ASSOCIATION, AGNOSIA, AND INTELLECT

By

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"O the mind, mind has mountains; cliffs of fall,
Frightful, sheer, no-man-fathomed."
Gerard Manley Hopkins.

THE importance of the anatomical basis of the simpler motor and sensory functions of the central nervous system, and their disorders, has been recognized for over a century (Brown-Séquard, 1855). But that more complex intellectual functions and their disorders should also have a relevant anatomical substrate is only slowly coming into recognition. Indeed, a still-fashionable theory of mental function, originating in Vienna towards the end of the last century, regards the study of brain structure as unnecessary and beneath contempt—for all that its founder began as a neuro-anatomist.

The advent of psychosurgery (Moniz, 1936), and, particularly, recent observations on the effects of surgical intervention in the temporal lobe, have re-awakened interest in the physical basis of mental function. Data gained from such experience have made it possible to attempt an integration of anatomical and psychological information to explain mental phenomena.

ANATOMICAL CONSIDERATIONS

Von Monakow (1914) was the first to point out that retrograde cell degeneration occurs in the thalamus following cortical ablation. Since then, this work has been extended in the macaque (Clark and Boggon, 1935; Clark, 1936; Walker, 1938), and in man (Feremutsch and Simma, 1959). It has been shown that the primary motor and sensory cortices receive specific (point-to-point, somatotopic) afferents from the ventral group of thalamic nuclei, which in their turn receive somatotopically-organized extra-thalamic afferents. The (sensory) association cortex may be defined as those regions of the parietal, occipital, and temporal neocortex which are not primary somatic, auditory, or visual sensory receptive areas (Penfield and Rasmussen, 1950), i.e. regions which are not the projection zones of the ventral thalamic nuclei; Rose and Woolsey (1949) have estimated that this is 85 per cent. of the human cortex (including frontal "association" areas). In terms of thalamic dependence, the sensory association areas may be divided into two groups: (*i*) the parieto-occipital and superior temporal association areas, which receive a point-to-point afferent projection from the lateral-pulvinar group of thalamic nuclei; afferents to these latter come from within the thalamus, chiefly (if not entirely) from the ventral group. As an example, the situation for somatosensory impulses is shown in Figure 1 overleaf.

These adjacent cortical areas are in fact joined by short cortico-cortical "association" fibres, but the experiments of Lashley (1952), as well as clinical experience of superficial cortical injuries in man, have shown that such fibres are not involved in the (mental) act of association.

(*ii*) The non-auditory temporal neocortex receives a small projection from the pulvinar, but it is not thalamically dependent in the same sense as the other

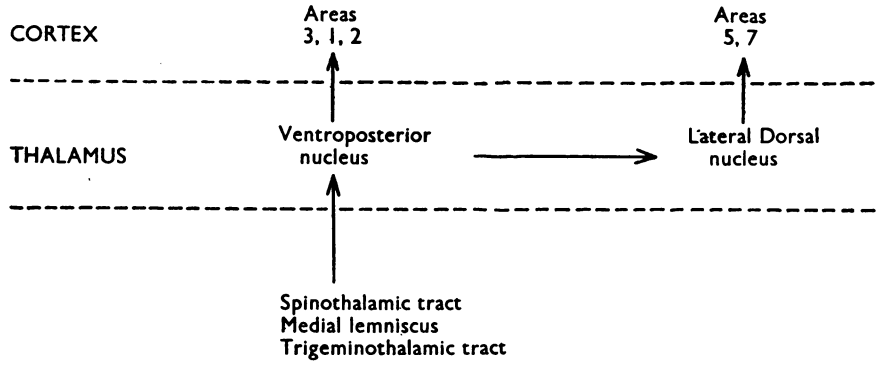

FIG. 1

cortical association areas. Its principal afferent connections are from (*a*) the hippocampus (Votaw, 1960, in the macaque), and (*b*) the thalamically dependent association cortex, by means of long cortico-cortical association fibres. These connections have been most fully explored in the macaque, by both anatomical and strychnine neuronographical methods; they are reviewed by Bonin and Bailey (1947) and Chow and Hutt (1953). Bailey and Bonin (1951) assert that these cortico-cortical connections are essentially similar in man. While the temporal neocortex is richly interconnected with other cortical "association" areas, it does not receive fibres from the primary motor and sensory cortices.

The relationship between the primary sensory cortex and the two types of "association" cortex, together with their functionally significant sub-cortical connections, is shown, in general terms, in Figure 2:

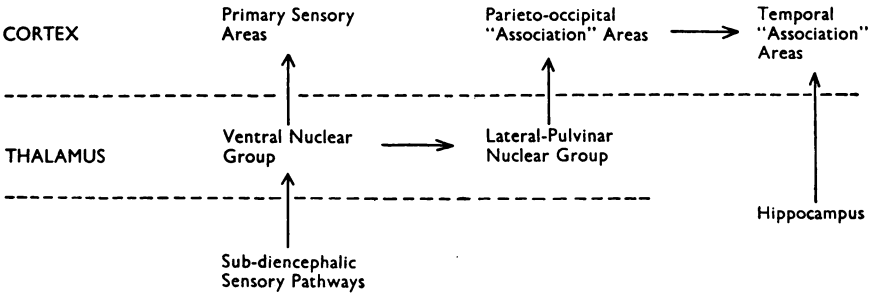

FIG. 2

Anatomically, then, there are two distinct types of "association" cortex, which differ considerably in their connections. The clinical evidence should be examined with reference to these connections, in an attempt to assess their functional significance.

