THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES DUE TO GUNSHOT INJURIES.

BY GORDON HOLMES, M.D.

INTRODUCTION

The functional defects which are produced by injury or disease of the cerebellum have been described by many physiologists and clinicians, and almost innumerable attempts have been made to analyse and solve their significance and thereby determine the normal functions of this
organ. But there is still a remarkable divergence between the symptoms attributed in various text-books and monographs to lesions of the cerebellum in man and the phenomena which physiologists have observed after its injury or ablation in animals. It is, however, obvious that a close correspondence must be established between them before clinical experience can contribute to the solution of the physiological significance of the cerebellum; and before we can determine the part which this plays in the human nervous mechanism.

The opportunity of making uncomplicated clinical observations is rare in civil life, since acute lesions of the cerebellum, comparable with those produced by physiologists, are uncommon; tumours and abscesses which develop in it are very liable to compress or influence the functions of other parts too; softening and hemorrhages are rarely wholly limited to it, and the degenerative and atrophic diseases which involve it practically never affect it alone. In warfare, on the other hand, wounds limited to the cerebellum and injuries of it of different extent and localization, can be frequently observed. During the present War I have been able to examine over forty men in whom it had been injured, but as wounds of this region of the head are notoriously serious a certain number died early, in others the symptoms were only slight or transient, and many were of necessity evacuated to England within a short time of the infliction of the injury. Twenty-one patients in whom the symptoms were pronounced remained, however, under observation sufficiently long to permit repeated examination and investigation. In several of these the wounds had healed and they were able to leave bed and walk before they were transferred; some of the more serious cases in fact remained under observation for two or three months.

My investigations have been consequently made chiefly in the early and acute stages of injuries of the cerebellum, but in many cases the recovery of function, or "compensation," could be also studied. I was particularly fortunate in being able to examine, before this paper was completed, two men in whom the lesions were of much longer standing. One, in an attempt to commit suicide eight years previously, had shot himself through his mouth and produced extensive destruction of one lateral lobe of the cerebellum, in which fragments of metal still remained. The other had been wounded by a fragment of shell-casing twenty months previously and had been trephined over one lateral lobe. Both patients still presented symptoms of cerebellar disturbance, but unfortunately the second man had developed signs of disseminated
sclerosis. In a third case, which also came under my observation, the characteristic symptoms of a very severe unilateral cerebellar lesion had come on suddenly while he was under heavy gun fire. I have not included these three cases in my series, but I have been able to control many observations made in acute cases on them. My observations have been made, therefore, on injuries which resemble closely those on which the classical physiological descriptions are based, and it will consequently not be surprising that a close similarity exists between them. The chief divergencies probably depend on the position of the subject in the phylogenetic scale, and on the relative importance or subordination of the cerebellum in the nervous system of different animals.

In the subsequent pages it will be my aim to describe those disturbances of function which constitute the symptoms of recent cerebellar lesions as objectively as possible, and to attempt to analyse complex symptoms into their simpler components. Names and terms applied to symptoms will be employed as rarely as possible, and as the defects of function can be often most easily understood by observing abnormalities in the ordinary or spontaneous activities of the affected parts, the usual clinical tests have been supplemented by further observations.

It is, however, impossible to deal with this subject without relying largely on Babinski’s masterly analysis of the symptoms of cerebellar disease, and on the careful descriptive work of other neurologists, especially André-Thomas, who has also attempted a valuable correlation of these clinical symptoms with the disturbances that occur in animals after experimental injury. If the references to their work are incomplete it must be attributed to the circumstances under which this contribution has been written.

In the majority of my patients who survived, the lesions were unilateral and involved only, or chiefly, one lateral lobe of the cerebellum; the descriptions of the resulting symptoms will be consequently based largely on these cases. Certain patients with extensive bilateral lesions were, however, also observed.

CHAPTER I.—UNILATERAL LESIONS OF THE CEREBELLUM.

§ 1.—Disturbance of Muscle Tone.

The effects of cerebellar injuries fall almost exclusively upon the motor system, and in the early stages of an acute lesion one of the most prominent symptoms is loss or diminution of tone in the muscles of the limbs, and to a less extent in those of the trunk, of the same side.
If a case in which there is an extensive unilateral lesion be examined within the first week or two, one of the most striking features is the flabbiness of the muscles of the homolateral limbs to palpation; as compared with those of the opposite side they are more easily compressed and displaced transversely, and they can be stretched or elongated to a greater extent without any discomfort to the patient. This is obviously the reason why the limbs often assume unnatural attitudes or are placed in postures which the patient would tend to avoid in a normal limb. Not infrequently, for instance, the hand may be observed lying for long periods prone on the bed with the fingers fully straightened out or even hyperextended, although this unnatural attitude can be easily corrected; and this is probably the explanation of the fact, that on attempting to rise into the sitting position in bed the patients occasionally help themselves by throwing their weight on the dorsum of the affected hand, so that the fingers and wrist are overflexed to an extent that would be uncomfortable or even painful in the normal limb. An officer, for instance, who was dull and restless for a few days afterwards complained that the knuckles and the wrist of his affected hand were sore, owing to the fact that in attempting to sit up in bed he usually placed this hand under him in the flexed position. This phenomenon cannot be attributed to loss of sensation, since repeated examinations have failed to reveal alteration in any of its modalities; it is analogous to the failure to correct instantly unnatural attitudes of the limbs, which Luciani has described after partial or total ablation of the cerebellum in animals.

If in such a patient both forearms are held vertically by the observer the wrist of the affected side usually falls passively into a position of extreme flexion, while in the normal limb the wrist is not allowed to become more than semiflexed owing to the tone of its extensor muscles (fig 1). Similarly, if as the patient lies in bed his arms are fully abducted and rotated outwards so that his hands lie behind his head, the affected wrist is more fully extended by its own weight than is its fellow, and the arm is often excessively rotated too.

When such a limb is handled and moved about passively it is at once obvious that there is loss of that slight but definite resistance that normal muscles offer to stretching. Further, if either the arm or the leg is seized and shaken it is found that the more distal segments of the affected limbs flop and swing about in an unnatural inert manner, like the arm of a flail. This can be easily seen, and the abrupter jar which the arrest of the freely swinging segments communicates to the
observer's hand is often even more unmistakable. Another striking feature brought out by this manoeuvre is, that while the oscillations of the distal segments of the normal limb are limited by the elastic tension of the muscles that are stretched, those of the affected limb can be felt to swing till the joints "lock," and their bony and ligamentous structures prevent their further movement. This "locking" of the joints can be most easily felt at the wrist, elbow, and the ankle. There is never, however, any evidence of articular or ligamentous relaxation which permits a greater range of absolute movement, such as occurs in tabes dorsalis.

The same phenomena may be observed by rotating or otherwise suddenly displacing the patient's whole body when he can stand erect, for the affected arm swings about inertly as though it were only attached to the shoulder by a string. (André-Thomas.)

In rapid voluntary movement, too, an excessive passive swing at certain joints not directly concerned in the action can be often observed. When for instance the patient is asked to flex and extend his supported elbows as quickly as possible, as in testing for adiadochokinesis, the affected wrist is successively overflexed and overextended by the momentum of the movement, so that the hand is flung against the shoulder or towards the bed. The fact that in this test the affected elbow is often raised from its support by the momentum of the flexing forearm is also due to deficient tension in those muscles that should fix the shoulder-joint.
And if the forearms are seized and suddenly flipped by the observer, as if he were cracking a whip, the wrists flex passively, but while the normal hand is immediately extended again owing to the elasticity of its extensors, the affected one remains flexed or only swings inertly. This may be seen at the ankle too if the relaxed leg is similarly jerked and arrested abruptly. Finally, if a patient in this stage holds his two arms horizontally outstretched and the normal limb is gently tapped by the observer's fingers, it is but little displaced and immediately regains its original position, but the affected arm swings more widely as a result of each blow, offers less resistance to displacement, is arrested less abruptly, and is slower in its return (André-Thomas). In this test the displacement is generally due to movement at the shoulder, but if the hand is more forcibly tapped the wrist and the elbow often flex too.

This diminution in elastic resistance of muscles to stretching can be often observed directly. If the fingers are seized and passively extended at the same time as the wrist they can be easily, and without the observer experiencing any resistance, bent back till further movement becomes impossible owing to the conformation of the joints and the tension on their ligaments; and even in this position the patient does not experience the dull pain or discomfort in the overstretched flexor muscles which he suffers when the unaffected wrist and fingers are forcibly extended to the same degree. This may be also observed at the elbow and shoulder, and in the joints of the lower limb. The affected heel can be for instance easily brought into apposition with the buttock, or the thigh can be so fully flexed that the knee touches the chest (fig. 2). Consequently, though the maximum possible range of passive movement may not be increased, the normal resistance offered to it by the muscles when they are passively-elongated is deficient, and definite resistance is felt only when the rigid ligaments or the apposed bones restrict the movements. As a rule this diminished elasticity of the muscles becomes more obvious about five to ten days after the infliction of the wound, and in cases of severe injury it may persist for several weeks at least.

We therefore find on palpation, passive movement, shaking and tapping of the affected limbs, that their muscles are soft and flabby, and that their tension, which normally tends to oppose passive movement and prevent that unrestricted swinging under the influence of gravity or displacement of their different segments, such as occurs in a hinged skeleton, is diminished. In other words, there is loss of that active tension which distinguishes normal muscles and is independent of their length, that is known as tone.
This flabby hypotonic state of the muscles suggested the examination of their reaction to direct percussion. This was done in several cases but no definite difference could be detected from the normal.

When an extensive unilateral lesion of the cerebellum exists this state of hypotonia is always limited to the same side, but it is usually more pronounced, or at least more easily demonstrated, in the upper than in the lower limb. Distal and proximal muscles are involved to relatively the same degree. It is less easy to demonstrate in the trunk muscles, but here too the stretching of those of the side of the lesion offers less resistance, as the patient sits or stands, than those of the unaffected side. It does not produce any appreciable asymmetry or other change in the face. In smaller lesions it is generally demonstrable in both the upper and lower limbs, but is less pronounced, particularly in the latter.

Its distribution, especially in slight cases, is a question of consider-
able importance in view of the conclusions arrived at by André-Thomas and Durupt from experimental work, which André-Thomas attempts to support by certain clinical observations. These authors have described as a result of small limited lesions of the cerebellum an affection of the tone of certain muscles or groups of muscles only, so that a condition of anisosthenia or loss of tone in some muscles and relative hypertonicity of their antagonists, results. It is to this disturbance that André-Thomas attributes many of the symptoms of cerebellar lesions.

Though I have intentionally searched for this condition in the majority of my patients, I have been unable to find any support for Thomas's observations. In the early stages of extensive lesions the atonia is certainly uniform, no matter what method is relied upon for its demonstration; and even during recovery, as well as in cases of circumscribed injuries, I have been unable to discover that it is ever limited to certain muscles. In my notes of one case, it is true, it is recorded that on the twenty-seventh day "there is perhaps relative hypertonicity of the adductors and inward rotators of the shoulder, and of the supinators," but in both the earlier and later notes it is emphasized that the hypotonia was general and uniform in the affected limbs. If such anisosthenia existed commonly it would probably be most obvious in cases with local and superficial lesions of the cerebellum; but though I have seen many of these I could find no evidence of it in them.

The one common symptom that might be advanced in support of Thomas's thesis is the tendency to deviation of the unsupported limbs, and the frequent error in projection revealed by Bárány's pointing test; but these signs will be dealt with later.

In cases which remained under observation for some time the hypotonia diminished gradually and uniformly, but in all it persisted and was easily demonstrable till the patients were transferred to England. In three cases it was still pronounced seventy-one, seventy-eight, and ninety days respectively after the infliction of the wound. Even in the case in which the injury dated from eight years previously the homolateral arm and leg swung about more inertly, and the patient, who was an educated man, described them as "floppier" than his normal limbs.
§ 2.—Disturbances of Voluntary Movement.

(i) Asthenia, and Slowness in Muscular Contractions and Relaxations.

The flabby toneless limbs of the affected side give an unmistakable impression of feebleness when they are handled or examined, and many patients have spontaneously complained that they are "weaker" or "more useless," or that they "have not nearly so much power in them," as they formerly had, or as those of the opposite side still possessed. This impression is, however, not wholly borne out by examination, for the degree of weakness is always moderate. But there can be no doubt that during the earlier stages of every extensive injury of one side of the cerebellum the strength of all the movements of the limbs of this side is definitely reduced, even when concomitant lesions of the cerebrum and of the chief motor paths can be excluded with certainty. Both the proximal and the distal muscles are affected and as a rule the former appear on the whole relatively weaker. The feebleness is always more pronounced in the arm than in the leg.

This feebleness is usually obvious even when the patient attempts any movement that requires the exertion of power, since more effort is apparently necessary to perform it. This may be seen when he is asked to hold his arms horizontally outstretched or when he tries to raise an object of moderate weight, and the longer the attempt is prolonged the more striking is his difficulty.

When in such cases the strength of various movements, as grasping, flexion and extension of the elbow, flexion of the hip, &c., is tested and compared with that of the corresponding muscles of the opposite side, the difference is usually definite and is approximately proportional in all movements of one limb. This can be easily demonstrated by dynamometric readings. With Dr. Castex's dynamometric apparatus, which he kindly lent me, I obtained the following measurements of the maximal force of several movements of the two sides, in two cases of severe unilateral injury.

<table>
<thead>
<tr>
<th></th>
<th>Case 1 (thirty days)</th>
<th>Case 2 (seventeen days)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unaffected side (R.)</td>
<td>Affected side (L.)</td>
</tr>
<tr>
<td>Grasp</td>
<td>12-4</td>
<td>5-8</td>
</tr>
<tr>
<td>Flexion of elbow</td>
<td>26-2</td>
<td>15-3</td>
</tr>
<tr>
<td>Extension of elbow</td>
<td>11-8</td>
<td>5-2</td>
</tr>
<tr>
<td>Supination</td>
<td>10</td>
<td>5-2</td>
</tr>
<tr>
<td>Pronation</td>
<td>20</td>
<td>14-5</td>
</tr>
<tr>
<td>Extension of ankle</td>
<td>17</td>
<td>15</td>
</tr>
</tbody>
</table>
In Case 1 there was a penetrating wound, from which a piece of shell casing was removed, over the middle of the left lobus gracilis; a considerable amount of softened cerebellar tissue escaped at the operation, and it is probable that the nucleus dentatus was involved. Healing occurred rapidly without any hernia formation.

In Case 2 a large fragment of metal entered over the lateral third of the right lobus gracilis and was removed from the mesial portion of this lateral lobe. There was considerable destruction of cerebellum and a hernia developed, but the patient eventually recovered.

These figures represent the average of several readings for each movement. Case 1 was examined on the thirtieth, Case 2 on the seventeenth day after the infliction of the wound.

The lower records on the affected side may be partly attributed to the difficulty and awkwardness of this hand in holding the instrument; this may be a factor especially when the power of grasping is tested by the ordinary dynamometer, but it can have little influence in estimating the power of other movements by Dr. Castex's apparatus.

When a series of records is made it is also found that the affected limbs tire more quickly than their fellows, and the power exerted by the contracting muscles then diminishes considerably. It is often obvious too, that when the patient is asked to grasp the observer's hand as strongly as possible, the grasp of the affected limb is less continuous and less well maintained than of the other; but this is not always so. It can be even more easily demonstrated when the patient attempts to depress the observer's hand with his extended arm; the exertion of power can be felt to be jerky and more or less intermittent. In other movements, too, a similar irregularity in the exertion of power can be often detected. If the lesion is severe the contraction of the muscles in every action against resistance may be less well maintained.

The sudden unexpected relaxations of contracting muscles that sometimes occur are closely allied to this; in standing, for instance, the leg of the affected side occasionally gives way under the patient, and some men complained that they were afraid to use the affected hand in taking food and in similar actions, as any object they grasped by it was liable to fall suddenly from it. But even when a limb is definitely weak in active movements its static strength—that is, the resistance it can offer to displacements which the patient attempts to resist voluntarily—is generally unaffected. If, for instance, he attempts to keep his elbows or his fingers flexed when the observer tries to extend them, the power necessary to overcome his resistance is approximately the same on the two sides.
Another feature, which is often very striking when the cerebellar injury is severe, is the apparent reluctance of the patient to move the affected arm. This may be frequently seen to lie for long periods in the same attitude, and though he may frequently bring the opposite hand to his face, arrange the bedclothes with it, &c., he more rarely employs the affected one. And when food or any object is offered to him it is almost always with the unaffected limb that he takes it. This may be partly due to the patient's knowledge that he cannot use the affected limb as efficiently or as safely as the other; but it is impossible to observe a patient with a recent cerebellar injury for any time without coming to the conclusion that there is an inherent reluctance to move the limb unless it is necessary. Even if his two hands are pricked by a pin the patient withdraws the affected limb less readily and less briskly than the normal. I could, however, never observe any difference between the reflex withdrawal of the legs of the two sides from a stimulus applied to the soles. In two cases it was noticed that during the early stage of chloroform anaesthesia the patient in struggling moved only or chiefly his normal limbs.

