These considerations were envisaged long ago by Jackson when he spoke of the influence of the "four factors of the insanities." The factors as he originally set them out in 1874 were: (1) the depth of the dissolution or reduction; (2) the rapidity of the reduction; (3) the kind of brain in which the reduction occurs; and (4) the influence of external circumstances and internal bodily states on the patient who is reduced. It is Jackson's third factor which augurs so well for future investigations.

Conclusion

According to Lenin, "one fool can ask more questions in a minute than twelve wise men can answer in an hour." None the less, I believe I am justified in trying to trace some of the steps which aphasiologists will take in the future. The problems I have outlined are all of such a nature as would surely have intrigued Hughlings Jackson were he still with us. These questions of to-day and to-morrow reflect the enduring influence of Jacksonian principles, and the stimulus of his ideas continues to guide our investigations and our thinking. So far as the subject of language and its disorders is concerned, the future of neurological research is most certainly assured for yet another century.

BRAIN, MOVEMENT AND TIME*

BY

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It is one hundred and three years since the auspicious day that Victor Alexander Horsley was born; and forty-four years have passed since he bowed to human frailty in the sixtieth year of his life in the midst of his last inspired and inspiring campaign for his country and for his profession. One hundred years have passed since the founding of the National Hospital that he served so loyally and with such distinction. We meet here in this hall, and under the chairmanship of the President of that professional association in which Sir Victor played so vital a part. We are met here to pay homage to a great man, a distinguished neurologist and a leader in surgery—Sir Victor Horsley.

I am persuaded that Sir Victor is not dead. The knowledge and the impetus that he gave to medical science and technique live on—increasing in significance as following generations build and will continue to build upon them. We may feel the joy that we believe he feels in the centenary celebrations of his hospital, the pride that he has in the magnetic influence of Queen Square, which he helped to increase in his mortal days, and which continues to increase in force and scope to this present. If I read his thoughts aright he views with favour and admiration the achievements of the British Medical Association in these days; and its Royal Presidency must give him the deepest satisfaction.

Sir Victor was a surgical neurologist. I am indebted to his successor at University College—the late Sir Wilfred Trotter—for the phrase. Sir Victor was a devoted clinician. It has been well said that Queen

Square was and is the cradle of neurology. Sir Victor brought the humane grace of surgical therapy to attend the infant in that cradle—the clinical science of neurology. He pioneered surgical neurology as a specialized clinical discipline. He was an experimental physiologist. The clear and purposeful planning of his investigations yielded to him important scientific knowledge in spite of the relatively crude techniques then available.

The living pathology and especially the mechanical pathology of the nervous system also received Sir Victor's attention and interest, as witness his work on the mechanism of papilloedema and on the effects of experimental cerebral compression. It is in this latter field—that of the living and moving pathology of cerebral displacements—that I propose to present some comments that I believe would interest Sir Victor and may, I hope, satisfy the Memorial Trustees.

The title I have chosen—"Brain, movement and time"—is intended to imply simply the effect on the brain of its abnormal movement in relation to its containing coverings; and the differing effects of the varying periods of time in which these movements may take place. In other words, I shall endeavour to survey the vital pathology of the brain under stress of displacement at various time rates, the clinical effects and the principles of their management.

Anatomy, Physiology, and Pathology

The containing coverings of the brain, the skull, the dura mater and its partitions, constitute two forebrain compartments and a hind-brain compartment connected with each other by the "narrows" at the archway under the falx cerebri and at the incisura tentorii. When the brain is displaced the linear displacement is greatest at these "narrows." The anatomical arrangement of the blood supply is such that the arterial circle of Willis is relatively fixed to the skeleton of the head. The perforating arteries of the base cross the subarachnoid spaces in their short course from the fixed arterial circle to the overlying brain; and here they are peculiarly vulnerable. Under certain pathological conditions the brain may move extensively in relation to the fixed circle. The delicate perforating arteries are stretched, attenuated, and their calibre is reduced or occluded. There ensues ischaemia, hypoxia, or infarction in their territory of supply. This includes the diencephalon and mid-brain, containing the reticular formation, the archipallium, and the hypothalamus. Ischaemia in this field finds clinical expression in impairment of consciousness, alterations of emotional state, and derangements of homeostasis in several fields—notably control of body temperature, of respiration, of visceral motor and secretory function, and of pituitary endocrine function. The long tracts traversing this zone may suffer hypoxia.

