' aphasia, which is due to large lesions and contains features of both of the syndromes. A few other, far rarer, syndromes also occur, and are of particular interest to neuropsychologists. *Conduction aphasia* can often fail to be detected unless specifically searched for. Production and reception are apparently normal, but the defect is in the ability to repeat sentences exactly; instead of exact repetition there is a paraphrasing of the sentence so that meaning is retained but exact form is not. The sentence is thus literally reconstructed from its meaning (which will only retain some of the surface structure of the particular words originally used in it), rather than being remembered verbatim. The classical account of this disorder is in terms of the Wernicke–Geschwind model of speech and language (although see Shallice, 1988, for a more modern view). The lesion is supposed to occur in the arcuate fasciculus, the large bundle of fibres connecting Wernicke's and Broca's areas. These two areas have become disconnected, and information cannot therefore pass directly from one to the other, but instead has to travel via other, more indirect routes, during which presumably the message loses its exact form, although retaining meaning, perhaps having been translated into and back from what Chomsky has called 'deep structure'. Certainly, the lesions of conduction aphasia are compatible with such a theory, but direct proof, in the form of sectioning of the arcuate fasciculus, is lacking, and in recent years there have been some cases reported which at first sight do not seem compatible with the theory (see Mendez and Benson, 1985).

Almost the exact opposite, or complement, of conduction aphasia, is *transcortical aphasia*, in which there is no evidence of spontaneous speech, or of comprehension, but the patient can (and does) repeat sentences (echolalia). Most intriguingly of all, the patient can modify grammatical constructions ('How are you today?' becomes 'How am I today?') and can complete conventional phrases ('roses are . . .'). The explanation of the syndrome in terms of the Wernicke–Geschwind model is that Broca's and Wernicke's areas, and the arcuate fasciculus connecting them, have become disconnected from the rest of the brain due to a diffuse lesion (typically carbon monoxide poisoning, or a multifocal dementia); in consequence the only possible route by which received speech can become spoken speech is by going directly from Wernicke's area to Broca's area, via the arcuate fasciculus, without any possibility of the intervention of thought or cognition *en route*.

The final major type of aphasia is anomic or amnesic aphasia. Comprehension, production and repetition of speech are entirely normal, and the patient is fluent in conversation. The problem is that they are unable to remember names of objects and things. The defect is not one of intelligence or thinking, since the patient is well able to provide circumlocutions, and to process ideas associated with the thing in question, but they cannot retrieve the name of the object from

memory. The syndrome can occur in a pure form, but is often found in association with other forms of aphasia, and also with agraphia and alexia (see below).

The account so far given of the classical aphasic syndromes (Broca's, Wernicke's, etc.) has come under attack in the past two decades from the emerging discipline of *cognitive neuropsychology*, whose main concern is with the dissection (or *fractionation*) of psychological functions into discrete processes (or *modules*—see Fodor, 1983), using the natural lesions produced by brain damage as its scalpel (see Shallice, 1988). It stresses the search for dissociation between different cognitive tasks and emphasizes that it is necessary to examine individual patients in great detail, looking for those key individuals who show dissociation of abilities (see Caramazza, 1986). The corollary is that the study of groups of patients is often not useful and indeed may be positively confusing or misleading, not allowing subtle differences within syndromes to emerge. Indeed when individual patients with conditions such as 'Broca's aphasia' are studied in depth, it becomes apparent that what in classical terms is a unitary syndrome can better be seen as comprised of many subsyndromes which are often dissociated (see Chapter 9 in Ellis and Young, 1988). Nevertheless, such theoretical strictures and problems apply principally to psychologists attempting to dissect function; to busy clinicians there is still little doubt that the classical syndromes provide an heuristically useful way of categorizing function in brain-damaged patients.

The model of language given above has also been attacked during the past decade for its emphasis upon the role of the *cortex* in controlling language. A growing number of aphasic patients have been shown in recent years to have subcortical, particularly thalamic, lesions, and it is thought that these are directly responsible for the aphasia (see Metter *et al.*, 1988).