Further, in the early stages of a cerebellar injury there is almost always a marked slowness of the movements of the affected limbs, and especially of the arm, as compared with those of the other side. If a rod is held in front of the patient equally distant from his two hands and he is asked to take hold of it with both, it is found that the movements start more slowly on the affected side, and that this limb seizes the rod an appreciable time after its fellow. Undoubtedly the slowness in reaching the object is partly due to the irregularity of voluntary movement, which is generally known as "ataxia," but this is certainly not its only cause. It may be felt when the observer's hands are placed in the patient's, and he is asked to grasp them, firmly at a given signal; the slowness of the affected limb in starting the action and in developing full power is often unmistakable. It matters not whether the patient attempts such simultaneous actions of the two limbs at the rate he chooses, or whether he tries to execute them as quickly as possible, the affected limb lags behind the other. In the early stages of severe injuries it is in fact difficult to make him hurry in movements of the affected limb.

Even in cases of less severe injury in which there was no obvious loss of power, this slowness in movement is also apparent. It may be partly attributed to the fact that experience has taught the patient that the more rapidly he attempts to make a movement the less well can he
control it; but that this is not the whole explanation can be easily seen by the investigation of simple movements, especially by the graphic method. Fig. 3 reproduces the tracings obtained when a patient with a right-sided lesion was asked to grasp simultaneously at a given signal against two carefully graded and equal steel balance springs. On the signal being given (1 and 1') he started immediately with his left hand, A, and attained full power in about five-tenths of a second; but in his right hand, A', there was a delay of two-tenths of a second in starting the contraction, and the exertion of power did not attain its maximum till one second later. Further, the power exerted was little more than one-half that developed by the left hand.

When the signal (2 and 2') to relax was given, his left hand opened at once and the tracing, B, quickly reached the base line, but on the right side relaxation did not commence till one-seventh of a second later, and the fall of the tracing to the base line was much more gradual even...
though the extension of the fingers was aided by the tension of the extended spring. The same features may be seen in the tracing reproduced in fig. 4; here the right hand, which was affected, was less slow in initiating contraction, but the full power of grasp was only attained after six-tenths of a second, and its force was only half that of the left hand. There was also an appreciable delay in commencing relaxation and a definite slowness in its completion.

In fig. 5 the same condition is seen when a third patient with a right-sided lesion attempted to flex his elbows simultaneously against resistance, and later relax on a given signal; in this case delay in initiating, and slowness in effecting, relaxation were the most prominent features.

The same facts may be easily observed in other movements of the affected limbs in the majority of, if not in all, patients with severe and moderate unilateral injuries. When, however, corresponding muscles of the two limbs are stimulated simultaneously by a faradic current no slowness in either their contractions or relaxations can be detected, (fig. 6); and there is no difference in the latent periods of the contractions of the muscles when these are excited reflexly.

We consequently find, (1) a delay in initiating muscular contrac-
Fig. 5.—From a case of injury of the right lateral lobe and probably also of the vermis of the cerebellum, six weeks after the infliction of the wound. Tracings, A and A', of an attempt to flex his two elbows simultaneously against the resistance of two equal springs, and later to relax them simultaneously on a given signal. 1 and 1' and 2 and 2' represent B and B', the simultaneous ordinates. Time in one-tenths of a second.

Fig. 6.—Tracings obtained from tambours placed on the tendons of the flexors of the fingers of the right and left hands when these were stimulated simultaneously by a faradic current, in a patient with an extensive right-sided cerebellar injury. There was in this patient a considerable delay in the initiation of both voluntary contraction and relaxation of these muscles. A and A' represent the corresponding ordinates. Time in fifths of a second.
tions; (2) a slowness in attaining the exertion of the full power; (3) a delay in commencing relaxation; and (4) a slowness in effecting relaxation, among the symptoms produced by cerebellar lesions. As a rule, there is delay and slowness in both contraction and relaxation, but occasionally only contraction, or more commonly relaxation, is slower than it is in the normal limb; a slowness in starting relaxation is probably the most common.

The affected limb also tires more rapidly than the normal, especially in movements that demand power and in those made against resistance. This can be well seen if the patient is made to hold his arms outstretched horizontally in front of him, or to keep his legs elevated from the bed. At first the affected limb is usually held very steadily, but it tires rapidly, and then a coarse jerky tremor often appears, owing
chiefly to his voluntary attempts to regain the position from which the
tired limb gradually falls away. This tendency of the affected limb to
tire more rapidly can be seen in most ergographic tracings. In these
the range of movement is also generally smaller and more irregular,
while its rate is slower. Occasionally, however, as in fig. 7, the most
striking feature is due to the fact that when the patient attempts
to perform such movements against resistance as quickly as he can
a second contraction often occurs before the previous relaxation is
complete, and consequently in many excursions the tracing does not
reach the base line. In this patient there was an obvious delay and
slowness in starting and in completing the relaxation of contracting
muscles.

The weakness of the limbs of the affected side is always most evident
in the early stages of the cerebellar injury and diminishes gradually.
In several of the cases in which the lesion was small or moderate in
size no difference could be detected in the power of the homologous
limbs after two to three weeks, but in more severe injuries the paresis
is more persistent. In the two cases referred to above in which
dynamometric readings were made, it was still obvious in the upper
limbs eighty and seventy days respectively after the infliction of the
wound, but the lower limbs were then equally strong. Even when
their strength had recovered the affected limbs still remain appreciably
slower in movement than the normal.

This form of weakness due to cerebellar wounds is distinguished
from that produced by lesions of the cortical motor area or of the
cortico-spinal tracts by its homolaterality, by the uniform and the
approximately equal affection of all groups of muscles, by the fact that
though all voluntary movements are weak none are limited in range,
by the absence of the characteristic alterations in the reflexes, and by
the fact that there is no tendency for rigidity or contractures to
develop. The term "asthenia" introduced by Luciani is consequently
more suitable for it than paresis.

(ii) *Ataxia.*

But the most commonly recognized sign of cerebellar disease is that
irregularity in voluntary movement which is generally loosely described
under the term "ataxia," though physiologists and certain clinicians
have tried to analyse it into simpler components.

This disturbance of voluntary movement may be most easily studied
separately, as it occurs in the early and in the later stages of extensive
THE: SYMPTOMS OF ACUTE CEREBELLAR INJURIES

lesions, since in the latter period the effects of cerebellar deficiency are complicated by the results of "compensation" by other parts of the nervous system, and by the voluntary efforts by which the patient attempts to control his disability.

In the early stages all movements of the limbs are affected, but the disturbance is more striking in the upper than in the lower extremity, and in complex than in simple movements. It must be emphasized that the control of movements by vision has no influence on their accuracy, and that they are quite as "ataxic" when the patient's eyes are open as when they are closed.

Let us assume we have a patient with a recent severe lesion of the right half of his cerebellum. When he is asked to touch an object with his left forefinger he does so promptly and accurately, and to reach it moves his hand in the most direct line practicable from the position in which it lay.

He raises his right hand from the bed less promptly, and as soon as the limb is unsupported it sways unnecessarily and aimlessly at its more prominal joints, and during the movement his finger deviates from the direct course by which it could most easily reach its aim. Further, he rarely succeeds in touching the object at once, but usually brings his index to one or other side of it, and projects it too far or more rarely stops the movement too soon. Errors consequently occur in both the direction and in the range of its movement. If his arm is fixed so that it is necessary for him to move only his forearm and hand, the irregularity and inaccuracy of the movement is less pronounced.

Similarly, if when his arm is extended vertically or held outstretched in front of him, he is asked to bring his index finger to his nose, it is seen that his finger does not take the direct and shortest course, and instead of coming accurately to his nose it often strikes his chest, chin or forehead. Further, it is often brought to his face with undue force.

If an attempt is made to obtain from the patient his explanation of this irregularity of his movements he merely describes, if he happens to be intelligent, that which the observer has already noticed. One patient said: "I do not seem to have the power to do what I want to with my hand, though if I take hold of anything with it I can grip it all right. If I want to bring it to my mouth I only hit my eye with it; it is drunk; it will not go straight." Another man described his disability by the statement: "I do not feel that I can get the right direction with this arm"; while a third patient when examined in a
This irregularity in direction and range is always more marked in rapid than in slow movements; in fact when the patient is asked to execute the movement slowly, or is urged not to hurry, there may be little or no obvious disturbance. Further, simple actions which require movement at one joint only may be fairly accurately performed, though complex actions are irregular and ataxic; the patient can succeed much better in touching with either his finger or toe an object that he can reach with the limb extended than when movements at the elbow, knee or other joints are also necessary.

The movements of all segments of the limbs are generally affected, and those of the distal joints are in fact usually the most seriously disturbed. If, when the patient’s eyes are closed, an unknown object is placed in his hands and he is asked to identify it, it may be seen that he handles it and moves his fingers over it irregularly and awkwardly; often he only grasps it firmly and rubs it between his fingers and thumb. The affection of his fingers is better demonstrated and more easily analysed if he is asked to bring the tip of each finger in succession to the top of his thumb; then he frequently fails to place finger and thumb in correct apposition, the finger often slides along the side of his thumb, and the relative degree of movement at the different joints of the finger are not so regular or appropriate as in the normal hand; sometimes only the metacarpo-phalangeal joint is flexed, or the bending of the interphalangeal joints may be excessive. The rate of movement, too, is not uniform, but is often jerky and intermittent. Further, while in the normal hand each finger is flexed separately, on the affected side all fingers are frequently flexed at the same time, though the patient wishes to bring one finger only to his thumb.

As a rule both the fingers and the thumb are moved, but many patients employ only the finger in the action and fail to oppose and flex the thumb when they wish to bring its point into apposition with the ulnar fingers.

When this disturbance of movement is more carefully examined several factors can be distinguished in it. These may be most easily considered separately, though the relative prominence of each may vary in different cases and even in different stages of the one case.

(a) Decomposition of movement.—If we again ask the patient as he lies in bed to extend his arm vertically over his face and then bring his
forefinger to his nose, it is seen that instead of depressing his arm and flexing his elbow simultaneously, he first brings his elbow towards the bed and only when it is there or near it does he approach his finger to his nose by flexing his elbow. In other words, he "decomposes" the movement into its separate elements (Babinski). This may be seen in various other actions of both the upper and lower limbs. One patient, for instance, when asked to place the heel of his affected side on his opposite knee always dragged the heel along the bed till it reached the knee and then only raised it to the proper height; other patients on the contrary have raised the heel unnecessarily before beginning to flex the knee. Or if the patient be asked, as he lies on his back, to bring his heel to his buttock, he may do it in two stages: first flexing his hip and only later flexing his knee. Similarly, if when able to leave the bed he attempts to place his foot on a chair, he frequently raises the foot too high by flexion of his hip before he bends his knee.

In other words, the patient tends to perform the separate movements that constitute an act "by numbers," as in a gymnasium or in military drill. This is the condition which Babinski has termed asynergia; he has defined it as "the inability to accomplish simultaneously the various movements that constitute an act."

The question arises whether this decomposition of movement is voluntary or not. I know of no test that can settle this point directly, but the fact that it may be observed in its extremest form when a patient, who is in a dull and stupid state on recovering from the early general effects of a wound, or after an operation which has entailed further damage of the cerebellum, first makes purposive movements, suggests strongly that it is not always a willed device by which he attempts to diminish the inaccuracy of his limbs.

(b) Asynergia.—The term asynergia can be more correctly restricted to the absence or disturbance of that proper synergic association in the contraction of agonists, antagonists and fixating muscles, which assures that the different components of an act follow in proper sequence, at the proper moment and are of the proper degree, so that the act is executed accurately and with the least possible expenditure of energy.

When the fingers are flexed the extensors of the wrist normally contract synergically with appropriate force in order to prevent simultaneous flexion of the wrist, but if a patient with a cerebellar lesion grasps a small object quickly it often happens that the wrist of the affected side is extended excessively or too early, so that the hand is bent backwards when the fingers are but half flexed: the normal
co-operation of the contracting muscles, on which the accuracy and precision of the action depends, is consequently disturbed. This can be often seen more definitely when the patient flexes and extends his fingers rapidly; then the wrist often flexes as he brings his fingers into his palm, or extends too much as he straightens them, or the finger movements and the synergic wrist movements may be partly dissociated or inaccurately combined in time or in degree.

A disturbance of synergia, which should keep the legs extended and thus bring the centre of gravity of the body as near the pelvis as possible, is also the explanation of the fact that in attempting to rise from the supine position by flexion of the trunk upon the hips the heel of the affected side often rises from the bed (Babinski). Frequently, however, only the knee is raised by flexion of the hip on the trunk and the heel remains on the bed owing to failure of the normally associated contraction of the quadriceps extensor. Babinski has also shown that the cerebellar patient, when standing, often falls when he throws his head backwards, owing to his failure to bring his centre of gravity over his base by flexing his knees, and I have observed this in several cases. When asked to sit down on a low bed or stool he is also liable to fall backwards, as he again fails to adjust his balance by flexing his trunk on his hips. Other examples are seen in walking, as the affected arm generally hangs inertly by his side and does not swing forwards when he advances his opposite foot; and in the failure of his head and eyes to move simultaneously when he looks to one or the other side.

In the majority of such movements the contracting muscles (agonists) have to displace the limb or some of its segments against gravity, and their antagonists only relax reciprocally; but in other actions, as when the elbow is fixed above his head and the patient brings his finger down to his nose, that is in the direction in which its weight would move it, the most important factor is the gradual relaxation of muscles which are the antagonists of the flexors of the elbow. The disturbance in the co-ordinated activity of agonists and antagonists suggested comparison of movements executed with and those performed against gravity, but though this was investigated in certain cases, as by making the patient bring his finger successively up to and down to his nose by merely bending his elbow, no significant difference could be detected in the characters of the movements.

(c) Dysmetria.—Another striking abnormality in the affected limbs is the fact that the range and the force of their movements are not correctly adapted or proportioned to their aim; they are ill-measured
or dysmetric (Luciani). In attempting to touch a point, for instance, the finger-tip often shoots past it, or more rarely the movement is arrested before it reaches it. And in bringing his finger to his nose, the patient may either strike his face too forcibly, or arrest the movement momentarily before he reaches it. Similarly, when asked to grasp an object, he throws his arm forward with excessive force, opens his hand unnecessarily widely, and finally seizes it too roughly and too forcibly. In other words, neither the range nor the force of the movement is accurately adapted to its aim.

This is particularly prominent in quick movements; when the movement is slow, and can be closely watched by the patient, it is more correctly graded, but even in slow and deliberate actions, as in carrying out Bárány's pointing test, dysmetria may be seen. Often, too, as he makes a deliberate attempt to touch his nose, he allows his affected hand to fall heavily against his face, as though he were too lazy or careless to pull it up.

It is noteworthy that the range of movement is most commonly excessive, as has been pointed out by other observers (Babinski, André-Thomas); but in the early stages of acute cerebellar lesions it happens frequently that the movement is arrested or slowed down before the point the patient wishes to attain is reached, and the hand or foot is then brought to it by a series of slow deliberate jerks. In a few cases the most striking abnormality when the patient tried to take hold of an object was the tendency of the affected hand to stop short of it, pause for a moment, and then swoop down on it with excessive force. In other actions, too, as when he tries to bring the tips of his fingers successively to the top of his thumb, the range of movement is often too small rather than exaggerated.

This dysmetria is particularly pronounced in attempts to perform accurate movements against resistance, as when he tries to raise a moderate weight, or to release his finger from the observer's grasp and bring it directly to the tip of his nose. It is under such conditions that the difficulty in arresting a movement that was correctly initiated is most obvious. These facts suggest that dysmetria may bear a relation to the symptoms described under the "rebound phenomenon."

(d) Tremor.—Voluntary movement is also often complicated and disturbed by the occurrence of tremor in the moving limb, but this is not such a prominent factor in the early as it is in the later stages of a cerebellar lesion. In the notes on many of my more serious cases it is stated definitely that there was no tremor during the movements of the limbs.
Frequently, however, and especially in quick movements, the finger or toe is approached at a fairly uniform rate to within a few inches of the point the patient wishes to touch, and is thence brought to it by a series of irregular brusque jerks, which in some degree resemble those of ordinary intention-tremor, though the pauses and interruptions of movement are usually less abrupt, and the projections more deliberate and larger in range. And even when the limb reaches the object for a time it is not held steadily in contact with it; after the patient has brought his finger to his nose, for instance, the tip of the latter is often intermittently depressed or displaced to one side or the other by the finger.