The time factor is important in relation to brain displacements. If the displacement is slow enough the dural partitions yield considerably and so mitigate the effect; and the arteries adapt themselves to the deformation without loss of calibre until displacement becomes extreme. On the other hand, the more rapid the displacement the severer the effects as regards herniation through rigid apertures at the "narrows" and as regards early arterial occlusion. Abrupt displacement adds another factor. Sudden stretching of an artery causes spasm of its walls, and the occlusion and ischaemia due

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to such spasm may last for seconds, hours, or days, according to the severity of the traumatic stimulus.

Brain Displacement Syndromes

The principal clinical groupings associated with what I may designate brain displacement syndromes may be conveniently considered in four categories arranged according to the rate of displacement. (1) Those due to slowly expanding, displacing intracranial lesions, for example, slowly growing neoplasms, chronic abscesses, chronic subdural haematomas. The duration of displacement is over a period of years to a few weeks. (2) Those caused by rapidly expanding, displacing lesions—for example, rapidly enlarging neoplasms, acute abscesses, acute intracranial haematomas. The duration of displacement is from a few weeks to a few days. (3) Those due to the abrupt and temporary displacement occasioned by basal subarachnoid haemorrhage from rupture of a cerebral aneurysm. The duration of displacement is a few minutes to a few seconds, but the effect may be prolonged for minutes or days by arterial spasm. (4) Those due to the sudden, temporary brain displacement occasioned by dynamic head injuries. The duration of displacement is a fraction of a second. The duration of consequent arterial spasm may be seconds or days.

The clinician recognizes a very similar clinical picture in each of these four categories: in the patient with brain tumour in whom coma has supervened; in the patient afflicted by the apoplexy of acute spontaneous subarachnoid haemorrhage; and in the unconscious victim of dynamic head injury. There is the same unconsciousness, overbreathing, raised temperature, intestinal stasis, oversecretion in pharynx, bronchi, and stomach, bilateral extensor plantar response, and sometimes a "decerebrate" type of rigidity. This is the syndrome of severe diencephalic ischaemia. No less striking is the similarity of the effects of lesser grades of diencephalic ischaemia in the four categories. The milder clinical syndrome is evidenced mainly in impairments of consciousness—defects of memory retention, restrictions of perception, and inability to sustain attention. It is seen in the progressive expanding lesions—as a late symptom in the slow forms and as an early symptom in the more rapid; it appears during their process of dissolution of function. It is seen during the process of resolution of function in patients recovering from the coma of apoplexy or of head injury; and, in them, as the last evidence of a resolving diencephalic ischaemia.

The Common Pathological Basis—Diencephalic Ischaemia

The pathologist recognizes a similar ischaemic pathology in each of these displacement syndromes. I think we have been tardy in recognizing the ischaemic nature of the pathology because ischaemia sufficient to abolish function may produce no changes recognizable by naked-eye inspection after death; and not even to microscopic scrutiny until it has lasted for at least 24 hours before death. Indeed, it is only when the ischaemia is so severe as to cause haemorrhagic infarction, with those characteristic mid-brain and diencephalic haemorrhages, that it is easily seen. Significantly, they are similar in pattern in cases of death from tumour displacement, from acute basal subarachnoid haemorrhage, and from severe dynamic head injury.