Language of course does not refer only to spoken speech (as is shown clearly in aphasic deaf patients who lose their ability to produce sign language), but it also includes written language. To a certain extent difficulties in reading (alexia or acquired dyslexia) or in writing (agraphia or dysgraphia) tend to coexist with aphasia. Nevertheless, the correspondence is sufficiently incomplete to produce interesting syndromes which throw light on brain organization. Déjérine, in 1891 and 1892, described the two major forms of alexia; alexia without agraphia often occurs in isolation, the only defect being an inability to read the written word. In particular the patient can write, and hence is in the apparently paradoxical position of not being able to read what they have just written. This syndrome is probably the best example of a disconnection syndrome (although this was not recognized by Déjérine). The lesion is usually in the left primary visual cortex (and hence there is a right homonymous hemianopia), and in the splenium of the cortex callosum. There is therefore no route by which the visual input can reach the dominant hemisphere, since information from the right visual field gets no further than the lateral geniculate nucleus, whilst information from the left visual field passes to the right visual cortex but is then prevented from passing to the dominant hemisphere by the lesion in the splenium.

The syndrome of alexia with agraphia involves defects in both reading and writing, and can be, but need not be, associated with a Wernicke's type of aphasia. The critical lesion seems to be in the angular gyrus of the dominant hemisphere,

at a position ideally placed to disrupt the integration of information from visual cortex and Wernicke's area. Pure agraphia (i.e. agraphia in the absence of aphasia or alexia), is rare and controversial. Exner, in 1881, suggested that it was due to lesions in what has since been called Exner's area, at the foot of the second left frontal gyrus (i.e. close to but separate from Broca's area), but this is by no means well accepted.

Finally a very rare syndrome, deep dyslexia, must be mentioned, not because it is clinically very important, but because these patients have been the subject of intense psychological research (Coltheart *et al.*, 1980). The symptoms are bizarre, and of the greatest importance in analysing the underlying mechanisms. The patients have problems in reading, and in particular are unable to read non-words (e.g. 'wux' or 'tud'), find difficulty in reading other words, in particular those which are of low imagability or are simply 'function words' (e.g. 'the' or 'is'), and make semantic errors while reading (e.g. 'play' for 'act', 'food' for 'dinner'). The final surprising thing about the patients, given the limit of their deficits, is the relatively large size of their lesions, which are always left-sided; indeed Coltheart has suggested that in the deep dyslexic we are observing the pure reading ability of the isolated right hemisphere.

Cerebral Dominance

In the mid-nineteenth century Broca and Dax showed convincingly, on the basis of the association between aphasia and right-sided hemiplegia, that language was preferentially associated with the left side of the brain (for an excellent historical account, see Harrington, 1987, who makes it clear that even at that time it was being argued that hemispheric asymmetry may explain psychiatric conditions —a recurrent modern theme: see Gruzelier and Flor-Henry, 1979). Since the time of Broca it has become clear that whilst a majority of the population have left-sided language dominance, a minority have right-sided language dominance; there is also the controversial possibility that some individuals have bilateral speech processes. It is probable that both handedness and cerebral dominance for language are under the genetic control of the same gene (see Annett, 1985; McManus, 1985).

The prediction of which individuals in a population have right-sided dominance is of theoretical and practical importance. Neurosurgeons wish to be able to operate through the non-dominant hemisphere in order to avoid producing post-operative aphasia, and psychiatrists wish to be able to give unilateral ECT to the non-dominant hemisphere in order to be able to minimize verbal memory disturbance. There are four basic methods for assessing dominance:

(1) *The Wada technique.* This involves the unilateral injection of sodium amytal into the one carotid artery and then the other, observing whether speech loss occurs after right or left injection. The method is indubitably effective, but has a sufficiently high morbidity to mean that it is only of use for pre-operative assessment where the risks are justified.

(2) *Unilateral ECT.* The administration of unilateral ECT to the two hemispheres, and the observation of verbal difficulties in the minutes after recovery of consciousness, may itself be used as an accurate means of assessment of dominance but is clearly difficult to justify in non-depressed patients.