The coarser forms of tremor may be associated with dysmetria, the movement being arrested too soon, and the limb then approached to the object by a series of jerks; or it may be due to voluntary arrest of the primary movement and the subsequent attempts of the ataxic limb to reach its aim. It cannot be attributed wholly to these causes. When simple movements, as flexion or extension of the forearm or leg, are carefully observed, it may be often seen that the limb is moved in a series of irregular jerks, and that the tremulousness usually increases towards its completion when accuracy is most essential. It is occasionally possible by palpation of the muscles to detect an irregularity or discontinuity in their contractions. Occasionally, however, the movement seems to be interrupted by irregular intervention of the antagonists.

Consequently, even from its commencement the movement is often irregular, and throughout its whole range it lacks the continuity and uniformity in rate which distinguish ordinary voluntary displacements of a limb.

(e) Deviations from the line of movement.—It has been already stated that the irregularity of movement in the early stages of a cerebellar lesion are partly due to the fact that the limb deviates from the direct line, and is not moved along the shortest course that it should naturally take; but this symptom needs more emphasis, as it has been overlooked by many clinicians who have studied chiefly the later effects of cerebellar injuries, or the symptoms of atrophic or degenerative diseases of this organ.

This deviation in movement was observed by Grainger Stewart and myself after the removal of tumours and surgical damage of the cerebellum, and I have seen it frequently since in similar cases. It is equally obvious in recent gunshot injuries. In the early part of the movement the limb sways about in a purposeless manner as soon as it
is raised from its support; during the movement it deviates from its proper course, and towards its completion it does not come straight to the object it should touch or seize; in trying to touch his nose, his finger, for instance, often comes to his cheek or eye. One patient declared that when he attempted to feed himself with the affected hand it was frequently to his ear that he brought the food, and another that he could not use this hand in smoking, as he was afraid of putting the cigarette into his eye. This error cannot be attributed to dysmetria, or to properly orientated movements which are executed with ill-graded force, as the hand which the patient wishes to bring to his nose often comes to his cheek or eye even when it is moved from directly in front of his face.

The slow swaying at the commencement of the movement is most pronounced when the limb is atonic, and it seems to be directly associated with loss of tone in the muscles that should fix its proximal joints; it often suggests an asthenic or paretic condition. But the deviation from the direct course during the movement and the failure to bring his hand or foot to the correct point, must be attributed to disturbance in the regulation of the force and sequence of the contractions of the different muscles employed in the act. Decomposition of a movement naturally produces errors in its direction, and a similar disturbance in the co-operation of the various muscles, even though it does not cause obvious decomposition, may lead to deviations from the correct direction.

It is an interesting question whether there is any constancy in the direction in which the limb deviates during and at the completion of the movement, and if so what its relation is to the direction in which it tends to deviate when unsupported and in Bárány's pointing test. No regularity in the direction of the deviation can, however, be observed in early cases, though later the frequent tendency to spontaneous deviation often influences the direction of the error. One man, twenty months after the infliction of a cerebellar wound but who now presented symptoms of disseminated sclerosis as well, in attempting to touch his nose when his eyes were closed always brought his forefinger to that side of it towards which his arm tended to deviate in Bárány's test, and the same fact was observed in another patient three months after he had been wounded. It is in such cases that Babinski rightly contrasts the failure of the cerebellar patient with that of the tabetic in attaining his aim.

After the first few weeks, according to the nature and the severity
of the wound, these disturbances of voluntary movement begin to alter in character. When the patient raises his arm or leg the limb sways about less aimlessly, in movement its deviations from the correct direction are less obvious, and it generally reaches its aim more directly.

At the same time the tendency to decompose or "two-stage" movements is less common in simple actions, as in bringing his finger to his nose, though it can still be detected in more complex actions. The disturbances in the co-ordination of agonists, antagonists and synergetic muscles also diminish; in closing his fingers quickly there is less tendency to over-extension or to flexion of the wrist, and the wrist and finger movements are more correctly associated when he rapidly extends or flexes the latter.

On the other hand, the deficient regulation of the range and force of movements, which has been described as dysmetria, persists or may be even more striking now. The arm is flung out from the shoulder and the hand tends to grasp every object with too much force. At this stage the range of movements is much more frequently excessive than short of their aim; dysmetria therefore generally takes the form of hypermetria. Movements consequently tend to be performed too abruptly and too forcibly, though they still start more slowly than those of the normal limb.

Now, however, voluntary correction begins to play a greater part; when the patient's movements and actions are carefully watched it may easily be seen that he tries to correct his errors. If on one occasion he overshoots his mark, in subsequent attempts he may stop short of it. The patient who stated that he could not pull up the affected arm quickly enough now began to arrest his finger short of his nose when he attempted to touch it, and then brought it in contact with it by a series of deliberate coarse jerks; and if the arm at first tended to strike his cheek or eye when the patient tried to bring his finger to his nose he now stops before he reaches his face and completes the action in the same intermittent, jerky fashion.

An irregularity in movement resembling intention tremor is consequently a prominent feature at this stage. This cannot however be attributed wholly to the intervention of voluntary effort in an attempt to control the errors, as even the jerks are irregular and inappropriate in range and force, though it is usually more pronounced the more rapidly the movement is performed and consequently the less opportunity the patient has to correct his errors voluntarily.

But in order to understand more fully these disturbances of voluntary
movement it is necessary to investigate the symptoms included here in
the rebound phenomenon, and to observe the affected limbs as the
patient attempts to perform alternate movements rapidly with them.

(iii) The Rebound Phenomenon.

Several years ago in a paper written in collaboration, Grainger
Stewart and I pointed out a sign of cerebellar involvement that is,
of considerable importance in the interpretation of the disturbances of
voluntary movement produced by this condition. The patient's elbows
are supported on a bed or table and he is asked to pull each hand in
succession towards his mouth against resistance offered by the observer
who grasps his wrists; when this resistance is suddenly released the
hand of the affected side flies to his mouth or shoulder, often with con-
siderable violence, but the movement of the normal limb is arrested
almost immediately by contraction of the antagonists (triceps), and
may even be jerked back or rebound. The rebound is excessive in
spastic limbs.

The absence of this rebound may be demonstrated in various other
actions, as extension of the elbow, elevation or depression of the arm at
the shoulder, flexion or extension of the hip and extension of the knee.
It occurs only on the same side as the lesion when this is unilateral,
but it is seen on both sides when the wound is bilateral. It is only
necessary to resist any movement which the patient attempts to
perform and remove the resistance suddenly; then the limb or the
segment of the limb which he wishes to move swings unchecked or
excessively in the direction of the movement attempted and is not
quickly arrested as the normal invariably is. If a hand is placed on
the antagonistic muscles it is found that these do not contract, or
come into contraction too late.

The occurrence of this "rebound phenomenon" in lesions of the
cerebellum has been confirmed by other observers (Bing, André-Thomas)
and it was easily demonstrable in all cases of my present series in which
there were moderate or severe lesions. As a rule it was equally
pronounced at all joints, though it was usually more definite in the
upper than in the lower limb, and more easily evoked at the proximal
than at the distal joints. It often happens that it becomes less easy to
demonstrate after it has been tested for several times, owing to a
voluntary effort on the part of the patient to suppress it, especially if
the unchecked movement may produce pain or discomfort.

A similar inability to arrest passive movement quickly may be seen
if the observer supports the elbow with one of his hands, while with his other he first moves the patient’s forearm about aimlessly and then unexpectedly throws the hand towards his face. The passive flexion of the normal arm is quickly checked, but the affected hand flies, often violently, into the patient’s face, just as a flaccid paralysed limb or a loosely-hinged rod would.

Further, if when his elbows are supported, the patient attempts to flex and extend his forearms alternately as quickly as possible the range of movement on the affected side is often excessive, and more than one patient has complained that he has actually jarred or hurt his elbow-joint by the excessive flexion, which is arrested only by the structure of the joint or by its ligaments, and not by contraction of the antagonistic muscles.

This phenomenon is always most pronounced in the early and acute stages of a cerebellar injury and diminishes gradually as improvement sets in. It is consequently more prominent when the limbs are hypotonic, but its relation to hypotonia will be discussed later.

(iv) Adiadochokinesis.

Babinski originally pointed out that a patient with cerebellar disease is almost always unable to execute alternate movements as quickly and correctly as the normal person, and he termed this symptom adiadochokinesis. It may be tested at any joint at which such movements can be easily and rapidly performed, as flexion and extension, or pronation and supination of the elbow, or flexion and extension of the fingers, ankles, or toes.

When a patient with a unilateral cerebellar injury is asked to pronate and supinate his forearms alternately and as rapidly as he can a very striking difference is noticed between the movements on the two sides, as the rate of the alternate movements of the homolateral limb is slower and much less regular, their range is less uniform, and both the slowness and the irregularity in rate and range become more pronounced the longer the effort is continued. In slight injuries the awkwardness of the attempt and the apparently greater effort necessary for it on the patient’s part are more striking than the slowness of its execution, and in very limited lesions or when recovery sets in this awkwardness and incompleteness only may be observable.

Further, while the other joints of the normal limb remain fixed, on the affected side various adventitious movements occur, as irregular flexion or extension of the fingers and more particularly flexion and
extension of the elbow, or adduction and abduction at the shoulder. These are generally obvious at once, but if not pronounced the observer by placing his hands under the elbows can feel that while that of the unaffected arm remains steady, the other is jerked about irregularly. Similar adventitious movements at the wrist and elbow can be often observed when rapid flexion and extension of the fingers are attempted, and in the knee and ankle when he is asked to move his toes only.

If the limb is hypotonic part of these adventitious movements may be attributed to flail-like flopping of its distal segments; when for instance the elbow is rapidly flexed and extended the hand swings about inertly, flexing as the arm is brought towards the shoulder and extending as the elbow is rapidly straightened; and in the same test the elbow is often raised from its support by the momentum of the flexing arm. They depend partly therefore on the failure of the fixing muscles to preserve the correct posture of those segments of the limb that should not move; this failure is associated with their loss of tone.

But there can be no doubt that they are largely due to the active contractions of muscles that are not directly concerned in the act desired by the patient; this can be easily determined if the arm and shoulder muscles are carefully palpated while rapid pronation and supination of the forearm is attempted; and if a hand is placed on the thigh as the patient flexes and extends his toes rapidly the irregular contractions of its muscles, which produce movement of the knee, can be felt. There is consequently a tendency to an irregular spread of the innervation to other muscles than those which under normal conditions execute the act. When the test is prolonged the movements which the patient wishes to perform may be in fact largely replaced by irregular aimless displacements of other segments of the limb.

Occasionally even the active muscles do not work together accurately and harmoniously, and this increases the awkwardness and irregularity of the patient's movements; in rapidly flexing and extending the fingers for instance, all these at first move together as they invariably do in the normal limb, but after a time they frequently get out of line and cease to flex and extend simultaneously or to the same extent. I have even seen the thumb bent involuntarily into the hand and thus interfere with the excursions of the fingers.

The slowness and irregularity can be equally well seen in more complex actions, as in shaking hands, clapping hands, rubbing a mark off a sheet of paper, stamping his feet, &c. When a patient attempts
to clap hands both arms generally move at first, but the affected one soon comes to a standstill and is then only passively displaced by the blows of the normal hand.

The causes of this disturbance can be more easily analysed if some simple action, as tapping a table or the observer's hand, is carefully examined. In the first place the range of movement is irregular; his hand is occasionally raised too high or brought down too firmly, but more commonly it is smaller than that of his normal limb. Often indeed the movement is arrested too soon and the hand may not actually come to the table. The slowness is due chiefly to delay at the turn and not to time lost in the movements themselves, for there is rarely much difference in the rate of these and of those of the normal limb.

The principal features of adiadochokinesis may be seen in figs. 8 and 9. Fig. 8 reproduces tracings of alternate pronation and supination of the forearms, in a patient with a right-sided lesion in whom this symptom was rapidly disappearing. The range of the excursions of the affected (right) arm was fairly regular, though on one occasion the movement was checked in semi-pronation, but it was considerably smaller than that of the normal limb; the slower rate was due chiefly to time lost at the turn and to slowness in the initiation of each sequence of muscular contractions. In fig. 9 tracings obtained from rapid flexion and extension of the fingers are reproduced; here the same features are present, but the delay at the turn of the movement, and especially in extension of the fingers, is more obvious.

The occurrence of adventitious movements certainly contributes to the awkwardness and slowness, since they confuse the patient in his efforts, and often put the contracting muscles at a mechanical disadvantage.

We can consequently conclude that the condition described by Babinski as adiadochokinesis is due to disturbances of the range of movement and to a slowness in initiating each excursion of the limb, while the occurrence of adventitious movements is partly due to lack of proper co-operation on the part of those muscles which should fix other joints of the limb, and partly to irregular purposeless contractions of other groups of muscles.

Frequently such alternate movements can be better performed by the affected limb when it is tested alone than when the homologous limb is examined simultaneously. The normal person generally finds it easier, or at least as easy, to perform such rapid alternate movements
Fig. 8.—Adiadochokinesis. Tracings of alternate pronation and supination of the forearms performed as rapidly as possible by the patient, in a man five weeks after the onset of a moderately severe right-sided lesion.

Fig. 9.—Adiadochokinesis. Tracings of alternate flexion and extension of the fingers performed in succession as rapidly as possible by the patient; flexion is represented by the downward lines, extension by the upward. From a man forty-one days after the infliction of a severe right-sided cerebellar wound.
simultaneously with his right and left limbs as with either separately, but when a unilateral cerebellar lesion exists the more rapid movements of the normal limb seem to confuse those of the affected one and disturb their sequence. The patient has, consequently, to attempt two different actions at once instead of the same action simultaneously with his right and left limbs.

Adiadochokinesis can usually be demonstrated at all joints of the affected limbs, even when only limited lesions exist, but it is usually more pronounced in the arm than in the leg and in complex than in simple actions. During recovery the slowness, irregularity and awkwardness diminish gradually, but adiadochokinesis is one of the most persistent of the ordinary signs of cerebellar injury.

The majority of the abnormalities of movement described above refer to those in which the larger and more proximal groups of muscles are concerned, but similar disturbances can be observed in the finer and more elaborate actions of the hand which are peculiar to the anthropoid and especially to man. The slowness, awkwardness and irregularity of the finger movements in handling objects, and the difficulty in bringing each finger of the affected hand separately and accurately to the tip of the thumb have been described above, but these defects are even more apparent when the patient attempts to use simple and familiar tools. When a man with a right-sided cerebellar lesion was given a pair of scissors he had, in the first place, difficulty in grasping them correctly, then failed to direct them properly and was unable to move the blades regularly and appropriately when he attempted to cut a piece of paper with them.

In writing, too, these disturbances are very obvious when the wound involves the right half of the cerebellum. The pencil is held incorrectly and insecurely, grasped too tightly, and its point is pressed much too firmly on the paper. The letters are frequently unequal in size and irregularly spaced; the individual letters are badly formed and their lines are often jerky and angular. Further, as he writes it can be seen that he pauses frequently, especially between the up and down strokes, and the movements of his pencil are consequently interrupted and discontinuous. If he uses a pen he is liable to run its point through the paper and make ink splashes; his attempt with it is therefore less successful (fig. 10).
§ 3.—Static Tremor.

I have never observed tremor while the limbs and body were at rest and fully supported, but it often appears during the maintenance of posture that requires muscular contractions. That which occurs during movement has been already described as a component of "cerebellar ataxia."

When a patient with a unilateral cerebellar lesion sits or stands there is frequently tremor of his head, which usually consists of fine or moderate irregular oscillations in any or all directions; it increases as he becomes tired. Similarly, when standing his whole body is often displaced by larger and more irregular swaying movements. This unsteadiness seems to be due to irregular and discontinuous contractions of the muscles that should maintain the attitude.

![Writing specimens](image)

Fig. 10.—Specimens of the writing of two men with right-sided cerebellar injuries. The upper is of the words, "Raymond, yesterday"; the lower, "I have been eight weeks ill."

Tremor is as a rule seen in the affected limbs only as they tire, which they do more rapidly than the normal, or when he strives to adopt a posture. When an attitude is once attained, as when his arms are outstretched horizontally in front of him, or his legs raised from the couch and flexed to right angles at the hip and knee, the affected limb is usually held for a time as steady as its fellow, and frequently even more so. It is noteworthy that the fine vibratory tremor which can often be seen in the normal arm, and even more distinctly felt, when it is held rigidly extended and unsupported, is generally absent in the affected limb; in one case its absence was so obvious that the photographer who exposed a plate as the patient held his arms in this position.
remarked how much steadier the affected one was. This condition corresponds to that which Babinski has described as *cerebellar catalepsy*, or the property of limbs remaining for a long time immobile under the influence of will; but otherwise I have seen no evidence of this symptom in the earlier stages of cerebellar lesions. When, on the contrary, a limb begins to tire, and especially if it is still asthenic, a coarse tremor often develops, which is partly due to failure of sustained contraction of the fatigued muscles, but largely to the repeated voluntary attempts of the patient to bring it back to the position from which it tends to fall away. The tremor which occurs in these conditions is comparable to nystagmus and consists of two phases, namely, slow displacements by gravity and quicker voluntary jerks back towards the original position.

