ANATOMY OF THE UNCONSCIOUS

DEAR SIR,

As one who has also attempted to write a neuropsychology (1) may I begin by commenting upon the latest translation of Freud's "Psychology for Neurologists" (2). Although in his editorial introduction, Strachey draws attention to the fact that this neurological document contains the nucleus of much of Freud's later psychological theory, understandably I find some of the neuro-physiological ideas of at least as much interest, especially if allowances are made for the essentially unfinished nature of the work and therefore for any inconsistencies which a final tightening-up might largely have excluded.

Freud appears to see in his system of "nerve-ending apparatuses" and omega neurones not only the way in which analysis of sensory qualities in consciousness takes place, but also a peripherally oriented mechanism damping the quantity of input received through each sensory modality. The functional importance of these two concepts experimentally demonstrated forms much of the substance of the Thirty-Second Maudsley Lecture (3) on Sensory Experience and Brain Structure, in which Le Gros Clark also refers to "the reticular formation stretching through the spinal cord and brain stem".

Freud similarly refers to the grey matter of spinal cord and brain when anticipating Sherrington (4)in trying to classify exteroceptive and enteroceptive systems of neurones, suggesting that from the biological standpoint of endogenous instinct impulsion, indispensable to Darwinian survival, the brain acts primarily as a sympathetic ganglion. This is reminiscent of current ideas equating the reticular formation with autonomic function (5) and identifying such a vegetative central nervous core with the origin of tides and rhythms which characterize animate matter.

Mott (6), writing on much the same general theme as Freud, also quoted Darwin's view that the difference in mind between man and the higher animals, great as it is, is certainly one of degree and not of kind; but Freud specifically draws attention to the subjectively unconscious nature of neuronal processes, which like other natural things can only be inferred indirectly, an association between real anatomy and consciousness nearer the kind adopted in modern psychophysiological theory (7) than that more usually referred to somewhat analogically in describing purely psychological structure.

Hill (8), for example, inclined to an electrophysiological view, apparently compatible with the idea that pseudopurposive antomatism in temporal lobe epilepsy may indicate a post-ictal release of the psychomotor (frontal) cortex from exteroceptive cortical (parieto-occipito-temporal) control temporarily paralysed by the discharging focus, and later (9) distinguished two major functional components of the sleep-walking mechanism—that concerned with consciousness and that concerned with motor activity. Partial disturbances of such exteroceptive sensory control over action include dyspraxia, which can follow parietal defect, and jargon dysphasia, which usually follows posterior temporal lesions, for dyspraxia and jargon dysphasia (10) may also arise as the result of deficit in pre-central i.e. frontal (motor) damage.

The posterior temporal sensory site of the exteroceptive control of jargon, indicates the importance of hearing in learning to speak, a control which may, at such a distance, be one stage even further removed from direct motor influence, producing its effect secondhand perhaps, via intermediary changes in exteroceptive touch and proprioceptive activity in the post-central gyrus, which must accompany speech movement activated more adjacent to them pre-centrally. If we accept, however, that consciousness is the kind of exteroceptive sensory accompaniment thus suggested (7), conversely it is possible to delineate, somewhat in Freud's sense, the cerebral structures whose activity does not so directly involve our awareness. These would include the frontal lobes with their associated thalamic nuclei and projections, the basal ganglia, hypothalamic structures, hemispheric autonomic representation and proprioceptive mechanisms.

Fulton (11) devoted 16 pages to a description of autonomic representation in the cerebral cortex. To highlight hemispheric autonomic representation therefore first, I would like to propose that the external and internal granular layers of the cortex be considered equivalent respectively to Meissner's and Auerbach's plexuses of the alimentary tract. Taken together with subcortical intracerebral supporting autonomic structure implied in thus relegating the cortex to a vegetative periphery, it may, as in Freud's sympathetic ganglion, then be possible to understand much that is involuntary (12), instinctive (13) and unconscious (14) in the processes of intellectual appetite, thirst, peristalsis, digestion, absorption and discard, on the same clearly defined somatic basis already accepted for other comparable bodily activities.

The agranular frontal cortex which orientates and directs our exteroceptive sensory receptors (15), might (in this arrangement) be equated with the oesophagus, which lacks intramural ganglionic plexuses (16) but nevertheless represents the initial common path for all material destined eventually for either nourishment or excretion; whereas the hypothalamus is centrally placed, much as the lumbosacral enlargement of the cord is central relative to the colon for example. Spinal anaesthesia can influence intestinal function for the better temporarily in megacolon and permanently in other alimentary conditions (17), much as psychotropic drugs may act beneficially on sub-cortical mechanisms of behaviour (5).