(3) *Dichotic listening and visual half-field studies.* If information is presented just to one ear, or to one visual field (by means of an exposure short enough to prevent saccadic eye movements, in a tachistoscope), then because of the corpus callosum the information will be transferred to both cerebral hemispheres. If, however, different messages are presented simultaneously either to the two ears or the two visual fields, then there is sufficient competition between the stimuli to mean that the message contralateral to the dominant hemisphere is perceived better (i.e. left language dominance is associated with a right-ear advantage or a right-visual field advantage). The method is easy to use and harmless, and hence has been much used for research purposes (see Hugdahl, 1988). However, it is insufficiently reliable or accurate to be of use for pre-operative assessment of language dominance.

(4) *EEG studies.* If the α rhythm is recorded from both hemispheres during verbal thought then there is a greater suppression on the side which is more active, which is the dominant side. Again, this is primarily a research tool, rather than a routine procedure.

A major advance in the study of cerebral dominance has been work on 'split-brain' patients. These individuals have had a cerebral commissurotomy, involving section of the corpus callosum, for the treatment of severe epilepsy. As a result it is possible, by mean of carefully controlled stimulus presentations, to study just one hemisphere at a time in isolation (thereby fulfilling the criteria for double dissociation within a single patient). From these patients it seems clear that the non-dominant hemisphere does indeed have *some* language but that it is far more restricted than that in the left hemisphere, being limited to concrete rather than abstract words, having a smaller vocabulary, and having poor command or syntax. By contrast, however, the right hemisphere seems to be better at tasks involving visuospatial problems, such as those requiring mental imagery or patterns. This insight is well supported by the clinical effects of right-hemisphere lesions.

Disorders of Perception and Action

The contrast between the cognitive modes of the left and right hemispheres is reflected in the difference between lesions affecting symbolic action (such as the aphasias, alexias, agraphias and some of the apraxias) and the lesions affecting spatial and perceptual processes, such as the agnosias and constructional apraxia. Relatively less is known about these latter syndromes and they appear to be somewhat less common, perhaps in part because they are less looked for.

At the lowest level, disorders of perception result in apparent absence of sensory function. It must, however, be remembered that in, say, the visual system, the primary visual cortex is not the only retinal projection. The projection to the

superior colliculus is of great importance in the unconscious detection of movement (whereas the visual cortex is of importance in the conscious recognition of form). Hence individuals with lesions of primary visual cortex cannot see in the conscious sense, but it can be demonstrated that they can use collicular information, albeit unconsciously, and in a way that they feel is random guessing; a phenomenon that Weiskrantz has called 'blind-sight' (see Weiskrantz, 1986).

At a slightly higher level in the visual system, we find a miscellaneous collection of conditions known as the agnosias, often involving lesions of the right occipitoparietal area. They were described originally in experimental dogs by Munck, in 1881, and he called them 'mind blindness', which emphasizes that there is no blindness in the strict sense of seeing lines, edges, etc., but rather there is a general failure to integrate the information. Some of the agnosias are general, whereas others, such as colour agnosia, and prosopagnosia (the inability to recognize faces), are highly specific, and are of great importance in suggesting that the brain has specific systems for dealing with certain types of stimuli. A common denominator of the agnosias, as recognized by Luria, is the inability to carry out the operation of separating figure from ground (or foreground from background), and this means that agnosics are peculiarly poor at disambiguating figures which are embedded in other figures. Agnosias can also occur in the auditory system (agnosia for sounds, in which non-verbal auditory stimuli are incorrectly recognized, and the amusias, which specifically involve defects of musical perception) and in the somatosensory system (such as astereognosia, in which objects can be recognized by touch alone, and asomatognosia, in which the patient fails to 'know' the position and state of their own body, and may even deny the existence of half of it—hemineglect—or apparently be unaware and indifferent to pain in a particular area—pain asymbolia). At the highest intellectual level agnosia can also manifest as a lack of understanding of the spatial environment (see de Renzi, 1982).

There is a sense in which the apraxias are the converse of the agnosias, being defects of motor rather than sensory function. Liepmann, in 1900, described patients who could perform simple actions, but could not integrate the actions into a well-organized sequence (ideomotor apraxia); there were also patients who could perform actual actions, but were unable to imitate them without the actual objects, as for instance in striking an imaginary match (ideational apraxia). There are also specific apraxias, such as buccofacial apraxia in which motor sequences with mouth and face are impaired, once more suggesting the possible existence of specific cerebral subsystems. The apraxias are controversial in that they are usually associated with left-hemisphere damage, and frequently coexist with aphasia; the question therefore arises of whether aphasia and apraxia are independent syndromes, or are perhaps indicative of a more widespread defect of symbolism and gesture (which is of course closely associated with language).