While each of the clinical conditions mentioned has the evidence of diencephalic ischaemia as the common feature and as its central feature, each has its own particular pattern of symptomatology grouped round the central feature in time and space. We may survey each briefly.

The Slowly Expanding Intracranial Lesions

In these conditions, progressing slowly over years or months, symptoms and signs originating at the locus of disease may be strikingly evident—for example, by local epilepsy, hemiparesis, dysphasia, or hemianopia—or they may be absent. Next come the train of symptoms and signs dependent on secondary hydrocephalus, including papilloedema and other patterns of neurological disorder that we have learned to associate with cerebrospinal-fluid obstruction and accumulation. The secondary hydrocephalus is usually the precipitating factor of the next, third, phase—signs and symptoms caused by cerebral displacement. Thus, late in evolution of the slowly expanding lesions cerebral displacement reaches a point at which dural yielding and vascular adaptation can no longer compensate it; and the arterial blood supply to the diencephalon and mid-brain begins to suffer.

Headache signalizes stretching of the perforating arteries. Consciousness begins to show impairment in defect of memory retention, restriction of perception, and failure to sustain attention. As these symptoms deepen to coma, respiration increases in rate and depth, the body temperature rises, there is excess of secretion in the pharynx, bronchi, and stomach, there is gastrointestinal stasis, and the plantar response becomes bilaterally extensor. As the brain-stem is thrust down, an abducent nerve palsy appears. A variable but common feature in these cases is evidence of hypopituitarism.

In these slowly expanding lesions I think we understand the mode of production and the expected patterns of symptoms, their significance, and the possibilities of misinterpretation. We have recourse to one or more ancillary modes of investigation when in doubt about the site and nature of the lesion and the direction of its management. With this knowledge of the problem and with these aids it is now rare for the victim of a slowly expanding lesion to reach the stage of coma.

Treatment aims, firstly, at relieving dangerous cerebral displacement and, secondly, at eradication of the underlying disease. In the rare cases in which severe impairment of consciousness has supervened, treatment aimed at combating the diencephalic ischaemia must be added—as is discussed below in relation to head injuries.

It is worth reflecting that when the patient with a slowly expanding intracranial lesion begins to complain of headache he is really complaining of stretching of his basal perforating arteries. When he mentions vomiting he signalizes hypothalamic ischaemia. Forgetfulness means archipallial ischaemia. Papilloedema portrays hydrocephalic distension of his optic-nerve sheath. Thus interpreted these symptoms of brain displacement are alarming and urgent, and properly so—for at any moment irreversible infarction in diencephalon or mid-brain may supervene.

Rapidly Expanding Intracranial Lesions

The rate of displacement is more rapid, extending over a few weeks or a few days. The perforating

arteries have less time in which to adapt to deformation; and the dural partitions remain relatively rigid.

Early headache signalizes tension in the arteries; and at an early stage vomiting and impairment of consciousness indicate diencephalic ischaemia. Any symptoms originating at the primary locus of disease tend to be lost in the deepening coma. Hyperpnoea, hyperpyrexia, and hypersecretion soon appear, and the bilateral Babinski sign and decerebrate rigidity may follow. Through the still rigid tentorial aperture the temporal lobe hernia is thrust, carrying the oculomotor nerve before it and thus causing the homolateral dilated pupil and external ophthalmoplegia. This is in contrast to the abducent-nerve palsy of the chronic case. Also in contrast is the greater frequency of homolateral spastic paresis in the rapidly expanding lesions—due to the thrust of the opposite crus against the rigid tentorial margin as the hernia displaces the brain-stem to the opposite side.

Again I think we have become familiar with the cerebral displacement syndromes in these more rapidly displacing lesions—with the proper interpretation of their clinical signs and with their possible misinterpretation.

