blood then results in an urgent attempt to prevent further accumulations of CO₂ in the tissues. This hyperpnoea is one of the clinical symptoms of acidosis.

(B) The acids which gain access to the blood are intermediate products in fat katabolism.

(C) The imperfect combustion of fats is associated with, and is perhaps due to the absence, or diminution, of, that product of carbohydrate metabolism which sets alight this combustion.

(D) A primary deficiency in carbohydrates is therefore an essential preliminary fault in cases of acidosis.

(E) This deficiency may be due to an insufficient supply of carbohydrates to the body, such as occurs in cases of, say, pyloric obstruction with copious vomiting, or to an imperfect metabolism of carbohydrates of which there is no lack. The anaesthetic chloroform probably interferes with carbohydrate metabolism by its direct action upon the liver cell.

(F) In cases of carbohydrate deprivation the deficiency may be at once made good by the administration of glucose. This cannot be given in adequate quantities by the mouth to patients afflicted with acidosis, on account of the vomiting which is so persistent. If administered by the rectum its absorption, when considered in relation to the urgency of the patient's need, may appear slow. If it is given subcutaneously the quantity taken up is small, owing to the enfeebled systemic and lymphatic circulations. When administered intravenously there is no reasonable limit set to the amount which can be given. Either a 5 or a 10 per cent. solution may be injected; an isotonic solution contains 4.15 per cent. of glucose. Matas of New Orleans, a surgeon to whom our debt of gratitude is very heavy, was the first to suggest (in 1911) that glucose might be given by continuous instillation. He has recently³ described an apparatus which renders easy the "continuous drip" intravenous administration of a glucose solution maintained at an equable temperature. About 8 pints may be introduced in the period of twenty-four hours; I have once introduced as much as 18 pints. A reaction is seen not infrequently—the patient shivers and then sweats, a miniature rigor occurring; no anxiety need be felt on this account. The cause of the reaction is doubtful; it may perhaps be the too rapid administration of the fluid.

(G) The mere accumulation of glucose within the blood is not, however, all that is necessary. The glucose must be utilized by the tissues. We owe to Thalhimer of Milwaukee⁴ the supremely important suggestion that this may be done by the injection of insulin.

My experience of the method is small, but it is enough to say that a new remedy of considerable importance is now at our disposal. Insulin must not be injected until there is a good store of glucose in the blood lest a hypoglycaemic reaction be caused; not less than 15 ounces of a 5 per cent. solution should have passed into the vein, and unless there is great urgency it is well to introduce an even larger amount, or to have a stronger solution. Lilian Farrar⁵ has used a 20 per cent. solution for intravenous administration. As she points out, glucose is not only a food and source of energy, but also a stimulant to the tissue cells and to metabolism; the liver function is conserved by it and the glycogen store increased.

Fisher and Snell⁶ record cases illustrating the value of the insulin treatment in pre-operative and post-operative non-diabetic acidosis.

Vomiting is often the warning symptom of impending acidosis. It is present along with great prostration and a considerable degree of air hunger, and with a sweet "new-mown hay" smell in the breath. The urine gives the characteristic reactions for acetone and diacetic acid.

Lavage of the stomach is the sovereign remedy for relief of the vomiting, which is most distressing. But in acidosis, as in cases of obstruction, or of adynamic distension of the stomach and duodenum, the lavage may need to be repeated so frequently as to be a tax upon the surgeon and a sore trial to the patient. Happily a most excellent method of lavage is made possible by the Jutte tube, or the Levin tube—tubes of about the same size as the Rehfuess tube. If passed through the nostril they may remain for hours without distress to the patient, who may then drink fluids freely.

I have, I fear, wandered discursively over a large field in dealing with the subject of cholelithiasis. There are so many problems that confront us as we cope with this very formidable disorder that it is not easy to choose among them. There is so much still to learn. The relationship of the liver to the spleen, of both to the pancreas, and of all three to the reticulo-endothelial system, and the effect which injury to the liver cell has upon the kidneys, are matters calling for fullest inquiry. Though we seem to know so little of any of these organs, and still less of their interplay, the work of the surgeon which has so marvellously contributed to our clinical knowledge may still hope to prove not unworthy of regard as one of the most fertile of all methods of research.

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Lectures

ON

THE SYMPATHETIC INNERVATION OF STRIATED MUSCLE.*

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LECTURE III.—PART II.

THE PRACTICAL APPLICATIONS (*continued*).

THE CONTROL OF TONE BY THE BRAIN.

THE SELECTION OF PATIENTS FOR RAMISECTION.

I SHALL now consider the reflex arcs concerned in maintaining the two elements of tone, and the scientific principles for the selection of patients for ramisection.

Plastic tone is enhanced in degree in the decerebrate animal. It may appear as a component of postural contraction immediately after the mid-brain is cut across, so that, normally, prespinal pathways are employed by the impulses maintaining it. When decerebration is performed, plastic tone is increased in degree as a result of the release of this prespinal sympathetic reflex arc from higher levels of control. In addition to this, contractile tone is enhanced in value because of the release of a somatic prespinal reflex arc. In animals recently decerebrated this increase of contractile tone selects the antigravity muscles, and their antagonists are reflexly inhibited. The increase in plastic tone rigidly maintains the extended posture, which is endowed by contractile tone upon all four limbs of the quadruped, and maintains the condition of "reflex standing" for long periods of time. When the relaxation from the posture of extension occurs, plastic tone still remains, as is proved by the presence of "lengthening and shortening reactions."

