Editorial.

THE LOCALIZATION OF PSYCHIC FUNCTION IN THE BRAIN.

A HUNDRED years ago the attack which science waged upon the surrounding darkness was little more than a raid carried out by a handful of soldiers of fortune who themselves forged the simple weapons of the fight. The battle front has extended since then, and the great engines of warfare have become complex. The adventurers of the past have become the professional army of the present. But the line of battle widens every day, and the soldiers in one brigade scarce know how it fares with their comrades in another.

The lack of co-ordination is particularly evident in the combined attack upon the problem of behaviour which is being made by psychology (as it were, from the air) and physiology (as it were, from the trenches). The information obtained by one seems often to be misinterpreted by the other. To lay our analogy aside, the problem of 'localization of function' in the brain is a case in point of what appears sometimes to amount to misconception.

The older view, that the optic thalamus is the chief 'seat' of sensation and the corpus striatum that of motion, received its death blow on the discovery that motor effects may be evoked by electrical stimulation of parts of the cortex cerebri. It was replaced by the physiological theory of localization of different functions in different parts of the cortex, to which was added the theory of 'subsidiary centres' at different lower 'levels' of the nervous system. Stimulation experiments revealed 'motor' centres in some areas of the cortex. Extirpation of other areas paralyzed general reactions to different forms of stimuli, even although stimulation of these other areas gave no motor reaction. The latter areas were therefore considered to lie further back on the path of conduction between receptive cell and cortical 'motor' area. The nerve fibres of the dorsal ('posterior') spinal roots had been called 'sensory' in the sense that they carried impulses which finally might subserve sensation (but not in the sense that they themselves felt); and in a similar manner these latter cortical areas were also labelled 'sensory'. This older terminology—largely abandoned in the case of the afferent nerves—has still been retained in the case of cerebral centres, in very much the same manner that
the cortical motor region for the lower limb is still called the Rolandic ‘leg’ area. But the retention of the word ‘sensory’ has given rise to misconception.

We must not suppose that the psychic phenomenon of sensation is localized by the physiologist in these so-called sensory regions. All that is claimed is that these different regions or centres form relay stations for nerve impulses derived from particular classes of stimuli; that these nerve impulses are perhaps rearranged there; and that they are transmitted thence to some more common motor mechanism. What is ‘localized’ is but the path of conduction of nerve impulses which may subserve sensation somehow or other; it is not the point in space at which sensation springs full armed as from the brow of Zeus.

The question whether or not psychic phenomena can accurately be localized at morphological points is insoluble in the present state of our knowledge. Perhaps it is a will o’ the wisp, scientifically absurd. We cannot even claim, because two different motor reactions (e.g., speech and gesticulation) may be evoked by the same stimulation, that therefore the seat of the psychic sensation must lie further back in the afferent path than the point of divergence. The possibility that a different individual sensation accompanies each of the different forms of response (although the stimulus is a common one) prevents us from claiming even this.

Head and Holmes have used the ‘thalamic syndrome’ of Roussy in an ingenious attempt to localize the “centre of consciousness for certain elements of sensation” (such as pain) in the optic thalamus. In one case they obtained a post-mortem examination which showed a lesion of the ventral and lateral regions of one optic thalamus, and this patient had, in life, over-acted to painful stimuli by speech reactions (as well as by other motor reactions). These observers suppose that ascending nerve impulses may pass into either (or into both?) of two paths when they arrive at the lateral nucleus of the thalamus. One of these paths is that towards the cortex cerebri; the other path carries them to a hypothetical ‘essential organ’ of the thalamus itself. They assume that the influence of the cortex upon the thalamus, through the known descending fibres of the former, is inhibitory; and that the over-action in the syndrome is caused by the removal of this inhibition where the lesion interrupts the path between cortex and thalamus.

If this is the case, destructive cortical lesions should also give the ‘thalamic over-action’ by a similar removal of the inhibition. But Head and Holmes point out that the fact that this does not occur may be due to the possibility that cortical lesions are not usually extensive enough to effect a sufficient reduction of the inhibition. The
fact that cortical lesions do not produce an effect upon sensibility to pain is used by them to support their theory that pain sensibility is not a function of the cortex, although it might of course be explained in a similar manner.

The importance of this well-known attempt to localize a psychic function in a subcortical region lies in the fact that the beauty and suggestiveness of the idea appear to be leading to its acceptance by psychologists as an ascertained fact. But the hypothesis is open to challenge.

We may leave aside the assumptions that the descending influence of the cortex upon the thalamus is one of depression, and of depression only, and that sensibility to pain is not affected by a lesion of the cortex however large it be. There remains the fundamental obstacle that the patient in question was able to over-act by *speech*.

If the thalamus itself takes part in the mechanism of the over-action, it *must therefore still be in connexion with the speech mechanism*. This fact forces us to one of two conclusions. Either the motor speech mechanism lies in the thalamus, and the thalamus in that case itself 'said' that it over-acted; or that mechanism lies in the cerebral cortex (or in some other region of the cerebrum than the thalamus)—in which case the 'essential organ' of the thalamus turns out to be merely a portion of, or a relay station in, the path between receptor and motor speech mechanism. In this latter case the site of the over-action *may* be in the thalamus (if the lesion removes an inhibitory influence of cortex upon *thalamus*); or it *may* be in the cerebral cortex (if the inhibitory influence removed is one of thalamus upon *cortex*).

Our knowledge of the physiological rôle of the thalamus in this over-action is therefore still indefinite. But even if we were able accurately to localize the site of the over-action, we should still be as far as ever from localization of the 'site of consciousness' for affective sensations. We may just as legitimately suppose it to be in the speech mechanism (or other motor mechanism) as in any preceding part of the path of conduction. We must return to our original thesis that we cannot at present localize psychic phenomena. All that we can say is this: *To judge from the characteristics of motor end-reactions, psychic phenomena may possibly accompany physiological activities which occur in certain (or in all) nervous paths of conduction.*

**REFERENCES.**

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