CLINICAL CONSIDERATIONS

It is known that lesions of the parietal somatosensory "association" cortex, or of its specific thalamo-cortical projection, produce a well-defined type of agnosia; it is in fact often called "nominal aphasia", in reference to the resultant motor (speech) response. The interesting thing about it, in the present context, is not that the patient cannot name, for example, a key introduced into his hand;

but that he can say, without giving the name, "This is for opening doors". In other words, the cognitive act required to name the object is impaired, but the interpretative act required to describe its function is intact. Similarly, lesions in the thalamically-dependent visual or auditory "association" areas lead to disturbances only of cognition of visual or auditory stimuli. Larger lesions, of course, may impair more than one of these "modalities" of cognition.

The temporal lobe syndrome cannot be so easily assessed by simple tests; this is particularly true of surgical temporal lobectomy, in which structures other than neocortex are usually damaged. Perhaps the simplest type of case to consider in the first instance is the aura produced by the discharge of an epileptic focus known to be in temporal neocortex. Such aurae consist of complex hallucinations involving the integration of sensory data from several different sources.

In the intact brain, it may be assumed that cognition in somatosensory, auditory, and visual modalities in the appropriate "association areas" occurs as the first part of the mental act of association. This information is transmitted by cortico-cortical pathways to non-thalamically-dependent temporal neocortex, where it is integrated and interpreted. Thus, Penfield (1958) has written: "The production of interpretative illusions by local epileptic activity or electrical activation [in temporal cortex] suggests that [this] cortex contains a mechanism which is normally employed in the interpretation of current experience. This interpretation includes comparison of the present with selected past experience."

Temporal epileptic aurae may also take the form of compulsive "memory patterns" (Penfield and Jasper, 1954). Surgical experience has shown that bilateral, or extensive unilateral, interference with the hippocampal formation leads to serious defects of recent memory (Scoville and Milner, 1957; Walker, 1957). Thus it would seem that the ability of the cortex to retain experience (memory) is dependent on the integrity of the projections from the hippocampus to the temporal neocortex.

It is a matter of definition whether or not memory constitutes a part of intelligence. But in general it may be said that intelligence is made up of a variety, or perhaps a succession, of anatomically localized cerebral functions. Cortical lesions at any site may affect "intelligence" in so far as they interfere with the subject's ability to appreciate (primary sensory), recognize (parieto-occipital associative), interpret (temporal associative), or respond to (motor) objects or problems presented to him. Naturally, if cortically-injured subjects are tested in unaffected parameters, their problem-solving abilities are not significantly affected compared with their pre-injury performance (Jarvie, 1960). Only 7 of Jarvie's 71 subjects showed intellectual deficit on such tests; and they all had extensive subcortical lesions in addition to their more superficial (cortical) injuries. Likewise, Milner (1958) has examined over 100 cases of temporal lobectomy before and after operation, and states that "intelligence as measured by the Wechsler-Bellevue I.Q. rating is not permanently affected by these operations".

Data of this kind show that it is no longer possible to attribute intelligence, as a unitary function, to the "mass action" of the cortex as a whole (Lashley, 1929); it must now be defined as the integrated compound of separable functions occurring in different and specific areas of cortex. While there is still every reason to believe that those purely intellectual properties, which so distinguish man from other animals, are a function of the neocortex, it must be remembered that the brain functions as a whole, not as isolated parts or systems. The intact cortex may act as a super-computer, which can be taken to pieces, but "value"

and "weight" are chiefly contributed by sub-cortical mechanisms and structures. The rhinencephalon would appear to be responsible for both the emotional correlate of sensory experience and (specifically as the hippocampus) the emotional "drive" which makes the performance of (cortical) intellectual activity possible.

Summary and Conclusions

"Association" occurs in two phases. The first is cognitive; it takes place in the "sensory association" areas of the cortex, and is anatomically dependent on a thalamo-thalamo-cortical circuit. The second phase is interpretative, and occurs in the non-auditory temporal neocortex; it depends, anatomically, on the integrity of cortico-cortical connections from other areas, and not (in itself) on thalamo-cortical projections. Isolated cortical lesions impair only those functions which are localized in the affected areas. Tests designed to obviate the defective functions therefore fail to demonstrate intellectual deficit. "Intelligence" represents the sum of a number of anatomically and psychologically discrete cortical functions. The ability of the cortex to perform intellectual work, and to retain data, is dependent on sub-cortical mechanisms.

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