The prespinal reflex arcs subserve the two components of tone of the normal skeletal muscle, and overactivity of these arcs accounts for the phenomena exhibited by the muscle in decerebrate rigidity. It was shown by Sherrington that cutting across the medulla oblongata led to flaccid paralysis of the limbs. More precisely, as Magnus has shown, the limits within which section of the brain stem produces decerebrate rigidity are as follows: The cranial limit is a plane passing dorsally at the level of the caudal limit of the superior colliculus and ventrally in the region immediately caudal to the mammillary bodies. The decerebrate rigidity persists until the level of entrance of the eighth nerve is passed. Obviously, therefore, the nuclei that constitute the cell stations of the prespinal reflexes responsible for decerebrate rigidity lie within these limits.

The work of Thiele in 1905, which is supported by the recent results of Bazett and Penfield,²² indicates that the integrity of the lateral vestibular nucleus of Deiters is essential for the development of decerebrate rigidity. It may be concluded that this nucleus takes part in the completion of one of the reflex arcs, the hyperactivity of which is responsible for decerebrate rigidity. The vestibulo-spinal tract (Fig. 13), which lies in the region of the emerging motor root fibres of the spinal cord mainly on the same side as the nucleus from which it originates, constitutes the descending limb of the reflex arc.

The nucleus of Deiters is a special afferent somatic nucleus, and the reflex arc in which it is incorporated constitutes, therefore, the somatic prespinal reflex pathway. It subserves contractile tone, which has a selective incidence upon antigravity muscles maintaining the body erect.

The question arises whether the nerve cells incorporated in the sympathetic prespinal reflex arc can also be identified. The levels between which section of the brain stem produces decerebrate rigidity suggest that the formative reticularis of the pons supplies the cell station of this arc. The reticulo-spinal tracts of the pons are anatomically of special

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importance. Collier and Buzzard have termed them the ponto-spinal tracts. The anterior ponto-spinal tract, which is uncrossed, descends in the lateral zone of the medulla oblongata and the anterior funiculus of the spinal cord. The lateral ponto-spinal tract crosses to the opposite side and lies in the lateral zone of the medulla oblongata and in the lateral funiculus of the spinal cord.

Sherrington's experimental results are compatible with the view that the vestibulo-spinal and ponto-spinal tracts convey impulses to the spinal cord that are responsible for decerebrate rigidity. Section of the lateral half of the bulb abolishes the rigidity on the same side as the lesion. Section of one ventro-lateral column of the spinal cord in the cervical region destroys the rigidity in fore and hind limbs of the same side, while section of one lateral column in the upper lumbar region abolishes the rigidity in the hind limb of the same side as the section. The ascending limbs of these arcs are also in the antero-lateral funiculus: for section of the posterior funiculus does not abolish decerebrate rigidity.

In an experiment to study this point I have found that plastic tone does not disappear after removal of the nucleus of Deiters. First, decerebrate rigidity was produced by cutting across the mid-brain between the superior and inferior colliculi. Extensor rigidity developed rapidly and well. Now, in a phase of relaxation, when plastic tone was well shown in the right hind limb, I isolated the region of the lateral angle of the fourth ventricle on the right side by a crescentic incision, removing Deiters's nucleus and severing the vestibulo-spinal tract by this procedure. But the "lengthening and shortening reactions" were still exhibited. A second section at the junction of the pons and medulla was then made. The limbs became flaccid immediately. The "lengthening and shortening reactions" were no longer to be elicited. Obviously plastic tone depends upon a reflex arc that attains a level between the mesencephalon cranially and the medulla oblongata caudally. This is compatible with the view that the ponto-spinal tracts subserve the function of plastic tone.

It is possible for an incomplete lesion of the spinal cord to affect the pyramidal and concomitant descending tracts, so releasing the primary spinal arc from control, without affecting in any considerable measure the vestibulo-spinal and ponto-spinal tracts. Riddoch and Buzzard²³ have recently described cases of quadriplegia from high cervical lesions of the spinal cord, in which the distinctive qualities of decerebrate rigidity were reproduced. Riddoch reports that *post-mortem* examination of one of these patients revealed a tumour in the region of the posterior and lateral columns of the fourth and fifth cervical segments of the spinal cord. The ventral portion of the cord was relatively little affected.

It is clearly to be borne in mind that the extensor posture is a relatively unimportant character in establishing an analogy between the condition of spastic paralysis in man and decerebrate rigidity experimentally produced. The posture assumed is due to the selective incidence of contractile tone upon the skeletal musculature. Riddoch and Buzzard recorded that, in their patients, the position of flexion was sometimes encountered. On these occasions the flexors were more spastic than the extensors. This condition could sometimes be induced by holding the limbs in the flexed position for a short time. Further, McAlpine reported a case in which rigidity was not confined to extensor muscles, and flexor rigidity of the knee-joint was sometimes obtained.

