SELECTED PAPERS
OF GORDON HOLMES

Compiled and Edited for
the Guarantors of Brain
by
C. G. PHILLIPS
Editor of Bridges
Dr Lee's Professor of Anatomy
in the University of Oxford

OXFORD UNIVERSITY PRESS
1979
V. THE CEREBELLUM

THE CROONIAN LECTURES ON THE CLINICAL SYMPTOMS OF CEREBELLAR DISEASE AND THEIR INTERPRETATION
Delivered before the Royal College of Physicians of London, 1922.

LECTURE I

My choice of subject has been determined largely by the opportunities I have had of observing the effects of local lesions of the cerebellum in both warfare and civil life. During the past three years I have been able to investigate the symptoms of 25 patients with cerebellar tumours, in each of which the localization was verified and the tumour was either wholly or partly removed by operation. The clinical material I had the opportunity of collecting during the late war was much greater, as during my service in France I was able to study nearly 70 cases of wounds of this organ. In 1917 I published an article on gunshot injuries of the cerebellum, but owing to the circumstances under which my observations were made my notes on the earlier cases were necessarily incomplete in many respects. During the later stages of the war, however, it was possible to keep a considerable number of the patients under observation for longer periods and under more favourable conditions, and since my return to England I have been able to follow up several of them and to examine other cases of gunshot wound of longer standing. These further observations, and the facts that the writings of physiologists, including Sherrington, Magnus, Piéron, Rossi, Simonelli, and Walsh, and of other clinicians, as André-Thomas and Noica, have influenced, and in certain instances modified, the interpretations of the symptoms which I then put forward, must be my excuse for returning to this subject. And as the main function of the cerebellum is the control of muscular contractions, I cannot but think that the subject is one which would have been of interest to the founder of these lectures, and the author of "de râisone motis musculorum."

Clinical studies demand in the first place an accurate recognition of the various abnormalities or disturbances of function which can be attributed directly or indirectly to a lesion or disease of the organ, or of the part of the organ, that is affected. These abnormalities form the "symptom-complex" or "syndrome" by aid of which we endeavour to diagnose and localize the pathological changes that interest us. But though the recognition of symptoms alone is often sufficient for practical purposes, a further analysis of them and their interpretations by simpler principles, invariably enables us to evaluate them more correctly and to employ them more accurately. And it is only by such an analysis of symptoms that we can utilize clinical observations to advance our knowledge of the functions and of the mode of working of the body and its parts under normal and pathological conditions. The value to physiology of conclusions obtained from a careful analysis of functional abnormalities in man is obvious in the cases of the central nervous system, as the complex evolution of the human brain distinguishes it in many respects from that of the animals usually employed in experimental research, and more particularly since the intelligent co-operation of our subjects enables us to investigate the anomalies produced by nervous lesions more intimately and more thoroughly than is often possible in animals. The study of the symptoms of cerebellar disease attempted here is devoted to these two objects: to a perfecting of our methods of diagnosis and localization, and to an inquiry into the normal functions of the cerebellum. These aims will make it necessary to deal with each symptom or a group of allied symptoms separately, and in that order which will throw most light on cerebellar physiology. The first to demand our attention, since it is one of the most important and fundamental, is disturbance of tone.

Since the experimental observations of Luciani, disturbances of muscle tone by cerebellar lesions have attracted much attention, and have also been a subject of controversy. Ferrier failed to detect any "atonia" in his animal's Babinski has stated that there was no "atonia" in most of the cases which he has examined, and even as late as 1911 André-Thomas wrote that he had never observed muscular relaxation or hypotonia, in the sense given to it by clinicians, in his patients. Myers has recently stated a similar experience.

The explanation of the many opposing statements in the literature of cerebellar disease obviously lies in the conception of what muscle tone is. The clinician usually applies the term to that slight constant tension characteristic of healthy muscles, owing to which the limbs when handled or moved passively offer a definite resistance to displacement, or require more force to move them than the hypotonic limb of the advanced tabetic or the limb in a state of flaccidity owing to injury or disease of its peripheral nerves.

To Sherrington we owe another conception of the nature of muscle tone and the part it plays normally in the motor activities of the animal.
Tone, according to him, is only postural contraction, that is, the tonic contraction of muscles owing to which a limb when brought into a new posture by active or passive movement remains in that new posture. It is excited reflexly from peripheral afferent end-organs, and mainly from those of the contracting muscles. Tone is therefore a proprioceptive reflex adapted to the maintenance of posture. This property depends on its plasticity, as the muscles fixing a limb at any joint adjust their length to that necessary for this attitude, and exert tension regardless of their length. Further, when acting statically—that is, preventing displacement of parts of the body by other forces—the muscles though in a state of tension do no mechanical work and are not subject to fatigue.

Another important function of tone is that it fuses with muscular contractions otherwise excited, reinforcing them, making them more tonic, and maintaining their contractions after cessation of the stimulus. The contractions of a toneless muscle are therefore more intermittent and clonic, and the muscle is more subject to fatigue than the normal when employed in continuous work or in the maintenance of an attitude. The importance of this conception of tone can be easily understood only when it is realized that every movement accurately adapted to its aim must not only start from a definite posture and end in a posture, but that the movement itself consists of a series of postures; and that its accuracy depends largely on the correct postural fixation of those parts of the body on which the limb, or segment of the limb, is moved.

In applying Sherrington's conceptions to clinical work we are met with the difficulty that there are few tests or "signs" by which disturbances of postural tone can be immediately recognized or measured; and that it may not be possible to distinguish it from voluntary or automatic muscular contractions in the conscious patient. All muscular contractions that maintain an attitude cannot be regarded as merely a manifestation of reflex postural tone; if a finger brought to the tip of the nose, for instance, remains there for a time, it is obvious that the arm is held in the appropriate attitude mainly by voluntary effort, and the maintenance of the erect attitude in standing is, to some extent, dependent on this too. In each of these instances plastic tone may stabilize the contraction initiated by volition, but we still lack clinical tests by which to distinguish it directly.

Method of Study

The disturbances of tone which result from a destructive lesion of the cerebellum are most easily studied in cases in which one side only of this organ is damaged. They are then limited to the homolateral musculature, and we are consequently able to contrast the states of the muscles on the normal and abnormal sides. On merely handling the affected limbs it is at once noticed that they can be displaced or moved passively more easily than the normal, and that no such resistance is encountered as that offered by the stretching of normally toned muscles. In severe affections flexion, extension, and other movements at any joint demand no more power than that necessary to bear the weight of the segments distal to this joint; it is as though each segment of the limb were tied loosely by a string to that proximal to it. To handling, the limb may suggest at once that inert flaccidity of the muscles of a person deeply under an anesthetic, or of a corpse recently dead. When the lack of tone is considerable the limb can be flung about as if it were merely a dead mass; the moving hand may strike the bed or even the patient's face or other part of his body with the full inertia of its movement. This lack of resistance to passive movement may be seen in the limb girdles too; a sharp blow on the scapula may cause it to slide forwards on the thorax in a manner never seen on the normal side. Passive displacement of any joint may be, in fact, restricted only by the articular ligaments that normally limit the range of movement.

The unsupported segments of the limb also lie more inertly than those of the normal side; if the upper arms are supported horizontally on a bar the affected one rotates inwards at the shoulder and flexes at the elbow owing to the weight of the forearm, and the hand and fingers hang more passively, and if the forearms are held vertically the wrist of the affected side may flex, owing to the weight of the hand, to a smaller angle than in

![Fig. 1](image_url)

Fig. 1.—A case of extensive injury of the left side of the cerebellum; photograph taken one week after infliction of the wound. When the forearms were held vertically the left wrist flexed under the influence of gravity much more than the right. (From Brath, 48, 601)
and it is often possible when the knee is extended to depress the patella further than on the normal side.

All these phenomena indicate that the elastic tension in normal muscles is so great that the elongation or stretching is effective on the side of the cerebellar lesion. This is the explanation, too, of the fact that the affected limbs do not frequently occupy an unnatural attitude, or attitudes which would be corrected quickly in the normal limb owing to the discomfort produced by an unusual degree of stretching of well-toned muscles. The hand may lie for long periods prone on the bed with the fingers fully straightened out or even hyperextended, and in attempting to rise into the sitting position the patient may throw his weight on the dorsum of the affected hand so that the fingers and wrist are flexed to a degree that would be naturally uncomfortable. It seems probable that owing to the lack of tension the sensory end-organs of the muscles are not stimulated by the stretching to the same extent as in normal muscles.

The lack of diminution of tone in the muscles can be also demonstrated by seizing one of the proximal segments of the limb and shaking it; then the distal segments flop or sway about inertly, like the arm of a flail, their passive displacements not being controlled or checked by the tension of those muscles stretched by each excursion. The freer and more inert swing of the affected limb is usually obvious to the eye, but it is even more unmistakable to the observer's hand, for while the oscillations of the distal segments of the normal arm or leg are arrested by the elastic tension of the muscles, the affected limb swings unchecked till further movement is prevented by the bony and ligamentous structures of its joints. The end of each movement is therefore more abrupt, and at the termination of each the joints seem to "lock" and consequently transmit a jar to the hand that shakes the limb.

The same unrestricted passive swaying of the affected limbs may be also observed when the patient's whole body is suddenly displaced or shaken, and in rapid voluntary movements a similar inert swinging may occur at joints not directly concerned in the action; in flexing and extending the elbow alternately, the affected wrist may be flexed and extended by the inertia of the movement, so that the hand is flung against the shoulder or to the bed, and the affected elbow may be jerked from its support by the momentum of the flexing forearm. These observations reveal a deficiency of the normal tension in muscles that should fix the joints in appropriate postures.

Deficient Muscular Elasticity

The elasticity of the muscles is often deficient too. If the pronated forearm is grasped firmly and suddenly jerked, as if the observer were trying to crack a whip, the hand of the normal side after flexing swings back in extension owing to the elastic tension of its extensors, but that of the affected limb sways passively, or recoils only when arrested by the inelastic ligaments of the wrist-joint. This lack of elasticity can be easily demonstrated in the lower limbs too; if the observer, placing his hand under the patient's thigh while he lies prone, jerks this suddenly and unexpectedly upwards, the normal leg is usually carried up since the knee-joint remains extended or extends again after a preliminary fixation, but the heel of the affected limb remains on the bed, or immediately falls inwardly, owing to deficiency of tone in the quadriceps extensor muscle. Similarly, on suddenly dorsiflexing the wrist the fingers may not flex, or flex to a less extent than those of the normal side, owing to lack of tension in their long flexors. Frequently, too, the heel can be approximated more closely to the buttock by passively flexing the hip and knee; more than one patient noticed this and commented on the greater ease of such a movement when actively performed, and the less strain experienced when it was passively done.

It has been stated that cerebellar hypotonia does not lead to an increase in the range of passive movement; in my experience that is not wholly true. In joints in which movement is normally arrested by ligaments or by apposition of bony surfaces, no increase of range can be found, since cerebellar disease does not lead to relaxation of the ligaments, but in those where the tension of the muscles normally checks movement the possible range may be definitely greater on the affected side.

These various tests were carried out with the muscles in as complete a state of voluntary relaxation as it was possible to obtain, but the same disturbances can be demonstrated, in cases with moderate or severe hypotonia at least, even when the patient attempts to control passive displacements of his limbs. The extended arm or leg oscillates more widely than normal as the result of a tap, even when the patient is requested to prevent the limb giving way to the blow; and when the arm is seized above the elbow and shaken its distal segments sway too widely even though the subject is asked to hold them steadily. It appears, therefore, that under these conditions voluntary muscular contractions cannot supplement efficiently the loss of postural tone, and that postural tone is a necessary adjuvant to those muscular contractions by which we voluntarily maintain our limbs and body in determinate attitudes. That this is so is also shown by the fact that even small lesions of one-half of the cerebellum may affect the maintenance of voluntary posture. When the two arms are outstretched horizontally the affected one tends to fall away under the influence of gravity and has to be repeatedly replaced by voluntary effort. In cases of severer damage the affected limbs are often unable to maintain any attitude if unsupported; they sway about in an inert purposeless manner, and in severer cases may fall unexpectedly.
as though the muscles that held them up had suddenly relaxed. This is particularly liable to occur if equal weights are suddenly dropped on the two limbs; the affected one “gives” under the increased load to a much greater extent than the normal.

Our observations consequently show that a lesion of the cerebellum diminishes the muscle tone which reflexly fixes the postures of the body and its parts, that it reduces the elasticity of the muscles, and that it disturbs the tonic element in the voluntary contractions of muscles directed to the maintenance of posture.

**Distribution of Hypotonia**

The next question that arises is the distribution of this disturbance of tone. When a lesion involves a large part of one-half of the cerebellum, there can be no doubt that the hypotonia is rigidly limited to the limbs of the same side, and it probably affects all the muscles of this side. In the limbs it is general, though its manifestations are often more pronounced at the proximal than at the distal joints. It is less easily recognized in the muscles of the trunk, but the abnormal attitudes that this assumes, and the more easy displacement of the patient or of his limb girdles in those directions which the tension of the homolateral trunk muscles resist, indicate that it extends to these too. It is rare to find any definitely abnormal asymmetry of the face in unilateral lesions.

Whether small local lesions of the cerebellum produce disturbances of tone in certain regions of the body, or in certain groups of muscles only, is important, especially in reference to the theory of cortical localization. I have had the opportunity of investigating a large number of cases of gunshot wounds with such circumscribed lesions, in both their earlier and later stages, and my experience has been that when small injuries disturb the tone the disturbance is never limited to one limb, or to a segment of a limb, or to one or more groups of muscles. If the arm or leg presents hypotonic symptoms too, though they may not be so easily demonstrated, and the relative affection of different segments of the limb follows that which occurs in more extensive lesions. I have been unable to detect that anisotonia, or hypotonia of some muscles with relative increase of tone in their antagonists, which Rothmann and Thomas and Duran have described in animals after removal of small areas of the cerebellar cortex, to which Thomas has called attention in man. It is a matter on which a definite opinion cannot be passed without careful deliberation, since it is by more or less indirect means only that such changes as Thomas has described can be detected, but even by employing Thomas’s own methods I have been unable to confirm his conclusions. The significance of this point of view of two well-recognized clinical phenomena, spontaneous deviation in Bärn’s pointing tests,

and excess of movement in certain directions will be considered later.

**Occurrence of Hypotonia**

Though some degree of hypotonia can be detected in probably all cases of local injury which present obvious symptoms, it must be admitted that it is not demonstrable in all affections of the cerebellum. I have seen a few warfare injuries in which no trace of hypotonia could be detected, but the number was small. That the cerebellum was injured in these cases was proved either by operative exploration or by radiographic examination of the skull. On the other hand, deficiency of tone, as far as we can judge from our methods of investigating it, is certainly not constant in the primary cerebellar atrophies; Babinski and others have denied its occurrence in such cases. This may be due to the gradual compensation or recovery that occurs pari passu with the slow progress of the disease, or to the coincident involvement of other parts of the nervous system that may have an influence on the tonic mechanisms, for in the cerebellar atrophies the disease is rarely limited to the cerebellum.

Purely cortical lesions of the cerebellum can undoubtedly disturb muscle tone; this is made certain by its occurrence in many of my cases with superficial lesions, in all of which it is impossible to assume that the central nuclei were also damaged. This experience conforms to many experimental observations in which hypotonia was observed after destruction of the cortex, though Rothmann failed to detect it after decortication of one lateral lobe, and therefore concluded that it occurs only when the central nuclei are damaged. That atonia is then a more prominent symptom there can be no doubt; the deeper and larger the lesion the greater and more permanent is the deficiency of tone. But this fact cannot be regarded as evidence that it is chiefly the central nuclei that contain the cerebellar tonic mechanisms, since all the efferent cerebellar paths, and consequently those through which alone the cerebellum can influence tone, pass through these nuclei.

Lesions of both the lateral lobes and vermis have the same effect on tone, but when the latter is involved the disturbances are more prominent in the trunk than in the limbs. As I have had no case in which the disease was limited to the vermis I am unable to say if it is merely on the musculature of the trunk that this excites its influence.