Before describing the abnormalities of standing and gait produced by unilateral cerebellar lesions, it is advisable to consider other symptoms which influence them. These are particularly the attitude assumed by the patient and the occurrence of vertigo.

§ 4.—Vertigo.

Many text-books include vertigo among the symptoms of cerebellar disease, but much doubt has been thrown on its direct relationship to cerebellar lesions; in fact, most authorities attribute it to affection of the labyrinth, or of the vestibular nerve or its nuclei.

Giddiness is an extremely common symptom after all gunshot injuries of the head, and almost all patients in whom the cerebellum is involved state on questioning that they became giddy immediately or after regaining consciousness. From many of these no accurate description of the sensation could be obtained; but this is not surprising as most men are dull, obfuscated and unobservant for some time after being wounded in the head. Seventeen men, however, described apparent displacement of self, or of external objects, or of both. In twelve of these self and the external world seemed to be simultaneously displaced; in three there was apparent movement of self only, and in two solely of the external world. In fourteen of these seventeen cases the apparent movement was rotation around the longitudinal axis of the patient's body; in two it was in the vertical plane; while one man stated that the walls of the room in which he lay seemed to be receding from him.

Among these fourteen patients the same difficulty was experienced in attempting to determine definitely the direction of the rotation, since the vertigo rarely persisted more than one or two days. In almost all
the subjective rotation of self and the movement of the outer world were in the same direction, but no constant relation could be discovered between the direction of the rotation and the site of the injury. In a series of cases of cerebellar tumour Grainger Stewart and I found that when vertigo occurred in patients with unilateral lesions the sense of movement of self and of external objects was from the side of the lesion towards the opposite side; but my present series, in as far as one can rely on the statements of the patients, does not confirm this rule.

It is interesting, however, that in the only three cases in which vertigo associated with actual movement occurred under observation, the patients rotated from the affected towards the opposite side (i.e., the shoulder of the affected side moved forwards) and the apparent movement of the external world was in the same direction. In the early stages of other cases, there was a noticeable tendency for the patients to lie on the affected side. Some expressed considerable reluctance to rest in any other position, though one man stated that if he fell asleep lying on this side he was liable to roll over on his face.

We can at least say that when "forced movements" were observed they took place in the same direction as they do in animals after ablation of one half of the cerebellum.

Further, the common attitude of a patient with a unilateral cerebellar injury, his head rotated towards the opposite side and the homolateral shoulder in advance of its fellow, may be looked upon as a latent tendency to rotation in this direction.

In the two cases in which during vertiginous attacks a sensation of vertical displacement only occurred, external objects seemed to arise in front of the patients, and one man complained that simultaneously his head seemed to be sinking back through the pillow. In both of these men the wound was over the middle of the posterior surface of one lateral lobe, which was also much the most common site in the series as a whole.

§ 5—Spontaneous Deviation of the Limbs and Bárány's Pointing Test.

These signs which Bárány has described, especially in connection with labyrinthine vertigo, may be dealt with here.

In order to examine for spontaneous deviation of the upper limbs the patient is asked to hold both arms extended horizontally in front of him and close his eyes. It is advisable for the observer to steady his hands and check any tendency to movement till his eyes are closed.
Then in the majority of cases with unilateral lesions the homolateral arm swings either slowly or abruptly away from the symmetrical position and comes to rest gradually. This occurred in all the thirty-three cases in which it was looked for. In thirty-two the arm swung outwards, and in one slightly inwards, but the latter case, in which one lateral lobe of the cerebellum was almost completely destroyed and death occurred on the third day, may be neglected as the arm was so asthenic that it could scarcely be raised. We may consequently assume that the involuntary deviation of the unsupported arm is almost constantly outward.

While the observer's hands are steadying the patient's arms he can often feel a definite tendency to active movement of the homolateral arm in this direction.

When in slight injuries this deviation is not obvious, it may be brought out by shaking or tapping the affected arm; this then generally moves in the direction towards which it tends to deviate.

Frequently the arm also deviates upwards or downwards from the level in which it was originally placed.

Bárány's pointing test confirms these observations. The patient's extended forefinger is placed in contact with some fixed object, as the observer's finger, which is held at some distance above the bed, and he is then asked, while his eyes are closed, to bring his finger down to the bed and slowly up again to the object; with his normal arm he can reach the mark accurately or approximately on each attempt, but in most cases the forefinger of the affected side deviates constantly to one side of it, and this deviation increases for a time if the test is continued. In order to avoid conscious efforts to correct the error it is advisable for the observer to allow the moving finger to strike a similar object that can be moved into contact with it; if a tape measure is held horizontally in such a position that the patient's finger must touch it the error in each movement can be easily measured. If the more proximal segments of the limb are fixed the tendency to deviation of its distal segments can be investigated in the same manner. In the early stages of an extensive injury, the homolateral limbs are often so asthenic and ataxic that the test cannot be easily employed.

In all the thirty-two cases of unilateral lesions, involving different parts of the cerebellum, in which the arms were tested, a tendency to deviation was observed in the limbs of the affected side, and in all the deviation was outwards from the vertical when the arm was moved at the shoulder only. In certain cases vertical movement at the elbow
and wrist were also examined, and in these, too, the deviation was constantly outwards, though the error here was much smaller.

In a few patients who suffered with extensive unilateral lesions the contralateral arm tended to deviate inwards, but the deviation was always slight, and neither constant nor regular.

In twenty-seven cases movements in the horizontal plane were similarly investigated; in nine progressive deviation upwards occurred, in seven the error was downwards, in six there was no tendency to deviation, and in the other five the direction of the error was irregular. The error in horizontal movements is not, however, always constant; in a few cases it was on one occasion upward and on another downward.

When this test is first seen these errors towards a fixed point in movements that are not controlled by vision suggest strongly a disturbance of the sense of position or of the appreciation of movement ("muscle sense"), in the affected limb, but it can be easily demonstrated that this does not exist; the patient can, for instance, while his eyes are still closed, bring his other forefinger accurately and promptly to the tip of that one which is actually several centimetres distant from the point he wishes to touch by it, showing that he is fully aware of its position in space. This can be confirmed by other tests too.

A tendency to deviation of the lower limbs is less common and the error is usually less pronounced when it exists. It is remarkable, however, that while the affected arm constantly, or almost constantly deviates outwards, the homolateral leg generally swings inwards (adducts), or is raised obliquely upwards and inwards over the opposite limb. In fifteen cases the pointing test was applied in vertical movements; in four there was no appreciable error, in two the leg deviated outwards, and in nine progressively inwards.

Another feature brought out by the pointing test is a remarkable tendency of the affected limb to overshoot the mark; while the normal limb is arrested at or near the position of the object which the patient wishes to touch, the affected one almost always swings past it, and often as much as 30° to 45° beyond it. This is obviously a manifestation of dysmetria; no disturbance of any form of sensation that could be responsible for it was demonstrable in any case.

§ 6.—*Attitude.*

Abnormal attitudes, which are such a prominent feature in animals after experimental lesions of the cerebellum, are much less constant and striking in man. Considerable importance has, however, been
attached to the position of the head in cerebellar disease, especially when the affection is unilateral; in gunshot injuries its significance is generally doubtful, since the wound frequently involves the cranial attachments of the neck muscles, and because the patient naturally assumes the position which is most comfortable or which gives him least pain. As a rule, however, the head tends to be flexed towards the side of the wound and rotated towards the opposite side, so that the chin approaches the contralateral shoulder and the occiput is approximated towards the shoulder of the affected side. This is particularly striking in cases in which the wound has healed.

Special attitudes of the trunk are less obvious, but in the early stages of an injury the body is often concave to the side of the lesion even as the patient lies on his back; this is usually more prominent when he sits up, but as he then inclines to and tends to fall in this direction the upper part of his trunk is often purposely flexed over towards the opposite side in order to maintain equilibrium.

In certain patients, especially in those who are still or were recently subject to vertigo, the upper part of the trunk also tends to rotate towards the unaffected side (i.e., the homolateral shoulder is advanced), especially when they close their eyes; this attitude can be made more distinct by gently shaking the bed or the chair on which they sit.

In some of my patients, and particularly in those in whom the vermis cerebelli was also involved, the face was stolid and expressionless, but I could observe no noteworthy change in the face when the lesion was unilateral.

The flaccid and hypotonic limbs generally assume any posture into which they fall or are moved, or which may be given them by gravity, but when the patient holds the arms outstretched that homolateral to the wound is frequently more abducted than its fellow, and in several cases it is described in my notes as more rotated inwards at the shoulder and pronated at the elbow. In a few cases the hand of this side tended to be saddle-shaped, with the wrist flexed and the fingers hyperextended at the metacarpophalangeal joints.

There is no common noteworthy attitude of the lower limbs as the patient sits or lies in bed.

§ 7.—Standing and Gait.

When a patient who is able to leave bed is placed on his feet for the first time he is shaky, uncertain and unsteady, his whole body sways irregularly, his head oscillates, and he is usually in considerable
danger of falling, especially towards the side of the wound and backwards. It often seems as if he were impelled to this side; several patients have, in fact, complained that they felt that they were pulled over, as though by an invisible hand, in this direction. It is, however, remarkable that even after being confined to bed for several weeks with a wound of one side of the cerebellum the patient can generally maintain his equilibrium, and make awkward though appropriate and often successful attempts to recover it if it is threatened. At this stage his attitude in standing is very striking; his head and trunk are both inclined to the injured side and his spine is concave to it, but his pelvis is so tilted that his weight falls chiefly on his opposite foot. The homolateral shoulder is generally higher than the other and it is almost always in front of it, and the trunk is occasionally rotated on its longitudinal axis towards the unaffected side. The homolateral leg is usually abducted and sometimes rotated outwards. His whole body is held stiff and rigid, and even when in danger of falling he does not flex it naturally in his efforts to save himself.

If he is gently pushed to either side, or if his shoulders are tapped firmly, it is found that he can be more easily thrown over towards the side of the lesion and that he makes less appropriate efforts to save himself when falling in this direction. When the force is applied to the point of his homolateral shoulder he offers an appropriate resistance to being thrown towards the normal side by flexing his trunk so that his spine becomes concave towards the affected side, but when the observer’s hand pushes him towards the side of the lesion, he generally falls at once without bending his vertebral column or making any natural effort to resist. Finally, he can usually stand on his unaffected leg alone, but when he attempts to place his weight on the homolateral limb, he sways and staggers so much that considerable support may be necessary to prevent him from falling. In cases of severe injury this leg also tends to give way suddenly under him as though it were too feeble to bear his weight.

It must be emphasized that the patient can stand as securely with his eyes closed as when they are open; there is consequently no tendency to Romberg’s sign. On the other hand, he becomes more unsteady and is in greater danger of falling if his attention is diverted, even if only by conversation; he obviously supplements the normal muscle reflexes that preserve equilibrium by voluntary effort.

When such a patient attempts to walk many remarkable features are seen in his gait. If the lesion is severe and extensive he has often
much difficulty in preserving his equilibrium and is frequently in danger of falling, though almost all men with unilateral lesions succeeded in walking unaided on the first day they left bed; this varied between eight and seventy-one days after the infliction of the wound.

The first and most striking feature is the patient's obvious fear of trusting himself on the affected leg. He throws his weight on it slowly and cautiously, and at each step hurries off it by bringing the other limb forward and to the floor, as quickly as he can. At the same time he stumbles, and tends to fall to this side. His steps are generally short, unequal in length, and irregular. In advancing the affected leg, the foot is occasionally dragged along the ground, but more frequently it is raised unnaturally by excessive flexion of his knee and hip, and is brought down with undue force. Further, it often comes irregularly to the floor, the whole sole falling on it simultaneously, or the toes before the heel; and, occasionally, either the inner or the outer margin of the foot alone may first come in contact with it.

This leg is usually abducted and rotated somewhat outwards, but this is not constant; often, in fact, the homolateral foot is brought in front of the other in one step, and in the next is abducted too much.

When the patient is able to walk safely alone, he still stumbles to the homolateral side, and reels abruptly from his proper course in this direction; in walking between two rows of beds, he is therefore liable to run into those on his affected side. He consequently takes a zig-zag course, the abrupt deviations being always towards the side of the lesion, except when he stumbles awkwardly, as he often does, towards the other side in an effort to save himself, or to correct the deviation. The stumbling is often due to excessive adduction of the affected limb, owing to which its fellow in being advanced trips on it; but it is most commonly a manifestation of the tendency to fall to the affected side.

In walking along a straight line he also deviates, or shows a tendency to deviate, towards the affected side. This is usually more obvious when his eyes are closed; then his course is often a parabolic curve concave to the side of the lesion. His attitude, with his head rotated towards the opposite side, the homolateral shoulder in advance of its fellow, and the transverse axis of his body consequently oblique, often gives the impression that his direction, even when he walks straight, inclines to the homolateral side of the line he intends to follow.

This tendency to deviation can be well demonstrated by making the patient walk round a chair or a small table; when the shoulder of his injured side is inward, he frequently runs into the chair, while he
THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES

constantly deviates outwards from it, so that he traces an opening spiral, in walking round it in the opposite direction. In walking backwards, especially when his eyes are closed, he generally deviates in a sharp curve concave towards his affected side.

The patient soon becomes aware of this tendency to stumble, reel, and deviate towards his affected side, and attempts to control it; occasionally, indeed, in his efforts to prevent falling to the one side, he actually stumbles to the other. These symptoms are usually most marked when he walks quickly; but a few patients got along better rapidly than slowly and deliberately.

Further, when walking quickly, he may have difficulty in stopping suddenly; when ordered to halt, he often cannot pull up for two or three steps; or, in attempting to do so, he may throw himself forwards so abruptly on to his toes that he is in danger of falling on his face. In certain cases the difficulty in stopping when walking backwards was even greater—one man, for instance, on taking a step or two backwards, could not pull himself up till he ran into some object, or was assisted, for several weeks after the infliction of the wound. In the former case a few patients walked much better when an attendant pulled them gently backwards, and in the latter case when they were pushed gently forwards by a hand placed on their backs. This difficulty in stopping resembles that seen in paralysis agitans, in so far as it is due to the legs having to move after the body in order to keep the centre of gravity above the base of support; but in cerebellar lesions it must be attributed to deficient synergia of the trunk and thigh muscles by which this should be promptly attained. Another example of asynergia is furnished by the fact that as the patient walks the affected arm hangs inertly by his side, and does not swing synchronously with the movements of the opposite leg; nor does he use this arm naturally in attempting to balance himself. Further, when he is in danger of falling, he does not move either his trunk or arms naturally or adequately in attempting to regain equilibrium, and if he is suddenly pushed in any direction he falls stiffly and rigidly like a doll.

In cases with unilateral cerebellar lesions the chief abnormalities in gait are therefore due to the irregular and incorrect placing of the affected foot, and to the tendency to fall, stumble, and deviate towards the affected side. The patient soon attempts to correct these disturbances by voluntary effort; he throws his weight as little as possible on the unsafe leg, and when he can control his direction by vision, he checks, though rarely completely, his tendency to deviate from the direction he wishes to follow.
The disturbances in gait are, it is true, usually greater in relation to the disorders of movement of the affected leg, when this is tested as he lies on his back, than might be expected; but the symptoms of cerebellar deficiency are always most obvious in the most complicated actions, and more complex synergias, involving both the trunk and limb muscles, are called into play in maintaining equilibrium and direction during gait than are required in movements of the leg alone. Many of the older physiologists and certain clinicians have regarded the cerebellum as an organ concerned mainly with the maintenance of equilibrium; but this hypothesis finds no support in the examination of men in whom one side of the organ only has been damaged; in fact, the successful efforts to maintain equilibrium, despite the patient's deficient control over the movements of the one leg, are often surprising.

When a man who has recently left bed attempts to progress on his hands and knees, similar disturbances are seen. The affected arm is abducted, this shoulder is held lower than the other, his trunk is concave to the side of the injury, the hip of this side is depressed, and the leg is usually somewhat adducted. In moving forwards, the affected arm is raised too high, and the hand swoops down too heavily to the ground; the homolateral knee, on the other hand, is generally dragged along the floor. The patient occasionally falls to the side of the lesion, but more frequently he merely slides over on to this hip, owing to its excessive adduction, or to the leg giving way under him. The similarity both in attitude and progression on the hands and knees between a patient with a unilateral destruction of the cerebellum and a dog or monkey after ablation of one half of this organ, is consequently very striking.

§ 8.—Disturbances of the Ocular Movements, and Nystagmus.