Graham Brown has demonstrated beyond all doubt that plastic flexor tone may be exhibited in the limbs of the

monkey. These observations are of added significance in the light of Bazett and Penfield's work on chronic decerebrate rigidity. These authors describe two kinds of decerebrate rigidity—extensor and flexor. Their observations prove that the somatic reflex arcs producing contractile tone may selectively act upon either the extensor or flexor muscles. In extensor rigidity the reflex, of which the vestibulo-spinal tract is the descending limb, is involved. In flexor decerebrate rigidity the primary spinal reflex is, no doubt, responsible for the activity, for severe injury to the spinal medulla produces paraplegia in flexion. In both types of decerebrate rigidity plastic tone, which is enhanced in degree, and coexists with contractile tone, is maintained by the sympathetic prespinal reflex arc. In each case it is inhibited in the antagonists of the muscles exhibiting the rigidity.

Inhibition in the antagonists is not so complete in man as it is in the condition of acute decerebrate rigidity in animals. Dr. Royle has called attention to this point. Riddoch mentions the fact that though the antagonistic muscles are relatively relaxed "they are certainly in stronger contraction than the corresponding muscles of a healthy individual."

I believe the explanation of this is that in an animal that has recently been decerebrated the extensor reflex arcs, chiefly through the nucleus of Deiters, immediately manifest increased activity. The isolated flexor spinal arcs regain activity only after a period of inactivity. When this occurs the selective action of contractile tone on the extensors is not so complete, for the spinal arc endows this aspect of tone upon the flexors also. Reflex inhibition is now not complete. Bazett and Penfield's experiences with the chronic decerebrate animal support this view. Moreover, in patients suffering from spastic paralysis, plastic tone is not inhibited in antagonistic muscle groups; which accounts, in some measure, for resistance to active, reflex, or passive movement of the limb.

I have commented on the work of Bazett and Penfield to emphasize the fact that the posture of the patient is not the all-important criterion of rigidity. Even the flexed position may be due to a combination of both contractile and plastic tone which together cause rigidity in the sense in which this term is employed in this lecture. Con-

versely, the extensor position may be wholly due to contractile tone. Sympathetic ramification in such a case would be of no avail. Extensor fits usually fall into this category. This is beautifully illustrated in a case recently reported by Walshe,²⁴ in which two stages of the illness were observed. In the first stage the patient exhibited a condition of decerebrate rigidity. The limbs were extremely "plastic" and tended to remain for a considerable time in any position passively imposed upon them. The lower extremities were extended and adducted and the feet plantar-flexed. As is usual in man the upper extremities were semiflexed at the elbows, with the forearms pronated and the wrists and digits flexed. This attitude often replaces that of extension of the fore limbs of quadrupeds because the upper extremities are no longer "simple locomotor props." Passive movement revealed the presence of the "clasp-knife" character of the spasticity. The tendon-jerks exhibited tonic prolongation, and in some instances in response to repeated taps "step-ladder" shortening of the muscle was observed. In recording these features, Walshe established beyond doubt the identity of this condition with decerebrate rigidity experimentally produced.

In the second phase of the illness this condition was replaced by the progressive failure of reflex activity and deepening coma. In this stage tonic fits appeared as a con-

FIG. 13.—Diagram to indicate the part played by the vestibulo-spinal tract (arising from Deiters's nucleus) and the ponto-spinal tract (arising from the reticular nucleus of the pons) in the two reflex arcs concerned respectively with contractile and plastic tone.

comitant of cardiac and respiratory irregularities. In these fits the respiratory muscles were fixed and the limbs became so rigid in their customary posture that no tendon reflexes could be elicited. In severe fits of this kind the head retracted forcibly. At this stage none of the essential qualities of decerebrate rigidity, enumerated as characterizing the first phase of the illness, were exhibited, for plastic tone was not apparently enhanced in degree. The second phase was in all probability due to hyperexcitability of the somatic reflex pathways due to asphyxia or anaemia (cf. Walshe, Stewart and Pike, Warner and Olmsted, Hunter and Royle). The fits are not comparable to decerebrate rigidity in which the enhanced tone is due to release in function of two prespinal reflex arcs—somatic and sympathetic. Yet the posture displayed in each condition is the same. Taking into account the results of tests for the presence of plastic tone in addition to posture Walshe and Riddoch have been able to establish the essential identity of decerebrate rigidity and the condition of spastic paralysis, such as that which occurs in hemiplegia, quadriplegia, and the extended form of spastic paraplegia.

The only patients who can be expected to benefit from the operation of ramisection are those whose voluntary control of their muscles is intact, but whose movement is hampered by excessive plastic tone. By cutting the sympathetic nerves plastic tone is eliminated and the limbs freed from the spasticity. The presence of "lengthening and shortening reactions" is an indication for the operation of sympathetic ramisection for the relief of spastic paraplegia. Slowness in execution of the tendon reflexes is an important indication for the operation.