Autonomic properties of hypothalamic nuclei have long been established (11), and quite apart from their indirect effects on reticular control of the level of general awareness (18), some have already favoured their efferent relay to the cortex via thalamic nuclei, e.g. the magnocellular dorsomedial anticlockwise spiral projection to the orbital surface (19, 20), a kind of intracerebral splanchnic nerve of worry or anxious anticipation (15). In some respects, however, a parallel may be closer between the hypothalamus and the cervical (C.3-T.2) rather than lumbosacral enlargement of the cord, for the former enlargement not only appears as capable as the latter of exhibiting local "central" autonomy, e.g. in Sudek's atrophy of the upper extremity, but also reveals an additional anatomical similarity to the hypothalamus.

Thus the cervical enlargement conveys downward one of the principal autonomic efferent projection systems of the hypothalamus (11), i.e. that from the posterior hypothalamic area to the lateral horns of grey matter in the thoracic cord which relay preganglionic sympathetic fibres, some of which then ascend the cervical sympathetic chain. The equivalent in the hypothalamic vicinity, presupposing some encephalization of vegetative function to give them cortical origin, would be the centripetal "doubling back" of prefrontopontine and temporopontine tracts which convey the psychogalvanic skin sweating response in mood change.

Overall autonomic structure subserving emotional conation may none the less be better revealed by the changing involuntary patterns which accompany the whole continuum of:

depression-fear-calm-rage-elation

In this, in one direction gradually increasing inhibition or suppression of a desire to flee or escape leads an individual to pass through anxiety into gloom, whilst in the other, as progressive motivation overcomes inhibition, the individual passes through anger toward cheerfulness. The continuum appears to be two-dimensional, since exceptionally it is possible to weep with joy and manic and depressive features can sometimes be observed concurrently in the same patient. The calm or tranquil node of balance is presumably therefore the point of origin of the two separate co-ordinates concerned, although rebound from one into the other and back again also seems to occur, e.g. when modified leucotomy in paranoid depression releases obscene rage which was pre-operatively presumably overcontrolled. Purpose may be served by the relevant autonomic substrate to each mood (21), for example the cephalad sympathetic activation of rostral hair and livid mien in anger (22) influences psychologically outward and facilitates forward progression by intimidating an adversary.

Finally, like the psychiatric textbooks of 1850 (23) which held the cerebral hemispheres to be as much the bodily organs of mind as are the lungs of respiration, may I suggest that the main dorsomedial ("parvicellular") thalamic nuclear spiral prefrontal projection is perhaps "somatic" rather than splanchnic in intracerebral character, i.e. comparable with the phrenic nerve rather than the vagus, being involved both in logical prediction (15) and probably an automatic unconscious control of "inspiration" (12, 14)!

As far as parapsychological factors may be concerned in all this, we nevertheless seem a long way yet from a unified oecological field theory in which they could be usefully applied to the extent physical and psychological field factors already can (24, 13, 25). Some progress may eventually be made here too, however, perhaps through an analogy with the proprioceptive system, which in cerebral photolenticular theory (7) appears to bridge the gap between exteroceptive sensory consciousness and motor activity across the central cortical divide.

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TWINS WITH EARLY CHILDHOOD AUTISM

Dear Sir,

I am engaged in a study of twin pairs, where at least one child suffers from early childhood autism. The main part of the survey consists in ascertaining twin pairs from the parent members of Societies for Autistic Children which are now in being in various parts of the world.

This method of ascertainment avoids bias of various types, providing all the twins represented in the membership of the Society are located.

However, it would be of interest from the genetic viewpoint to study also twin pairs not collected in this way. I would therefore be extremely grateful if any doctor knowing of such a twin pair could let me have information on:

- (a) whether the twins are monozygotic or dizygotic, and
- (b) whether one or both twins is affected.

Diagnostic criteria could be discussed at a later stage for each notified pair.

Would any doctor knowing of such a twin pair please contact me; address, 91 Corton Road, Lowestoft, Suffolk, England, if such a notification has not already been made.

M. P. CARTER, D.M.

Area Organizer (East Anglia),

National Society for Autistic Children.

104