**Appearance of Atonia**

A diminution of tone appears almost immediately, or at least very rapidly, after the occurrence of an acute destructive lesion of the cerebellum. I have observed it within a few hours of a gunshot wound and of a softening, and have seen a great increase of hypotonicity immediately after recovery from the anaesthetic in a patient in whom it was necessary to excise a portion of the cerebellum in the removal of a tumour. The
flaccidity of the muscles, as tested by clinical methods, increases, however, during the first week or ten days after the onset of the lesion, the limbs becoming more flail-like on handling and the elasticity of their muscles diminishing, but the change is rarely great.

The persistence of hypotonia depends largely on the site and severity of the lesion. When this is small and superficial the muscles may regain an apparently normal condition within a few weeks as the other symptoms pass off, but when the destruction is more extensive the loss of tone may persist as a permanent phenomenon; I have observed it two and three years after apparently small gunshot wounds, and several years after the removal of cerebellar tumours.

Lesions involving the inferior cerebellar peduncles and the efferent cerebellar connections passing through Deiter's nucleus, certainly produce a loss of tone in man, and it is probable that injuries of the middle peduncles may have a similar effect. There are, however, many facts that suggest that destruction of the superior cerebellar peduncles may have a different influence on muscle tone. The fact that the transaction of the brain-stem which produces decerebrate rigidity, a state which is the paradigm of postural tone, divides either them or their mesencephalic connections, suggests that the cerebellar impulses which pass by them may be inhibitory of tone rather than tone-exciting. The stiff hypertonic state of the musculature of one side of the body when one superior peduncle is destroyed in man, or when its termination in the red nucleus is injured by such lesions as produce Benedict's syndrome, point to the same conclusion. Unfortunately, Ferrier's classical experiments on the superior peduncles throw no light on this matter, and, as far as I am aware, the condition of tone has not been carefully investigated in other such experiments. In two of my cases in which one superior peduncle was damaged, in one by a tumour and in the other by a softening, there was hypotonicity rather than atonicity of the homolateral musculature.

A series of other symptoms of cerebellar disease which can be attributed directly to disturbances of tone, may be most conveniently considered here before we deal with the irregularities of active movement.

ATTITUDE

The abnormal attitudes assumed by animals after experimentally produced lesions of the cerebellum have attracted much attention, but these are rarely prominent symptoms in man, and when they occur, it is often impossible to exclude the influence of concomitant affections of the vestibular nerves or of their central connections. The importance of the labyrinths in determining attitudes of the body and of its parts is well known, and has acquired a special significance since the investigations of Magnus and de Kleijn and their pupils have shown that a number of reflex attitudes can be excited from them, and the importance of these reflexes in the adjustment of the body in equilibrium and in motion. These workers have, however, demonstrated that these tonic labyrinthine reflexes persist unchanged after removal of the cerebellum, which cannot therefore be regarded as their reflex centre. There can be, however, no doubt that pure cerebellar lesions may produce abnormal attitudes which can be most easily explained by disturbances in the tone of those muscles which act normally together to preserve natural postures.

We have already seen that owing to the tonelessness of their muscles the limbs may assume strange attitudes when lying passive, and that even when held in position by volitional effort certain of their segments may droop under the influence of gravity or other forces. These are usually the only postural abnormalities they present. The only two frequent exceptions are a tendency for the outstretched arm to swing in that direction in which it deviates in the pointing test, and abduction and outward rotation of the affected leg in standing. As the latter is usually observed only when the patient is erect on his feet, it may be attributed to an attempt to compensate his instability, while the spontaneous deviation of the arm can scarcely be regarded as a disturbance of an attitude, since it occurs under certain conditions only, and has not a constant influence on the posture of the limb. Occasionally there is some inward rotation of the arm with pronation of the forearm, or outward rotation at the shoulder, but these attitudes frequently vary from day to day and may alternate. It is, however, common to see the shoulder of the affected side depressed and often lying in front of the opposite one; this attitude is certainly due to passive displacement by the weight of the limb, the hypotonic muscles failing to counteract the influence of gravity. It sometimes happens that the affected shoulder is the higher, but this is common only when the patient is sitting, or when he makes a voluntary attempt to overcome the tendency to incline to the affected side.

Special attitudes of the trunk are more common. They are rarely observed when the patient is lying supine and fully supported, but even then the vertebral column is sometimes concave to the affected side, especially in recent and severe injuries. On standing this curvature of the spine is more pronounced, and it is often associated with a slight rotation, the homolateral shoulder being carried forwards. These attitudes can be explained by the unequal distribution of tone in the muscles of the two sides of the body. The effect of hypotonia of the trunk muscles is most obvious in cases of severe bilateral injury. One man with such a lesion was noted to "sit into any attitude into which he was put"; as he sat on a chair unsupported, he usually inclined forward and developed an extraordinary degree of antero-posterior curvature, or he might incline to either side until arrested by some support.

The typical position of the head in cerebellar disease has given rise to some controversy. My cases of gunshot injury can scarcely be used as an
argument in favour of any of the opinions expressed; for, in the first place, the damage to the muscles inserted into the base of the skull, either by the original wound or by the subsequent operations, must have exerted some influence on it; and in the second, each patient naturally kept his head in the position in which the damaged tissues were most relaxed, and the discomfort of the wound or injury least troublesome. There is, however, a general tendency in unilateral cerebellar injuries for the head to flex to the homolateral side and rotate so that the chin is carried towards the opposite shoulder. It is, however, doubtful if this is a true cerebellar attitude; Horley has attributed it to vestibular disturbances, and it is noteworthy that it was pronounced in six cases of tumour in the cerebello-pontine angle, most of which probably originated from, and all of which injured, the vestibular nerve.

From these observations the conclusion is justified that abnormal attitudes assumed by the trunk or limbs are due either to the passive displacement of hypotonic parts by external forces, or to an unequal distribution of tone such as occurs in the trunk muscles as a result of unilateral cerebellar lesions.

**ASSOCIATED MOVEMENTS**

The excessive associated movements that frequently develop in the homolateral limbs on strong muscular effort of either an opposite limb or the other limb of the same side, can be explained by defective postural fixation of the affected parts. In certain cases, particularly in those with considerable disturbance of tone, these associated movements are very vigorous and wild. When, for instance, one patient with an extensive wound of the left side of the cerebellum grasped a dynamometer strongly with his right hand, his left arm became rigid, all the muscles of the arm and forearm stood out in firm contraction, the thumb straightened out, the fingers flexed at the proximal and extended at their inter-phalangeal joints, the elbow extended, and the arm was abducted and rotated outwards at the shoulder. At the same time the left knee and thigh flexed so that the foot was raised and drawn under the chair in which he was sitting. There seems to be no constancy in the type of these associated movements; they vary from patient to patient and often in the same patient from day to day. They are more intermittent, more clone, and more irregular than those which occur in spastic conditions, and usually require more effort to evoke them. Further, they subside more abruptly when the effort that has excited them ceases.

Normal limbs have a tendency to develop such associated movements, distinguished by their apparent purposelessness and by their slow tonic character, on strong intervation of the muscles of some other limb, but their excursions are restricted by the tone that fixes them in their natural postures. When this postural tone is deficient the limb or any of its parts is displaced excessively by the shortening of those muscles that contract, or rather, by predominance of contractions in certain muscles or groups of muscles. The excess of associated movements in cerebellar disease is consequently another manifestation of deficient tone.

**THE REFLEXES**

It is generally stated that the reflexes are unaffected by lesions of the cerebellum, but a careful study usually reveals certain changes in the tendon-jerks that are more or less characteristic. I have detected no abnormalities in the cutaneous or superficial reflexes, but after acute and extensive lesions there may be a less brisk withdrawal of the homolateral foot on stimulation of the sole, or the withdrawal movement may be more irregular and unstrained than in the normal limb; the plantar reflexes, however, are invariably of the normal type, and the abdominal and cremasteric remain unchanged.

The alterations in the tendon-reflexes can be best studied in the knee-jerk. For some time after the occurrence of an acute unilateral lesion the homolateral knee-jerk is often depressed or difficult to elicit, or a response may be obtained from one or two only of a series of taps on the patellar tendon, though the range of the jerk when it occurs is usually large and ample. It was generally in patients with large infected gunshot wounds that the jerks were absent or much depressed, and in such cases it is impossible to ascribe the change to destruction of the cerebellum only. I have, however, found the jerks also diminished after extensive operation wounds and within a few days of the occurrence of a vascular lesion of the cerebellum, but as this diminution persists for a short time only it may be regarded as an effect of "shock."

When the patellar tendon is tapped at the patient sits on a high stool so that his legs can swing freely, the response of the affected limb lacks the decisive, forcible character of the normal jerk, though its range may be as large or even greater. But the most characteristic change is that while the normal leg (fig. 2a) comes quickly to rest on falling to its original position, the affected limb (fig. 2b) continues to swing inertly like a pendulum, oscillating to and fro around its position of rest. The jerk has, therefore, the pendular character described by Thomas. A similar pendular jerk can be obtained in the upper limb if, while the elbow is flexed to a right angle and the forearm hangs vertically, either the tendon of the triceps muscle or the lower end of the radius is tapped; then the forearm continues to swing to and fro after the reflex contraction has ceased.

It will be remembered that Sherrington has shown that the knee-jerk, and the same must hold for the other tendon-jerks, consists of a simple twitch contraction to which there is added a plastic shortening reaction persisting after the twitch has ceased. The initial twitch contraction is
extensor and flexor muscles. When, however, the extensors of the knee lack their plastic tone the leg, after being extended by the twitch contraction, drops unimpeded and swings to and fro. Sherrington has described this type of knee-jerk in states where the reflex excitability of postural contraction is low, "the relaxation fall of the curve is full, drops to the zero abscissa freely and is usually followed by some pendular oscillations."

That this is the explanation of the pendular jerk in cerebellar disease can be easily proved by myogram tracings of the contraction of the quadriceps muscle in the jerk. The normal curve (fig. 3a) shows a definite plateau, or a second rise corresponding to the appearance of after-shortening, which is absent in the cerebellar patient where the tracing (fig. 3b) is identical with that of a simple twitch. Tracings of the reflex contractions of other muscles excited in the same manner as the gastrocnemius, biceps, triceps, and hamstrings, show identical abnormalities.

---

b.—Tracings of two knee-jerks of a man with a right-sided cerebellar lesion of eight years' duration. Tracing is less reduced than in a. Read from right to left. (Brain, 40, 461, Fig. 12)

generally unaffected by lesions limited to the cerebellum; to it is due brisk extension of the leg. Under normal conditions the leg in falling again to the vertical under the influence of gravity is restrained by the after-shortening of the quadriceps, and its fall is consequently less rapid and abrupt than that of a pendulum or an inert body, and its momentum on reaching the vertical is therefore not sufficient to make it swing beyond it. Further, oscillations are also checked by the plastic tone of both

---

Fig. 2. a.—Tracings of three knee-jerks of a normal man taken on a slowly revolving drum. Read from right to left. The slight secondary swing seen in these tracings does not occur in many normal men. (Brain, 40, 461, Fig. 11)

---

Fig. 3. a.—Tracing of the knee-jerk of a normal man on a rapidly revolving drum. Read from right to left. Time by tuning fork of 128 vibrations per second. (Brain, 40, 461, Fig. 15A)

b.—Tracing of the right knee-jerk of a man who received a severe injury to the right side of the cerebellum eight years previously. Time and direction as in a. (Brain, 40, 461, Fig. 15)
It must be, however, pointed out that the pendular jerk is not pathognomonic of cerebellar disease; it is occasionally found in persons in apparently normal health, and it is common in hypotonic states, as in early tabs when these reflexes are not abolished. There is frequently another abnormal feature in the knee-jerk of the affected side. If the observer places a hand across the hamstring tendons behind the normal knee he can generally feel a brisk tightening of them at a short interval of time after the patellar tendon has been struck, provided this excites a jerk of sufficient range. Palpation of the flexor muscles shows that this tightening of the tendons is due to their active contraction, and not merely to passive stretching by the extension of the knee. On the affected side no such contraction of the hamstrings may be felt, no matter in what position the knee is placed, or how great the amplitude of the jerk. The result of this is that if the two limbs are fully supported on the bed with the hips and knees semi-flexed and the thighs rotated outwards so that the heels are at the same level, the foot of the unaffected side, after moving abruptly towards the bottom of the bed by each jerk elicited by a tap on the patellar tendon, tends to return immediately to its original position, while on the affected side the foot generally remains in the position into which it has been moved by the contraction of the quadriceps, and the knee can be consequently fully extended by a series of taps.

We cannot, however, assume that the failure of the hamstrings to contract in cerebellar disease is due to their inexcitability, for this can be shown to exist by other means, as by a sudden blow on their tendons. The probable explanation is that in the absence of postural tone the tension within the muscles is insufficient to permit their end-organs being adequately stimulated by that degree of stretching which normally evokes a reflex contraction. This explanation would also account for the difficulty sometimes experienced in states of marked cerebellar atonia, in eliciting the knee-jerk and other reflexes by a gentle tap.

**Static Tremor**

As tremor in portions of the body that are not fully supported can be attributed to disturbance of muscle tone it can be most conveniently considered here. But all tremors occurring in cerebellar disease are not of the same nature.

If a patient with a unilateral lesion holds both arms extended horizontally in front of him, or raises his lower extremities from the couch on which he lies, the limb of the affected side may at first maintain its attitude as steadily as its fellow. Indeed, for a time it may appear even more steady, since the fine vibratory tremor that is commonly observable in the normal arm when it is extended rigidly is often absent in the affected limb. But as the muscles holding the limb up begin to tire an irregular tremor develops. Its oscillations are mostly in the line of gravity, and can be seen on careful inspection to be due to a failure in the tonic contractions of the muscles that maintain the attitude, with the result that the limb falls with gravity and is replaced by voluntary efforts. This form of tremor may occur at any joint, but it is more common at the proximal. It is frequently seen in the head too when the patient is in such a position that muscular effort is necessary to fix it. In cases with bilateral lesions of the cerebellum a similar tremor of the whole body may be seen when the patient stands erect.

In all these instances the unsteadiness of the trunk or limbs is obviously due to failure of those muscles that should fix the attitude to remain in a state of steady tonic contraction.

Another type of tremor, characterized by more regular oscillations of a limb or some of its segments, occurs when the patient attempts to maintain the limb accurately in certain positions, or in postures necessary for the performance of some act. On holding his finger near to, but not in contact with, the tip of his nose or some other point, or his heel above, but not actually touching, his opposite knee, a tremor may develop that is sometimes so regular in rate and range as to resemble that of paralysis agitans. This form of tremor is frequently seen too when the patient employs tools that require delicacy and precision; on attempting to write the point of the pen may oscillate from side to side, or swing around in irregular circles before it reaches the paper; and on beginning to sharpen a pencil the hand that holds the knife may swing to and fro until the blade touches the pencil, then it ceases, or is replaced by the more irregular jerks that disturb voluntary movement.

It is usually only in attitudes determined by the tonic contractions of opposing groups of muscles that this regular form of tremor develops; in other words, only when both the agonists and their opponents are both contracted. A limb can be maintained in any position by the action of the anti-gravity muscles only, but if it is required that the fingers shall be held steadily, 1 mm, for example, from the tip of the nose, other muscles, or perhaps all those of the limb, contract tonically in order to assure the accuracy of the fixation. This can be ascertained by handling or palpating the limb, particularly if a special effort is necessary owing to it being unsteady. But this tonic contraction of the opposing muscles is no longer required when the finger comes in contact with the nose, or the pen with the paper, or the blade of the knife with the pencil; then the action of the prime movers against the resistance of the object touched is sufficient, and then this regular tremor usually ceases.