Conversely, rapidly executed reflex movements usually constitute a definite contraindication. The greater number of spinal injuries are accompanied by this type of reflex and plastic tone is at a minimum. This is due to the fact that a prespinal reflex is essential in higher forms for the maintenance of this property of muscle, and usually, though not always, as will be proved subsequently, this reflex is rendered inactive when the spinal cord is injured.

HIGHER LEVELS OF THE BRAIN CONTROLLING THE TONIC REFLEX ARCS.

Magnus has shown that in animals, such as the rabbit, cat, dog, and monkey, the whole of the cerebral hemispheres, including the corpora striata, may be removed and the tone of skeletal muscle remain of normal intensity. Nor is the tone altered when the brain stem is cut across behind the thalamus. Such a "mid-brain" animal, unlike the decerebrate preparation, is capable of a great variety of reflex activities—as walking, running, and jumping. It can right itself if overturned, the head at once resuming its normal posture. These are the "postural reflexes" of Magnus. Obviously, therefore, in these animals, the mid-brain provides an important part of the nervous mechanism utilized in the regulation of posture. It is when this region is taken away that release of the prespinal reflex arcs takes place to produce decerebrate rigidity. For instance, in higher forms, at least, as Magnus's observations upon the monkey reveal, the connexions of the visual pathway with the mid-brain play an important part in the nervous regulation of posture. Tecto-bulbar connexions therefore control the prespinal reflex arcs in these forms. Moreover, decerebrate rigidity develops after the removal of the red nuclei, and, as Walshe mentions, "it seems as though removal of the greater part of the red nucleus is an essential preliminary to the development of rigidity." In this process rubro-reticular fibres, which in man have attained special importance, are severed. The cells of the

substantia nigra are also known to send axones to the tegmental region of the brain stem.

In view of prevailing conceptions it is noteworthy that in the animals described by Magnus the postural reactions are carried out by the mid-brain in the absence of the corpus striatum. It would, however, be unwise to argue that in man the mid-brain structures exhibit this independence of the corpus striatum, for the principle of the shifting forward of function in the nervous system must not be lost to sight in this connexion. Moreover, it may well be that the physiological value of the corpus striatum is only adequately expressed in the presence of voluntary activity. In such a case the symptomatology of lesions of the corpus striatum, with the cerebral cortex intact, will be different from the experimental results of complete removal of both the cerebral cortex and corpus striatum.

For these reasons the observations of Magnus that "thalamus" and "mid-brain animals" exhibit tone of normal intensity and distribution cannot unreservedly be applied to man. It is possible that the corpus striatum in man, with the cortex intact, controls through the intermediation of subpallidal nuclei, as the substantia nigra, the sympathetic prespinal arc subserving plastic tone. To study this point I have removed the corpus striatum on one side in birds, employing the domestic fowl and the sea-gull for this purpose. Birds represent a group widely divergent from mammals. In birds the corpus striatum has attained a relatively enormous importance, both structurally and functionally, and in certain respects its influence may present a closer analogy to that of man than is the case in most mammals. The operation is easily performed on birds because the corpus striatum constitutes, except for the septum and a small superficial area of cortex, the entire cerebral hemisphere. Further, in removing the corpus striatum, which is readily accessible because it forms the greater part of the surface of the hemisphere, the cerebral cortex may be lifted up and left in communication with the septum. In this way the tractus cortico-septo-mesencephalicus is left intact.

This operation produces rigidity of all the limbs. Standing is not evenly maintained owing to rigidity of the hind limbs. On this account in sea-gulls a tendency to rise on the toes is constantly exhibited and the bird does not maintain any given position for long. Birds in this condition often squat down, presumably to avoid balancing on the rigid limbs. The wings are also rigid, and here the nature of the rigidity may readily be studied. The wings are sometimes maintained in a position somewhat higher than usual, so that the adjacent borders are almost in contact with one another. The rigidity maintains its intensity during passive movement, and on account of its rigidity the wing remains fixed in the new position assumed. This occurs even though the position be so abnormal, as when the wing is folded over the mid-line of the back, that the fibrous structures contained within are put upon the stretch. Dependent positions are maintained equally well. Both wings exhibit this phenomenon after the unilateral operation, but the rigidity is greater on the same side as the injury. The rigidity is responsible for slowness of movement, especially on the same side. This is readily seen on watching the movements of the wing during attempted flight while the bird is held captive in the hand. A greater effort is necessary to carry the wing through a smaller range of upward movement than on the less affected side; increased effort marks downward movement also, and the wing seems to overshoot the position at which it normally

FIG. 14.—Diagram to represent in an entirely tentative way the influence of the cerebral cortex (acting via the cerebellum), corpus striatum, substantia nigra, and red nucleus upon the activity of Deiter's and the reticular nuclei. Only those tracts which are mentioned in the text have been represented. The relays in the ascending spinal tracts have not been indicated.

ends. These alterations in the nature of the movement of the wings are greater on the side of the injury, so that the flight of such a bird, when released, is always in a circle, the bird tending to fly toward the more affected side.