In animals subjected to cerebellar injuries disturbances of the ocular movements have been observed, but little emphasis has been laid on the occurrence of nystagmus; this is not surprising, as it can be properly investigated only on voluntary movements and fixation of the eyes, and with the co-operation of the subject. Many physiologists have even refused to admit that the nystagmus is dependent on the cerebellar lesion (Munk, Rothmann). It is more remarkable that even from most clinicians these symptoms have not received the attention they deserve; the reason, probably is that they are particularly prominent only early after acute lesions.
For some days after a unilateral gunshot wound of the cerebellum the eyes while at rest are generally deviated towards the opposite side, especially if the patient happens to be unconscious, and at first it is often difficult to make him move them conjugately towards the injured side. When he attempts this the range of movement is occasionally incomplete, but usually a more striking feature is its slowness and the effort necessary to execute it. In one patient at least in whom it was carefully investigated this conjugate paresis was associated with erroneous projection towards the homolateral side. The paresis diminishes gradually but the difficulty in movement to this side, in comparison with deviation in the opposite direction, frequently persists for weeks. The vertical movements of the eyes and convergence are never similarly affected.

In five cases the position known as "skew-deviation" was observed, that is, the homolateral eye was directed downwards and inwards while the other looked upwards and outwards. This lack of parallelism in the optic axes disappeared however on fixation when this could be obtained, and consequently diplopia did not result. Skew-deviation was observed only during the first week or so after an injury of or an operation on the cerebellum, except in two cases in which rapid destruction and compression of this organ occurred owing to abscess formation.

Nystagmus is a much more common symptom; it was present in fact in almost every case in which injury to the cerebellum produced any trace of functional disturbance, and its chief characters are so constant that it must be regarded as a clinical symptom of the highest importance. It is essentially a fixation nystagmus, that is, it can be seen as a rule only when the patient fixes an object.

It is advisable to describe it first as it most commonly occurs in the early stages of a severe unilateral lesion. In such a case some nystagmoid jerks can be usually observed when the patient fixes an object in any part of his visual field, except in the region of that point, usually 10° to 30° to the unaffected side of the middle line, towards which his eyes when at rest tend to deviate. We shall speak of this as the "rest point."

When he looks at an object held directly in front of him his eyes tend to deviate slowly towards the unaffected side and are brought back to the middle line by sharp jerks of small or moderate range. These movements are usually slow in rate and at first regular, but when the patient's effort or attention begins to tire his eyes may move more
widely before their deviation is checked, and the correcting jerks consequently become less regular in rate and larger in range.

It is on looking towards the injured side however that the nystagmus is most pronounced; then it consists of wide, slow deviations towards the middle line, or more correctly towards the "rest point," and forcible jerks of large amplitude, slow in rate and fairly regular in rhythm, towards the point to which the eyes should be voluntarily directed. Its rate was estimated in several patients and in all varied between twenty-three and thirty oscillations in ten seconds. Both movements are as a rule strictly horizontal, but occasionally slight rotation, generally downwards, may be seen in the quick phase. This nystagmus generally remains regular in rate and amplitude for a short time, but as the patient tires his eyes are less frequently and less completely pulled back towards the point he should fix.

In this stage too there is almost always nystagmus on fixing an object to his unaffected side, but it is more rapid, finer in range and less regular. Here too the slow deviation is towards the "rest point" and the movements are most commonly horizontal. Frequently in looking towards this side the nystagmus consists of series of rapid jerks with short pauses between each series.

Nystagmus is less constant and less regular on vertical movements. In the majority of early cases, however, the eyes on being directed upwards tend to deviate slowly towards the horizontal and are brought back by sharp quick jerks of small range, which are rarely quite regular in rate or well sustained. Almost always there is either some rotation of both eyes upwards and towards the side of the injury in the quick phase, or the quick movement is more or less obliquely upwards and to this side. This occurs especially when the point fixed is brought to the affected side of the mesial sagittal line. On looking downwards regular and sustained nystagmus is rare, but very commonly slow deviations towards the horizontal line occur, which are corrected by quick, relatively small jerks vertically downwards, or more frequently obliquely downwards and to the side of the lesion, or with rotation to this side. The nystagmus that occurs on vertical movement always increases, or it may appear only when the object fixed is brought to the homolateral side of the middle line.

Finally, on convergence both eyes often tend to deviate away from the side of the lesion and are brought back to their proper position by irregular jerks of small range.

Unless there is much paresis of conjugate deviation towards the
injured side the movements are more regular, larger in range and better sustained the farther the point fixed is from the primary central position, or rather from the "rest point," and the jerks increase in range and accelerate in rate when the point is moved farther from this position. The nystagmus also tends to become less regular and less rapid as fixation tires, but it can be evoked again by renewal of fixation, especially if the object fixed is moved slightly farther from the middle line.

This is the characteristic form of nystagmus produced by recent cerebellar injuries, but divergences from it are common. Little or no disturbance may be, for instance, seen on movement of the eyes in one direction, though characteristic nystagmus is associated with all other movements; or the relative amount that occurs when the eyes are moved in different planes may not be typical. These divergences may be partly dependent on the localization of the cerebellar lesion, though I am not yet able to state definitely that this is so. It varies, however, according to the severity of the injury; when this is recent and extensive and the eyes deviate spontaneously towards the unaffected side and movement to the contralateral is difficult, no nystagmus, or only an occasional jerk, may occur when the patient looks to the normal side, and on fixing to the side of the lesion only a few slow irregular jerks of large range can be seen before the effort at fixation becomes ineffective.

If the lesion is slighter, or if some time has elapsed since the infliction of the wound, and there is no obvious deviation of the eyes, or at the most only slight difficulty in conjugate movement towards the injured side, the nystagmus is also less regular and less characteristic. On central fixation the eyes generally remain steady, or only an occasional deviation of small range towards the unaffected side occurs, which is at once corrected by a sharp jerk. On looking towards the side injured a slow, coarse, forcible nystagmus, with its slow phase towards the "rest point," still develops, but it is usually less regular and less well sustained. After a time the oscillations become more rapid and smaller in range before the true nystagmus disappears or is replaced by occasional irregular jerks of the eyes. On fixing to the opposite side the excursions of the eyeballs are still smaller in range and more rapid in rate, but they are less regular and persist for a shorter time. Vertical movements are now less commonly complicated by nystagmoid jerks, and these are in fact rare on downward deviation. In a few severe cases there was at this stage more rotation upwards and to the side of the lesion when the patients looked directly upwards,
though in one man, in whom the rotation had been previously to that side, it was upwards and in the opposite direction four weeks after the injury was received. Here, however, the mastoid process and the internal ear probably, too, was damaged.

Finally, in cases with very slight injury, or at a later period after the infliction of a severe wound, the only abnormality in the movements of the eyes may be occasional irregular slow deviations towards the middle line and sharp rapid jerks, irregular in rate and in amplitude in the opposite direction, on prolonged fixation towards the injured side; or there may be only slight irregularity of the eyeballs when they are fully deviated in this direction.

It is obvious that the nystagmus produced by a unilateral cerebellar lesion is essentially a fixation nystagmus, that is, it is seen chiefly when an attempt is made by the patient to fix accurately a point in any part of his visual field, but it may also occur when accurate fixation is impossible. In some of the cases spectacles with high convex lenses, such as have been employed by Wilson and Pike, were placed in front of the patient's eyes, and it was then found that when he moved them to order the oscillations were considerably less marked, or did not occur on deviation in certain directions.

Nystagmus of cerebellar origin is therefore characterized by the facts that it occurs chiefly on fixation, that the slow phase is always towards the primary central position or the "rest point," and that it is more constant, more regular, better sustained, and the oscillations of the eyeballs are slower in rate but larger in amplitude, when the patient looks towards his injured side. Finally, it persists for weeks, or even months, after the onset of the lesion, though it gradually becomes less regular, or is replaced by irregular nystagmoid jerkings.

The question as to how far this nystagmus is dependent on coexisting labyrinthine lesions at once arises, more especially since labyrinthine disturbances, as Moutier and others have shown, commonly result from gunshot injuries of the head. It cannot be denied that the internal ear was possibly damaged in certain cases of my series, and particularly in those in which the missile entered through or near the mastoid process, but in the majority it certainly escaped, and no essential difference in the type of the nystagmus could be detected in the two groups. Further, the nystagmus has not the character of that produced by destructive or irritative labyrinthine lesions. We owe valuable contributions on this subject to Gordon Wilson and Pike, and to the former I am indebted for the following distinguishing features of the
nystagmus which results from destruction of one labyrinth: it persists at the most only two or three days; it is increased when fixation is cut out by suitable lenses placed in front of the eyes in a spectacle frame or by other means, and the slow phase is always towards the injured side, whether the eyes are at rest or deviated voluntarily towards either side.

If the features of that produced by cerebellar lesions are compared with these, it will be seen that nystagmus of cerebellar origin differs in many important particulars from that due to destructive labyrinthine lesions, and we may consequently conclude that injury of the labyrinth plays no direct part in its pathogenesis.

The nystagmus associated with cerebellar lesions has also been attributed to injury or disease of neighbouring centres, and especially to affection of Deiters' and Bechterew's nuclei. This factor can be certainly excluded in the majority of my cases, and the regularity with which the characteristic nystagmus occurs, even when the wound is small and relatively superficial, and in the absence of all signs of medullary and pontine involvement, makes it extremely probable that it is due to damage of the cerebellum alone.

§ 9.—Disturbances of Speech.

Apart from the oculomotor; the only cranial nerves of which the functions are obviously affected by unilateral cerebellar lesions are those which are concerned in phonation and articulation:

Speech is abnormal in most cases in which the lesions are recent and severe; it is usually slow, drawling and monotonous, but at the same time tends to be staccato and scanning. This gives it an almost typical "sing-song" character and makes it indistinct and often difficult to understand. In a few patients speech was in fact quite unintelligible for a time. In many cases the utterance is remarkably irregular and jerky, and that of many syllables, especially, as Marie has pointed out, of those that end a sentence, tends to be explosive.

Phonation is as a rule more affected than articulation, though both vowels and consonants are slurred and uttered unequally and irregularly. All classes of consonants too are affected, but articulation sometimes has a special nasal character and the labials particularly tend to be explosive.

Another striking feature is the apparent effort necessary to utter a series of syllables or a sentence; the attempt is associated with excessive facial grimacing and speech has consequently a laboured character that often recalls a pseudo-bulbar paresis. A few of the patients also showed a tendency to burst into explosive and excessive laughter when amused.
These abnormal features subside as a rule rapidly, but in several cases the speech was not yet natural two or three months, or even longer, after the infliction of the wound.

§ 10.—The Reflexes.

Striking alterations in the reflexes is not a prominent or very obvious symptom in the clinical examination of patients with cerebellar lesions, but they are frequently abnormal, or when the injury is unilateral they may be unequal on the two sides. The change can be best studied in the knee-jerks in cases in which the injuries are limited to one-half of the cerebellum, but they can also be seen in the arm- and in the ankle-jerks.

![Tracings of three knee-jerks of a normal man taken on a slowly revolving drum.](image)

Fig. 11.—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.

When the knee-jerks are elicited as the patient either lies in bed or sits with his legs hanging freely, that of the healthy side seems unaffected, but the homolateral jerk is at first often feebler, less brisk and less easy 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 is then generally large and ample. In a few cases the jerks were wholly absent during the acute stages of the illness, unless reinforcement was employed.
THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES

When the patellar tendon is tapped as the patient sits on a high chair so that his legs are unsupported and can swing freely, the jerk of his homolateral leg when compared with that of his other limb appears less brisk, often slower, and it lacks that decisive, forcible character of the normal knee-jerk, though its range may be as large or even greater. Further, while the normal limb falls deliberately to its original position and quickly comes to rest, that of the affected side often continues to swing inertly to and fro for a time like a pendulum; the jerk has consequently the pendular character described by André-Thomas. This feature can be seen by comparing fig. 11 and fig. 12.

Fig. 12.—Tracings of two knee-jerks of a man with a right-sided cerebellar lesion of eight years duration. Tracing is less reduced than that in fig. 12. Read from right to left.

But in order to study more fully this change in the reflexes it is necessary to record the movement of the leg on a more quickly moving drum.1

By this means I found that though from unaided observation the jerk was frequently described as slower or less brisk, there was no

---

1 My records were obtained by attaching firmly to the leg, at a fixed distance below the axis of rotation of the knee-joint, a properly shaped splint which carried a rod attached to it by a joint at which only slight vertical movement was possible. This rod was connected by
increase in the latent period as recorded by the apparatus I have employed. In several normal persons this, when measured from the instant the tendon was struck to the commencement of the movement of the leg, varied between 0.038 sec. and 0.054 sec., being in the majority 0.043 sec., which agrees approximately with the latent time obtained in man by Franz, by Guillain, Barré and Strohl and by others by tambours placed on the quadriceps extensor. In the patients with cerebellar injuries in whom I have similarly recorded the jerk the latent period always lay within the same limits; in one man with a very severe lesion it constantly approached the lower figure. The apparent slowness is consequently not due to a delay in the response or to a slower movement of the limb; it is probably the subjective interpretation of the inertness in the swing of the leg.

![Fig. 13.—Tracings of the knee-jerks of two normal men recorded in a rapidly revolving drum. Read from right to left. Time by a tuning-fork of 128 vibrations per second.]

But such tracings (figs. 14, 15) show other important changes. When that of a normal jerk is examined (fig. 13) it is obvious that the fall of the leg is considerably slower than its rise, and the curve does not therefore correspond with the oscillations of a pendulum. Evidently then a tonic contraction, or state of tone, in the extensor a ball-and-socket joint with a longer bar which could move accurately and with a minimum of friction through three guides in the plane of the movement of the leg: a suitable marker fixed to this bar recorded the movement directly on a revolving drum. The moment of the tap on the patellar tendon was registered by an electro-magnet, the circuit being closed when the metal hammer, to which one terminal was connected, came in contact with a copper wire placed in close contact with the skin over the patellar tendon. The latent time of this electro-magnet was 0.001 sec., and it is assumed that it was constant. Time was recorded by a tuning-fork of 128 vibrations per second (C°).
muscles prevents the immediate fall of the normal leg to the abscissa. This fact is already known from the records obtained by various methods in experimental animals and in man. I have found the time

![Diagram](https://academic.oup.com/brain/article-abstract/40/4/461/273305)

**Fig. 14.**—Tracing of the right knee-jerk of a man who received a severe injury to the right side of his cerebellum eight years previously. Read from right to left. Time by a tuning-fork of 128 vibrations per second. Signal on the lowest line.

**Fig. 15.**—Tracing of the right knee-jerk of a man with an extensive injury of the right side of his cerebellum, obtained ten weeks after the infliction of the wound. Read from right to left. Time by a tuning-fork of 128 vibrations per second. Signal on the lowest line.

of the rise in relation to that of the fall in several controls to vary between 1 to 1.2 and 1 to 2.2. In the curves obtained from the affected limbs of men with cerebellar injuries the fall to the abscissa is on the
other hand almost invariably more rapid than the rise (fig. 14); in the cases in which I have measured it the relation of the time of the rise to that of the fall of the curve averaged 1 to 0.85. Here there is consequently no evidence of tone or muscular contraction impeding or delaying the fall of the limb under the influence of gravity, and the falling limb acquires sufficient velocity to make it swing, provided its oscillations are not resisted by the tone of the muscles that move the knee-joint. These oscillations give regular curves which have all the features of those of a pendulum; it can be easily ascertained that they are not associated with any active contractions of the extensor or flexor muscles of the knee.

There is another feature in the knee-jerk of the affected side which can be easily detected by careful observation. If the observer places one hand across the hamstring tendons behind the normal knee he can generally feel a brisk tightening of these tendons, due to contraction of the flexors of the knee, at a very short interval of time after the patellar tendon has been struck, providing that this produces 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, as a rule, no such contraction of the hamstrings can be felt, no matter in what position the knee is placed, or how great the amplitude of the jerk may be. One result of this may be seen if, while the two limbs are fully supported on the bed with the hips and knees semiflexed and the thighs rotated outwards so that the heels are at the same level, the patellar tendons are tapped in succession; the foot of the unaffected side is moved abruptly towards the bottom of the bed by each jerk that is elicited, but it returns, or 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 in fact often fully extended by a series of taps on the patellar tendon. This failure of the contraction of the hamstrings and of the consequent pull back of the leg was present in all my cases of severe injury, and its occurrence was repeatedly confirmed by independent witnesses. It usually persists as long as there are obvious disturbances in the movements of the limb; it was very striking in a patient who had received his injury eight years previously.

The ankle-jerk is less commonly abnormal, though when the knee-jerk is depressed or difficult to elicit this reflex is usually more so. In five of my cases, however, in which there was no evidence of involve-
ment of the pyramidal tracts, a short ankle-clonus could be obtained on the affected side; it resembled that due to organic disease but did not persist on continued pressure of the sole; in fact, as a rule it consisted of a few jerks only.

In many cases the flexor and extensor reflexes of the elbow appeared less brisk than on the normal side. In these reflexes it is less easy to detect a concomitant reflex contraction of the antagonists of the contracting muscles. When the forearm is allowed to hang unsupported it frequently tends to swing in the pendular manner described by André-Thomas, especially after the triceps-jerk has been elicited.