The oscillatory tremor that occurs in these circumstances can be attributed to alternate contractions of certain groups of muscles and their antagonists. This is probably due to the loss of postural tone, the failure of fixation by muscles that move the joint in one direction allowing their contracting antagonists to displace it in the opposite sense, and as these
do not immediately adapt their length to the fixation of the limb in its new posture, it is again moved by the opposing muscles towards and beyond its original position. Consequently, the part oscillates around the position it should occupy.

A similar regular tremor occurs more rarely in portions of the body which are not fully supported. The affected arm of one of my patients always developed a tremor, regular in rate and amplitude and of about four oscillations a second, when it fell over the arm of a chair on which he sat; and in another an identical tremor was seen in the leg when it hung 

over the edge of the bed. It is more common in the head than in the limbs. Its resemblance to the tremor of paralysis agitans is unmistakable; it is apparently similar to that which Ferrier and Turner observed after section of the superior peduncles, and which is frequently associated with mid-

brain lesions that involve these peduncles. In my own two cases of gun-

shot wounds in which it was a prominent feature, the course or position of the missiles made it probable that the superior peduncles were injured. It occurred also in a man lately under my observation in whom an en-
tensive tumour was found by the operation to extend towards the anterior margin of the cerebellum, where it probably involved one superior peduncle.

THE REBOUND PHENOMENON

Another symptom frequently found in cerebellar disease, which shows a further role probably played by muscle tone, is the rebound phenomenon. If the resistance that effectively prevents a movement of a normal limb in response to a strong voluntary effort be suddenly released, the limb, after moving a short distance in the desired direction, is arrested abruptly by the action of the antagonist muscles, or it may even jerk back or rebound. This sudden arrest fails frequently in cerebellar disease. It can be demonstrated at most joints of the limbs, but is most conveniently examined at the elbow. The arm is preferably supported and the patient is asked to pull his hand towards his mouth against the resistance offered by the observer who grasps his wrist; when the grasp is suddenly relaxed the hand of the affected side swings violently towards his face or shoulder, and if the limb be very hypotonic, the elbow may be at the same time jerked off its support with the result that the arm may be flung above his head. Similarly, if the string by which he attempts to raise a weight is unexpectedly cut, the arm flies upwards, unchecked by the antagonists. There is often a corresponding inability to arrest passive movements quickly; the affected hand, for example, can be often thrown violently against the patient's face, as though the limb were paralysed or an inert body, in a manner which is never possible on the normal side.

Several factors must be considered in attempting to explain the failure of the antagonists to check active and passive movements of the limb promptly. If we assume that the arrest is brought about by their voluntary contraction, it might result from either defect or delay in this. But it appears doubtful if it is effected only by a voluntary contraction, and even though this does take a part it may be that the deficiency of tone in the opposing muscles prevents them from withstanding the sudden strain to which they are subjected by the momentum of the moving limb. It is, however, more probable that a contraction of the antagonists, sufficient not merely to arrest the limb, but also adapted to fix it in its new attitude, is normally assured by some subcortical reflex mechanism in which the cerebellum, owing to its importance in the postural functions, takes an important part. The contraction of the flexors of the knee normally excited by a brisk knee-jerk, which may be absent in cerebellar disease, is an allied phenomenon, and there can be no doubt that it is produced reflexly.

LECTURE II

In the first lecture I dealt with the disturbances of muscle tone produced by lesions of the cerebellum, and certain symptoms that can be attributed directly to them; today we will consider the associated irregularities of movement, beginning with a study of the simplest possible acts.

DISTURBANCES OF MUSCULAR CONTRACTIONS

Various disturbances can usually be detected in the simplest muscular contractions in the affected limbs, but as it is not possible to study the functions of a single or isolated muscle in clinic work, we must be content to investigate them in simple movements employing as few muscles as possible. The abnormalities usually persist as long as signs of cere-

bellar deficiency remain, but like the alterations of tone they are always more prominent soon after the onset of an acute destructive lesion.

Ataxia

There has been a good deal of discussion as to whether cerebellar lesions ever produce a diminution in the force of muscular contractions, in the sense that the strength of the limbs is diminished. Luciani included among the effects of ablation of the cerebellum ataxia, meaning that there is a deficiency in the energy of voluntary, automatic, and reflex actions, but other physiologists, and many clinicians, have denied that cerebellar disease has any influence on muscular power. In the earlier stages in a severe unilateral lesion it is, however, easy to ascertain that the power of the limbs of the same side is diminished. Many patients complain spontaneously of this feebleness, and in the more intelligent it is frequently easy to ascertain that there is subjectively a definite weakness, and not merely a uselessness of the limb due to its irregularity in movement. This subjective paresis is usually transient and disappears as the acute symptoms subside, but it occasionally persists for many months. It is
often associated with a reluctance on the part of the patient to move the limb, and particularly the arm; he may not use it to arrange the bed clothes, to rub his eye or his face, or even in gesture when he is conversing. This might result from the lack of control of voluntary movements of which he is conscious, but I have repeatedly observed it in semi-conscious subjects, and in a few patients struggling while going under an anesthetic. The affected arm or leg may be also withdrawn less promptly than the normal from a prick or other noxious stimulus, even though careful examination reveals no subjective change in, or alteration in the threshold of, any form of cutaneous sensibility. Russell and others have observed a similar slowness in the reaction of the affected limbs to painful stimuli in animals after extirpation of one side of the cerebellum, but their interpretation of it as an effect of loss of sensation is certainly incorrect.

The investigation of muscular power by a dynamometer presents considerable difficulties even when the simplest movements only are tested. Those disturbances which are usually included under “cerebellar ataxy” naturally reduce the motor efficiency of the limb, especially in actions necessitating the cooperation of several groups of muscles, and failure to grasp the ordinary dynamometer as efficiently as in the normal hand may influence the result. But when adequate precautions are taken, and the tests are so arranged that only the muscles which act together on one joint are concerned, the readings are frequently lower than on the normal side. This paresis is always more obvious in the upper than in the lower limb, and it is usually greater in the proximal than in the distal muscles. In acute lesions strength may be reduced to half that of the normal limb, but though asthenia may persist for months it diminishes gradually or becomes less apparent. The following figures represent the averages of several measurements obtained from two patients with gunshot wounds of one side of the cerebellum. In one the measurements were made nearly ten weeks after the wound was inflicted (Holmes, 1917).

<table>
<thead>
<tr>
<th>Case 1 (thirty days)</th>
<th>Case 2 (sixty-eight days)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Unaffected side</strong></td>
<td><strong>Affected side</strong></td>
</tr>
<tr>
<td>Grasp</td>
<td>12/4</td>
</tr>
<tr>
<td>Flexion of Elbow</td>
<td>26/2</td>
</tr>
<tr>
<td>Extension of Elbow</td>
<td>11/8</td>
</tr>
<tr>
<td>Supination</td>
<td>10</td>
</tr>
<tr>
<td>Pronation</td>
<td>20</td>
</tr>
<tr>
<td>Depression of Arm</td>
<td>10</td>
</tr>
<tr>
<td>Flexion of Ankle</td>
<td>17</td>
</tr>
<tr>
<td>Extension of Ankle</td>
<td>15</td>
</tr>
<tr>
<td>Depression of Leg</td>
<td>15</td>
</tr>
</tbody>
</table>

This feebleness is often recognized by patients; many complain that the arm particularly “feels weak and heavy.” “I have to put more effort into it to move it,” “I can’t put force into it,” “I can’t lift the same weight with it.” I have never observed any asymmetry of the face in voluntary or expressional movement, when involvement of the facial nerve or its nucleus could be excluded; it is scarcely possible to say if the muscles of the trunk are affected owing to the difficulty in testing their strength accurately. Muscular asthenia is certainly not present in all cases of cerebellar disease. I have frequently failed to detect any difference in the gross strength of the homologous limbs in cases of unilateral gunshot injury, and frequently in patients with slowly growing tumours. It seems to be most prominent when extensive and deep lesions exist which probably involve the nuclei. Although it is immediately after the onset of acute lesions that this asthenia is usually found, my notes on several warfare injuries indicate that, like atonia, it may increase during the first week or two after the infliction of the wound. Another striking feature in many of the severer injuries was the greater effort that strong movements of the affected limbs required. This was frequently evident to the most casual observer, and many patients speak of it spontaneously; one man, for instance, stated that not only was his right arm weaker than his left, but that it was more difficult for him to exert power with it, and that every vigorous movement demanded more effort from him and was harder to accomplish.

**Fatigability**

The affected limbs also tire more quickly than the normal. Many patients complain of this when an examination is prolonged, or when they are engaged in any task that demands the continued exertion of power. Even when the unloaded arms are held horizontally the affected one begins to droop more quickly, and may even drop. One man, seven months after a right-sided gunshot wound, allowed his right arm to fall to his knee after holding it outstretched for 60 seconds, explaining that it was so tired he could no longer keep it up. This exhaustibility becomes more prominent when the patient is asked to perform muscular work against resistance; in holding up equal weights placed in the unsupported hands, or in pulling against a graded spring, the affected limbs give out more rapidly. This can also be seen in ergogram tracings, but the most striking feature of these is not a gradual fall in the range of the movement, but a sudden cessation of the effort, the patient complaining that the limb is so tired he can no longer use it.

**Astaesthesia**

Another abnormality in the muscular contractions of the affected limbs is their jerky, intermittent, or clonic character. This may be seen in the simplest possible movements, as in flexion of the index finger or of the forearm at the elbow (fig. 4). It occurs even when the more proximal segments are firmly supported, and is more obvious in slow than in rapid
movements. Flexion of the forearm with the elbow resting on a table may, for example, consist of a series of irregular jerks and pauses rather than a uniform continuous movement, and if during it a hand is placed on the biceps this can be felt to contract intermittently. The clonic broken character of the movement must be therefore attributed to irregularity in the shortening of the agonists, and not to other factors, as spasmodic intervention of the antagonists. That this is so is proved by the fact that it is more irregular when the muscles act against resistance, as then the patient attempts to pull against a spring using one group of muscles only; while the normal arm draws the spring out at a fairly uniform rate and maintains it at the tension that is desired, the affected one extends it by a series of jerks and is unable to keep it steadily extended at any tension (fig. 5). The intermittent, clonic form of the contractions can also often be felt when the patient grasps the observer's hand; instead of exerting a steady pressure his grip is discontinuous and irregular.

To this discontinuity in the contractions of muscles—which he attributed to imperfect fusion of single twitch contractions—Lanciani gave the title astasia. Its effects are more obvious when several segments of the limb are employed simultaneously in an act. Not infrequently sudden relaxations of muscular contractions occur in both opposed and unopposed movements; they are most common when the muscles tire, but even light objects may fall from the hand, or the leg or the arm supporting the patient may suddenly give way.

**Interpretation**

It will be most convenient to consider here the cause of these three phenomena: the astenia, the fatigability, and the clonic character of the muscular contractions of the affected limbs. As the latter will throw light on the other two we will discuss it first.

A voluntary contraction is a tetanus due to the summation of individual twitch contractions, and though such a tetanus can by the progressive shortening of the muscles produce a movement, any failure in the fusion of its component twitches must produce irregularity of it. It has already been pointed out that, according to Sherrington, postural tone fuses with muscular contractions excited otherwise, making them more continuous and sustaining them. Lack of tone alone must consequently lead to the clonic and discontinuous character of movements met with in cerebellar disease. The astenia may also be directly due to this, for if the successive twitch contractions do not fuse, each twitch shortening the muscle must start at a lower level than that reached by its immediate predecessors, and the power exerted by the whole contraction must consequently be less than that of the corresponding normal muscle, in which, owing to the fusion of the successive twitches, each twitch can start at a higher level and reach a higher level. And as some or all of the twitch contractions of a muscle excited by a voluntary impulse start at a lower level and have consequently more mechanical work to do in shortening the muscle, continued movements are more likely to produce fatigue in the affected than in the normal limbs. Further, postural tone is indefatigable, while a muscle is easily exhausted by a series of such twitch
contractions as produce a tetanus; the patient is therefore liable to tire when in maintaining an attitude he has to supplement or replace the indefatigable tone by voluntary effort.

**Delay in Contraction and Relaxation**

A third disturbance of muscular function to which relatively little attention has been paid by either the physiologist or clinician is a slowness in contraction and relaxation as compared with the normal side. If a patient with a unilateral cerebellar lesion be asked to grasp with his two hands an object equidistant from them, or to bring his two forefingers to his nose or to his ears, the homolateral limb usually arrives at its aim later than the normal. There can be no doubt that in such tests the so-called ataxia is partly responsible for the delay, but if simpler movements, especially those requiring only the action of a muscle or a simple group of muscles, be tested, a similar delay as compared with the normal limb may be often detected. On asking a patient with a severe right-sided lesion to grasp the observer's two hands simultaneously with his own, or to flex his elbows on a given signal, the delay or relative slowness of the right limb may be detected. When such simultaneous acts are more carefully examined, especially by graphic methods, it is found that the contraction starts later and that power is developed more slowly. There is frequently a similar delay in commencing and in effecting relaxation of the contractions, but the time lost is rarely so great. It is usually most obvious after strong voluntary actions, which may therefore persist longer than is necessary. The slowness in muscular contractions and in the initiation of movement may be seen in the ordinary spontaneous activities of the patient; it is, in fact, usually more prominent when he is unaware of its existence than when his attention is directed to it by specific tests. Several of my patients were unable to catch a coin dropped on the affected hand, since they failed to close their fingers quickly enough on it when it touched the palm, as they invariably did on the normal side. Often, too, they cannot jerk up a coin or other object from the palm, since the hand, not being arrested abruptly enough, follows the object. The slowness in relaxation also occasionally causes inconvenience to the patient. One man, for instance, complained that as his fingers continued to clench a cup or any object he wished to place on a table he was liable to knock it over or raise it again on withdrawing his hand.

I am not at present able to suggest a satisfactory physiological explanation of this delay in initiating contractions and relaxations, or to determine if it bears any relation to disturbances of tone; experiments have, in fact, shown that the latency of the tonic muscle is not altered, and that it relaxes on cessation of the stimulus more abruptly than the normal. The slowness in getting up strength in opposed movements may be, however, due to the defective summation of the individual twitch contractions, owing to which each successive twitch starts at a lower level and reaches a less height.

**Disorders of Movement**

Ataxia, inco-ordination, and asynergia are the terms generally applied to those irregularities of movement occurring in cerebellar disease, but the exact connotation of all these terms has become so vague and indefinite that it is inadvisable to employ them. Probably all forms of movement, automatic and reflex as well as voluntary, are disturbed, but as it is in voluntary movement that the disorders are most obvious and most easily studied, they will be dealt with first. It is obvious that a careful analysis of these disorders must be made if we wish to understand their nature and their significance. But the investigation of movements presents great difficulties, since they are usually so rapid and complex that it is impossible to observe to what the errors in rate, range, or direction are due. Tracings obtained on a plain surface, as on a kymograph drum, can help us considerably, but unfortunately they represent errors in two planes of space only; a more serious objection is that the moving limb must be either supported on the surface that records the movement, or complex apparatus which may influence it are necessary for its transference to this surface.

**Light-line registration.**—A much easier, and in many ways an ideal, method is the light-line registration developed by Gilbreth. It is only necessary to fix a small electric bulb or a series of bulbs to the moving part, with the patient placed in such a position that the movements to be recorded lie more or less parallel to the plate of a camera, which is exposed in a darkened room. By employing a stereoscopic camera errors in all the three planes of space can be permanently recorded, and by interrupting at regular intervals the current that lights the bulbs the rate of every stage of the movement can be accurately measured. I have employed an electrically driven tuning-fork of 24 vibrations per second for this purpose, and consequently each dot or dash of light shown in my photographs represents approximately 0.04 second (cf. fig. 6).