This diffuse muscular rigidity is strongly reminiscent in an exaggerated form of the condition of the wing already described, in which, after the removal of contractile tone, plastic tone is accessible for independent study. I cannot avoid the deduction that increased plastic tone in both flexor and extensor muscles of the wing is responsible for the diffuse muscular rigidity which follows the removal of the corpus striatum in birds. This element of tone is so enormously increased that manifestations of the presence of the contractile component are in the background.

The condition of rigidity of the wing of birds above described is remarkably similar to Parkinsonian rigidity in man. Its diffuseness, resistance to stretching, and the tendency for fixation of the muscle at any length passively imposed upon it, are common to the two conditions; and in both conditions poverty and slowness of voluntary movement, which are probably due in part to the underlying phenomenon of rigidity, are notable concomitants.

Removal of the influence of the sympathetic innervation by section of the cervical sympathetic trunk caudal to the brachial plexus abolishes the rigidity of the wing. This property, like normal plastic tone, is subserved therefore by the sympathetic system. It is difficult to escape the conclusion that the basis of the rigidity following lesions of the corpus striatum is increased plastic tone, which is exhibited in all muscle groups, and that this is due to removal of control from the sympathetic prespinal reflex arc which maintains the plastic tone of normal skeletal muscle.

The different manifestations of excess of plastic tone in those cases of spastic paralysis which resemble experimental decerebrate rigidity, and in the condition of Parkinsonian rigidity, are capable of reconciliation. The fundamental difference between the two conditions is that, while plastic tone is increased in both, an excess of contractile tone is also exhibited in spastic paralysis, while it is not greatly in evidence in Parkinsonian rigidity. Contractile tone is selective in its incidence, and the plastic tone of the antagonists of the muscles selected by it is, to a very varying degree, reciprocally inhibited. In Parkinsonian rigidity this selective incidence upon muscles, and consequently the selective inhibition of plastic tone in the antagonists, are absent. In spastic paralysis resistance to passive movement is considerable till the position imposed upon the limb by contractile tone is overcome, when the resistance becomes less. A combination of contractile and plastic tone is responsible for the initial position in spastic paralysis. The posture assumed in Parkinsonian rigidity is due to plastic tone only exhibited in agonists and antagonists, and therefore resistance to passive movement is the same throughout the whole range of movement. The fact that postural reactions and intercurrent reflex movements are not exhibited in Parkinsonian rigidity as in spastic paralysis is a further indication that somatic activities, which include contractile tone, are in abeyance in this condition. The mechanism which inhibits plastic tone in order that a movement may be performed or to allow a new posture to be assumed is no longer active. This effect is probably the cause and not the result of the rigidity—that is to say, a predominant function of the corpus striatum is to regulate plastic tone. From a consideration of the other effects of corpus striatum lesions it seems that this regulation is especially in the interests of automatic and associated movements, and of emotional expression. These functions are no doubt impaired by the lesion; the rigidity itself may limit these activities still further.

It remains to be proved that the rigidity of paralysis agitans and allied conditions is subserved by a proprioceptive mechanism in order to establish completely the identity of the tendency in this condition to fixation of the muscle at any length imposed upon it, with the "lengthening and shortening reactions."

In the first place it must be borne in mind that the sympathetic reflex arc subserving plastic tone is not necessarily at the same level as the somatic connexions. There-

fore, in posterior root section, contractile tone may be affected without removing plastic tone.

Surgical operations upon the posterior nerve roots have necessarily been less complete than is possible under experimental conditions.

Leriche's cases, in which posterior root section was performed for the relief of paralysis agitans, are evidence of this. In one case the fifth, sixth, and eighth cervical posterior nerve roots were divided. In this case the rigidity was undiminished. Obviously, however, the sympathetic afferent connexions of the skeletal muscle of the limb were left intact by this operation because the posterior root of the first thoracic nerve was not attacked. Therefore this case does not disprove that rigidity is maintained by a proprioceptive mechanism. The first principle of posterior root section in man should be to include the posterior root of the first thoracic nerve in the case of the upper limbs, and of the first or second lumbar nerve in the operation for the lower extremities.

However, Walshe has recently brought forward strong evidence that the rigidity of paralysis agitans is due to the activity of a proprioceptive reflex mechanism. He has shown that injection of 1 per cent. novocain into the muscle at its motor point causes rigidity to disappear. While admitting the possibility that this effect may be due to direct action upon the muscle, he shows convincingly that the balance of evidence is in favour of the view that the afferent nerve terminations are affected.

Walshe's investigations upon the rigidity of paralysis agitans beautifully illustrate the fact that the tremor exhibited in this condition and the rigidity are subserved by two different nervous mechanisms. For although novocain abolishes rigidity the tremor is totally unaffected. The indications are that impulses through the cerebro-spinal tracts are involved in the production of spontaneous tremor, for it disappears after hemiplegia. This is in accord with the results of sympathetic ramisection in this condition. The rigidity is markedly diminished, voluntary power is increased, and movements are more ample and rapid, but the tremor is not appreciably affected when well established. But a patient with Parkinsonian rigidity without tremor, as for instance as a sequela of encephalitis lethargica, may derive sufficient benefit to justify the operation.

