Editorial

A GAP IN NEUROPSYCHIATRIC KNOWLEDGE

The rivalry and antagonism between Neurology and Psychiatry are happily passing away, and it has been part of the mission of this Journal in welcoming both subjects within its purview, to assist in their rapprochement. Most of those who are interested in these two subjects would adhere to Huxley’s dictum, ‘No psychosis without a neurosis,’ and therefore it behoves every psychiatrist, when describing the vagaries of his patients, to give a thought to what is going on in the brain, and every neurologist to ponder what variation of cerebral function—temporary and reversible as it may be—may underlie the delusion, the manic excitement, the anxiety or the hysterical fugue of the psychotic or psychoneurotic patient.

Knowledge of how the brain works is increasing rapidly and older conceptions of localization of function are being revised and corrected. It is being realized more and more that in many respects the brain works as a whole, yet that certain areas, possessing certain cytoarchitectural structure and arrangement, have the duty of organizing and controlling special functions. As Kuntz remarks in comparing the cortex of man with that of animals: ‘As the cortex increases in expanse it also undergoes regional differentiation, with the result that more and more cortical areas can be mapped out on the basis of structural differences. These structural differences are not without functional significance; consequently new cortical areas may in a very real sense be regarded as new cortical organs.’

Nor is it only in the cortex that new knowledge of functional localization is being obtained. In that crowded area the
diencephalon and mesencephalon considerable exactitude is beginning to emerge. We know that centres in the diencephalon regulate and control autonomic activity, while those in the mesencephalon are intimately concerned with tonic variations in skeletal musculature. It is established that nuclei in the diencephalon control the muscles of the eye, the contraction of the bladder and uterus, the tonic contraction of blood-vessels, regulate body temperature, secretory activity of salivary, lachrymal, sweat, sebaceous and other glands, govern carbohydrate and protein metabolism, elimination of water and the trophic regulation of the skin and subcutaneous fatty tissue. But in the same area are centres which play an important rôle in the affective life of the organism. It has been shown that in animals normal expressions of fear and rage depend on the integrity of the diencephalon. This area, moreover, is concerned not only in automatic activity but takes a hand in the regulation of skeletal muscle control. We may laugh voluntarily (cortical) or involuntarily (thamic), and to some extent we can control involuntary laughter if the stimulus is not too strong or too 'relevant,' though we cannot control the acceleration of our hearts or respiration or our carbohydrate metabolism.

In the realm of Psychiatry we are dealing primarily with distorted and confused affective impulses which no doubt secondarily give rise to distorted ideas and distorted behaviour. It is now generally admitted that however involved is the delusional system of the paranoiac, however grotesque the behaviour of the manic, the schizophrenic, or the hysterical, the basis of the whole trouble is emotional. At the same time, in our studies both of the psychoses and psychoneuroses, we constantly meet with physical manifestations of disturbances of diencephalic and mesencephalic centres. The lowered activity of all bodily functions in depression, the intense metabolic and visceral activity of the manic, the 'frozen' muscle-tone of the catatonic and the baffling and conflicting autonomic activities of the anxiety case all exemplify this. It is clear therefore that diencephalic and mesencephalic disturbance is a dominant factor if we are to interpret these conditions in terms of disturbance of neurological function. In attempting such inter-
interpretations it must be recognized that practically all of these symptoms are reversible, that we are therefore dealing with alterations of function and not of structure, and that in consequence we are likely to find a clue not in the diencephalic centres themselves but in inhibitions and releases which are impingeing upon them from above. Moreover, we are investigating behaviour disorders of an essentially cortical nature, and consequently our final quest must be to discover exactly how cortical influences are conveyed to diencephalic centres. Here is a gap in neurological knowledge.

We know that there is a large bundle of fibres, probably both afferent and efferent, connecting the frontal lobe with the thalamus, and that this body also has connections with parietal, occipital, and temporal lobes; but we know little about the exact function of these tracts. We infer that there must be cortical connexions in both directions with the hypothalamus and that these chiefly subserve inhibitory mechanisms, but we know little of their anatomical paths or how they work.

Hunsicker and Spiegel, experimenting on cats, showed that some cortical impulses governing autonomic function travel in close association with pyramidal fibres, but although division of these tracts produced alterations of autonomic activity, further division of extrapyramidal tracts produced a further degree of such alteration. This led them to conclude that at least two paths of cortico-autonomic control existed, one pyramidal and the other extrapyramidal. White dismisses our knowledge of cortical representation of the autonomic nervous system thus: ‘Attempts to define a clear cut autonomic representation in the cerebral cortex have not been very successful. Fulton and his colleagues have found a certain degree of vasomotor and intestinal localization in the premotor cortex, but much more work remains to be done before any definite facts can be established.’ Kennard, Viets and Fulton, describing a case of a removable tumour in the premotor cortex (Brodmann’s area 6), pointed out that in addition to the frequently described components of the syndrome of premotor lesions—forced grasping, emotional instability, confusion and slowing of mental activity—vasomotor changes in the affected extremities occur, but have seldom received notice.
In this case temperature and sweating changes were noted, and the authors refer to the old observations of Eulenberg and Landois, who held that irritation of centres near the motor areas for particular limbs produces pallor and coldness, while hyperæmia and increased warmth are probably the result of a loss of the central influence and may be due to disease of the cortex or of the path from it which seems to pass in the posterior limb of the internal capsule. Their conclusions are that while changes in skin temperature and sweating on the side opposite the lesion indicate that representation of the autonomic nervous system exists in the cortex, further analysis is needed of the anatomical position of this vasomotor area. All that Kuntz can say on this subject is: 'The mechanisms through which cortical impulses exert their influence on visceral functions as yet are not fully known. Most of the data available at present support the assumption that the cortical impulses in question are conducted downwards through fibres of cortical origin which terminate mainly in the hypothalamus; consequently, the cortical influence probably is exerted mainly through the diencephalic autonomic centres.'

It is quite clear from all this that there is little exact knowledge of the anatomy and physiology of cortico-autonomic connexions. Further, when we come to the more vague and general subject of the cortical factor in emotional behaviour we find a welter of confusion and contradiction. Since Bianchi described the ‘loss of social sense’ and ‘sentimentality’ of his monkeys deprived of their frontal lobes, many authorities have thought that the cortex in general and the frontal lobes in particular exercise a ‘restraining influence’ on instinctive behaviour and primitive emotional impulses, but we find Brickner attributing the lack of ‘emotional restraint’ (observed in a patient from whom almost the whole of both frontal lobes had been removed) to a failure in intellectual synthesis and categorically stating that ‘There is nothing in A.’s record to indicate the primary participation of the frontal lobes in emotional function.’

It is clear therefore that here is a gap in neurological knowledge which must be filled if a true wedding of neurology and psychiatry is to take place. Such a wedding need not disturb
the field of interest of either of these branches of medicine, but, on the contrary, would curb the too frequent wild flights of fancy of the purely psychical psychiatrist and quicken the equally undesirable dry-as-dust materialism of the structural neurologist.