The demand for treatment is still more urgent, for at any moment the diencephalic ischaemia may advance to irreversible ischaemic haemorrhagic infarction. The primary object in treatment is the timely correction of the cerebral displacement, and so the restoration of an adequate blood supply to the diencephalon and mid-brain. The clot is removed, or the abscess is tapped, or local contusional swelling of brain tissue is afforded generous decompression. This is often not enough; for with a rigid tentorium and an impacted hernia the displacement requires more positive restitution. This is achieved by the rapid injection of normal saline solution into the theca while the head is still surgically open. By this manoeuvre the hernia is pushed back through the tentorial aperture. Often the patient awakens dramatically from coma as this is done. One can visualize the pleasant pink blush from restored arterial supply that pervades his mid-brain as this occurs. We call the procedure "positive disimpaction."

When, in severe cases, the effects of basal ischaemia persist, those measures are pursued to promote an effective circulation and to minimize the effects of ischaemia, as detailed later in connexion with head injuries.

Abrupt and Temporary Expanding and Displacing Lesion of Basal Subarachnoid Haemorrhage from Aneurysmal Rupture

The rate of displacement is still more rapid—a few minutes or a few seconds. As the basal cisterns rapidly fill with fluid arterial blood, the brain is displaced from the base of the skull and from the fixed arterial circle of Willis. The slender perforating arteries are rapidly stretched and attenuated and their territory is rendered ischaemic. The patient may cry out complaining of severe headache as his arteries are stretched; and he collapses in apoplectic coma as diencephalic ischaemia supervenes. The blood, which remains fluid in the subarachnoid spaces, soon disperses and the displacement subsides. The sudden stretching of the arteries has, however, induced spasm of their walls, so that the duration of ischaemia is prolonged; while it lasts,

perhaps for minutes, perhaps as long as 12 days, the patient remains comatose. As the arterial spasm relaxes and the diencephalic circulation recovers coma lightens to a state of impaired consciousness or confusion. Recovery may be rapid and complete. In the severer cases when coma is prolonged it is usually accompanied by clinical evidence of hypothalamic ischaemia and dysfunction—hyperpnoea, hypersecretion, gastric haemorrhage, gastro-intestinal stasis, hyperpyrexia; often a bilateral Babinski response develops. The blood-pressure usually rises initially—a reflex attempt to compensate hypothalamic ischaemia. Later it may fall and prove difficult of maintenance—an evidence of pituitary and suprarenal insufficiency.

Exact localizing evidence of the underlying aneurysm is usually absent, as the haemorrhage occurs into a free space. Symptoms and signs peculiar to acute basal intracranial haemorrhage include subhyaloid or vitreous haemorrhages from acute distension of the optic-nerve sheath by the extravasation; and when consciousness has been regained the features of meningeal and sensory nerve root irritation are evident. Blood is found in the cerebrospinal fluid on lumbar puncture.

Not infrequently some part of the extravasation penetrates into the brain substance. In this environment the extravasated blood clots. It then constitutes a rapidly expanding, displacing, intracranial lesion, which requires appreciation from its clinical syndrome and from angiography.

Management of these cases is largely controlled by the progress of restitution of consciousness—that is, of evidence of recovery of the diencephalic blood supply. Until this is restored angiography is dangerous as it may aggravate the ischaemia. In the mildest cases, when confusion clears within a few hours, angiography can then be proceeded with, and treatment of the aneurysm to prevent further bleeding can follow it at once. In the severer cases treatment to mitigate the effects of diencephalic ischaemia and to promote diencephalic blood supply must be instituted and maintained until consciousness is regained and confusion has largely recovered. Angiography and treatment of the aneurysm must await this recovery in spite of the risk of further haemorrhage during the waiting period, which may vary from a few hours to several weeks.

Intracerebral or subdural haematomas require treatment, in their own right, as rapid expanding, displacing intracranial lesions. It is worth noting that infarction of any large cerebral territory, which may arise in connexion with the treatment of a cerebral aneurysm, is associated with severe swelling of the affected brain tissue and may require adequate surgical decompression.