In a recent paper McAlpine²⁵ has described a patient who, as a sequela of epidemic encephalitis, developed Parkinsonian rigidity on the right side and a condition strongly resembling decerebrate rigidity on the left side. According to views advanced here, the diffuse muscular rigidity on one side was due to a generalized increase of plastic tone, lack of selective incidence being due to the absence of increase in contractile tone. The rigidity on the other side, however, was sometimes flexor and sometimes extensor. Therefore contractile tone was present and acted selectively on different groups of muscle at different times. Plastic tone was also enhanced in the upper and lower limbs of this side. It was exhibited by the presence of the "lengthening and shortening reactions." It was shown to the greater extent in the flexor and extensor muscles at any particular time according to the selective incidence of the contractile tone at the time. In the erect posture tone predominated in the extensor muscles of these limbs. Since the tone responsible for this decerebrate attitude consists of the two normal components of tone, the term "plastic tone" alone is clearly inadequate to describe the condition. Sherrington's term "postural tone" may be used for this purpose. Contractile tone reflexly imposes the position upon the body and plastic tone maintains this position once it is assumed. On passive movement the characteristic "lengthening and shortening reactions" indicating the presence of plastic tone are elicited.

In a natural attitude such as standing contractile tone is responsible for selective action upon the antigravity muscles and plastic tone fixes the length of the muscle fibres necessary to maintain this position. To accomplish voluntary movement from this position the prespinal reflex arcs responsible for postural tone must be controlled by the cerebral cortex. Evidence is accumulating that the cerebral cortex not only causes contraction of the appro-

priate muscle groups by way of the cerebro-spinal tracts, but inhibits the reflexes responsible for postural contraction. It effects this purpose through the intermediation of the cerebellum (Figs. 13 and 14).

An inhibitory pathway leads from the cerebral cortex to the anterior part of the superior vermis of the cerebellum through the medial part of the crus cerebri and the pontine fibres. Stimulation of the crus in a decerebrate cat leads to complete rigidity in the limbs of the same side and diminution of rigidity in those of the other side. Warner and Olmsted²⁶ have traced the origin of this inhibitory tract to the frontal region of the cerebral cortex. When the motor area was cut away no increase of extensor tone was observed on either side of the body. Definite rigidity developed at once in the opposite fore and hind limbs when the frontal area was removed. This observation is of great significance in view of the fact that, in man, pure cortical lesions of the pyramidal tracts do not produce spasticity. On the other hand, lesions of the area of the frontal lobe from which the fronto-pontine tracts originate (Campbell) are associated with a condition of "tonic innervation" expressed as inability to relax a contracted muscle group.

Warner and Olmsted have elucidated the course of this tract arising in the frontal region by studying the inhibition of rigidity obtained by electrical stimulation of the cut surface of the brain in various situations. They found that the inhibitory effect of the tract is bilateral, but predominantly on the crossed side. The tract passes from the frontal lobe through the medial part of the internal capsule. Below the level of the superior colliculi it crosses, in greater part at least, to the other side of the brain stem. It continues to the cerebellum by way of the middle cerebellar peduncle. The inhibitory tract is therefore fronto-ponto-cerebellar in its connexions. (See Figs. 13 and 14.)

Warner and Olmsted state that when this tract is stimulated a limb exhibiting extensor rigidity goes "limp and flaccid." Miller and Banting describe the effect of stimulation of the anterior surface of the cerebellar vermis as being "immediate and general relaxation of the rigidity." Obviously the inhibition affects both contractile and plastic components of tone which are enhanced in degree to produce decerebrate rigidity. The fronto-ponto-cerebellar tract, therefore, inhibits the effect produced by the somatic vestibulo-spinal and sympathetic ponto-spinal tracts. When this tract is ineffective the power to relax the muscles after being employed in muscular effort is defective. In the absence of a case purely of this nature in Dr. Royle's series it is interesting to note that the effect of sympathetic ramisection improves the power of relaxation of contracted muscles in spastic paralysis. It is to this fact that the increased control and rapidity of effecting voluntary movement are due. The patient need no longer inhibit plastic tone. His cortical control is mainly concentrated on executing movement.

It is worthy of note that tumours of the middle lobe of the cerebellum give rise to fits of a tonic character in which the extensor attitude of decerebrate rigidity is assumed (cf. Hughlings Jackson). Sherrington found that a rigidity somewhat similar to that ensuing on removal of the cerebral hemisphere set in after median section or ablation of the cerebellum. He was not convinced that the two conditions were identical. Thiele,²⁷ however, regarded the bilateral rigidity which followed removal or bisection of the vermis as being like decerebrate rigidity. He explained it as being due to loss of the inhibitory control of the vermis over the nucleus of Deiters. No doubt also the ponto-spinal tracts are released from control. It will readily be seen that the only effect of sympathetic ramisection upon tonic extensor fits following interference with the fronto-ponto-cerebellar pathway is to diminish the degree of rigidity during the extensor spasms. Since contractile tone remains practically unaffected after this operation the extensor attitude will still be assumed. This is another instance of posture alone not being an indication for ramisection.