No difference in the superficial reflexes, the abdominal, cremasteric and plantar, of the two sides could be detected in even the severest unilateral lesion, and in every case they presented their normal characters.

§ 11.—Sensation.

Finally we have to consider the state of sensation in patients with cerebellar lesions. This is an important question, since Lussana regarded the cerebellum as an organ of the "muscular sense," and Lewandowsky has attributed many of the motor abnormalities to disturbances of muscle sensibility, meaning thereby those sensations evoked in consciousness by the state of contraction of the muscles, and by movement and the position in space of different portions of the body.

I have, however, examined every modality of sensation in many cases but have never found disturbances of any form, nor have I detected any evidence that would point unequivocally to any alteration of it. No matter how irregular the movements may be, or how far the affected limb deviates from the point to which it should be moved, the patient always has a full and accurate recognition of its position in space.

It is true that, as Luciani observed in animals, the withdrawal of a limb that is pricked and the reaction to the prick, are occasionally less brisk on the homolateral side when the lesion is early and extensive, but no alteration in the threshold of, or diminution in the acuity to, tactile and painful stimuli, or even subjective differences between the sensations similarly evoked on the two sides, ever existed in my cases.

Lotmar has described a disturbance in the appreciation of weights by the affected hand, and Maas and Goldstein have supported his statements, but in none of their three cases was a lesion limited to the cerebellum demonstrated anatomically; there was for instance a diffuse cysticercus meningitis with hydrocephalus in Goldstein's patient. All three authors base their conclusions on the fact that when weights were
placed simultaneously in his two hands the patient usually underestimated that on the affected side. I have investigated the appreciation of weights in eleven cases with extensive unilateral lesions, and in some of them on several occasions. In two no disturbance existed, but in the others it was found that if while the arm was still asthenic identical weights were placed in his two hands, his eyes being closed, the patient frequently did not recognize that they were equal, and in almost every case stated that the heavier was in the affected hand. When unequal weights in which the difference was relatively small but appreciable, as 80 grm. and 100 grm. or 200 grm. and 240 grm., were placed in his hands, his replies were correct when the heavier lay on the affected palm, but often wrong when the normal limb carried it. Consequently the ability to recognize the identity or inequality of weights lifted simultaneously by the two hands was affected. In all these cases the tendency was to call that on the affected palm the heavier, except one man who at first occasionally described that borne by this hand as lighter, though in all subsequent examinations when the weights were equal or approximately so, that in the affected hand seemed to him the greater.

But obviously these observations cannot be accepted as evidence of disturbance in the appreciation of weight, since by every paretic limb weights are adjudged heavier than they actually are, even though sensibility is unaffected. The greater effort which a patient suffering from a unilateral cerebellar lesion must put into all attempts to move the homolateral limbs suggest strongly that he will necessarily overestimate, or estimate wrongly, the resistance which these movements encounter. Whether any disturbance in the appreciation of weights exists can be decided only by testing his ability to discriminate between two weights within the normal threshold of difference which are placed successively on this hand. This was done in all my eleven cases. The difference-threshold for the unaffected hand was first carefully ascertained and then a series of observations was made by placing the same two weights in succession in the affected hand. By this method no loss or defect in the appreciation of weights could be detected on either side; in one patient in fact the difference-threshold for the affected limb was considerably smaller than in the other and in several normal persons who were tested at the same time, but this was explained by the fact that he had been a mica dealer in Canada and accustomed since childhood to "weigh" his wares in his left hand.

It must be admitted, however, that when a severe degree of ataxia
exists, the appreciation of weight may not be so accurate in the affected hand as in the normal one, owing to the difficulty which the patient has in alternately raising and lowering his hand regularly as he attempts to estimate the relative heaviness of the test objects.

The following records, selected from a long series of observations which were obtained nineteen days after the infliction of the wound, in a man with an extensive lesion in the right half of his cerebellum, illustrate these facts.

<table>
<thead>
<tr>
<th>Right hand</th>
<th>Left hand</th>
<th>Reply</th>
</tr>
</thead>
<tbody>
<tr>
<td>200 grm.</td>
<td>200 grm.</td>
<td>Right heavier</td>
</tr>
<tr>
<td>200</td>
<td>200</td>
<td>Equal</td>
</tr>
<tr>
<td>200</td>
<td>200</td>
<td>Right heavier</td>
</tr>
<tr>
<td>150</td>
<td>150</td>
<td>Left heavier</td>
</tr>
<tr>
<td>150</td>
<td>150</td>
<td>Right heavier</td>
</tr>
</tbody>
</table>

But when the discrimination of weights was tested by placing two weights successively in the one hand he gave the following replies:

<table>
<thead>
<tr>
<th>First weight</th>
<th>Second weight</th>
<th>Right hand</th>
<th>Left hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>80 grm.</td>
<td>100</td>
<td>Correct</td>
<td>Correct</td>
</tr>
<tr>
<td>100</td>
<td>100</td>
<td>Correct</td>
<td>Equal</td>
</tr>
<tr>
<td>80</td>
<td>100</td>
<td>Equal</td>
<td>Correct</td>
</tr>
<tr>
<td>200</td>
<td>150</td>
<td>Correct</td>
<td>Correct</td>
</tr>
<tr>
<td>150</td>
<td>200</td>
<td>Correct</td>
<td>Correct</td>
</tr>
<tr>
<td>200</td>
<td>150</td>
<td>Correct</td>
<td>Correct</td>
</tr>
</tbody>
</table>

We must therefore conclude that no form of sensation is disturbed by cerebellar disease, and that though the patient often cannot compare correctly weights placed at the same time in his two hands, this does not depend directly on a disturbance of the faculty of appreciating and discriminating weights.
All the evidence available from the careful investigation of sensibility in cases of cerebellar lesions in man, in whom alone it can be properly tested, shows conclusively that this organ is not concerned in the transmission, or in any modification or elaboration, of those afferent impulses which give rise to conscious sensations.

CHAPTER II.—SYMPTOMS DUE TO LESIONS OF THE VERMIS.

I have not yet seen a case in which a gunshot wound produced a lesion limited to, or affecting mainly, the vermis of the cerebellum, though this was undoubtedly involved in some of the unilateral and in most of the bilateral injuries. Consequently the only conclusions on this point that can be drawn from my material must be by comparison of the symptoms in cases where one lateral lobe only was affected, with those present in which the vermis also was injured. But unless the differences in the symptoms of these two groups of cases are essentially distinct in one or more particulars they cannot be forthwith regarded as indicating a special physiological significance of the vermis, in view of the evidence of functional localization in the cerebellum which has been brought forward within recent years.

No essential difference can be detected when the symptoms due to lesions strictly limited to one lateral lobe are compared with those of cases in which the vermis is in addition involved, though in relation to the extent of the damage the functional disturbances are somewhat greater and recover less rapidly in the latter. The hypotonia, the abnormalities in the voluntary movements of the limbs, the changes in the reflexes and the nystagmus differ in no essential particular. Even the affection of gait and of equilibration, which have been attributed by certain authors (Thomas, Rothmann) to injury of the vermis, were not more pronounced when the lesion included this, provided it did not extend sufficiently beyond the middle line to produce symptoms in the opposite limbs. Two patients, for instance, were under observation at the same time—in one a piece of metal was removed from the lateral part of the left lateral lobe and considerable destruction of tissue was found around it, while in the second a large piece of shell-casing was extracted from deep in the mesial portion of the right lateral lobe, practically through the paramedian fissure, and a hernia which must have involved the vermis developed. The first man attempted to walk 58 days, and the second 71 days, after the infliction of the injury and
both succeeded equally well in walking and in maintaining equilibrium. Further, there was no striking difference in the character of their gaits.

When the injury extends so far over the middle line as to produce also disturbances in the opposite limbs, gait and the maintenance of equilibrium are naturally more seriously affected than when one side only is involved.

The only special features of vermis affection that I have observed are greater disturbances of phonation and articulation, and more pronounced tremor and greater difficulty in movement of the head and trunk when the patient sits up, but even in these respects the symptoms do not differ in nature from those produced by injury of both lateral lobes.

CHAPTER III.—SYMPTOMS OF BILATERAL LESIONS OF THE CEREBELLM.

In seven of my cases both sides of the cerebellum were injured; in four the missiles had passed transversely or obliquely through the organ, and in the other three there were tangential wounds which fractured and depressed the occipital bone over it. Four of these patients died; in one, who had a penetrating wound, the posterior margins of both hemispheres (lobi semilunares superior and inferior) were extensively damaged, but the vermis was injured only superficially in the region on the declivum; in two cases there were depressed fractures with extensive haemorrhages and superficial softenings in each lateral lobe, but the vermis escaped, while in the fourth large portions of the vermis and both lateral lobes were destroyed by haemorrhage.

All these cases presented the same symptoms, and no striking difference in nature could be detected between those in which the vermis was involved and those in which it escaped. The limbs of both sides were affected, but the disturbances of their movements differed in no essential particular from those that result from unilateral lesions. Speech was, however, very much disturbed; it was slow, drawling and scanning, and many syllables were uttered explosively. In two men it was so indistinct as to be scarcely intelligible. The muscles of the trunk and neck were very hypotonic, and each patient had difficulty in holding his head in any attitude if it was unsupported, and in sitting up if unaided. The power of standing and walking could be
observed in only one of these patients, and in this man gait particularly was more severely affected and the maintenance of equilibrium was more difficult, than in any case with a purely unilateral lesion.

CHAPTER IV.—SYMPTOMS PRODUCED BY LESIONS OF THE CEREBELLAR NUCLEI.

It is extremely difficult to determine the part played by damage of the nuclei in the symptomatology of these cerebellar lesions. Most penetrating gunshot wounds produce diffuse damage owing to the occurrence of haemorrhage, oedema, softening and septic infection in the parts around them, and the nuclei, the dentate in particular, are consequently very liable to be involved even when the wound of the cerebellum is relatively superficial. On the other hand, when the nuclei are involved by such injuries a portion of the cortex is also destroyed. We can consequently decide which functional disturbances are associated with nuclear lesions only by comparing the symptoms of superficial with those of deep wounds.

In the first place we find that when the lesion is so superficial that the nuclei cannot have been directly injured the symptoms are less intense, less regular, and that they disappear much more rapidly. Several such cases could be cited, but two will suffice.

Case 3.—Captain J. was wounded by a fragment of shell and became immediately unconscious. When he was seen two days later a contused penetrating wound was discovered 3 cm. below and 2½ cm. to the left of the inion. An X-ray photograph revealed a piece of shell-casing, which had been driven through the skull, immediately under the wound. This and pieces of depressed bone were removed next day; a moderate area of destruction existed in the cerebellum, but it seemed to be superficial. By measurement it was found to correspond to the inner third of the lobus gracilis.

When he was first seen he presented all the symptoms of a severe cerebellar injury. He had difficulty in deviating his eyes to the left, and when he brought them over there was coarse, slow, regular and well-sustained nystagmus of large amplitude; on looking to his right the movements were smaller, more rapid, less regular and less well sustained. No nystagmus occurred on looking downwards, though intermittent but fairly regular jerks developed when his eyes were deviated upwards. His speech was slow, forced and scanning. His right limbs were unaffected, but the left were very hypotonic (all groups of muscles were equally so), slow in all movements though not appreciably weak, and their voluntary actions were disturbed by dysmetria, aimless deviations from the direct line of movement, dissociation into their component movements, and by the occurrence of tremor towards the end of the action.
Adiadochokinesis and the rebound sign were marked in both arm and leg, and the left arm tended to swing outwards when outstretched, and deviated outwards in Bárány's pointing test.

Yet twenty-four days after the infliction of the wound this patient was able to walk and use his limbs quite well, and did not complain of any abnormal symptom. On examination, however, it was found that some nystagmus still developed when he looked fully to the left, hypotonia was still demonstrable in the left arm, but this limb was now only slightly slow and awkward in attempting rapid alternate movements, and it still deviated outwards in the pointing test.

Case 4.—Private R. was wounded by a piece of shell-casing. He did not become unconscious, but in attempting to stand up staggered like a drunken man and fell to the left. When he arrived in a base hospital two days later a gutter wound was found extending from the middle line 2 cm. below the inion towards the tip of the left mastoid. An X-ray examination showed fracturing with depression of bone fragments to a depth of 1½ cm. under the inner portion of the wound. (This corresponded to the inner part of the lobus semilunaris inferior and the upper margin of the lobus gracilis.) These fragments were removed from the cerebellum and the wound was cleaned and drained.

When he entered hospital he had slow, coarse, sustained nystagmus on looking to his left, fine and more rapid movements on deviation to the right, and hypotonia, slight paresis and considerable ataxia of his left limbs. Twelve days later, however, he could use his limbs quite well, walk safely and on examination he presented practically no abnormal symptoms.

Such cases, in which the lesions were in all probability superficial, suggest strongly that the ordinary symptoms of a destructive injury are usually transient and recover rapidly if the deeper parts of the cerebellum, and especially the nuclei, are not directly or indirectly involved. Certainly such rapid improvement is never seen when the damage extends to the neighbourhood of the central nuclei.

On the other hand, these superficial lesions produce all the symptoms that are found when the nuclei too are injured, and they differ from these only in degree.

Consequently, as far as my present observations permit, it must be concluded that the effects of nuclear and of cortical lesions of the cerebellum differ in no essential particular, though those produced by the latter are less intense, less regular, and recover more rapidly.

These statements can refer to the nucleus dentatus only, as I have seen no case which survived long enough to permit thorough examination in which there was reason to believe that the roof nuclei were involved. If a wound extends so deep, it is very liable to open the fourth ventricle.
and lead to an early fatal termination. The dentate nucleus, however, is only 2 or 3 cm. deep from the posterior surface of the cerebellum, which is the site of most gunshot injuries.

CHAPTER V.—THE NATURE OF THE SYMPTOMS PRODUCED BY CEREBELLAR LESIONS.

A study of the symptoms of destructive lesions and a determination of their constancy and relative importance will form a sufficient basis for the clinical diagnosis of diseases of the cerebellum, but in order to obtain an insight into the normal functions and the physiological properties of this organ it is necessary to analyse and attempt to resolve these numerous functional disturbances into their simplest components.

This is unquestionably a difficult task; in the past it has been the subject of much discussion and controversy among physiologists, who can deal with simpler factors and possess means of measurements that are not available to the clinician; the clinician, however, has the advantage that he can rely on the intelligent co-operation of his patients and consequently employ more numerous and better adapted methods for observing the effect of various lesions. Certain problems indeed, as the occurrence of sensory disturbances, can be decided only by clinical observations.

The first point which demands attention is the nature of the symptoms already described. Are they to be attributed to destructive or to irritative lesions of the cerebellum? Their constancy, their regularity, their persistence for long periods, and especially their nature, suggest strongly that they are directly due to destruction and that they are consequently negative or defect phenomena. In the case of many of them, as the muscular atonia, this cannot be doubted, and indeed careful observation makes it probable that irritative effects are minimal after gunshot injuries of the cerebellum in man. Those forced movements and attitudes which Luciani has tentatively termed "dynamic phenomena," that occur so commonly after experimental injuries in animals, are very rarely seen in man, or occur at least only as immediate symptoms. Vertigo, if it is to be included among the true cerebellar symptoms, is more probably of irritative origin; its inconstancy, its variability, and the facts that it occurs only early after the infliction of the injury and is rarer in gunshot wounds than in cases of tumour or abscess, favour this view. It may be consequently safely assumed
that the functional disturbances with which we have to deal are produced only by negative or destructive lesions of the cerebellum.

It is worthy of note that, as von Monakow has pointed out, symptoms referable to the effects of shock or diaschisis on other parts of the nervous system are rarely obvious.

Atonia, or diminution of that slight constant tension which is characteristic of healthy muscle, is such a constant and striking feature of all early injuries, and persists for such long periods when the wounds are large, that it is obviously a primary and direct result of the lesion.

The state of the tendon-jerks has been generally taken by clinicians as a measure of tone, and yet it is the rule that even in the toneless limb of a cerebellar patient the knee-jerks and similar reflexes are not abolished, and the excursion of the jerks may be even larger than on the normal side. Physiologists too (Risien Russell, André-Thomas) have found the homolateral reflexes exaggerated after unilateral experimental ablation. But tone is dependent not only on the activity of the direct spinal reflex arc, the integrity of which is essential for the presence of the knee-jerk, but on other factors too, as on labyrinthine and cerebellar influences; or rather that state we call tone is influenced by several factors and the presence of the knee-jerk indicates the activity of one only, that is of the spinal reflex arc. The presence of the knee-jerk is consequently not an argument against the existence of atonia in cerebellar disease.