**Complexity of motor acts.**—In studying irregularities of movement it is necessary to keep before our minds the complex nature of even the simplest motor act. In the first place few movements are normally executed by the agency of one muscle only, several agonists or prime movers usually co-operating, and it is necessary that each of these contract at the proper time and to the proper degree. Secondly, the antagonists may either relax reciprocally as the prime movers contract, or by an increase of tone they may steady the movement by opposing graduated resistance to it. In the third place it is often necessary that other muscles contract synergically in order to prevent unnecessary displacements at those joints at
which the agonists or antagonists act. Finally, that the movement may attain its end directly and accurately it is essential that other segments of the limb, and especially that with which the moving part is articulated, should be fixed by a tonic contraction of the muscles around their joints. The loss or disorder of any of these components disturbs the regularity and precision of the action attempted. The complex combination of muscles required in the simplest act may be illustrated by what occurs on flexing the fingers. The distal and middle phalanges are bent by the long flexors and the proximal by the interossei and lumbrical muscles, the contractions of all of which must be accurately correlated; the long extensors with the interossei and lumbricals, acting as antagonists, either relax or by their tone regulate and moderate the movement; the three carpal extensors prevent the flexing of the wrist that would otherwise result from the shortening of the long flexors of the fingers, and if the other segments of the limb are unsupported they must be fixed by the tonic contractions of the muscles acting on them in order to avoid their active or passive displacements. When in cerebellar disease a voluntary movement that requires accuracy and precision is carefully examined it is seen that errors occur in its range, rate, and in its direction. The movement may be either excessive so that the limb passes its aim, or it may stop short of it. This abnormality, to which the title dysmetria has been given, was observed by Schiff, and has been more carefully investigated by Luciani in animals, and by Babinski, Thomas, and others in man. The movement may be also abnormal in its rate, being either too rapid or too slow in some portion of its excursion, or jerky and intermittent. Finally, the moving limb may not follow the shortest practicable route, or deviate from its course.

Disorders of Simple Voluntary Movements

We can most conveniently begin our study of the disorders of voluntary movement by investigating those which occur in simple actions, and later deal with the disturbances of more complex ones. It will also be advisable for purposes of description and analysis to examine separately the errors which occur in range, direction, and rate.

(1) Dysmetria.—Disturbance in the range of movement is one of the most frequent disorders in cerebellar disease. The most common and striking abnormality is an excessive range, so that the limb overshoots the point aimed at. In attempting to touch his nose the patient's finger may pass it and strike his cheek violently, and the same may occur in bringing his finger to any point outside him. Sometimes the limb seems merely flung in the desired direction and continues to move of its own inertia until it strikes an object or reaches its full possible range. Or in some part of the movement the limb may appear to fall passively with gravity; on the patient's attempting to touch his nose as he lies in bed the hand may be allowed to fall on his face, or in placing the heel of the affected leg on the opposite knee the foot may simply drop to or beyond it. This hypermetria, or excessive range of movement, may be observed in all ordinary activities; in drawing a line and in writing the point of the pencil is frequently moved too far in one or all directions; in attempting to seize an object the hand may pass beyond it or strike it with undue force, with the fingers opened more than is necessary, or it may be grasped with too great vigour. Neither the range nor the force of the movement is therefore accurately adapted to its end. When this condition has persisted some time the patient generally learns to control the excessive range of movement by arresting his limb before it reaches its aim. The finger or toe is then brought to the point it wishes to touch either slowly and cautiously, or by a series of abrupt jerks, some of which may also be excessive. Hypermetria is generally more prominent the more rapidly the movement is performed, but is frequently present in slow and deliberate movements, though the patient's effort at correction may then obscure it. Voluntary movements are less commonly arrested before their aim is reached, but it sometimes happens that in approaching his nose or any other point the finger stops some distance short of it, and is then brought to it by a second movement or by a series of jerks. This is not always due to voluntary arrest to avoid overshooting, for it may occur before the patient has experienced the necessity of such correction. In a man at present under my care, in whom the removal of a large tumour from the right lateral lobe of the cerebellum was followed by characteristic irregularities of movement, the finger always halted before reaching its aim, even on the day after operation, before experience could have shown the necessity of correction.

(2) Errors in direction.—As a rule voluntary movements are well directed in cerebellar disease, but the limb frequently deviates in one or other direction from the straight course. Records of such movements show that the deviations are usually gradual and their corrections more abrupt. There is no constancy in the directions of the errors, and though they are often most pronounced towards the termination of the movement they may occur in any stage of it. An error is repeated regularly in successive movements only when there is a tendency for the moving part to follow a curve rather than a straight line; the finger, for instance, may be brought from the nose to the elbow through an arc of a circle, but this occurs only when the elbow is fixed firmly to the side and the movement is executed by flexion of the forearm alone. More often the deviations are due to a lazy inert swaying of the limb around the line of movement. This is often prominent when the extended arm or leg is moved, the finger or toe then describes an irregular spiral. These errors in direction are easily seen if the patient traces a circle or square in space with his forefinger. The circle is irregular and is frequently outlined by a broken zigzag line, while the sides of the square may
be represented by similar irregular lines, or by curves which deviate chiefly at the angles owing to dyssmetric termination of each movement and voluntary effort to correct it. They also come into prominence in actions that do not consist merely in movement in a straight line; if the observer's finger is shifted while the patient is trying to touch it, his efforts to alter direction are remarkably slow and clumsy, and at each turn there are purposeless deviations as well as irregularities in range and rate.

Other types of deviation are due to what Thomas has called "decomposition of movement," the various components of the act not being performed in their proper sequence or measure. If asked to bring his finger from above his head to the tip of his nose, the patient may depress the arm at the shoulder before beginning to flex his elbow; and in placing his heel on the opposite knee he may complete the flexion of the hip before bending the knee, with the result that the heel is raised too high and then lowered to the knee. Sometimes this decomposition of movement seems to be a purposive device to control its irregularities; on attempting to feed himself the patient often fixes the elbow firmly to his side and then brings the spoon to his mouth by simplyflexing his forearm. Others, in trying to touch an object, extend their arm or leg rigidly and then swing the limb from the shoulder or hip towards it. These types of deviation from the direct line occur in both slow and rapid movements, but are usually more pronounced in the latter, chiefly since in them voluntary correction is less easy. Frequently, however, the limb in a rapid movement follows a fairly straight or regular line, though it fails to come accurately to the point at which it aims, while in slower movements deviations from side to side occur, but as they are corrected the finger comes more correctly to the point. Deviations from the line of movement are also often prominent when a rapid movement is suddenly arrested, the finger swaying from side to side before reaching its object.

(3) Disturbances in rate of movement.—Not uncommonly the rate of movement of the affected limb differs from that of the normal, either at its commencement or termination, or during its course. When a movement of a normal limb directed accurately to its aim is examined by a graphic method it is found that its rate is quicker during the middle of the excursion that at either its commencement or termination; in other words, the movement starts slowly and ends slowly, but the alteration in rate is always gradual. In cerebellar lesions the commencement is frequently slower than normal, and usually less uniform in rate; the speed at which it is carried out varies considerably, being frequently less during the whole movement no matter how strongly the patient be urged to hurry, but in other cases the limb is lunged out or projected like an inert body, and acquires in its course an abnormal velocity. It is at the termination, however, that the main disturbance in rate is seen; often there is no retardation, and the finger reaches or overshoots at its maximal velocity the point it should touch; this occurs chiefly in movements in which hypermetria is a prominent feature. In other cases the velocity is checked abruptly before the termination of the movement, and the finger then approaches its aim intermittently, in a series of irregular jerks which constitute a terminal tremor. This is most obvious when hypometria is a dominant feature, but excessive movements also may be arrested suddenly. Slow and deliberate movements generally show much more disturbance in rate than do the ordinary spontaneous movements of the patient. In moving his finger from one point to another, or in attempting to outline a square or circle, as slowly and accurately as he can, the patient is unable to maintain a uniform rate; the movement may, in fact, consist of a series of jerks separated by considerable intervals of time, and the slower he attempts to perform it the more irregular does it become (fig. 6). Con-
the result that it is the palmar surface of the second or third phalanx that comes in contact with the thumb; and owing to errors in direction of the thumb or finger, or of both, they do not come so accurately together as on the normal side, or after coming in contact the finger may slide down the thumb. The functions of the thumb are equally irregular; it may fail to move, or may be opposed too far, or flexed so much that the finger comes in contact with its dorsal surface, or to such an extent that its tip touches the basal joint of the finger. An even more striking feature is the inability of the patient to move each finger alone or individually; two or more generally flex at the same time as if he were trying to close his fist rather than bring finger and thumb together. These adventitious movements of the other fingers make the opposition of each finger and thumb more difficult; they impede the moving finger, and if the attempt is sustained the patient may become hopelessly confused in the sequence of movements. The cause of these disorders becomes obvious when the patient tries to move one finger alone or to hold it immobile at the other joints while he flexes or extends it at one. On attempting to bend his forefinger, for example, the other fingers flex or jerk about in irregular alternate flexion and extension; and the patient may be unable to keep the inter-phalangeal straight when flexing the metacarpal-phalangeal joint only is desired. There is consequently a lack of the tonic muscular contractions by which the postures of the fingers and of their separate segments are determined. Since there may be similar defects in the fixation and movements of the thumb the disorders when finger and thumb move together are naturally greater. Another unnatural feature is the occurrence of irregular movements of the whole limb if it is unsupported, or of some of its parts, as the patient brings finger and thumb together, the arm away from the shoulder-joint, and the hand may move at the wrist.

Similar disturbances are found in all delicate actions of the fingers. When the patient is asked to determine the shape of an object placed in the hand he fails to move his fingers over it accurately, or he may simply grasp it. He may be quite unable to use even a simple tool, as scissors, or to button up his clothes, since he cannot employ his fingers separately. A professional musician with a tumour of the left side of the cerebellum complained that with his left hand he “could not unlike the four notes of a chord in proper sequence or time” on the piano, and one of my patients with a long-standing gunshot wound was no longer able to play the flute, although the movements of his arm were apparently normal to other tests.

FACTORS CONTRIBUTING TO DISORDERS OF MOVEMENT

Our next task must be to determine as far as possible the causes of these irregularities of movement. In attempting this it will be advisable to consider separately those more elementary components into which we
have analysed ataxia. Many explanations of dysmetria, and especially of excessive range of movement, have been put forward, but none is entirely satisfactory. One hypothesis, originally suggested by Babinski, and lately adopted by several German writers, is that the cerebellum has a "braking" action on the effector motor mechanisms, and that when this is removed voluntary impulses tend to evoke a movement too great in force and range. There are no arguments, clinical or experimental, in favour of such an action of the cerebellum, nor is it necessary to explain hypermetria.

One cause of excessive movement is certainly deficiency of tone in the antagonists, so that the resistance normally opposed to the contracting muscles is diminished or absent. There can be no doubt that this is an important factor; it explains partly the violent, inert, lunging movements often seen, but it is certainly not the only one, for hypermetria may exist under conditions in which the antagonists are normally relaxed, as in raising a weight or moving the limb against gravity, and when the opposition of the antagonists is replaced by an elastic resistance, such as a spring, or a band of India-rubber, against which the patient pulls in the movement. I have employed this device in a series of patients. If in a man whose finger flies past his nose on attempting to touch it a spring attached to his wrist opposes the movement, it is found that if the tension is not so great as to impede it the finger is still liable to overshoot, though certainly not to the same extent. This still occurs even though the action be limited to movement at one joint, as it may be by fixing the elbow. But the patient's difficulty in touching his nose may be so less, since in approaching it an irregular tremor frequently develops, the limb swings to and fro owing to the alternate predominance of the spring and of the contracting muscles. This observation reveals another factor in dysmetria—namely, the lack of proper measure and sustenance in the contractions of the power movers when their tone is deficient; this also may naturally disturb the range of movement. It must be remembered that the momentum of a rapidly moving limb, which is the product of its mass and velocity, is so great that considerable force is required to arrest it. The ordinary tension of the antagonists alone would not be sufficient unless reinforced by a more powerful tonic contraction, and the form of this contraction must be such that the moving part is not jerked back by it as the agonists relax; in other words, it must be adapted to fixation of the posture, and it therefore comes within Sherrington's definition of reflex tonic. We have a similar phenomenon in the failure of the antagonists to check the movement in the rebound phenomenon, and in the frequent absence of contraction of the flexors of the knee on extension of the leg by the knee-jerk. But, we have already seen that in cerebellar disease there is frequently a delay in initiating and completing the relaxing of contracting muscles, and when this occurs in a rapid movement the limb must necessarily be carried beyond its aim, no matter how slight the delay is, while in slower movements the error in range would be less or may disappear. The strongest cut argument in favour of this explanation is that in my experience a delay in the relaxation of voluntary contractions is invariably found when there is marked hypermetria, and that hypermetria is always associated with such a delay. This slowness in relaxation, associated with a diminution in the plastic tone of the antagonists which should moderate and steady the movement, are sufficient to account for hypermetria.

It is less easy to offer a satisfactory explanation of hypometria, or diminished range of movement. It may be due partly to voluntary correction when the patient has learned that his movements tend to be excessive, but this is obviously not its origin in every case; nor can defective innervation be assumed to be the cause, as hypometria is often not present in all movements of the limb, and it is not in anesthetic limbs only that it occurs. The assumption of Schilder that it is due to disorder of a physiological apparatus closely connected with the cerebellum which "puts a brake apparatus in the limb and brings about an anisotonia," or unequal distribution of tone in opposing groups of muscles, could explain the premature arrest of movement by the hypertonic antagonists, but I have been unable to detect any evidence of such anisotonia, and hypometria may result from lesions destroying the greater part of one lateral lobe, or from disease so gross that such a limited disturbance of tone could be scarcely expected. Schilder, who has given the phenomenon the unmagically name bradytelytelsykes, has rightly observed all the movements of a limb may be prematurely arrested, and all the muscles are never hypertonie.

It is more easy to explain the occurrence of errors in the direction of movement. The most important factor is certainly defective fixation of the joints of the moving limb, owing to which its different parts fail to maintain their correct postural relations, or are displaced passively by the inertia of the movement. We have already seen that a sudden flexion of the forearm may jerk the arm in the same direction, that movement of the wrist or fingers may disturb the attitude of the elbow, and that when the wrist or fingers are in the segment a joint is not sufficiently supported by the muscles around it the segment of the limb moving at it may sway about. Such displacements of any part of the limb necessarily lead to deviations of the moving point, which is generally its distal extremity, from its proper direction. Another factor is disturbance in the time and spatial relations of the separate muscular contractions that execute the movement. If in an action that requires simultaneous movements at two or more joints, one segment for any reason moves before the other so that there is a decomposition of the movement, the finger or toe does not follow a straight line to its aim;
and if the range of any of its components be inaccurate a similar irregularity in direction results. Disturbances in the direction of movement consequently depend partly on defects in the tone of the contracting muscles, of their antagonists, and of those which should fix other segments of the limb, and partly on disturbances in the time and spatial relations of the separate components of the act.

The very large part played in the production of cerebellar ataxia by defect of postural tone may be easily seen in other tests. Flexion of the forearm, for instance, is executed more correctly when the elbow is fully supported than when the position of the arm is dependent on the tonic contractions of the shoulder muscles only, and as Noice has pointed out the irregularity of other simple movements becomes less when the part of the body on which they occur is rigidly fixed. The influence of defective postural fixation is often obvious in tracings of such simple movements as flexion and extension of the forearm when the elbow is unsupported; owing to lateral swaying at the shoulder, and at the elbow, the hand traces irregular ellipses instead of following a straight line. The greater ataxia in actions requiring simultaneous movements at two or more joints than in those consisting of a single movement is partly due to defective postural fixation at each of these joints.

The inability to keep the other fingers extended while one flexes is also dependent on defective fixation by tonic muscular contractions, since it is by this that we normally hold in position the fingers we do not wish to move; when postural tone fails all fingers are flexed by the common action of their longer flexors. It is to this that the disturbances seen in the finger-thumb test can be largely attributed, and since the accuracy of this test depends on the correct adaptation of the movements and postures of both finger and thumb the irregularity in it is necessarily greater than when the finger only moves.