The fact that the cerebral cortex employs the cerebellum as an intermediate organ through which it exerts an inhibitory influence over the postural reflex arcs of the brain stem is of great general significance. It confirms the

suggestion of Walshe that "the cerebellum is the organ through which the cerebral motor cortex influences postural activities and regulates posture in the interests of co-ordinated purposive movement." "It may be," he proceeds, "the subordinate mechanism employed by the cerebral cortex in the refined correlation of posture and movement." This view indicates that the cerebellum is employed by the cortex in the performance of active movements also. It reinforces postural tone in order to maintain the attitude of the body assumed during the performance of the movement initiated and executed by motor impulses which have their origin in the cerebral cortex.

In 1900, discussing the significance of the cerebro-cerebellar pathway which passes through the brachium pontis, Sherrington said: "Thus one and the same cerebral discharge may perhaps pour upon the motor root nerve cells a combination of cerebral and cerebellar impulses." It is on account of the influence of the cerebral cortex in the regulation of posture through the intermediation of the cerebellum that lesions of the cerebellar hemisphere cause inco-ordination of voluntary movements.

When a voluntary movement is completed cortical impulses reinforce contractile tone to determine the attitude assumed by the body. Plastic tone aids in maintaining this position because the muscle fibres exhibiting it are fixed at their new length. After sympathetic ramisection this accessory contribution is lost, but the influence of the cerebral cortex on contractile tone remains. In this connexion it is well known that tone is increased by attention. Dr. Royle has noticed that during the first period of re-education after sympathetic ramisection when the patient's attention is focused on the performance of a voluntary movement, under orders, the cortex greatly increases the amount of contractile tone exhibited in the limb. This increases the difficulty of the patient in performing the movement. On this account gently executed movements are far more effective than considerable effort in this stage of the re-education process.

Evidence is not wanting that plastic tone is of use also during the performance of voluntary movement. In ordinary circumstances a patient who has been subjected to the operation of sympathetic ramisection cannot detect any disability in consequence of the loss of the sympathetic nerve supply of his voluntary muscles. However, to test this matter closely I have observed the effect of sympathetic denervation after the exercise involved in the flight of a bird. During the flight of a sea-gull in which the unilateral operation has been performed, no obvious difference is to be detected in the movement of the two wings. After completing a flight of one hundred metres, however, the position of the wing on the operated side is in marked contrast to that on the normal side (see second lecture). It is drooped and abducted to a remarkable degree, although hardly any deviation from the normal position may have been visible prior to undertaking the flight. The explanation of this observation seems to be that plastic tone enables the fixing muscle fibres to follow up the contraction of the active fibres and assist in maintaining the intermediate positions assumed at all stages of the movement. When plastic tone is removed, voluntary effort is increased in order to maintain these intermediate positions against the action of gravity as well as to perform the movement.

It appears, therefore, that in voluntary movement postural tone, made up of a contractile and a plastic component, is first inhibited; the "movement" muscle fibres then contract and are assisted in maintaining the advantage gained by the plastic tone of neighbouring "fixing" fibres which are passively shortened; and finally cortical impulses determine that contractile tone is exhibited in the muscles whose contraction is necessary to confer the required position upon the body; the length of the muscle when this position is attained is maintained by plastic tone exhibited by "fixing" muscle fibres.

In certain cases of spastic paralysis plastic tone is in excess. This interferes with voluntary activity, because it is imperfectly inhibited at the outset of voluntary effort. When excessive voluntary effort initiates movement the exaggerated degree of plastic tone, which is present in both agonists and antagonists, as Dr. Royle has shown, tends to provide resistance to the execution of the movement by

fixing, to an abnormal degree, the length of the muscles of the limb at all the intermediate positions through which the moving part passes. In this way the effects of diminished voluntary power are enhanced in degree on account of excess of plastic tone, which is no longer adequately controlled by the cerebral cortex. In fact this defective control is not infrequently the greater disability, and on account of it the voluntary motor pathway may be unable to function to the full extent of which it is capable. This desideratum is attained by removing the plastic tone entirely, when the rigidity of the muscles disappears. The result is that the initiation and the execution of voluntary movement are both improved.

Dr. Royle found that one patient, with a gunshot wound of the cerebral cortex, who illustrated the presence of excess of plastic tone to a striking degree before the operation, was able to inhibit the tone of the extensor muscles of the knee following ramisection. He was unable to flex the knee voluntarily, but he could inhibit the tone of the muscles sufficiently to let the leg fall into the flexed position. This was impossible prior to the operation. In other words, this patient was unable to inhibit plastic tone. After ramisection he was able to inhibit the contractile component of tone which alone remained. This patient illustrates that the cerebral cortex normally inhibits both components of postural tone, and that injury may affect the control over the plastic component to a greater extent than that of the contractile element. This accounts for the marked improvement in control which follows sympathetic ramisection.

Consideration of these facts reveals the far-reaching influence in the interpretation of the functions of the cerebrum and cerebellum the further investigation of the central control of muscular tone is likely to exert.