During local operations for cerebral aneurysms we may find the artery in a state of spasm or the surgical manipulations of the artery may induce severe spasm of it. Papaverine solution applied direct to the exposed arterial wall causes visible relaxation of this spasm in about five minutes. Indeed, we have sometimes left a small plastic tube in place so that we can inject papaverine solution to bathe the arterial wall at suitable intervals after such operations and so guard against recurrence of spasm. I do not know of any substance that can be administered systemically that has a similar effect on this traumatic type of local arterial spasm. If such a substance were available, I believe many of our problems of the diencephalic ischaemia associated with cerebral displacement could be resolved.

Abrupt Temporary Displacements of Dynamic Head Injuries

Concussion

By the qualification "dynamic" we indicate those head injuries associated with abrupt acceleration or deceleration of the head in space, with their transient deformation of the skull at the site of impact and their associated forces of momentum and of inertia. These factors cause the brain to move extensively within the skull. The displacements occupy but a fraction of a second. Consequent arterial spasm may last for seconds or for days. These head injuries are of increasingly frequent occurrence, and constitute an important social problem and a surgical challenge.

The transient deformation of the skull drives the brain to the opposite side under the falx and also down through the tentorial aperture. The anterior and posterior groups of perforating arteries are abruptly stretched. The elastic skull rebounds to its original form—in the absence of comminuted fracture—and the arteries are jerked back. The force of momentum now takes over and displaces the brain towards the site of impact, and the arteries are tugged in this direction quite extensively. There results almost immediate diencephalic ischaemia and loss of consciousness. As this abrupt tugging of these arteries induces spasm of them the effect on the diencephalon is less or more prolonged. In the mildest cases there may be only impairment of consciousness for a few seconds. In the severer cases spasm can probably continue for a week and unconsciousness for much longer. I suggest, then, that diencephalic ischaemia, so occasioned, is the pathological basis of clinical concussion. It may recover rapidly and completely. It may leave residual infarcts of greater or lesser extent. In the severest cases it is rapidly fatal, and may show the characteristic haemorrhagic infarction of severe ischaemia in the perforating artery and mid-brain territory, quite similar to that seen in expanding lesions. I believe, then, that in treating a patient with concussion we are, or we should be, treating him for diencephalic ischaemia.

In the severer cases the accompanying hypothalamic disorders, expressed as hyperpnoea, hypersecretion, vomiting, gastro-intestinal stasis, gastric haemorrhage, hyperpyrexia, bilateral Babinski sign, and decerebrate rigidity, soon follow the initial loss of consciousness.

The management of this state resolves itself into measures designed to promote an adequate diencephalic blood supply on the one hand, and to mitigate the effect of diencephalic ischaemia on the other.

Thus oligaemia, if it exists from external or internal blood loss, must be corrected; and effective fluid intake to maintain the blood volume must be ensured. The quality of the blood supply in respect of oxygen content requires a clear airway from nose or mouth to lung alveoli; and a sufficiency of appropriate nutrition must be maintained.

Mitigation of the effects of diencephalic ischaemia requires artificial regulation of the body temperature. In most cases the effect of the hypothalamic disorder is to interfere with the reflex mechanisms for thermostasis by loss of heat, and hyperpyrexia results. This increases tissue demand for oxygen which is already in short supply to the ischaemic diencephalon. A vicious circle is established and the temperature soars. The temperature must be reduced by artificial cooling; and heat production must be minimized.

In practice the application of these principles requires transfusion of blood if necessary and the administration of sufficient fluid and nutrition daily, intravenously at first, and by gastric tube as soon as the gastro-intestinal tract is available. The gastric tube is inserted early to control gastric accumulation and prevent dangerous regurgitation. There must be no impediment to pulmonary ventilation, for the slightest fall in blood oxygen aggravates the local diencephalic hypoxia. This is secured as may be required by posture—snoring in these circumstances can be fatal—by temporary tracheal intubation, or by tracheostomy. Unfortunately many patients with head injury vomit and aspirate gastric contents into the airways before they come under medical care. This may require early bronchoscopic clearance; and later this measure may have to be repeated to clear bronchial hypersecretion. If the available lung tissue has been reduced by atelectasis oxygen administration and antibiotics to prevent infection are required.

