VEGETATIVE NEUROLOGY.

PHYSIOLOGY has such a direct bearing on clinical neurology that any new knowledge gained in the researches of the former must of necessity give a fresh impetus to the study of the disorders of function which fall within the domain of the neurologist. For the truth of this assertion we need look no further than to Sherrington's researches on the physiology of co-ordinated movement, without which the interpretation of disorders of motility and of muscle-tone might never have been rescued from the vague conceptions and loose terminology so conspicuous in most of the more early work.

In the field of the sympathetic nervous system our debt to the physiologist is equally great. It was Gaskell who first showed that the slender nerve-fibres connecting the spinal cord and the sympathetic chain subserve the functions of organic life; and it was he who stimulated the researches of other workers in this field, particularly Langley and Anderson, whose brilliant investigations culminated in the revelation of the plan and working of the sympathetic nervous system with a degree of accuracy which has left little for others to add. Yet the clinical bearings of these researches have been but tardily recognised, and it is unfortunate that there has arisen upon this secure physiological basis a maze of highly speculative hypotheses in which the clinician is in some danger of losing his way. Of these hypotheses perhaps no better example can be given than the conception of 'vagotonia' imagined by Eppinger and Hess. They started with the well-established antagonism between the two parts of the autonomic nervous system and endeavoured to prove that there are two corresponding types of individuals in which the normal balances between the sympathetic and the parasympathetic systems are disturbed in one or other direction. They suppose that if the parasympathetic system is unduly irritable a condition of 'vagotonia' is
produced; while a similar increase of irritability in the sympathetic is supposed to produce 'sympathicotonia.' Combination in different degrees of these two hypothetical opposite states is made to explain the origin of a range of symptoms which covers practically the whole field of medicine from asthma to vitiligo.

This speculation seems to be largely based on two assumptions: (1) that the sympathetic is tonically and continuously stimulated by adrenalin; and (2) that the hypothetical substance performs an analogous function in respect of the cranial and sacral autonomic nerve-fibres. It is unfortunate that neither of these two assumptions is supported by unchallenged evidence. Not only is there no satisfactory evidence of a constant secretion by the suprarenal bodies in normal conditions, but the hypothetical hormone 'autonomin' has never had the good fortune to be detected. Moreover, the hypothesis, thus weakly based, is in conflict with established facts, such in particular as those of the innervation of the sweat-glands. This in itself should be sufficient to discredit the whole speculation, yet 'vagotonia' has nevertheless become firmly established in medical terminology, and clinical records are rife with descriptions of 'vagotonic' and 'sympathicotonic' states.

The criticism and rejection of such ill-established speculations must nevertheless not be allowed to blind us to the importance of the vegetative nervous system in the genesis of nervous disease. There is, indeed, abundant evidence to suggest that that system plays an important rôle in the metabolism and economy of the individual. A system which regulates motor and secretory functions in all parts of the body, which regulates organs essential to life, and is intimately related to and connected with the glands of internal secretion, can hardly be supposed to escape the more morbid influences which attack the central nervous system itself. The contraction of blood-vessels, with its resulting effect upon the outpouring of sweat, the quickening of the pulse, and the dilatation of the pupil, are all manifestations of an emotional excitement which finds a ready explanation in involvement of sympathetic nerve-cells; but while the facts concerning the interrelationship of emotional factors and mechanistic alterations of the vegetative nervous system are incontrovertible, the evidence that certain nervous diseases are due to structural or functional changes of the sympathetic system is of the most vague and fragmentary
character and continues to be largely a repetition of the intelligent guesswork to be found in monographs as far back as the seventies of last century. Epilepsy, migraine, exophthalmic goitre, the myopathies, and many other diseases, have all been attributed to disease of the sympathetic nervous system; but where are the pathological data for such assumptions?

It is surely a singular reflection that while the pathology of the cerebrospinal nervous system has been dealt with in a manner so exhaustive that the smallest nerve-cell or fibre may be said to have its clinical significance, the morbid anatomy of the sympathetic nervous system remains an unwritten chapter.

It is true that tumours arising from sympathetic nerve-cells have been accorded some attention, and that the effects of traumatic lesions of the main sympathetic chain are given a time-honoured place in textbooks of neurology. But concerning morbid changes in the larger visceral sympathetic system little or nothing is to be found there or elsewhere. Scattered throughout neurological literature are references to congestion, round-celled infiltration, and nerve-cell atrophy of the larger plexuses; but one may search it in vain for any attempt to bring into something like systematic order the significance of these changes in the production of disease.

There are many factors to account for the barrenness of this field. In part we may note the failure of experimental transection of ganglia or nerve-fibres of the visceral sympathetic system to produce symptoms endowed with the obtrusive character of those produced by similar lesions of the brain and spinal cord; in part we may note the technical difficulties which attach to the study of degeneration in the sympathetic nerve-trunks and ganglia. Many of the latter are placed in most inaccessible positions, or are so scattered in the wilds of the body that they are difficult to find. None the less, in spite of these technical difficulties, we have here a large, important, and scarcely explored field in the mapping of which the neuropathologist might well expend a little of the energy which he devotes to other and possibly more recondite problems. The future of knowledge in this field gives promise that such labour would not be without its reward.