[Hunter was in the midst of his experimental work when he received the invitation from the American College of Surgeons to deliver the Murphy Oration in New York. He and the team of collaborators who were inspired by his enthusiasm were preparing to investigate a host of problems—anatomical, physiological, pharmacological, and clinical—suggested by this work; and it was with the utmost reluctance that he left his laboratory last September. But he realized the value of a personal exposition of his results, and felt it his duty to accept the invitation from America. He fully realized how profound a revolution his work was destined to create in anatomy and physiology; and it is unspeakably tragic that he was not permitted to develop these investigations himself.

I refer to these matters to call attention to the fact that circumstances forced Hunter to make the two announcements of his results at times when he realized their incompleteness. One of these I have just mentioned—the call to New York just at the moment when he had got a clear vision of the host of fascinating problems, the solution of which seemed to be made feasible by his working hypotheses. The first announcement of his work on the sympathetic was made in 1923, during the visit of Sir William Macewen to the British Medical Association in Australia. The work was then still in the early experimental stage; but as the results were being applied to surgery it became necessary to explain the purpose and the principles underlying it for the guidance of those surgeons who felt it their duty to make use of a procedure that seemed to afford a certain hope of alleviating the chief troubles of the spastic paralytic patient.

Hunter had become keenly interested in the problems of the influence of the sympathetic upon visceral functions (which have not been discussed in these lectures); and if he had lived he had intended to embark upon a far-reaching inquiry as to the similarities and differences between the non-striated, cardiac, and striated muscles, and to correlate the actions of the sympathetic system on all three kinds of muscle.

In preparing these lectures for publication I have made free use of the work of Dr. T. K. Potts, published in the *Journal of Anatomy* (January, 1925), and the reports of the Murphy Oration in *Surgery, Gynecology and Obstetrics* (December, 1924); and I have to express my thanks to the editors of these journals (Sir Arthur Keith

and Dr. Franklin H. Martin) for their generosity in allowing me to quote freely and copy illustrations. The drawings illustrating these lectures were made by Mr. T. Poulton, mostly from illustrations selected by Hunter himself. Dr. John Beattie and Miss Audrey Russell made special dissections for me to check some of the anatomical points in the relations of the sympathetic rami in man and the fowl. Professor Hunter is in no sense responsible for Figures 2 and 14, which I constructed to give pictorial expression to the implications suggested in his argument.—G. E. S.]

REFERENCES.

²² *Brain*, 1922. ²³ *Ibid.*, 1921, p. 307. ²⁴ *Lancet*, 1923, p. 644. ²⁵ *Brain*, 1924, p. 178. ²⁶ *Ibid.*, 1923, p. 189. ²⁷ *Journ. Physiol.*, 1905, p. 369.

THE EARLY DIAGNOSIS AND TREATMENT OF PYLORIC STENOSIS IN INFANTS.*

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A CLEAR understanding of the nature of pyloric stenosis in infants and the recognition of the importance of early diagnosis, and so treatment, are most essential. We are quite sure that if the condition could be recognized early many lives would be saved. It is more common than is usually thought. By pyloric stenosis we mean a great hypertrophy of the circular and longitudinal muscles which control the passage of food from the stomach to the duodenum. The muscle here becomes enormously thickened and hypertrophied, so as to block the pyloric canal. The consequence of the block is that the stomach wall also increases in thickness and size as it tries to force its contents through the constricted opening into the duodenum. The food, not being able to pass through the pylorus, is forced the opposite way up the oesophagus, and we get the forcible vomiting so characteristic of the disease.

DIAGNOSIS.

Sex.—In the great majority of cases the infant is a boy, but the cause of this is quite unknown. This fact, however, is a help in diagnosis. Eight or nine out of ten cases are boys.

Place in Family.—Many cases occur in the first-born; this looks as if the mother had not been able to produce a perfectly normal infant, but one different in some way. There is, by the way, a special liability of first-born children to abnormalities.

Age.—The condition is often called "congenital hypertrophic pyloric stenosis." This term is somewhat misleading and wrong, for the infants are, for a time anyhow after birth, perfectly well, and they show no symptoms till they are a few weeks old.

Symptoms do not appear till the infant is at least two weeks old, and the commonest time for the vomiting to start is from two to four weeks after birth. The vomiting seldom starts for the first time after the age of six weeks.

Vomiting.—This is perhaps the most characteristic sign, but only when the vomiting takes on the character peculiar to the disease. The vomiting is forcible, and the food is shot out of the mouth, even also down the nose, and finishes well away from the infant, as he lies in his cot. The infant does not vomit after each feed, but usually twice or three times a day, but when it does vomit more than one feed comes up at a time. The infant who vomits after each feed does not usually have pyloric stenosis. The vomit often has much mucus mixed with the food, showing that the stomach has become inflamed.

Constipation.—As the vomiting begins the infant becomes constipated to a marked degree. The bowels are only moved by enemata, and then only a little at a time. The reason being that little food gets through the pylorus. The intestines become atrophied from disuse, and this is a bugbear after operation, when the food gets through the pylorus

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