The important part that the efforts to correct these irregularities of movement plays in the clinical picture of cerebellar ataxia must not be forgotten. It has been already pointed out that a movement which tends to be excessive in range is often arrested before its termination, and that the subsequent jerky approach of the limb to its aim makes the end of the movement tremulous; that in bringing a spoon to the mouth the elbow may be fixed to the side before the forearm flexes, so that the movement is decomposed; and that the slowness in the rate of the movement may be purposeful. Correction certainly influences all movements in cerebellar disease, especially in cases of long standing. There is evidence of this in the fact that movements of the affected limb become more irregular if the patient's attention is by any means diverted from them, as by making him perform actions by the two limbs at the same time. Failure of attention is probably also the reason that a dose of alcohol or morphia increases the ataxia, and that it becomes more striking when the patient tires. The irregularities in the rate of movement, and the tremor that is occasionally seen during it, can be partly explained by the abnormalities we observed in the simplest muscular contractions. The slow starting of the movement is obviously due to the delay and slowness in initiating contractions, while the lack of uniformity in rate depends on the failure of steady contraction of the prime movers and on the absence of the steady influence that the normally toned antagonists exert. The excessive velocity of the movement and its inertness towards its termination are partly results of deficient opposition by the antagonists, owing to which the unopposed agonists move the limb at too great a speed, but the delay in the relaxation of the contracting muscles may also contribute to it. The terminal tremor is in part due to the errors that occur towards the end of the movement and their correction by extracerebellar mechanisms, in part to inability to maintain steadily the attitude into which the limb has been brought, owing to deficient tonicity of those muscles that should fix it.

Lecture III

The last lecture dealt with the disorders of simple voluntary movements produced by cerebellar lesions; to-day we must consider the disturbances in more complex actions and will begin with those seen in alternate movements.

Adiadochokinesia

Babinski originally pointed out that a patient with cerebellar disease is frequently unable to perform rapid alternate movements as quickly and as accurately as normal persons, even though each individual movement alone can be properly executed. To this disability he gave the name adiadochokinesia. A careful analysis of this symptom is necessary, since the functional disturbances to which it is due throw considerable light on the defect phenomena that result from cerebellar lesions. A record of the alternate movements by a graphic method is essential, since it enables us to recognize the more elementary abnormalities. One of the most convenient tests is rapid pronation and supination of the forearm at the elbow, but other alternate movements, as flexion and extension of the fingers, of the forearm, or of the foot at the ankle joint, may be employed; the same disturbances may be observed in more common and habitual acts, as shaking hands, clapping hands, tapping a table, stamping the foot, etc. When a patient with a unilateral cerebellar lesion pronates and supinates his unaffected forearm as rapidly as possible the successive excursions are remarkably regular in both rate and range, and other segments of the limb not actively concerned in the act, as the upper arm, hand,
and the fingers, are adequately fixed at their proximal articulations, and consequently maintain their proper postural relations to the moving forearm. On the affected side, on the contrary, the primary movements are irregular in rate and range, and various accessory movements occur in segments of the limb proximal and distal to the moving part.

Generally the most obvious feature is the slowness of the movements; their rate may be reduced to a half or third of that of the normal limb, but slowness is not constant. It often happens that when the action is first attempted the limb moves fairly quickly, but if continued the excursions become progressively slower until the patient may give up the attempt.

![Graph showing movements](image)

(Fig. 7). Irregularity in rate may be equally prominent; instead of alternating rapidly and without an appreciable delay, pronation and supination may be separated by appreciable intervals which vary constantly. Frequently the limb is arrested abruptly in some position, often at the end of an excursion, until the patient by a special effort moves it again. The slowness results mainly from this delay at the turn, each individual movement being usually performed with normal rapidity, though tracings frequently show a delay in the commencement of each.

The range of movement is also irregular; occasionally some or all of the excursions are excessive, or an excursion may be arrested before it has reached its natural limit. There can be no doubt that the rate of movement is closely related to the range, the larger excursions being the quicker, but the abnormality of the one is certainly not dependent on that of the other. The irregularity in range also becomes more prominent when the limb tiring. The movements of the affected arm are almost invariably more inaccurate and less complete when the patient attempts to perform them simultaneously with both limbs; this is partly due to insufficient attention being devoted to the affected one, but partly to the confusion that naturally arises when, owing to lack of synchronicity, different acts are at the same time attempted by the two limbs.

**Inappropriate Accessory Movements**

Inappropriate accessory movements are frequently an equally prominent feature, though they have attracted less attention. If the elbow be not firmly supported or held in position the upper arm sways about owing to displacements at the shoulder, every possible movement occurring, though abduction and adduction generally predominate. Similarly the hand, and frequently the fingers too, swing about inerely; one of the most common displacements being ulnar deviation of the wrist with flexion of the ulnar fingers as the forearm supinates. But there can be no doubt that in addition to these inert ammonous movements more regular acts may replace that which the patient attempts; the forearm, for instance, may begin to flex and extend, and I have repeatedly seen opening and closing of the fingers. This is more likely to happen when pronation and supination is continued for a time; then as the limb tires the effort apparently degenerates into other movements.

Some of these adventitious movements, as the inert flopping of the wrist and fingers, can be attributed wholly to the toneless state of the muscles that normally fix them. This is also the origin of some of the purposeless deviations at the shoulder, but if the muscles around the latter be carefully palpatred active contractions can be easily detected in many of them. They can be also demonstrated if the observer grasps the arm and attempts to displace it while the patient is engaged in the action, for owing to these contractions it offers more than normal resistance to displacement. This led me to believe that in attempting rapid pronation and supination there might be an irregular spread of the innervation to muscles which should not be actively concerned in the act, but further observations have made it probable that these active contractions represent a voluntary attempt.
to fix those segments of the limb which are displaced passively owing to absence of fixing tone in their muscles. These adventitious movements have a considerable influence on the regularity of the actions that are attempted, since these require adequate fixation of the arm on which the forearm moves, and the inert swaying of the distal segments of the limb must also disturb the equilibrium of the moving parts. An attempt to flex and extend the forearm at the elbow reveals disturbances of the same type. Irregularity in the range is often striking owing to the larger angle of movement possible; this is not uncommonly the forearm is flexed until it is checked by the ligaments of the elbow joint, the sudden stress on which may give pain or discomfort. The distal segments may also flop about inertly, the wrist flexing as the hand approaches the shoulder and extending as it is jerked away from it. The elbow is often jerked up by the momentum of the flexing forearm, and if not supported it may wobble about owing to passive displacements at the shoulder.

Abnormal Finger Movements

In flexion and extension of the fingers additional abnormalities can be seen. Though the fingers are all moved together at first they soon fall out of alignment, or fail to flex and extend simultaneously; in fact, extension of one finger may commence before some of the others are fully flexed. The fingers often spread, too, and assume weird attitudes, and the thumb may be adducted either over or under them. This naturally leads to a confusion that may make the act apparently impossible. One intelligent patient explained that "after I have done it for a time I seem to lose the power of opening and closing my hand properly," and another with a right-sided lesion said, "I seem to do it subconsciously on the left side, but on the right I have to think out each movement; the arm comes to a dead stop and I have to think before starting again." Other patients state they have to "drive" the affected arm. In the second place, there is often a disturbance in the synergic movements of the wrist and fingers. Normally the hand is extended at the wrist by active contractions of its extensor muscles as the fingers flex, but in a limb subject to cerebellar disorders this synergic extension of the wrist may fail so that the fingers and hand are both flexed by the long flexors of the fingers, or the wrist and fingers may not move synchronously.

Flexion and extension of the foot at the ankle may be equally irregular in rate and range. When this is attempted on the normal side as the patient lies in bed the knee is usually raised a little as the foot dorsiflexes by a slight degree of flexion at both hip and knee; on the affected side these synergic movements are generally much more irregular, they may be either absent or excessive. If he attempt this action while sitting on a high stool with his legs hanging free the affected limb may swing about

widely owing to movement at the knee. Similarly, though the patient can keep the normal foot fixed at the ankle while he flexes and extends his toes, the foot of the affected side flexes and extends or turns in and out as the toes move. In attempting to tap a table as rapidly as possible it frequently happens that the hand on reaching the table slides along it before rising again; this is apparently due to accessory movements at other joints, and especially at the shoulder. Light-tine records of such movements also show that the moving point, instead of following a straight line, describes curves or swings around in irregular ellipses. Similarly, on attempting to stamp his foot on the ground the limb often sways about in space before reaching it instead of moving vertically.

Most of these disturbances result from the simpler disorders of movement we have already studied, and can therefore be largely attributed to defect of the postural tone which normally controls the range of movement, and ensures the correct fixation of those parts on which the moving segment is articulated. This is shown by the fact that the irregularities diminish when the proximal segments are firmly fixed, and also by an experiment I have carried out in several of my recent cases. A light elastic rubber band is tied at its middle around the segment of the limb that is moved and its two ends are held slightly stretched in the plane in which the movements should be made, so that each excursion is performed against its elastic resistance. In testing flexion and extension of the forearm, for instance, the band being fixed to the wrist, one end is held near the shoulder and the other in the plane in which the forearm moves in extending; its two portions consequently represent the tonic resistance of the two groups of antagonistic muscles. I have invariably found that the movements are then executed more accurately than when the arm is free, and the patient feels that it is easier to do them. Dysesthesia, or disturbance in the range of the individual movements, is naturally a latter too.

Dissociation in Time and Force of Muscle Contractions

The failure in the proper synergic associations of the fingers and wrist, and of ankle and knee, cannot, however, be explained wholly by lack of tone. Deficiency of this in the extensors of the wrist may account for the simultaneous flexion of hand and fingers by the long flexors of the fingers, but not infrequently the wrist is over-extended as the fingers close. We are consequently compelled to assume a dissociation in the time and force of the contractions of the separate groups of muscles that should act synergically; this may be due to a relatively greater delay in initiating the contractions in one group than in others, and this we cannot yet attribute to hypotonia only.

The confusion which appears when alternate movements are continued
for a time is a natural result of these elementary disturbances. Normally such simple alternate movements are controlled automatically by sub- conscious mechanisms; when this control is defective it must be replaced by conscious efforts which are more liable to tire, and when tired to become confused, especially when the mechanisms through which they act are disordered. The influence of attention on the movements of the affected limb is shown by the increased irregularity when it is diverted by simultaneous use of two limbs or otherwise. The erroneous but purposeful movements that may develop when the patient is forced to persist in an action, as the substitution of flexion and extension of the fingers for pronation and supination of the forearm, may be probably explained by a subconscious effort to escape from an action in which both muscles and attention have tired. It is certainly not due directly to the cerebellar defect.

**STANDING AND WALKING**

The abnormalities in standing and walking depend partly on the primary defect phenomena of the cerebellar disease, and partly on the automatic and voluntary measures by which the patient attempts to correct or maintain his balance. It is not always easy to separate these two groups of symptoms, or to determine which disturbances are primarily due to the lesion.

**Unilateral Lesions**

When the lesion is unilateral the attitude in standing is frequently unnatural. The trunk and head are generally inclined to the affected side with the spine concave in this direction, but by tilting its pelvis the patient moves as much of his weight as possible on the normal leg. The homolateral shoulder is generally in front of the other since the body is rotated on its longitudinal axis towards the normal side. The arm usually hangs inertly and is not used in efforts to preserve equilibrium, but occasionally it is either adducted to the side or separated from it. The attitude of the head is not constant; it is most commonly inclined to the affected side with the face rotated towards the opposite shoulder.

In recent and severe injuries the patient may be unable to stand erect without support or assistance; he inclines towards the side of the lesion, and may fall to it passively without making adequate attempts to avoid doing so. Many are unable to explain why this happens, others say it is as though a magnet or some unseen agency drew them in this direction. When the symptoms are less severe and the patient can stand without support he can be more easily pushed over to the affected side; a gentle tap on the contralateral shoulder may be sufficient to disturb his balance, though he can resist more forcible blows that tend to displace him to the normal side. As he can be also thrown over to the affected side when he sits unsupported, the instability must be partly due to disturbances in the functions of the trunk muscles. In falling he often holds himself straight and rigid, not flexing his trunk to the normal side in a natural manner. The homolateral leg is usually abducted and often rotated outwards; this attitude is probably either a voluntary or a reflexive device to assure equilibrium. Further, the upright attitude is not maintained steadily, the whole body frequently sways about, the head is tremulous, and irregular contractions may be seen in the muscles of the leg.

The danger of falling is in part at least due to the instability of the affected leg: many patients state that this gives way under them and that they consequently collapse suddenly, and inspection may show that the contractions of the muscles around the knee and ankle joints are irregular or intermittent. Almost invariably the patient has more difficulty in standing on the affected leg than on the other; many who keep their balance easily when resting on both feet fall at once when on this alone. It is often due to failure in extension of the knee which flexes under the weight, or to lack of stable fixation of the ankle owing to which his foot turns under him. The leg seems to be used as a passive support as a still would be rather than as an active member, and even when standing at rest the ab ducted limb often gives the impression that it is employed mainly as a shore or prop to prevent him falling to this side. Its unsteadiness usually increases when deprived of the support of the boot; then irregular contractions of the tendons on the dorsum of the foot may be visible, and toes do not attempt to clutch the ground as they normally do when balance is threatened. The patient is also unable to readjust his balance promptly or correctly on the displacement of any part of his body that shifts his centre of gravity; he may, for instance, fall if his head is suddenly tilted in any direction, or if the observer's hand placed in his axilla throws his arm unexpectedly from his side. If placed on the middle of a plank that is raised at one or other end the patient falls more readily, especially when tilted towards the affected side.

**Gait in Unilateral Lesions**

The gait is generally so characteristic in a unilateral lesion of severe degree that it can be recognized at once. The movements of the homolateral leg are irregular; the foot is often raised too high owing to excessive flexion at the hip, and may either fall to the floor inertly or be brought to it with undue force. Sometimes it hovers over the floor and hesitates for a moment before coming to it. In some patients, however, the affected limb is in some steps dragged forwards or raised so little that the toes scrape along the floor. The placing of the foot is frequently incorrect; most commonly it is abducted too much, but it is often brought in front of the other, so that in advancing the latter he may trip over it and fall towards his normal side. Sometimes the whole sole falls flat on the floor,
or there may be an excessive interval between the placing of the heel and the dropping of the toes, or it may come in contact with the ground in either an inverted or overlapped attitude. Occasionally while the limb is advancing the foot flops about inerently, or the limb may be brought forward extended rigidly at the knee with the foot dorsiflexed. The steps are also irregular in length; some are too long, others too short, but when there is much disturbance of equilibrium they tend to exceed those of the normal limb as the patient, afraid to trust his weight on the affected leg, takes short shuffling steps with the other. This irregularity in placing the foot is more obvious if the patient, even when supported, attempts to walk held to toe along a line. In walking sideways with the normal limb in front the affected one may be merely dragged along the floor, but when it leads its steps are unequal and irregular, and the foot may fall either in front of or behind the other.

There are two more or less characteristic features in the gait; in the first place the patient reels, lurches, or stumbles towards the affected side, in the second he tends to deviate in this direction, and if his eyes are closed he may wander at a considerable angle from the line he intended to follow. One result of the tendency to stumble towards the affected side is that the patient jolts into anyone on this side of him, and he consequently prefers that his attendant walks on his normal side and merely places a steadying hand on his arm. The tendency to deviate becomes more obvious when he walks around a chair; when his affected side is towards the chair he is constantly stumbling and falling to it, but he deviates more and more from it, following the course of an opening spiral, when he walks around it in the opposite direction. These features are, however, frequently obscured or complicated by voluntary efforts to maintain balance; the patient may, for instance, learn to incline and deviate to the opposite side, and though he is more liable to fall in the direction of the lesion the excessive abduction of the affected leg may throw him towards the other side, or he may stumble in this direction owing to over-correction.