In contrast to the left-sided apraxias described above, there is constructional apraxia, in which the patient is unable to construct objects from building blocks, or is unable to draw, or even to copy simple line drawings. Here the defect is primarily one of the mental reconstruction of the object, rather than in the motor control *per se*, and it is almost always associated with right-sided parietal damage.

By now it must seem that there is an infinity of different syndromes involving specific defects. As a cautionary note on the overmultiplication of syndromes, it is worth considering Gerstmann's syndrome, which was described in 1924, on the basis of a single patient, and consists of the tetrad of finger agnosia (the inability to name or recognize the fingers), acalculia (inability to carry out arithmetic), agraphia and right–left confusion. It is now controversial as to whether there is any fundamental psychological similarity between these heterogeneous defects (see Benton, 1961), and current opinion probably favours the suggestion that these defects had come together by chance, perhaps due to the lesion affecting several different adjacent cortical areas, particularly since patients may be found with just one, two or three of the tetrad of symptoms.

Disorders of Memory

Two very different types of lesion cause permanent amnesia (see Whitty and Zangwill, 1977): bilateral hippocampal removal (as shown in the much studied patient HM), and Korsakoff's syndrome, in which the major anatomic lesions seem to be in the mammillary bodies and the anterior thalamus. In both conditions, despite the lack of coincidence of the lesions, the symptoms are strikingly similar: a loss of the ability to form new memories (anterograde amnesia) and an inability to access old memories (retrograde amnesia). In neither condition is there any deficit of short-term memory. The major psychological interest in these cases has concerned whether the defect is one of the creation of new memories, the storage of memory, or the retrieval of memories from store. Warrington and Weiskrantz have shown that with suitable assistance during recall (using 'fragmented stimuli') such patients can 'learn' (although they are not consciously aware of it), suggesting that the major defect may be in the *retrieval* of memory. For more recent ideas on the defects in amnesia, see Mayes (1987) and Hintzman (1990).

Disorders of Personality and Emotion

Thus far the majority of deficits described have been intellectual or cognitive deficits. In this section we must consider the non-cognitive, or affective and personality effects of lesions. Unlike previous work, much of this area is dominated by particular lobes of the brain (temporal and frontal), partly because it is easy to remove the lobes, in both animals and man, and partly because localized diseases, such as tumours, can readily simulate such experimental and surgical lesions. It has also become apparent in recent years that the two sides of the brain are not equally involved in emotional responses (see Heilman and Satz, 1983).

Temporal lobes

In 1937 Kluver and Bucy bilaterally ablated the temporal lobes of a rhesus monkey. The previously aggressive monkey became tame, and also demonstrated

hypersexuality, as well as evidence of visual agnosia. The crucial question of course is whether similar symptoms occur in man, and the answer is, yes. However, the symptoms are also associated with defects in language, and in memory, and these factors necessarily modify the picture. Animal studies now make it clear that the decreased aggression of the Kluver–Bucy syndrome is due to lesioning the amygdala; that the hypersexuality may be due to lesioning of the septal system; and that the visual agnosia is due to lesioning the inferior temporal gyrus. In man the defects of language and memory are probably due to involvement of Wernicke's area and the hippocampus, respectively. The 'syndrome' is thus an artificial collection of symptoms which bear no obvious psychological relationship to one another; nevertheless, gross lesions of the area, due to tumour or trauma, or gross stimulation of the area (due to temporal lobe epilepsy), will necessarily affect them all in parallel.