But even when septic infection and the effects of increased cerebrospinal pressure can be excluded the knee-jerk and the other tendon reflexes are in some cases depressed, or they may be even for a time absent, on the side of an early and extensive lesion. As far as clinical observations go this seems to be generally associated with an extreme atonicity of the muscles, but their relation is probably not direct. The depression of the reflexes may be due to transient shock or diaschisis as it is an early hemiplegia; but the more probable explanation is that when the muscles are so atonic a tap on the tendon does not produce a sufficient increment of tension in them to be an adequate stimulus. The common observation that a reflex response can be frequently elicited by only one of a series of taps, or when the knee is in a certain position, and that the jerk is then quite brisk, favours the latter hypothesis.

This is probably also the explanation of the failure of the "rebound phenomenon" in the homolateral limbs. The excessive range of the after-movement may be due to absence or to too long delay of the
voluntary contraction of the antagonistic muscles—the fact that the patient's hand can be flung forcibly into his face shows that prompt voluntary arrest is not possible—but it must be ascribed mainly to failure of the immediate reflex contraction of the antagonists when they are suddenly stretched. The absence of contraction of the hamstring muscles when the knee-jerk is elicited under certain conditions, and the failure of the forearm muscles to contract when they are suddenly elongated by an abrupt jerk of the limb, are similar phenomena; the muscles do not react normally to a sudden putting on stretch.

Recent physiological investigations (Sherrington, Langelaan) have shown that tone is a more complex condition than has been generally realized by clinicians; it may indeed be a compound state, its separate components being perhaps dependent on distinct elements of the muscle fibres, and possibly on separate innervation.

If these views prove correct, it will be the task of experimental physiology to determine what component of tone is affected by cerebellar injuries. The study of the knee-jerk by the graphic method shows that the tonic contraction of the quadriceps, which prevents the free and immediate fall of the extended leg under the influence of gravity, is absent or diminished when the homolateral side of the cerebellum is injured; the jerk has then the character of that described by Sherrington when the reflex excitability of postural contraction is low, or when what Langelaan calls plastic tone, that is the slow yielding of the extensor muscles to a continued stress, is absent or diminished.

Langelaan has indeed already suggested that decerebrate rigidity is partly due to a spasm of the muscle sarcoplasm of sympathetic origin owing to the prevailing influence of the cerebellum, but Horsley and Clarke found that decerebrate rigidity persists after destruction of the cerebellum, unless the paracerebellar nuclei too are injured, and Sherrington also states that the cerebellum can be removed without the posture in this condition being annulled.

It seems that the various symptoms of atonia described above can be attributed to this loss or diminution of postural contraction or plastic tone; its absence abolishes the normal resistances of the various segments of the limbs to passive movement, leaves the muscles less elastic to sudden stretching, and makes them soft and flabby to palpation.

Clinical experience, therefore, fully confirms the statements of Luciani and other physiologists that atonia is a constant, important and
striking result of acute cerebellar destruction. It diminishes gradually in time, and may, like all the other symptoms, disappear, at least if the lesion is not very extensive. I have, however, at present no definite observations on this point, nor will it be possible to obtain satisfactory conclusions until we possess a reliable clinical method of measuring tone.

The remarkable fact that atonia as a symptom of cerebellar disease has received little attention from clinicians is probably due to its gradual decrease after the onset of acute lesions, and to its compensation pari passu with the progress of degenerative and atrophic diseases. Babinski, for instance, states that even in particularly severe cerebellar affections he has found no atonia, and André-Thomas in 1911 wrote that he had never observed muscular relaxation or hypotonia, in the sense which is given to it by clinicians, in any patients he had examined.

The latter author, however, in 1914, elaborated in conjunction with Durupt a hypothesis, foreshadowed by Rothmann, according to which focal lesions of the cerebellar cortex produce a condition of anisosthenia, or hyposthenia of certain muscles and hypersthenia of their antagonists. Of this condition I could find no evidence either when only local circumscribed lesions existed, or as the symptoms of more extensive injuries cleared up.

The next group of symptoms that claim our attention are those seen in voluntary movement. Certain of them are most easily studied in simple actions against resistance. It has been shown that when a man with a cerebellar lesion attempts to grasp the observer's two hands simultaneously: (1) the power exerted by the affected limb is defective; (2) the initiation and the execution of muscular contractions and relaxations are slower than on the normal side; (3) the grasp is often intermittent and irregularly maintained; and (4) the affected limb tires more quickly than its fellow. These symptoms can be equally well demonstrated in other actions.

**Asthenia.**—This term, which Luciani has applied to the diminished functional energy of the affected limb, may be conveniently used to describe their lack of normal power in movements that demand its exertion. It differs so definitely from the paralysis and paresis produced by the diseases of the motor system that it is advisable to designate it by a distinct word.

That any degree of feebleness is ever produced by cerebellar lesions has been denied by many, and others who have observed it have attri-
buted it to pressure on the motor tracts (Rothmann), to co-existing cerebral foci, to atonia, or to ataxia (Mann). In the early stages of every severe injury it is, however, easily determined that the homolateral limbs are feebler than their fellows, and when their strength is measured a very considerable reduction of power may be found (p. 469) in all groups of muscles, though it is usually more obvious in the arm than in the lower extremity. The greater effort necessary to deviate the eyes fully towards the side of the lesion, and the frequent defect in the range of their movement in this direction, is another manifestation of this asthenia. It has been already emphasized that the asthenia is not due in any large part to ataxia or awkwardness of the limb and a consequent misdirection of its energy; and though it is usually most definite when the limbs are very hypotonic it cannot be attributed to loss of tone, as these two symptoms bear no relation to one another. Asthenia must be consequently regarded as another primary and immediate symptom of cerebellar injury. It is probably pronounced only when the cerebellar nuclei are involved. The abnormal fatiguability of the affected limbs is associated with, and may be regarded as a result of, this asthenia.

Slowness in movement has been noticed by both clinical and experimental observers. It is certainly not willed or intentional as André-Thomas states, nor can the delay be attributed to time lost in “taking up slack” in the atonic muscles, since the latency of toneless muscles is very short (Sherrington), and this explanation would not account for the closely associated delay in relaxation. And since it occurs in the simplest actions, as in simple flexion of the fingers or elbow, it is obviously not dependent on the inco-ordinate and inappropriate muscular associations which disturb voluntary movements. Finally there is no evidence that a contraction or state of increased tone of the antagonists delays or impedes the shortening of the contracting muscles.

In order to understand its nature it is necessary to study simple actions, and if possible by the aid of the graphic method. It is then seen that the slowness is due both to delay in starting the contraction and to slowness in completing it. Further, careful examination shows that there is usually associated with it a similar delay in commencing and completing the relaxation of the same muscles, which is occasionally even more pronounced. This slowness may be due to retardation of the impulses that excite voluntary muscular contractions; or to the fact that certain nervous mechanisms concerned in the production of
voluntary movement react too slowly to these cerebral impulses; or it may be dependent on a state of the muscles owing to which their contractions and relaxations are retarded. As there is no evidence or probability that the cortical motor centres from which these impulses come are in any way affected the first of the hypotheses may be excluded. And as there is not a corresponding delay in the contractions and relaxations of muscles that are excited electrically or reflexly there is not sufficient basis for the third possibility. It consequently seems that the cerebellum exerts an influence on the nervous mechanisms, most probably on the spinal, immediately concerned in the execution of voluntary muscular contractions, by virtue of which these react promptly to cerebral impressions.

The cerebellum might be therefore regarded as a motor reinforcing organ, in the sense in which Luciani and others have used this term. It seems, however, probable that it takes no direct part in the processes, whether initiated reflexly or voluntarily, that produce motor effects, and that it does not augment these, but that it "sets" or "tunes," or regulates the activity of certain motor mechanisms, most probably spinal, so that the response to a volitional stimulus is immediate, effective and proportional to the intensity of the cerebral impulse.

Closely associated with asthenia is the discontinuity and irregularity in the maintenance of muscular contractions. This disturbance can be observed occasionally in voluntary movements and in the contractions of muscles concerned in maintaining posture (static tremor), but it is most easily studied in actions that demand the exertion of power; in these the contractions reach their maximum slowly and intermittently, and while in the normal limb a forcible contraction can be maintained regularly for some time, the grasp or other action which the patient attempts with the affected limb is often discontinuous and irregular, and it is frequently interrupted by sudden relaxations. The outstretched arm if unsupported often, for instance, falls suddenly, and in walking the affected leg frequently gives way under the patient without any apparent cause. The tremor that occurs in maintaining an attitude and in voluntary movement is due to this defect in the regularity and stability of the muscular contractions. Luciani has described this condition as astasia, and has attributed it to the imperfect fusion and summation of the single twitch contractions. Patrizi has indeed shown experimentally that cerebellar lesions lead to an incomplete fusion of the elementary twitches in muscles.

Astasia is not, however, such a prominent symptom in most local
lesions of the cerebellum in man as it is in animals after experimental destruction, though it varies in degree in different cases. It does not seem to stand in any close relation to the atonia and it is probably not in any way dependent upon it; it is, on the other hand, intimately associated with asthenia. Consequently, in addition to that function by virtue of which it assures that the motor response to a voluntary cerebral impulse shall be immediate and proportional to the impulse, the cerebellum also exerts an influence on the efficiency of this response by determining the complete fusion of the elementary muscle twitches.

Clinical observations consequently confirm Luciani's conclusions that atonia, asthenia and astasia, the triad of symptoms to which he attributes all the functional disturbances, result from cerebellar lesions.

On analysing the complex condition often called cerebellar ataxia, we meet with other facts that cannot be explained by atonia, asthenia and astasia alone. The kinetic tremor is, it is true, chiefly a manifestation of astasia, but the disturbances termed asynergia, decomposition of movement, and deviation from the line of movement, must be considered more fully.

The term asynergia here implies a defect in that accurate functional combination of the muscles which participate in a movement, that is the agonists, antagonists, synergic and fixating muscles, on which the precision and correct adaptation of the movement to its end depends. We have seen that on closing the fingers the wrist is often hyper-extended, or it extends too little or too late, and when the fingers are rapidly flexed and extended the appropriate wrist movements do not occur simultaneously. Here the co-operation of the agonists and their synergies is obviously disturbed.

Again, when the patient attempts to bring each finger in succession to the tip of his thumb all or several of his fingers flex simultaneously, and much the same may be seen when he handles a small object; this is due to the agonists and antagonists not working properly together as they must to permit the flexion of an isolated finger. Similarly, in rapid alternate movements one or other excursion is often abruptly arrested owing to disturbance in the reciprocal relations of agonists and antagonists.

And as the patient attempts to pronate and supinate his forearm quickly, various irregular and inappropriate movements often occur at the shoulder, elbow and even at the wrist, owing partly at least to defective fixation at these joints.
Finally, when—for example he flexes and extends his ankle alternately, the knee and hip often flex and extend simultaneously, owing, not to defective fixation but, as may be easily determined by palpation, to active contractions of the thigh and pelvic muscles. Here the intervention of muscles that should not be concerned in the action disturb its precision.

Other instances can be easily cited of all these forms of disturbance.

This affection of the normal harmony and correct co-operation in time and degree of the various muscular contractions concerned in movements and in the maintenance of posture is such an important factor in cerebellar symptoms that it claims careful attention. It is essentially an inco-ordination or ataxia (i.e., absence of discipline or arrangement) of the active muscles, though these terms have become so vague and have given rise to so much confusion that they must be reluctantly employed. Parenthetically it may be pointed out that ataxia, when employed in its literal sense, is more correctly applicable to these disturbances than to that component of the irregularity of movement seen in tabes dorsalis, which depends upon or is influenced by loss of the sense of position, and which is consequently most pronounced when the movements of the limbs are not controlled by vision.

A disturbance in the co-ordination of the muscles engaged in individual movements must be therefore accepted as a symptom of cerebellar disease, and its acceptation does not involve the “creation of an abstract and fictitious entity, the principle of co-ordination.” All actions are the product of the activity of several cortical and subcortical centres, and though it is the cortex which initiates voluntary movement and probably selects and integrates the adequate impulses for individual acts, the elaboration and co-ordination of the numerous factors that are concerned in each must depend largely on subcortical centres. We have learned from Sherrington’s work the elaborate integration of which even the isolated spinal cord is capable in reflex acts. His experiments have demonstrated that it is on the afferent impressions of the proprioceptive system that the control and regulation of the spinal reflexes chiefly depend, and numerous experiments and clinical observations have shown that this system exerts similar functions in voluntary movements. Sherrington has happily described the cerebellum as “the head ganglion of the proprioceptive system,” and if this view, which conforms to the anatomical connections of this organ, is accepted it would be natural that one of its chief functions should be the
exertion of a regulating and co-ordinating influence in more complex movements.

Babinski has applied the term asynergia particularly to the inability to perform simultaneously the various movements that constitute an act, and we have seen that this condition, which has been referred to here as decomposition of movement, does occur, especially in recently wounded patients. André-Thomas and Durupt attribute it partly to voluntary dissociation and partly to dysmetria. That it is often voluntary there can be no doubt; one ataxic patient for instance when given a spoon and told to place it in his mouth always brought his elbow down to his side and held it firmly applied to it before moving his hand towards his face, in order, as he explained, to control as well as possible the disorderliness of his movements. But my observations convince me that it is not always intentional, at least in its milder forms, and that it may be a result of faulty association in time and degree of the various muscular contractions that are concerned in the action.

The symptoms described under the term dysmetria are more difficult to interpret. Luciani, who introduced this term, regarded it as a natural consequence of atonia; according to him the excessive elevation of the leg is due to a too sudden relaxation of the extensors as the flexors contract, and he therefore refused to accept it as a primary cerebellar symptom as Schiff had previously considered it. Babinski, who has pointed out its importance in clinical symptomatology and the fact that the movements are generally excessive in range, admits he does not understand its nature, though he suggests that it may be due to removal of an inhibiting or braking function ("action frénatrice") of the cerebellum. André-Thomas regards dysmetria as the most important factor in the disturbance of movement; he formerly assumed (1911) that the influence of the cerebellum is manifested in moderating the voluntary impulse, and not in causing the antagonistic muscles to intervene, but later he has attributed it, as well as most of the other symptoms, to anisosthenia; the movement is excessive because the hypersthenic muscles contract too vigorously, and because it is arrested too late or insufficiently owing to the hyposthenic state of their antagonists. I have observed no facts that support this explanation, nor is it intelligible to me how hypermetria would, on this hypothesis, result from extensive lesions or widespread disease which must involve numerous pairs of his reciprocally functioning "dynamogenic centres." Further, movements in opposite directions at the one joint are generally both hypermetric; and as Babinski has pointed out excessive movements
Finally, when—for example he flexes and extends his ankle alternately, the knee and hip often flex and extend simultaneously, owing, not to defective fixation but, as may be easily determined by palpation, to active contractions of the thigh and pelvic muscles. Here the intervention of muscles that should not be concerned in the action disturbs its precision.

Other instances can be easily cited of all these forms of disturbance. This affection of the normal harmony and correct co-operation in time and degree of the various muscular contractions concerned in movements and in the maintenance of posture is such an important factor in cerebellar symptoms that it claims careful attention. It is essentially an inco-ordination or ataxia (i.e., absence of discipline or arrangement) of the active muscles, though these terms have become so vague and have given rise to so much confusion that they must be reluctantly employed. Parenthetically it may be pointed out that ataxia, when employed in its literal sense, is more correctly applicable to these disturbances than to that component of the irregularity of movement seen in tabes dorsalis, which depends upon or is influenced by loss of the sense of position, and which is consequently most pronounced when the movements of the limbs are not controlled by vision.

A disturbance in the co-ordination of the muscles engaged in individual movements must be therefore accepted as a symptom of cerebellar disease, and its acceptation does not involve the "creation of an abstract and fictitious entity, the principle of co-ordination." All actions are the product of the activity of several cortical and subcortical centres, and though it is the cortex which initiates voluntary movement and probably selects and integrates the adequate impulses for individual acts, the elaboration and co-ordination of the numerous factors that are concerned in each must depend largely on subcortical centres. We have learned from Sherrington's work the elaborate integration of which even the isolated spinal cord is capable in reflex acts. His experiments have demonstrated that it is on the afferent impressions of the proprioceptive system that the control and regulation of the spinal reflexes chiefly depend, and numerous experiments and clinical observations have shown that this system exerts similar functions in voluntary movements. Sherrington has happily described the cerebellum as "the head ganglion of the proprioceptive system," and if this view, which conforms to the anatomical connections of this organ, is accepted it would be natural that one of its chief functions should be the
exertion of a regulating and co-ordinating influence in more complex movements.

Babinski has applied the term asynergia particularly to the inability to perform simultaneously the various movements that constitute an act, and we have seen that this condition, which has been referred to here as decomposition of movement, does occur, especially in recently wounded patients. André-Thomas and Durupt attribute it partly to voluntary dissociation and partly to dysmetria. That it is often voluntary there can be no doubt; one ataxic patient for instance when given a spoon and told to place it in his mouth always brought his elbow down to his side and held it firmly applied to it before moving his hand towards his face, in order, as he explained, to control as well as possible the disorderliness of his movements. But my observations convince me that it is not always intentional, at least in its milder forms, and that it may be a result of faulty association in time and degree of the various muscular contractions that are concerned in the action.