It is usually ascertained that vision has no influence on these disturbances of gait and equilibrium; this is literally true, but many patients become nervous of falling on closing their eyes, and the voluntary efforts they make to avoid it may disturb their balance. Some patients keep their balance better when moving quickly than when walking slowly and deliberately; it seems that the momentum they acquire in rapid movement is of more service than the voluntary control they can exercise when walking slowly, just as it is more easy to ride a bicycle at a fast than at a slow rate. It is particularly on turning that equilibrium is in danger owing to failure to control or modify the inertia of the body by proper movements; even a sudden turning of the head may upset the balance. Similarly, the patient may be unable to halt suddenly on a word of command when walking either forwards or backwards; when advancing he may totter forwards by a few shuffling steps or throw himself on his toes, or on walking backwards may be unable to arrest himself without assistance. For the same reason he may fail to attain equilibrium immediately on rising to his feet from a low seat, and on sitting down quickly he may fall backwards. Irregularity of the ground increases the unsteadiness, since the patient fails to adjust his foot to any slight obstacle or unevenness it may encounter, but on a polished or slippery floor the affected foot is liable to slide from under him. Ascending and especially descending an incline is also more difficult than walking on the level, and going up and coming down stairs even more so; in ascending the foot is often elevated excessively, or the toe may strike the rise of the next step or come to it too forcibly, while the patient usually descends one step at a time owing to fear of trusting his weight on the affected limb, when his hip, knee, and ankle flex.

In walking the homolateral arm usually hangs inerently by his side and is not swung actively forwards as he advances the opposite foot; sometimes, however, it is held against the thorax or abducted from it with the elbow partially flexed.

Gait in Bilateral Lesions

The maintenance of equilibrium in the upright position is much more difficult when both sides of the cerebellum are injured, especially when the vermis is involved. Then the patient generally stands with feet widely separated, his body swaying from side to side and generally crouched forwards as though lack of tone in the vertebral muscles made it impossible to hold it erect. He may fall in any direction or lose his balance immediately if his centre of gravity is suddenly displaced. Certain patients with lesions of the vermis have a tendency to totter backwards or forwards for a few steps and then fall without making any adequate attempts to save themselves; in less severe cases this may happen only when the patient is suddenly pushed in one direction. On attempting to walk he sways, staggers, and reeling in all directions like a drunken man, and though able to maintain equilibrium when at rest, may fall on attempting to walk. The movements of his legs have the same irregular characters as those seen in cases of unilateral injury, but the steps are more hurried as the patient fears to throw his weight on either foot alone. When a fall occurs it may be either due to the displacement of the feet, or failure to adjust the weight of the body to the unnatural positions into which they fall, or to a sudden “giving way” of the legs. The difficulty in keeping the centre of gravity over the base of support is a striking feature in many cases; most commonly the body, instead of being carried forward with the legs, lagging behind so that the patient falls backwards unless supported by pressure from behind, or pulled forwards. When he stops suddenly, or attempts to walk backwards, he totters in this direction with short shuffling steps, his
heels alone perhaps bearing his weight, or rocking to and fro in the anteroposterior plane on heel and toes. In such cases the absence of appropriate efforts to maintain equilibrium is very remarkable; the patient may fall inertly like a doll. More rarely there is a tendency to incline forwards and carry the centre of gravity in front of the toes; then the patient may be unable to halt suddenly when walking, or may totter and stumble forwards when pressed from behind.

The large part played in the compensation of the cerebellar defect phenomena in gait by voluntary effort, or by the intervention of other portions of the nervous system, is shown by the effect of training and education. The symptoms produced by an acute lesion diminish more rapidly when the patient is systematically trained to walk correctly, and the disturbances are, in relation to the severity and extent of the lesion, never so great when the disease develops so slowly that compensation can occur pari passu with its progress. Voluntary correction probably plays the most important part, since gait invariably becomes more unstable when the patients tire, or are dulled by a dose of alcohol or opium, or when their attention is diverted.

Factors of Instability

On analysing the instability in standing and walking and the tendency to fall, we find they can be attributed to (1) errors in moving and placing the affected leg, (2) instability of the leg making it liable to flex under the patient's weight, and (3) a failure to adjust promptly the centre of gravity to the base on which the patient rests, or to the line on which he moves. The inaccuracies in moving and placing the foot are identical with those which occur in voluntary movements of the individual limbs and must be due to the same causes. It is true that the "ataxia" of the leg in walking is usually greater than in movements performed when the patient lies on his back, but this is natural since the trunk being firmly fixed in the latter position the limb has a firm base on which to move, while in walking the pelvis is constantly changing its position in relation to the floor, and the limb must consequently adapt its movement to constantly varying relations of space. The sudden flexing or "giving way" of the leg under the weight of the body can be also attributed to lack of adequate postural fixation. We have seen that analogous disturbances occur in the arm, which is often unable to maintain attitudes, and in which sudden relaxations of the muscles may allow objects to fall from the hand. When standing on the affected limb alone the axis may invert under the patient's weight, or the knee may not remain rigidly extended, or, what is more important, the trunk may sway about irregularly on the hip which bears it.

The failure to assure equilibrium by adjusting the centre of gravity to the base of support is partly due to the same causes. In locomotion the body is in a state of unstable equilibrium, since its base of support is small and its centre of gravity is constantly shifting in all planes of space. Walking requires not merely the correct movements of the legs, but also the proper adjustment of the rest of the body to the positions in which they are placed. The advancing leg is at first extended, but in order to carry the body forward it must flex at the knee and ankle, and at the same time rock on its sole from heel to toes. In these movements the flexing segments can no longer depend on the formation of their joints and ligaments, but must be adequately fixed by the tonic contractions of their muscles, and if this fails the centre of gravity is further displaced and its adjustment to the supporting foot becomes more complicated. The exaggerated oscillations of the body, due to the atomic state of the muscles that move and support the vertebral column, and the irregularity of the steps increase further the difficulty in correction.

That equilibrium is always more disturbed by bilateral than by unilateral lesions is due to the fact that the muscles on both sides of the body being affected the postures and the stability of the trunk in movements are more disturbed; and both lower limbs being involved the patient has no stable base on which to move, as he has in alternate steps when the disease is one-sided. Though muscular hypotonia plays a very large part in the disturbances of equilibrium they cannot be wholly explained by it alone. A careful investigation of gait in cerebellar disease reveals an irregularity in the accurate combination of movements necessary to assure balance, analogous with that which we have seen in the affected arm and due to the same factors. Clinical observations certainly lend no support to the theory originally propounded by Magendie and later adopted or modified by many authors, that the cerebellum is an organ for equilibration, since the loss of balance in cerebellar disease is no greater than might be expected to result from the disturbances of the more elementary movements and postures of the limbs and trunk.

Disturbances of Speech

As a rule only relatively extensive lesions of the cerebellum, and particularly those that involve the vermis, disturb articulation and phonation seriously, but speech may be affected by disease strictly limited to one lateral lobe and not bilateral lesions only, as Jelgersma, Brunner and Coenen assert. My experience also disproves Stenvers' hypothesis that the right side of the cerebellum has a more special relation to it.

The most striking features are the slow, drawing, and monotonous character of the voice, the unnatural separation of the syllables, and the slurred, jerky, and often explosive manner in which they are uttered. The indistinctness is usually due to defective combination of the consonants, but the vowel sounds are often slurred too. Articulation and phonation are equally affected, and the voice is frequently too loud or is
not properly modulated to the circumstances. The patients and their friends usually describe the change as a "stuttering," or a "thickness," or a "jumbling" of speech. It generally becomes more indistinct and sometimes scarcely intelligible when the patient tenses. Many with recent and severe lesions complain that they can often not use the proper word, if it is a long and difficult one, by which to express their ideas. In severe bilateral lesions speech may be unintelligible for a time; in one man, who had a transverse bullet wound through both sides of his cerebellum, it was merely a slow dribbling in which only isolated syllables could be distinguished. As he improved it became a succession of widely separated syllables, each pronounced slowly in an unnaturally high-pitched voice.

The effort apparently necessary in such cases to utter even a few syllables or a sentence is another remarkable feature. It is as though the utterance of every sound had to be controlled voluntarily. Patients occasionally complain of this difficulty or of their inability to utter words without a special care, especially after speaking quickly for a time. There is frequently, also, a marked excess of grimacing or purposeless facial movements during speech, and the expressional gestures normally associated with it are often irregular and inappropriate. A tendency to unnatural explosive laughter, in which the patient may eject saliva from his lips, is not uncommon in bilateral disease.

Speech improves, as a rule, rapidly when the affection is unilateral, but recovery is slower, and may never be perfect in patients with bilateral lesions. In the more chronic forms of cerebellar disease, as the atrophies, its disorders are usually permanent. These abnormalities of speech can be explained by the same factors that disturb the finer movements of the limbs. The delicacy and precision of the individual movements of the larynx, palate, tongue, lips, and of the chest, and the accurate manner in which they must be associated to produce normal speech is remembered, the disturbing effect of defective relations of the separate parts in posture and movement is obvious.

**Disorders of the Ocular Movements and Nystagmus**

There are few cases of severe cerebellar injury which do not present, especially in their acute stages, disturbances of the ocular movements. Some of these are transient unless the disease be progressive, while others, and particularly nystagmus, may persist indefinitely. In the early stages of any acute lesion the eyes while at rest are generally directed slightly to the contralateral side of the primary central position. This deviation is never so great as that which occurs in a conjugate ocular palsy, and is never associated with loss of the power of movement to the affected side, though this is often more difficult than deviation to the normal side. Even when it is affected there is commonly an inability to maintain them in this attitude as they tend to recede towards the middle line. This

relative weakness of deviation to the affected side usually disappears rapidly. The vertical movements and convergence are not affected in this way by unilateral lesions, but they may be executed less easily than is normal when acute bilateral disease exists. True ocular palsy never result from lesions limited to the cerebellum, their frequent occurrence in cases of tumour and abscess being due to extension of the disease to, or compressions of, the brain-stem or of the extra-medullary trunks of the ocular nerves. The only disturbance in the parallelism of the optic axes which occurs is known as "skew-deviation," and it is rare. In it the homolateral eye is directed downwards and inwards, and the contralateral upwards and slightly outwards, but as the visual axes generally become parallel when fixation is attempted, diplopia does not usually result; two of my cases, however, complained of double vision. Skew-deviation is always a transient condition, disappearing frequently within a few days of the occurrence of the lesion. It is identical with the deviation originally described by Magendie in animals, in which it appears chiefly after section of the vestibular nerve or injury of the inferior or middle cerebellar peduncles. In man it probably occurs only when the lesion extends down to the neighbourhood of the para-cerebellar nuclei.

**Nystagmus**

This is a much more common symptom; in fact, very few lesions of the cerebellum fail to show it in some form or other. It was absent in only three of the gunshot injuries I observed, and it occurred in all the cerebellar tumours included in this study. It is always more marked immediately after the development of a severe lesion, and in slighter cases it may subside quickly or disappear. Its degree and the form it assumes vary considerably in different cases, and it is therefore advisable to describe first the most characteristic type which is seen in the acute stages of a unilateral lesion. The eyes almost invariably remain steady in the position they assume spontaneously, whether it be the central or to the normal side of it, but any movement from this position evokes the characteristic nystagmus. These are always pronounced when the patient moves his eyes laterally towards the side of the lesion. Then the nystagmus consists of slow oscillations of relatively large range, which are more or less regular in both rate and amplitude, and usually persist as long as accurate fixation is maintained, but when this tiring and the eyes deviate towards the middle line they cease or become irregular or intermittent. Each oscillation consists of a quick phase which is always in the direction of the voluntary movement of the eyes, and of a slow phase during which the eyes recede towards their position of rest. Both the rate and the range of these jerks vary considerably in different cases; sometimes the rate may be as slow as three jerks in two seconds, but it is usually more rapid; in 12 acute cases in which it was counted it varied between 15 and 30
oscillations in 10 seconds. The range of the jerks is variable, too, and is usually larger through less regular when the deviation of the eyes tires. Both the rate and range of the oscillations usually increase on moving the object fixed further towards the affected side.

On turning the eyes towards the normal side the oscillations are typically smaller in range, more rapid in rate, and usually less regular. Each consists of two phases—a rapid jerk in the direction of the voluntary movement, and a slow recession of the eyes towards their primary position, but the difference in rate is usually less than on deviation to the affected side. The nystagmus evoked by this position is often represented by a short series of fine oscillations separated by pauses, and it rarely persists so long as the eyes are turned to the opposite side. In many cases the jerks occurring on lateral deviation of the eyes are purely horizontal, but there is often some rotation of the eyes, too, most commonly in the direction of the rapid phase and downwards—that is, clockwise rotation on movement of the eyes to the left and anti-clockwise when they are turned to the right. Vertical nystagmus is less constant, but on looking upwards there is frequently an occasional jerk of the eyes, more rarely a regular sequence of oscillations, upwards or obliquely upwards and towards the affected side. Occasionally some rotation occurs, too, which is generally upwards and towards the affected side. Nystagmus on downward movement of the eyes is much less common; when the cerebellar lesion is unilateral it is generally represented only by intermittent downward jerks. On central fixation nystagmus occurs as a rule only in cases with recent and severe lesions. Then, on moving the eyes into the primary central position from that which they spontaneously assume, more or less regular jerks develop, the quick phase being towards the side of the disease. In certain of these cases the eyes are at complete rest only when directed at an angle of from 10 degrees to 20 degrees towards the contralateral side. We shall refer to this as the “rest point.” Similar nystagmoid jerks occur sometimes on convergence, the slow phase in both eyes being towards the normal and the quick towards the affected side.

This characteristic form of nystagmus is certainly not present in every case of unilateral lesion; but the almost invariable rule is that the jerks are most easily elicited by deviating the eyes towards the side affected, and the jerks occurring on movement in this direction are slower and more motion is seem those which appear on deviation towards the normal side. The quick phase of the jerk is invariably in the direction in which the eyes are voluntarily moved. Frequently no vertical oscillations can be obtained, and those on lateral movement may be irregular or represented by intermittent jerks only. Remissions frequently occur, particularly on contralateral regard and when the patient fails to fix a point accurately owing to inattention or tiring.

The nystagmus produced by lesions of the cerebellum is essentially a fixation nystagmus—that is, it is elicited only by, or is most pronounced on, accurate fixation of some point by the eyes; and though it may occur under conditions in which fixation is impossible, as when the patient wears spectacles with high convex lenses and blinik or when an opaque screen is held within a short distance of the eyes, it is less marked and less regular. Then the most striking feature may be the failure of the eyes to remain in that position to which the patient turns them, especially when it is to the side of the lesion. It is also less pronounced when the patient is simply told to look to the right or left and not given some point to fix. Change in posture of the eyes, as when the point fixed is moved, temporarily increases the range of the jerks; in mild cases, in fact, absence of steady fixation when the eyes take up a new posture may be the only abnormality.

Nystagmus is generally more symmetrical in bilateral injuries of the cerebellum, but the same rule holds, that the quick phase is in that direction in which the eyes are moved voluntarily. Vertical nystagmus occurs more commonly when the vermis is involved, though it may be seen when the injury is limited to one-half of the cerebellum.

Nystagmus of cerebellar disease is consequently a well-defined and systematized disturbance. It cannot be regarded as simply an “ataxia” of the ocular movements or a tremor of the eyes. An observation I have recently made throws light on the nature of the oscillations. A girl in whom a tumour was removed from the left side of the cerebellum eight years ago has had since then a complete paralysis of the left external rectus muscle, but on attempting to look to the left characteristic nystagmus appears in both eyes. The jerks of the left eye, which are identical in time and range with those of the right, can be explained only by periodic relaxations of the left adductor muscles, and each jerk must be consequently regarded as a co-ordinated act in which the antagonists relax as the prime movers contract. Kahlo has made a similar observation in animals after division of an ocular muscle.