The temperature should be maintained at normal or a little below it. Cooling is achieved, as required, by exposure of the body surface at an open window, by the application of a moist sheet corresponding to artificial sweat, or the use of actual surface refrigeration. Heat production is minimized by chlorpromazine to control shivering and physical restlessness, and pethidine to control overbreathing. In extreme cases, and especially when there is extensive muscular spasm or rigidity, the use of the potent relaxants and of the artificial respirator are necessary.

Hypersecretion and gastric haemorrhage are dealt with by administration of atropine. Failure to maintain blood-pressure in spite of adequate fluids may indicate acute pituitary deficiency and require a maintenance dosage of A.C.T.H. Direct steroid therapy is to be avoided on account of the liability of these patients to gastric haemorrhage and to pulmonary infection. Occasionally gastric stasis may require the prolonged use of gastric aspiration and of intravenous administration of fluid, electrolytes, and nutrition. The care of the skin, lungs, and kidneys of these unconscious patients requires frequent change of their posture. Mouth hygiene is important; the eyes may require protection from undue corneal exposure; and the convenient disposal of urine and faeces requires attention.

It is important to bear in mind the aggravating effect that pre-existing cardiac, pulmonary, or renal disease may have upon diencephalic ischaemia; and to institute appropriate treatment concomitantly with that for the brain condition. Similarly, uncontrolled diabetes, hepatic insufficiency, or ill-advised "sedative" drugs may aggravate the effect of cerebral ischaemia by their interference with cell metabolism; and suitable counter-measures are required.

It may be noted that the hyperpnoea associated with the local diencephalic hypoxia of dynamic head injuries is what one might term an erroneous reflex. The body has only one response to oxygen lack—to breathe more; and this is a valid response to natural circumstances of increased oxygen consumption and to the pathological circumstance of impediment of the airway. As the diencephalon contains the neural mechanisms for this reflex, local hypoxia of the diencephalon from local cerebral ischaemia sets reflex hyperpnoea in action quite erroneously and uselessly. As the muscular effort of hyperpnoea gives rise to heat production it is actually harmful and should be artificially suppressed.

Similarly, the shivering reflex, spontaneous or provoked by surface cooling of the patient in hyperthermia, is a deranged reflex due, in this instance, to derangement of the diencephalic thermostatic mechanism. The threshold for its activation becomes set too high. Thus the patient begins to shiver with a body temperature of 103° F. (39.4° C.) or more. Again the doctor must step in to abolish this harmful heat-producing reflex activity and permit reduction of body temperature to a normal level.

Secondary Expanding Lesions

In addition to the diencephalic ischaemia of concussion, the abrupt brain displacements of dynamic head injuries may inflict damage that initiates the later development of a rapidly expanding, displacing, intracranial lesion. These secondary lesions may show their presence in a few hours, days, or weeks after the injury. Such are the extradural haematomas, initiated by the rebound of the indented skull from the underlying dura or by fracture of the skull and rupture of the middle meningeal artery; the subdural haematomas from the venous ruptures of "contre-coup"; or the lacerations and venous ruptures as the moving brain encounters the sharp sphenoidal ridges or tears a vein crossing the subdural plane; the local cerebral contusion and oedematous swelling occasioned by similar injuries or by the spread of traumatic phlebo-thrombosis. If such a complication arises in a patient who has suffered relatively brief concussion and has already regained consciousness, we have the classical syndrome of the "lucid interval" before the secondary lesion again causes clouding of consciousness by its secondary displacement effects. If unchecked it rapidly produces severe diencephalic ischaemic effects, and at any moment may cause irreversible haemorrhagic infarction. In these cases the correct assessment is clear and the indications for relief are urgent.