The symptoms described under the term dysmetria are more difficult to interpret. Luciani, who introduced this term, regarded it as a natural consequence of atonia; according to him the excessive elevation of the leg is due to a too sudden relaxation of the extensors as the flexors contract, and he therefore refused to accept it as a primary cerebellar symptom as Schiff had previously considered it. Babinski, who has pointed out its importance in clinical symptomatology and the fact that the movements are generally excessive in range, admits he does not understand its nature, though he suggests that it may be due to removal of an inhibiting or braking function ("action frénatrice") of the cerebellum. André-Thomas regards dysmetria as the most important factor in the disturbance of movement; he formerly assumed (1911) that the influence of the cerebellum is manifested in moderating the voluntary impulse, and not in causing the antagonistic muscles to intervene, but later he has attributed it, as well as most of the other symptoms, to anisosthenia; the movement is excessive because the hypersthenic muscles contract too vigorously, and because it is arrested too late or insufficiently owing to the hypothenic state of their antagonists. I have observed no facts that support this explanation, nor is it intelligible to me how hypermetria would, on this hypothesis, result from extensive lesions or widespread disease which must involve numerous pairs of his reciprocally functioning "dynamogenic centres." Further, movements in opposite directions at the one joint are generally both hypermetric; and as Babinski has pointed out excessive movements
are common in chronic cerebellar disease in which there is little or no demonstrable atonia.

The main cause of hypermetria is the pathological slowness in commencing and in completing the relaxation of the contracting muscles. While delay and slowness in starting contractions may cause no pronounced disturbance of voluntary movement, a delay in the arrest of the contraction and a slowness in effecting relaxation must necessarily prolong the movement or continue it too far, and consequently make its range too great. In several of the latter cases of my series this explanation was carefully tested and in all it was found that when there was a definite tendency to hypermetria in any movement the muscles which effected it relaxed more slowly than their homologues in the normal limb.

It often seems, however, that the exaggerated movements are to some extent due to the failure of their prompt arrest by the contraction of their antagonists; when, for instance, towards the completion of the action the limb moves with gravity it is often allowed to fall inertly on the object it wishes to seize. The timely intervention of the antagonists is then absent, as it is in the rebound phenomenon, but this cannot be attributed to their hypotonia only, as André-Thomas suggests.

But the mismeasured movements are occasionally too small. This may be due to intentional arrest, which we have, in fact, seen often occurs, or to an underestimation of the effort necessary to move the slow and asthenic limb, but it is chiefly a result of the defective co-ordination of the various muscles concerned in the act, which necessarily disturb its range as well as its direction.

Deviation from the correct line of movement, which is prominent chiefly in early injuries, is a natural result of the functional disturbances which have been already considered. The limb deviates, especially in the early part of the movement, because the muscles that should be employed in fixing certain of its joints and in maintaining its correct posture contract inadequately or too late. Further, since the co-operation of those concerned in it, whether they contract or relax, is no longer accurate, the moving limb is not brought in the most direct line to its object, even though the general direction of its movement is correct.

All these disturbances are well brought out by Babinski's tests for adiadochokinesis. Rapid alternate movements are slowly, awkwardly, and irregularly performed, owing to the slowness in the initiation of each, the irregularity in their range and the disturbance in the normal
co-ordination of the muscles that should contract, those that should simultaneously relax, and those that should assure the correct posture of the limb. The more rapidly the movements are performed the greater is the need of accurate co-ordination, since any irregularity or defect in one must also influence the proper execution of the succeeding excursion of the limb.

The same factors are responsible for the affection of speech; its slurred, indistinct, and scanning character results from the imperfect harmony of the movements and attitudes necessary in normal phonation and articulation, while the tendency to explosiveness may be regarded as a manifestation of dysmetria. The unnatural effort which the patient puts into his attempts can be more easily interpreted as an attempt to control voluntarily the disturbances of the complex and highly specialized mechanism of speech, rather than to asthenia of the muscles of phonation and articulation. The grimaces and unnecessary contractions of the facial and neck muscles in a patient with a severe cerebellar injury often recall those that occur on the inco-ordinate attempts of a mild stammerer to speak.

We must consequently conclude that, in addition to its influence on tone, and that by which it assures the regularity and maintenance of muscular contractions and the immediate and effective response of subcortical mechanisms to cerebral impulses, the cerebellum also exerts a regulating and co-ordinating influence on the motor centres that effect voluntary movements and by this means assures their harmony, precision and correct range. This does not mean that the cerebellum puts into play the muscles necessary for the accomplishment of complicated movements. It is an organ which has evolved on the afferent rather than on the motor side of the central nervous system. But it receives and integrates proprioceptive impulses from all parts of the body, and by virtue of these it keeps the motor mechanisms in such a state of "tone" that they can react promptly and efficiently to voluntary impulses, and it thus assures the correct co-operation of the separate motor centres that are concerned in individual acts.

After a recent injury of one side of the cerebellum the eyes when at rest tend to deviate towards the opposite side, and nystagmus occurs on movement and especially on accurate fixation. This deviation of the eyes, the greater effort necessary to move them towards the injured side, and the frequent defect in the range of movement in this direction, must be attributed to loss of an influence, probably of a reinforcing nature, which each half of the cerebellum exerts on conjugate deviation.
towards the same side. The effect of this loss is most evident in voluntary movement, but it may be seen, too, in the adjustment of the eyes when the head is passively rotated, and when they move reflexly to a sudden visual or auditory stimulus. As an analogous deviation is produced by unilateral labyrinthine extirpation it is probable that this function of the cerebellum depends largely on its labyrinthine afferents, but since in this condition the deviation of the eyes is towards the injured labyrinth, and the associated nystagmus differs in type, it is evident that the eye symptoms of cerebellar disease are not due merely to interruption of labyrinthine impressions; it has, in fact, been found by experiment that the impulses by which the labyrinth influences the ocular movements do not pass through the cerebellum. (Wilson and Pike.)

Further, both the nystagmus and the paretic deviation produced by cerebellar lesions, though not permanent, are more persistent than when the vestibular apparatus only is destroyed. It is probable that it is to the combination and integration in the cerebellum of labyrinthine and other proprioceptive afferents, especially those from the ocular, and possibly from the neck muscles too, that this influence on the movements and position of the eyes is due.

The nystagmus is closely related to this symptom. When a patient attempts to look towards the injured side the eyes are brought over quickly, but they soon recede more slowly towards the middle line as though the muscles were too weak to maintain the position, and are then jerked back again in the desired direction. A series of these recessions and corrections constitute nystagmus. It consists of a slow phase towards the position which the eyes assume when at rest, and a quick phase in the direction towards which they should be moved voluntarily. The range of both excursions increases the farther the object to be fixed is from the middle line, that is, it is proportional to the effort necessary to bring the eyes into, and keep them in the correct position. The essential feature is the slow recession, which is only a manifestation of the spontaneous deviation, while the quick phase can be regarded as an attempt at correction. It has been shown by Wilson and Pike that in labyrinthine nystagmus the quick jerks are of cerebral origin, and the same is probably also true of cerebellar nystagmus. There can be no doubt that this is always more marked when the patient makes a voluntary attempt to fix an object than when his eyes are at rest or moved to order. Nystagmus is apparently more pronounced and more persistent in man than in animals; this
is probably related to the greater development of the forebrain, and to
the larger part that this takes in the execution of ocular movements.

The nystagmus that occurs on central fixation is similar. The eyes
still tend to recede towards the healthy side, and their recessions are
corrected by similar sharp abrupt jerks. It is important in support
of this explanation, that nystagmus on central fixation is pronounced
only when a tendency to spontaneous deviation exists.

On full deviation towards the unaffected side and on vertical move-
ments a similar nystagmus may also occur, the slow phase being
towards the primary central position, or more correctly towards that
point in space to the healthy side of it on which the eyes tend to
devote when at rest. It consequently seems that each half of the
cerebellum has an influence, not only on conjugate movements of the
eyes towards the same side but also on vertical movements and on full
deviation towards the opposite side. This influence is probably closely
allied to that by which the normal labyrinth assures the adjustment
of the eyes to change of the position of the head in space, and is
adjunct or reinforcing to the processes which effect voluntary deviations
and reflex ocular movements of other origin. That this reinforcing
action should be more pronounced in the ocular movements than in
those of the trunk and limbs is not surprising when the greater extent
to which the ordinary movements and adjustments of the eyes are
dependent on subcortical mechanisms, and especially on labyrinthine
impressions, is realized:

This type of nystagmus cannot be regarded as an ataxia of the
ocular muscles; it is usually a well co-ordinated phenomenon in which
the co-operation of the various muscles concerned in movement and
fixation is undisturbed, though it may be occasionally seen that the
adjustment of the visual axes to the object that should be fixed is
imperfect.

The mode in which lesions of the cerebellum influence the posture
of the head, trunk and limbs is more obscure. Those abnormal postures
that result from unilateral lesions are much less marked and less
persistent in man than in animals, and even in these physiologists have
not yet reached any definite or generally accepted hypothesis as to
their nature.

The most striking and constant of the abnormal attitudes is that
assumed by the head. This is as a rule inclined towards the injured side
and rotated to the opposite, so that the occiput approaches the homo-
lateral shoulder. It is noteworthy that this position also occurs after
THE SYMPTOMS OF ACUTE CEREBELLAR INJURIES

destruction of one labyrinth, and Horsley has indeed suggested that it should be regarded as a labyrinthine rather than a cerebellar attitude. It may be that it is through the cerebellum that the labyrinth exerts this posture influence.

The deviation of the limbs when unsupported and in movements uncontrolled by vision is a more important problem, since Bárány and André-Thomas have laid much emphasis on it in discussing the normal functions of the cerebellum. I have not been able to observe the anisosthenia to which Thomas and Durupt attribute it, and to explain it by the relative hypertonicity (that is, excessive tonic state) of certain groups of muscles and hypotonia of their antagonists, is, unless this condition of tone can be detected by other tests and in other movements, merely to restate the question. Since analogous deviations are produced by labyrinthine lesions, Bárány’s conclusion that they are due to the interruption of labyrinthine impulses in the cerebellum is plausible.

The tendency to fall and to deviate to the injured side in standing and walking are analogous symptoms, which may be attributed to loss of motor balance between the two sides of the body owing to the removal of an influence which each half of the cerebellum elaborates mainly from labyrinthine impressions. The other disturbances of equilibrium and of gait are due to those abnormalities of movement seen in all actions of the affected limbs, and which are more pronounced the more complicated and delicate the action is, and the greater the number of muscles it employs. My observations lend no support to the view held by many physiologists and clinicians that the predominant function of the cerebellum is the maintenance of equilibrium.

CHAPTER VI.—FUNCTIONAL LOCALIZATION IN THE CEREBELLUM.

The experiences of this war will probably settle the question of localization in the human cerebellum, or rather in its cortex.

It must be admitted that the results of physiological experiments are strongly in favour of it. Ferrier’s electrical stimulations, Bolk’s anatomical researches, and the observations of numerous physiologists as van Rynberk, Rothmann, André-Thomas and others, on the symptoms produced by small circumscribed lesions, seem at first sight to prove conclusively that a localization of function does exist in the cortex. But when they are more carefully examined so much
discrepancy is found, even between the results of experiments made in the same animal class, that they are less convincing.

Further, Horsley and Clarke's careful researches have shown that the cerebellar cortex is inexicitable to electrical stimuli; and many of the local lesions from which the most definite conclusions have been drawn extended to or involved the nuclei. And even a constant correlation between structure and functional adaptation is an unsafe argument, though a valuable guide, for the physiologist.

My own observations are of only negative value. I have attempted to determine the position of the injury in every case by careful observation at the time of operations, by a study of radiographs which revealed penetrating fractures of the skull or the presence of foreign bodies in the cerebellum, and by post-mortem examinations. For this purpose it was necessary to learn the relation of various points on the surface to underlying parts of the cerebellum; this was done by modelling plasticine to represent the covering scalp and soft tissues, on to a dried anatomical skull in which a cerebellum was placed. When the position of the entrance wound and its direction were known, an approximate idea of the region injured could be then obtained.

In many cases the primary wounds were large, and the softenings, hæmorrhages and septic processes that so frequently accompany such injuries undoubtedly increased the extent of the destruction. In others, however, only small local lesions probably existed; in three men, for instance, the missiles, a small shrapnel ball and fragments of shell casing respectively, were merely embedded in the skull, but on removing them small lacerations were found in the dura mater through which some softened cerebellar tissue escaped. In several other cases similar circumscribed lesions were produced by small depressed fractures.

On investigating the sites of the wounds it was found that the majority involved the posterior-inferior surface of the cerebellum, most were in fact referred to the lobus gracilis, but practically every region except the anterior-superior margin was affected in one or more cases. In two patients, for example, depressed fragments of bone were driven along the under surface of the tentorium, so that the injury was almost limited to the superior surface, and in several others this surface was wounded by missiles which had entered through the occipital or parietal lobes and had penetrated the tentorium. In a few cases missiles of higher velocity had passed through different portions of the cerebellum. In some patients the lesions were more or less mesial, in others they involved its lateral part only.
Final conclusions on localization can be drawn only from cases controlled by complete anatomical examinations, and I have not yet had the opportunity to complete the histological investigations of the brains which were obtained by autopsy. But, on the other hand, if there is a focal localization of function in the cortex some definite evidence of it should be obtained from such material as has been available to me for investigation. Of such localization I could, however, find no certain evidence. When small superficial lesions existed, they produced only slight and transient symptoms which were never limited to one segment of a limb, or even to one limb. On the other hand, unilateral lesions produced symptoms which were always limited to the same side, and we can consequently assume that the functions of each half of the cerebellum are exerted to the same side only of the body.

It is true that the muscles of the head, neck and trunk, including those concerned in phonation and articulation, are more seriously affected when the vermis is injured, and disturbances of their function are usually more obvious in mesial than in lateral lesions of one lateral lobe; it is therefore probable that these activities, which require the co-operation of homologous bilateral muscles, are represented in or near the vermis.

A special interest has been given to the question of functional localization in the human cerebellum by Bárány's publications. According to his views there exist centres for the direction of movement in the cortex, and a further representation of muscles, according to the articulations they move, within these centres. He has come to this conclusion after investigating the deviations of the affected limbs when they are unsupported and in movements which are not controlled by vision, and from the fact that when local lesions exist the deviations in certain directions which are normally produced by stimulation of the labyrinth no longer occur. I have found however that a unilateral injury of any part of the cerebellum almost invariably causes deviation, both spontaneous and in the pointing test, of the homolateral arm outwards, while the errors in horizontal movement are frequently inconstant. Rothmann and others have also drawn attention to the frequency of outward deviation of the upper limb. Since in my cases many separate regions of the cerebellum were involved these facts are difficult to assimilate with Bárány's hypothesis of distinct focal centres for movement in different directions.

As the majority of my patients remained under observation for only
relatively short periods after the infliction of their wounds it was rarely possible to test the rotation or caloric reactions. In three cases, however, both were examined, and in these it was found that immediately after the appropriate labyrinthine stimulation the homolateral arm, which had previously deviated outwards, no longer showed any tendency, or less tendency, to deviate in vertical movements; in other words, the normal reactions were not abolished, but instead of producing deviation inwards they merely corrected the outward deviation which previously existed.

The same condition was found in two other men whose cases are not included in this paper. One had been wounded in the head twenty months previously and had been operated upon. No information on the nature of the injury or on the extent of the operation could be obtained, but there was a trephine opening over the right half of the cerebellum. He came under observation with unmistakable symptoms of disseminated sclerosis and had probably a large plaque in the region of Deiters' nucleus. There was a marked tendency to spontaneous deviation of his right arm inwards and to progressive deviation in this direction in vertical movements, which increased after rotation to the left and on irrigation of his left ear with cold water, but disappeared on rotation to the right and on irrigation of his right ear. The irrigation of his left ear with cold water in the case of a second patient whose right half of the cerebellum had been extensively injured eight years previously also only corrected the constant deviation of his right arm outwards.

If these observations are confirmed Bárány's conclusions cannot be accepted that local cerebellar lesions abolish certain deviations of the limbs which are normally produced by stimulation of the labyrinth, since these can still be elicited, though the error in movement is masked by an actual or by a latent tendency to deviation in the opposite direction, which is due to the cerebellar wound.

But though my observations lend no support to the theory of focal localization of function in the cerebellar cortex they cannot be accepted as proof that such localization does not exist.

There can be, however, no doubt that the relative prominence of different symptoms, as tremor, slowness and inco-ordination of movement, as well as nystagmus, varies with the site of the lesion. I hope to deal with the question in a later communication.
REFERENCES.