The frequency with which nystagmus occurs in all forms of cerebellar disease leaves no doubt that it is directly from lesions of this organ, and is not dependent on affections of the neighbouring structures, like Deliria’s nucleus, as Rothmann and Marburg assume. On analysing the nystagmus we find that it is composed of slow deviations of the eyes from the position to which they are moved voluntarily towards their rest point, constituting the slow phase, and the correction of this deviation by quicker and more abrupt jerks in the direction of the voluntary movement. The essential component of nystagmus is consequently the slow phase of the jerk, and this is obviously due to a failure of the eyes to maintain postures. Consequently nystagmus can be attributed to a disturbance of the tonic contractions of the ocular muscles by which postures are maintained. Simonelli has also regarded it as a manifestation of defective postural activity. We must therefore conclude that each half of the cerebellum
has an influence on the maintenance of all postures of the eyes, and that this influence is greater when the eyes are deviated towards the same side, less when turned towards the opposite. Each side of the cerebellum is also to some extent concerned in the vertical postures of the eyes, though this probably is mainly a function of the vermis.

The quick phase by which the eyes are replaced in the position from which they had receded is probably not of cerebellar origin, but the central mechanism on which it depends is not definitely determined yet. It is improbable that it is of cortical origin, as Barlow, Wilson, and Pike assume, since Magnus and de Kleijn have found that it is possible to elicit nystagmus in animals after removal of the fore-brain and thalamencephalon; the reflex centre of the quick phase must consequently lie in the mid-brain. It is, however, noteworthy that in one of my patients in whom a large missile, after passing through the left side of the cerebellum, entered the left hemisphere of the brain, producing a complete right hemiplegia probably owing to destruction of the internal capsule, no nystagmus occurred on deviation of the eyes to the right, although characteristic slow, coarse, forcible jerks developed when he fixed an object to his left side. In another patient with a left-sided cerebellar lesion and right homonymous hemianopia due to a gunshot wound no nystagmus developed on deviation of the eyes to the right, though there was typical nystagmus when he looked to the left. This suggests that injury of different fibres from the left hemisphere may abolish the quick phase on deviation of the eyes to the right.

Nystagmus can consequently be entirely explained by a defect in the postural tone of the muscles which should maintain the eyes steadily in postures to which they are brought by voluntary effort. And on the regulation and control of this postural tone the cerebellum has evidently an important influence.

LECTURE IV

There remain two symptoms of destructive lesions of the cerebellum which we must consider before we put together our conclusions on the nature of the defect phenomena dealt with in the preceding three lectures, and the theory of cerebellar function to which these conclusions lead us.

BÁRANY'S POINTING TEST

The importance lately attached to Bárány's pointing test in cerebellar diseases makes it necessary to record fully the results obtained in my cases. As is well known, Bárány discovered that the deviations of the limbs in certain directions which are evoked by appropriate labyrinthine stimulation may be absent when the cerebellum is affected, and that deviations in the opposite direction may occur when the limbs are unsupported.

In most of my cases spontaneous deviations only were investigated, since it was neither possible nor advisable to excite the labyrinthine reflexes by rotation or irrigation of the ear in the patients with recent gunshot wounds. This was, however, carried out in a certain number of those with more chronic affections.

Direction of Deviation

If a normal person holds his arm outstretched horizontally in front of him the limbs usually remain in the position in which they are placed, even though his eyes be closed. In unilateral disease of the cerebellum the contralateral arm also maintains its position, but that of the side of the lesion generally swings or deviates in one direction or the other. This is not merely a passive falling away, such as occurs in anatomic or asthenic limbs, since the observer's hand, if placed on the limb, may feel that this is forced in the direction in which it deviates. Deviation can be often evoked or made more pronounced by shaking the patient or tapping the arm.

Lateralward deviation is the most common. It occurred in 37 of the 41 successive cases of my series in which specific observations are recorded; in 35 of these the movement was outwards, in one it was inwards, and in another variable in direction, while in 4 none was ever observed. As a rule, this deviation from the position of rest occurs in the homolateral limb only when the cerebellar lesion is one-sided; the other arm occasionally swings a little in the same direction, but probably only when the injury is extensive. Owing to the tendency of the affected limb to fall under its own weight spontaneous deviations in the vertical plane are less definite; of 23 successive cases in which passive falling could be excluded the arm deviated upwards in 7, in 10 downwards, in 4 the direction of deviation was variable, and in 2 no deviation occurred. Deviation is generally more obvious in movement. It may be tested at the shoulder by making the patient, while his eyes are closed, move his finger in either the vertical or horizontal plane from a fixed point and back to it again, with his arm steadily extended at all joints. The presence or absence of deviation at other joints of the limb may be similarly determined by fixing the more proximal segments. As there is frequently a conscious effort to correct the errors it is advisable to allow the forefinger to touch a similar object. I have generally used a linear measure on which the amount of angular error can be measured.

My notes contain observations on lateral deviation of the arm in vertical movement from the shoulder joint in 58 cases; in many of these the test was repeated several times. In 45 of these patients, outward deviation of the arm (abduction) occurred either constantly or predominantly; in 2 there was inward deviation, in 0 the direction of error was variable, and in 5 no deviation was at any time noticed. Vertical movements of the index-finger, of the hand, and of the forearm were separately tested in some of
these cases; but the results were the same, the error was almost constantly
outwards. Occasionally, the movements of the arm are not purely vertical;
it may be raised obliquely upwards and outwards, or deviation from the
vertical may occur towards the end of the movement, the finger then turning
abruptly outwards or inwards. Errors in horizontal movement of the arm
are less striking and tend to be more variable. The notes of 41 cases
record upward deviation in 10, downward in 12, while in 10 the direction
of error was variable, and in 9 none was observed.

Several factors seem to influence the deviations of the arm in movement;
probably the most important of these is the position of the head. If, for
instance, the right arm deviates outwards owing to right-sided cerebellar
lesion when the head is straight, the deviation increases when the head is
flexed to the right and diminishes or disappears, or may even be replaced
by an inward error, when it is approximated to the left shoulder. Similarly
forward flexion of the head tends to increase downward deviation and
abolish upward errors. This influence of the attitude of the head extends
to the normal limb, which also tends to deviate in the direction in which
the head is moved.

The pointing test was applied to the lower limbs in a smaller number of
the cases. My notes refer to observations on 15 patients only; in 10 the
leg moved vertically from the hip deviated inwards, in one only it deviated
outwards, and in 4 no constant errors were observed. It consequently
seems that although outward deviation is the usual error in vertical move-
ment of the arm, inward deviation is more common in the leg. In the few
cases in which the effect of labyrinthine stimulation by rotation or by the
caloric method was tested it was found that stimuli which normally
inhibit the arm in the direction in which it deviates increase the error,
while those which move the limb in the opposite direction diminish or
annul it.

Theories of Spontaneous Deviation

The nature of this spontaneous deviation and the causes to which it is
immediately due are still obscure. According to Bárány the cerebellar
cortex contains four "centres of direction" determining movement at
each of the joints of the limb in the corresponding planes of space;
normally the two centres for vertical and those for horizontal movement
balance one another, and when one is destroyed its antagonist prevails.
André-Thomas and Durupt have put forward a somewhat similar hypo-
thesis, that local lesions of the cortex produce an anisokinesia or unequal
distribution of tone in opposing groups of muscles, and that the limb
deviates in the direction of action of the group possessing more tone.
Each of these hypotheses would explain the deviation, but there are objec-
tions to each. I have never been able to detect the anisokinesia described
by Thomas. If it occurred so frequently as to cause deviation in the
majority of cerebellar lesions a more constant error in direction than
occurs might be expected in ordinary movements; even Thomas has stated
that this can be observed only occasionally. And it is scarcely credible that
such gross lesions as existed in many of my cases could have produced
alterations of tone limited to one set of muscles, leaving their antagonists
undisturbed or relatively hypertonic.

The fact that gunshot wounds, tumours, and other affections of one
lateral lobe of the cerebellum produce outward deviation of the arm in
the great majority of cases, regardless of their situation, is difficult to
explain by either theory. The greater frequency of outward deviation has
been observed by others, and various explanations of it have been put
forward. Acerbi's hypothesis that the adductor mechanisms are more
frequently damaged owing to their wider representation in the cortex, and
that the intact abductor centres then displace the arm outwards, can scarcely
account for it. Nor is Rothmann's assumption that the adductor centres
are more developed satisfactory, especially in view of the accurate localiza-
tion of these centres claimed by Bárány.

The frequency of similar deviations in lesions of the fore-brain, even in
conditions which from their nature cannot disturb the functions of the
cerebellum, complicate the problem, and make it difficult to offer any
explanation of general application. It is not yet possible to decide definit-
ively whether it is through the cerebellum that labyrinthine impressions
act in exciting deviations as Bárány assumes; or whether deviation is
an independent function of the cerebellum which can neutralize the
effects of the labyrinthine excitations, but the fact that Magnus and de
Kleijn have been able to elicit their tonic labyrinthine reflexes in absence
of the cerebellum is in favour of the latter view.

Sensation

The question whether any form of sensation is disturbed by destructive
lesions of the cerebellum has been revived by recently published observa-
tions. Magendie first suggested that the cerebellum is an organ of "muscle-
sense." Lussana elaborated this view, assuming that the position of the
centre of gravity of the body and of its different parts is perceived through
the cerebellum by means of the affrent fibres which it receives from the
dorsal columns of the cord. Though physiological investigations as well
as numerous clinical observations failed to lend any support to this view
of the cerebellum as a sensory organ, it has been again put forward by
Lewandowsky, who regarded the disorders of movement resulting from
cerebellar disease as a "sensory ataxia," which is always accompanied
by disturbance of "muscle-sense." Rothmann has also described an
affection of "deep sensibility," or of the sense of position, after cortical
lesions in animals. Munk, Hitzig, Martin, and more recently, Jegher have,
on the other hand, regarded the cerebellum as an organ of "un-
conscious sensation" (unconscious Sensibilität), the afferent impressions that reach it remaining below the threshold of consciousness, or reproducing representations of a lower order that can be consciously perceived only when transmitted to the fore-brain. Others, as Risien Russell, have described an affection of cutaneous sensibility by acute cerebellar disease, since animals subjected to experimental lesions may not react normally to superficial stimuli.

But as no sensory disturbances have been found regularly associated with even the severest affections of the cerebellum in man, these views have attracted little attention from clinicians. Within the last few years, however, more definite changes have been described. First Lotmar, and later Maas, Goldstein, Reichmann, and others have discovered a disturbance in the appreciation of weight by the affected arm. Goldstein, Reichmann, and André-Thomas have also described defects in the sense of position, while the twoder and another record diminished sensibility to pressure and vibration, and even disturbances of spatial and temporal perception. I have repeatedly examined all forms of sensibility in patients in whom, as far as could be determined from their symptoms, the lesions were limited to the cerebellum, but in no single instance did I discover any alterations of cutaneous sensibility or of the sense of position. It is true that, as Luciani has pointed out, the withdrawal of the affected limb from a painful stimulus may be less prompt than on the normal side, especially when the lesion is acute, but there was never any alteration in the threshold of, or subjective difference in, the sensations evoked by tactile or painful stimuli. Nor have I been able to detect any diminution or alteration of the sense of position by any of the ordinary clinical tests. My patients with their eyes closed have always been able to bring the normal hand immediately and correctly to the affected one, after this had been moved passively in space; they could reproduce accurately with the normal limb attitudes given passively to the affected; and they never presented any sign of diminished appreciation of shape or size. I have investigated the threshold of pressure sensibility in a few cases only, and in these no difference existed between the normal and affected sides.

The Appreciation of Weight

There is, however, no doubt that a patient with a unilateral lesion may be unable to compare accurately equal weights, or weights in which the difference is small, placed in his two hands. In 18 of my cases this test was used, and in many of them on several occasions over long periods. In 4 cases equal weights were always adjudged equal, in 3 the results were so irregular that no conclusion can be drawn from them, and in 11 the weight placed on the affected hand was constantly estimated by the patient as heavier, when they were actually equal or the difference was small. Con-

sequently, the majority of my patients over-estimated the weight on the abnormal side. Some of the more intelligent recognized this subjectively, particularly when heavy weights were employed; one man three months after receiving a gunshot wound in the right lateral lobe of the cerebellum, stated, "every weight I try to lift seems heavier to my right hand." The only patient who, when identical weights were employed, ever judged the weight on the affected hand as lighter, was variable in his replies during the first four weeks after the wound, and during the next two months generally described the weight on this hand as heavier.

Such observations do not, however, necessarily indicate a disturbance in the appreciation of weight since the tendency to over-estimate on the abnormal limb might be due to an affection of mobility rather than to a sensory defect. Every paralyzed limb exaggerates the load it carries if its sensation be normal, and the affected cerebellar arm may similarly over-estimate the resistance that the test object opposes to its movement. The difficulty in raising and lowering the hand regularly as is normally done in attempting to estimate a weight, may also contribute.

We can speak of a disturbance in the appreciation of weight only when there exists an inability to discriminate between two weights above the normal difference threshold which are placed successively on the limb. In each of my cases where the comparison of identical weights placed on the two hands revealed any alteration, the threshold was carefully ascertained in the normal limb, and then a series of observations were made with the same two weights on the affected side. No difference in the accuracy of discrimination of the two limbs could be detected in any of these cases, and we are consequently forced to the conclusion that the appreciation of weight is not affected directly by cerebellar disease. The tendency to over-estimate weight on the affected side may be partly explained by the ataxia of the limb. Another possible factor is the toneless state of its muscles, owing to which the proprioceptive nerve-endings within them may be less easily excited when they contract or are subjected to strain by the resistance of raising the weighted limb. It is a priori possible that, as Goldstein suggests, this factor would also influence the recognition of the position of the limb in space, but I have been unable to detect such disturbance. Goldstein and Reichmann have attempted to differentiate symptom-complexes due respectively to irritative and destructive lesions of the cerebellum, the chief feature being over-estimation of weight in irritative states, and under-estimation in destructive. It seems extremely improbable that any such distinction can be made. Certain of my patients who invariably judged the weight in the affected hand the heavier had long-standing quiescent lesions in which persistent irritation was very improbable, while others suffered with recent gunshot wounds. In fact, this and the other symptoms of cerebellar disease
present no qualitative differences in the acute and the chronic stages of the lesion which can permit us to distinguish irritative from destructive effects. The cases in which Goldstein and Reichmann observed sensory disturbances were particularly unsuitable for the investigation of sensation; in three the condition was diagnosed as meningitis serosa, which is rarely a local or circumscribed affection; in a fourth there was a history of a gun-shot wound, but as no scan was visible a diffuse concussion injury that possibly involved the brain-stem was more probable; in one of Reichmann's cases sensory disturbances which were regarded as hysterical complicated the picture, and in the other there was a labyrinthine concussion. Other portions of the nervous system were probably involved in all these cases, and it is consequently impossible to conclude that the defective discrimination of weights and the other disturbances of sensation observed by these authors were due to cerebellar lesions.

**FUNCTIONS OF THE CEREBELLUM.**

As we have described the chief symptoms of destructive lesions of the cerebellum, and discussed as far as was possible their nature and significance, we can now consider what light our observations and analysis throw on the normal functions of this organ. Attempts to determine physiology by arguing from pathological manifestations are, however, fraught with danger, for the functions of any organ cannot be defined in terms of defect phenomena; and clinical symptoms are not always merely defects of normal function, since compensation by unaffected parts of the organ or other parts of the nervous system may influence them. But when we keep before our minds these possible fallacies, observations of the symptoms of its disease in man can offer contributions of value to the physiology of the cerebellum.

The interpretations offered of the chief disturbances of function in our cases show that they can be explained by a small number of elementary disorders. The flaccidity and lack of elasticity of the muscles are obviously due to diminution or loss of that state of normal tension in them which clinicians speak of as tone. The inability of the limbs to maintain attitudes in opposition to gravity or to the action of external forces, and the tremor of outstretchedness which develops under these influences, are a result of loss of that tonic component which stabilizes and sustains the voluntary contractions of the muscles. The excessive associated movements of the affected limbs during vigorous muscular effort of other parts of the body also result from deficiency of the muscular tone that determines and maintains their normal postures; and in nystagmus and in the knee-jerk we have given direct manifestations of the effect of loss of postural tone. The relative asthenia and fatigability of the limbs, and the chronic or intermittent character of the contractions of their muscles in even the simplest movements, are a natural consequence of the lack of fusion of the twitch contractions of the muscles and of the replacing of inexhaustible postural tone by voluntary effort which tires more quickly.