On the other hand, I know of no clinical problem more difficult than the diagnosis of a secondary, expanding, displacing process in a patient who is still unconscious from the initial concussion. The secondary process by its displacement intensifies the signs already present from concussion. To ascertain an intensification of signs in a comatose patient with a severe diencephalic ischaemic syndrome is no easy matter, for it is a repetition of the same cerebral insult before the effect of the first has passed off. It should be noted that the cerebral tissue is much less tolerant of a second insult than of the first, and this accentuates the need for early recognition and treatment. In circumstances of doubt it is obligatory to ascertain the absence or presence of such a secondary expanding, displacing lesion at once, usually by direct surgical inspection through multiple exploratory burr-holes. Treatment of secondary expanding lesions in head injuries varies with their nature. It may consist in the removal of clot or wide decompression of an oedematous part of the brain. One should err on the side of generous decompression, for there is often a phase of oedematous swelling of compressed cerebral tissue after clot removal which should be anticipated. In most of these cases "positive disimpaction" is advisable as the final step of the operation before the head is closed.

Fibre Ruptures

In addition to the diencephalic ischaemia of concussion which may recover spontaneously or may do so

under appropriate management, dynamic head injuries may cause direct rupture of axons in the cerebral white matter, especially of the corpus callosum and the centrum ovale, as has been shown by Dr. Sabina Stritch at Oxford. Extensive damage of this nature accounts for many of the early fatalities from severe head injuries. Some cases show gross, visible disruptions; but in others the lesions are not obvious and require careful microscopic elucidation by special methods. These lesions may account for the occasional cases in which signs of basal ischaemia soon settle but which remain with more or less spastic paresis, and with varying degrees of apathy or stupor. Curative treatment of these lesions would appear at present beyond our reach.

Variants of the Concussion Syndrome

I have mentioned the clinical syndrome most frequently associated with severe concussion; and I have expressed the view that its cause is diencephalic and mid-brain ischaemia. There are variants from the typical syndrome whose causes we do not understand in terms of the particular parts of the brain affected or spared. Occasionally, with profound coma, there may be an abnormally low body temperature, and respiration may be normal or slow. Sometimes the blood-pressure may remain low in spite of a sufficient blood volume. Sometimes the initial gastro-intestinal stasis, which usually resolves spontaneously after a day or two, may persist for several weeks. These, and other variants, require further classification, analysis, and pathological correlation.

Conclusion

I would make a plea for adoption of the term "brain displacement" in preference to "cerebral compression" as more expressive of the living pathology that exists. I would urge the use of the term "dynamic head injury" in classifying head injuries, to identify those in which the forces of momentum and inertia play a major part in damage to the brain. In some quarters this generation still seeks a physical sign upon which to base decision and action; and insufficient attention is paid to the psychological manifestation of impairment of consciousness as a neurological sign. The endocrine glands and hormones should be regarded as part of the effector mechanism of the nervous system. The neuronal communications may be compared to a telephone communication system—and the endocrine glands to transmitters, and their hormones to emanations, of a broadcast section of the communication system.

Sir Victor Horsley shows us how he, throughout his life, sustained by faith and courage, without aid of large research appropriation, combined an active surgical career with continued laboratory research work. He sustained an exacting surgical hospital practice; but, at great sacrifice, he restricted it to the sphere of action he had chosen. Throughout his life he prosecuted related laboratory work. He teaches us that clinicians, who meet the problems, must personally devote a substantial part of their endeavour to their solution in the laboratory. He would insist that in our hospital staffing arrangements to-day ample provision should be made for part-time clinical work combined with part-time laboratory investigation, both for young men in training and also throughout life for those with aptitude in both fields; and that in the administration of our ample research funds, sufficiently attractive means should be devised to sustain such combined research and clinical activities.