Frontal lobes

Jacobsen, in 1936, showed that frontally lesioned monkeys showed impairment on delayed response tasks, in which an animal is shown where to find a reward, but is prevented from taking the reward for a few seconds. Similarly, the animals are poor at tasks involving alternation of responses (e.g. the reward is under the left cup and then the right cup on alternate trials). The problem seems not to be one of memory but of the inhibition of responses, a conclusion which is supported by the failure of animals with frontal lesions to habituate as rapidly as normal, and hence they continue to produce responses long after a normal animal has ceased —giving the appearance of stereotypy. Frontal lobe damage in humans is far more dramatic, but may be regarded in part as a continuation of the effects found in animals. Frontal patients show a tendency to produce bizarre antisocial behaviours, involving a lack of awareness of social niceties, and with a child-like interest in repeated stimuli and responses—behaviour which has been termed 'pseudopsychopathic', and may be explained in terms of a lack of response inhibition. An alternative personality change in frontal lesions is 'pseudodepression', in which there is a lack of spontaneous activity or drive, a general loss of emotional expression, and reduced speech. Except for the last feature these symptoms may also be found in animals, where spontaneous behaviours may be reduced, facial expressions absent, and social drive diminished (as shown by the animals tending to fall to the bottom of the hierarchy, or 'pecking order').

The deficits of response inhibition and spontaneous behaviour are not the only problems found in frontal lesions. There are also motor difficulties (shown in the inability to imitate movements), difficulties in control of eye movements (due to lesions of the frontal eye fields), and deficits of memory and spatial orientation. It is this heterogeneity of deficits which makes it difficult to know whether there is a simple unitary psychological defect, or perhaps, as in the temporal lobes, there is a series of independent deficits which are associated merely due to neuroanatomical contiguity. It is somewhat difficult to see how complete removal of the frontal lobes could be therapeutically useful, and it is worth stressing that

Moniz introduced the operation of frontal lobotomy on the basis of a single 'neurotic' chimpanzee which appeared to be more 'relaxed' after its operation.

Conclusions

In a survey such as this, in which coverage is necessarily superficial, some omissions are inevitable. However some omissions are intentional, and perhaps need stressing. One such is the absence of reference to developmental aspects; the organization of the normal adult brain is far from understood, and that of the individual damaged in childhood is still less well understood, and may indeed be qualitatively different from that of the normal adult. Similarly, all references to alexia and dyslexia are to acquired adult dyslexia; the syndrome of childhood dyslexia is controversial, and almost certainly unrelated to the adult condition. And in a similar vein, there is no detailed discussion of the development of recovery from lesions; again too little is known and the questions are perhaps too complex to answer at present.

Perhaps a surprising omission in a work on cognitive function is any reference to 'intelligence'. Thus there is little evidence that intelligence is reduced in aphasia or after brain lesions of small or even moderate size, including, for instance, bilateral frontal lobotomies. Similarly there is little evidence that certain areas are of particular importance in determining intelligence; intelligence as a whole is robust in the face of damage, although specific abilities are not, perhaps thereby justifying in part Lashley's concept of mass action (or equipotentiality). Certainly there is no need as yet for us to postulate a centre which the mediaeval neuroanatomists would have labelled *ratio*.

An exception to the above rule is in the differentiation of right- and left-sided lesions. In general left-sided lesions produce a relatively greater impairment on the verbal subtests of IQ batteries, and right-sided lesions produce a relatively greater impairment on 'performance' (or visuospatial) subtests, and a difference of 10 or more IQ points on the two scales is probably of importance.

Finally, in a book of this sort, which hopes to provide the relevant scientific background for a student of psychiatry (and there seems little doubt that in the future brain imaging and localization will be of growing importance in psychiatry: see Andreasen, 1988), there has been little direct attempt to provide immediate and practical applications of the information given. Rather it has been assumed that any knowledge of the brain and its actions must be of relevance to the concerned psychiatrist, particularly when dealing with bizarre effects of lesions which can simulate psychiatric problems (see e.g. Benson and Blumer, 1975), and that psychiatrists in training will rapidly make such associations for themselves. There is, however, also a more serious problem; while in principle there should be much relevance, that is not always the case. I will conclude therefore with a quotation from Geschwind, who until his death in 1985 was probably one of the most eminent and influential neurologists to address psychological questions:

> While it has become fashionable to acknowledge the existence of an area of overlap between neurology and psychiatry, this common ground unfortunately bears more resemblance to

> a no-man's land than to an open border . . . Unfortunately, few members of either group have in fact really interested themselves in the borderland area, and too frequently interactions between them are educationally disappointing . . . Hopefully, this situation will be corrected in the next few years, but until then, both psychiatrists and neurologists [and one would add, psychologists] will often have to acquire the necessary knowledge themselves.

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