On investigating the disorders of movement we discovered that hypotonia plays a large part in these too; errors in range can be attributed partly to deficiency of that tone in the antagonists which should steady or moderate the action of the prime movers, and partly to lack of tonicity in the muscular contractions which should not merely arrest, but also fix, the limb in the position in which it is arrested. Errors in direction are mainly due to the failure of adequate fixation of the moving segments or of the parts on which they move; the irregularity of rate of movement and tremor result partly from the intermittent and irregular manner in which the prime movers contract owing to lack of an adjuvant tone, and partly from absence of the steadying influence of normally-toned antagonists. The influence of disturbances of tone is still more prominent in complex actions; the disorders of gait are largely due to inadequate postural fixation and incorrect postural relations of the different segments of the limbs to one another, and of the trunk to the limbs; while we have seen that the irregularities in the movements of the individual limbs are mainly a result of hypotonia. The abnormalities of speech, too, can be explained by disturbances of the relations in posture and in movement of the separate organs concerned in it. In the inability to perform alternate movements rapidly, and in the irregular adventitious movements of the limbs which accompany attempts to do so, lack of fixation tone in the muscles also plays a very large part. Finally, the only sensory disturbance that exists in cerebellar disease—that is, inaccuracy in the comparison of weights—depends on deficient tone in the muscles, and not on a primary defect in any sensory quality.

But in these descriptions we are employing the word tone in two different senses, or we are dealing with at least two different manifestations of tone. In the first place we apply it to the tension or elasticity of normal muscles which opposes or restrains their sudden elongation; in the second place we signify by it that property of muscles owing to which they exert a certain and indefeasible tension regardless of their length, and consequently fix those segments of the body on which they act in attitudes into which they are brought either actively or passively. This same tone, according to Sherrington, fuses with, reinforces, and stabilizes muscular contractions otherwise excited. It is a reflex state excited mainly from the proprioceptive organs of the muscles themselves, and is subject to the same laws of excitation and inhibition as reflex muscular contractions. But while these can be excited, maintained, and coordinated by the lowest levels of the nervous system alone, postural tone seems to require the cooperation of higher nervous levels, and, as we shall see, the cerebellum is actively concerned in it.
Disturbances of Coordination.

It has not been possible, however, to explain all the symptoms of cerebellar disease by disturbances of tone alone. Delay and slowness in starting and executing muscular contractions and relaxations are prominent features in many cases, and they cannot be attributed wholly to atonia. They also are certainly an important factor in some of the motor disorders. A slowness in evoking relaxation of contracting muscles may make the movement attempted excessive or hypermetric; and a greater delay in the contraction of one set of muscles than of others with which it should act synchronously may lead to decomposition of movement and errors in direction. The slowness and irregularity in performing rapid alternate movements are also due chiefly to the time lost at the turn of the movement, owing to failure of the one group of muscles to relax immediately and of the other to contract at once. We have also seen that the explanation of the disorders of gait and the inaccurate coöperation of agonists and synergic muscles in other actions requires the assumption of a disturbance in the measure and time relations of the contractions of muscles that should work together harmoniously. We are consequently compelled to admit that the disorders of movement in cerebellar disease are in part due to lack of harmony and coöperation of the various muscular contractions and relaxations that are required in an act, and there is consequently an incoördination or ataxia of movement in its literal sense. But a fuller recognition of the part that postural tone plays in both active and passive attitudes and in movement has cut down enormously the symptoms in the explanation of which it is necessary to invoke incoördination. At the same time a more careful analysis of the motor disorders makes it possible to account for the ataxia by simpler factors, particularly by a lack of synchrony in the actions of muscles that should contract at the same time, or a disturbance in the time relations of those that should contract in succession.

The nature of the influences exerted by the cerebellum on the motor system can probably not be settled by clinical investigations alone. There are two possibilities.

The first is that the cerebellum is concerned only in the maintenance of a proper state of tone in the musculature as a whole, that it has a general and unvarying influence only, and does not participate in the separate stages of a movement or action. The disorders of movement in cerebellar disease would be, therefore, if this assumption were correct, due to defects in the lower mechanisms through which the higher levels of the nervous system exert control. There are, however, many facts which make it difficult to explain all cerebellar disorders by this view. In the first place, ablation of the cerebellum does not abolish the tonic reflexes of labyrinthine and muscular origin which have been studied by Magnus and his collaborators, and the postural reactions that may be excited in the decerebrate animal also persist after its removal. The explanation may be that the labyrinthine acting alone are able to maintain a tone sufficient for the reflexes elicited from them, and that the medullary mechanisms through which the decerebrate reactions are evoked are also tone exciting for their own purpose. That this is so is shown by the fact that Sherrington, Horsley, and Lowenthal have proved that stimulation of the cerebellum may inhibit decerebrate rigidity.

The other possibility is that the accurate combination and correlation of the phasic and tonic elements of every movement demand the active interplay of the cerebellum, and that the tonic supplement to tetanic contractions as well as the modifications of tone in the antagonistic, synergic, and fixing muscles are excited or controlled through it in every stage of movement. We have found it necessary to invoke this hypothesis in explanation of many of the symptoms, and there are consequently two factors contributing to the disorders of movement in cerebellar disease; in the first place a deficiency of postural tone in the moving parts, in the second place a lack of harmony and coördination in its modifications in the muscles engaged in the movement. And since postural tone and its regulation is a reflex function we speak of a disturbance of 'reflex adjustments,' to adopt the term Ferrier employed nearly 30 years ago, as one of the factors in cerebellar ataxia.

Though there can be no doubt that the distribution and regulation of tone is dependent largely on the lower reflex levels of the nervous system, this does not exclude the possibility that the cerebellum takes a part in determining its grading or measure in the separate groups of synergic muscles concerned in every act. Our investigations, therefore, point to the conclusion that the cerebellum plays two distinct though closely allied roles in movement: 1. It is a part, and probably the controlling part, of those central mechanisms which maintain and regulate the distribution of postural tone in the muscles. In this rôle the cerebellum has a continuous and unvarying function. 2. During movement it regulates and coördinates the modifications of tone that accompany the relaxations and contractions of all groups of muscles directly or indirectly concerned in the movement. In this function its activity is discontinuous and variable.

Functional Localization

The question whether a localization of function exists in the cortex of the cerebellum is not yet settled; even among those who maintain the existence of localization there is no general agreement on what is localized, or on the topographical representation of either special functions or separate segments of the body. The earliest investigations devoted to this subject were the experimental studies of Sir David Ferrier, who, by faradization of various points of the surface, elicited lateral and vertical move-
ments of the eyes and head, as well as less definite movements of the homolateral limbs, from which he concluded that the cerebellum is a complex arrangement of individually differentiated centres. These results were to some extent confirmed by Nothnagel and others. From later observations, however, Horsley and Clarke concluded that the cortex of the cerebellum is inextensible, and that the motor reactions obtained by earlier observers were probably due to spread of the current to the brain-stem or to the trunks of the cranial nerves. But more recent investigations have thrown some doubt on Horsley and Clarke's conclusions, as they have shown that under certain conditions motor reactions can be obtained from the cerebellar cortex by the use of moderate or even weak currents. The modern search after a functional localization can be traced to Bolk's comparative anatomical study of the cerebellum. By examining this organ in a large series of mammals, he discovered a principle of subdivision by means of which all its various parts can be compared, and their homology ascertained in different species. On making such a comparison he found a parallelism between the relative size of certain parts of the cerebellum and the development of groups of muscles which are associated together for special functions. Then arguing that form is determined by function, and that morphological details are in some way correlated with the functional, he concluded that the cerebellum is a complex of organs, each with a distinctive physiological duty. The logical outcome of these observations was an attempt to determine a localization, or rather to map out the physiological representations of different portions of the body in the cortex of the cerebellum. Bolk's conclusions were: That the lobus anterior, the unpaired segment in front of the primary sulcus, contains centres for the eyes, tongue, jaw, face, larynx, and pharynx; that in the lobulus simplex immediately behind it the neck muscles are represented; that the lobuli ansiformes and paramediani, which represent the larger portions of the lateral lobes, contain paired centres for the movements of the individual limbs; while in the middle part of the vermis, or lobus medianus posterior, there is an unpaired centre for the bilateral synergic movements of the limbs, and in the inferior vermis a centre for the trunk musculature.

Bolk's investigations at once attracted attention, and several physiologists naturally sought to test his conclusions by experimental methods. The most important of the earlier researches were those undertaken by van Rijnberk, who removed small portions of the cortex in several of the functional areas described by Bolk. His results appeared to confirm the conclusions which Bolk had already drawn, but the anatomical examination by Binnert of the brains on which he had operated showed that in many of them the lesions extended deeply into the white matter of the cerebellum, and in some cases injured the central nuclei. Vincenzoni, Marussini, Lima, Poli, and others repeated these experiments, but though they all claim to have found evidence of localization their conclusions diverge considerably.

The experimental investigations of Rothmann, and especially those of Thomas and Durupt, in which the extent of the lesions were controlled more accurately by anatomical examinations, also tend on the whole to confirm Bolk's theories, though their localization differs from his in many details. Thomas concludes that the vermis contains centres for the synergic movements of the head, trunk, and limbs, while each lateral lobe is concerned in the regulation of movement of isolated parts of the body and possesses separate areas for the homolateral limbs. These areas are made up of secondary centres for each articulation, and in these articular centres there is a separate representation of muscles according to the plane of space in which they move the limb. This attempt at an extremely intimate localization is surprising when we remember that there is far from being any general agreement as to the representation of even larger segments of the body in the cerebellar cortex. The discrepancies in the conclusions of even the most recent observers may be illustrated by the fact that while according to Ingvar the lobus posterior (inferior vermis) contains centres for those muscles that prevent the animal falling backwards, Simonelli, repeating his experiments, could find absolutely no confirmation of this. Another excellent instance of the present state of the theory of localization is, that while Bolk assumed that the formatio vermicularis contains centres for movement of the arm, according to Rothmann its destruction produces disturbances of movement at the head, Thomas and Durupt found that lesions of it were followed by symptoms in the lower limb, and Bárány that it influences the movements of the eyes only. Rossi, from his recent studies, has concluded that there are not absolute or circumscribed cortical centres, but probably at the most only a "prevalence of representation" of segments of the body in certain extensive areas of the cortex.

Another interesting scheme of cortical localization has been put forward by Bárány, who assumes that each limb is represented in two or more distinct cortical centres, in each of which there is a further representation of the muscles according to the segment of the limb on which they act and the direction in which they move it. The centre for the outward deviation of the arm, for instance, lies in the lobuli semilunares superior and inferior, that for inward deviation in the lobulus biventer, and in this the hand is represented most medially, the arm more laterally. Bárány's theory is based upon the spontaneous deviations which occur in the pointing test in local lesions of the cerebellum, and on the absence of reactional deviations on labyrinthine stimulation. It must be noted that the representation of the different segments of the body according to his scheme differs considerably from that assumed by Bolk and obtained by several physiologists; for instance, Bárány places some of the arm centres caudal to the centres
for the lower limbs. It is these great discrepancies in both the topography of functional representation and in the views of what is localized that make it so difficult to accept any of the conclusions that have been put forward.

**Facts Unfavourable to Localization**

Investigations by other methods do not strengthen the case of localization. Rossi, for example, finds that electrical stimulation of one side of the cerebellum lowers the threshold of excitability of the opposite cerebral motor cortex; and if one point of the latter is selected, as that which evokes flexion of the arm, its excitability can be modified equally from several regions of the cerebellum; these experiments therefore do not indicate a more intimate connection of one point than of any other in the cortex of the cerebellum with a focal motor point of the opposite cerebral hemisphere. Conversely, Beck and Bielsch have found that the electrical variations in the cerebellum produced by stimulation of the motor cerebral cortex show no local tendency. Finally, according to Miller and Banting, paralyzation of a point in the anterior portion of the vermis may inhibit decerebrate rigidity in both the homolateral limbs and excite complex bilateral movements.

The lesions in my own cases of gunshot wounds involved diverse portions of the cerebellum. Some were large and deep, in others there was probably at the most only a very limited destruction of the cortex. In the majority the posterior and inferior surface was injured, but in several the damage was limited to the superior surface. The tumours observed by me also occupied different regions of the cerebellum. Consequently my material, extending to nearly 100 cases of local cerebellar lesions, affords an excellent opportunity of testing the reliability of the various theories of localization that have been put forward. There can be no doubt that the movements of the head, neck, and trunk, and in fact of all movements that require the co-operation of bilateral muscles, are more severely disordered when the vermis is injured than when the lateral lobes only are damaged, and we must consequently accept a representation of those parts of the body that act bilaterally in the vermis. Certain facts indicate that there may be a further localization of function within the human vermis; I have, for example, observed a tendency to fall or stagger backwards in tumours and other lesions of its posterior portion, and a falling forwards in one case at least of tumour in its anterior part.

My observations do not lend support to the theory of circumscribed functional representation of different parts of the body in the lateral lobes. I have invariably found that the symptoms produced by a small lesion were never limited to one limb or a segment of a limb; that if any abnormality could be detected it occurred in the arm the leg was the more frequently it there was in addition some disturbance in the func-

...tions of the cranial nerves. In a certain number of cases, however, the one limb seemed to be relatively more affected than the other, the leg being more involved by caudal and mesial lesions, and the arm by the more anterior. It is possible, then, that there is a "preponderance of representation" in Rossi's sense, the arm being represented more anteriorly and the leg more posteriorly, but this localization is so indefinite that I would not regard it as a trustworthy guide in the diagnosis of a small lesion, such as a cortical tumour.

There are two clinical facts brought out by my observations that tend to invalidate many of the schemes of cortical localization that have been put forward. In the first place, Bolk and many of his followers have claimed that the ocular movements are represented in the lobus anterior or superior vermis, but, as will be remembered, I have observed nystagmus or other disturbances of the ocular movements in practically all local lesions, no matter what their situation was. In the second place, Bolk placed the representation of the organs of phonation and articulation in the lobus anterior, and Rothmann and Kallmann and others have described a cortical centre for the larynx here too, but in my experience speech may be affected by lesions of any portion of either the vermis or lateral lobes, though it is more liable to be severely disturbed when the vermis is injured. My observations consequently lend no support to a circumscribed or focal representation of different portions of the body in the cortex of the cerebellum, apart from the fact that the vermis is concerned mainly with movements requiring the co-operation of muscles on the two sides of the body, and the lateral lobes mainly with the functions of the homolateral limbs.

Note.—No Bibliography was appended to this paper. Full lists of references are to be found in the papers in Brain of 1917 and 1939.

**NOTES**

The Croonian Lectures, as republished in the Selected Papers of 1956 and as reproduced here, differ from the original Lectures in the following respects.

Illustrations. Lecture I. Figures 1, 2a, 2b, 3a and 3b are taken from Brain, 40, 461 in which they are numbered 1, 11, 12, 13A and 14. The last two figures replace figures numbered 5A and 2B in the original Lecture I.

Lecture II. Figures 3, 4, 5, 7 and 8 are omitted. Figure 6 from Brain, 52, 1 (Fig. 13) is a re-orientation of Figure 6 in the original Lecture.

Lecture III. Figures 2, 3 and 4 are omitted. Figure 7 (Fig. 6 in Brain, 62, 1) is essentially the same as Fig. 1 in the original Lecture.

Text. Lecture II, Section headed ASTHENIA: The passage beginning 'In acute lesions' is omitted. "'I can't lift the strength may be reduced to half that of the normal limb' and ending "... with it" is not in the original Lecture. The enclosed Table also differs in that Case 1 is taken from Brain, 40, 461 and Case 2 is introduced de novo.

Lecture IV. The section headed Functions of the Cerebellum as Revealed by Clinical Observations, which in the 1956 version was replaced by a section from the Juddings' Observations, which is in the 1936 version was reinstated; it makes splendid points.