INJURIES OF THE SUPERIOR LONGITUDINAL SINUS.

[WITH SPECIAL PLATE.]

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SYMPTOMS OF INJURY OF THE LONGITUDINAL SINUS.

EXPERIENCES in the present war have made us acquainted with many conditions which are rarely seen in civil practice, and have especially presented to us groups of uncommon neurological symptoms or clinical pictures; these are also often less complicated and more sharply defined than those due to the ordinary pathological lesions with which we were previously familiar.

The vascular lesions of the brain met with in civil life, for instance, are most commonly due to arterial disease, to thrombosis, haemorrhage or embolism, while primary affections of the cerebral veins are uncommon; on the other hand, in gunshot injuries of the head, especially when tangential or superficial, disturbance of the cerebral venous circulation by depression of fragments of the skull is frequent, owing to the superficial course of the cerebral veins and the fact that their thinner walls and the lower pressure of the blood that flows through them make them more liable to be blocked by pressure than the arteries.

The most striking clinical effects are, however, produced when the cranial sinuses into which these cerebral veins flow are affected, and in our experience much the most common of these to be involved is the superior longitudinal sinus. This receives on either side the veins which drain the mesial aspect, as well as those which carry blood from the superior half of the lateral surface of each hemisphere, while the veins from the lower part of each lateral surface pass through the Sylvian system which opens directly, or through the sinus sphenoparietalis, into the cavernous sinus. There is often, however, a fairly free anastomosis between these two sets of lateral cerebral veins, and, consequently, complete occlusion of the one set may not necessarily produce permanent blocking of the venous outflow from the area naturally drained by it. A sudden blockage may, however, lead to circulatory disturbance sufficient to abolish, temporarily at least, the functions of a part of the area normally drained by the venous system affected.

The manner in which the superior cerebral veins open into the longitudinal sinus is important in relation to the symptoms produced by lesions in its neighbourhood. This arrangement has been fully described by one of us (P. S.). Although there is no strict constancy, the superior lateral veins usually unite into four principal trunks—a frontal,

a precentral, a post-central, and an occipital; of these, the post-central is usually the largest, and, as it drains the central gyri, the most important. As a rule these veins do not open directly into the sinus, but into thin-walled lacunae that project from it over the lateral as well as over the mesial surface of the hemisphere. A small frontal lacuna receives the frontal vein, a large parietal lacuna the pre- and post-central veins, and an occipital lacuna the occipital vein. Sometimes these lacunae are more or less continuous. The larger veins either open directly into the floor of the lacuna, from which the blood finds its way into the sinus by several small openings (Fig. 1), or they may pass beneath the lacuna and open directly into the longitudinal sinus (Fig. 2). The parietal lacunae are often more extensive than is generally supposed, and may spread at least $2\frac{1}{2}$ cm. on to the convexity of the hemisphere; consequently any depressed bone or direct injury in their neighbourhood within this distance of the middle sagittal line may block the venous circulation of the upper parts of the central gyri, and, owing to the thinness of their walls, this is more easily produced by pressure on the lacunae than on the rigid sinus.

Different groups of symptoms are produced by lesions of the different lacunae, but we intend to limit our description to those due to disturbance of the circulation through the parietal lacunae, when they are predominantly those of disturbance of motion and sensation.

We have up to the present seen over 70 cases in which the longitudinal sinus was injured or the circulation in its venous tributaries disturbed in its immediate neighbourhood, that is, in the lateral lacunae, or where the veins enter it.

In many of these cases there was no direct damage to the brain, either by the projectile or by depressed fragments of bone, but in others the disturbance of the venous circulation was associated with gross cerebral lesions.

The symptoms in these cases have naturally varied very much according to the severity of the injury and the region in which the sinus was damaged, but the chief features of the condition can be best conveyed by describing a typical case; other types and other symptoms which occur will be considered later.

CASE I.

Private J. H. was wounded by a bullet on December 12th, 1914. He became unconscious at once, and was unable, on admission to the base hospital two days later, to give any accurate information about himself. He was dull and apathetic, but answered questions quite readily.

There were two separate penetrating wounds, 4 cm. apart, and equidistant from the mid-line, and about 17 cm. behind the nasion—that is, slightly behind the mid-point. The entrance was on the left and the exit on the right, slightly larger and slightly anterior to it; the skull between them was comminuted.

His speech and the functions of all his cranial nerves were unaffected, but the retinal veins were swollen and the inner margins of the optic discs were blurred and indistinct.

His arms lay adducted to his side, flexed and pronated at the elbows, and were very rigid at the shoulders and elbows, but only slightly so at the wrist and in the fingers. He was unable to perform any voluntary

Fig. 1.—a, Superior longitudinal sinus laid open by cutting away its roof. b, A lateral lacuna, laid open in a similar manner, showing a Pacchionian tuft projecting into its floor, and at c the valve-like opening of a large cortical vein. d, Dura mater turned back to show the cerebral convolutions. e, Glass rods showing the channels by which blood reaches the sinus from the lacuna. (From a drawing by D. C. Bluett.)

movement with the right, but could flex and extend the left fingers feebly. The abdominal muscles were rigidly contracted and respiration was mainly thoracic.

Both lower limbs were very rigid and fully extended at hips, knees, and ankles, and rotated inward and adducted at the hips, so that the patellae came in contact with one another; owing to their position and their extreme rigidity they resembled strongly the lower limbs of a severe case of Little's disease. This extensor rigidity was not constant, as occasionally the limbs were found rigid in flexion, but as a rule it was so great that the limbs could not be passively flexed or separated from one another by any reasonable force. The knee and ankle jerks were much exaggerated and the hamstring-jerks were present and brisk; the flexor-jerks in the arms were also exaggerated, but the triceps-jerks were feeble; both plantar responses were extensor and the abdominal reflexes were absent. When admitted his mental state was too dull to permit a proper examination of sensation.

Three days later he had a prolonged left-sided Jacksonian fit which commenced in the face. Ten days after receiving the wound he showed definite signs of improvement; his lower limbs were still completely paralysed and extremely rigid, fully extended, adducted and rotated inwards, but he was now able to move his fingers freely, and perform feeble movements at the elbows; the shoulders were, however, still rigid and their movements paralysed.

Twelve days later all movements of the upper limbs were possible, but the proximal muscles, especially of the right, were very feeble and all efforts he made were very ataxic; both arms were still rigid at the shoulders, and the right at the elbow, and constantly lay closely adducted to the sides and flexed.

The legs, too, remained extremely rigid, extended, adducted, and rotated inwards, but he occasionally had strong flexor spasms in them, especially when turned on his side. No definite voluntary movement of either was possible, and any effort only resulted in a general contraction of all their muscles, and a slow vigorous extension if any segment of the limbs were flexed. Stimulation of either sole produced a vigorous withdrawal movement of the limb without any contralateral effect.

All the tendon-jerks were greatly exaggerated, the plantar responses were of the Babinski type, and the abdominal reflexes were abolished.

The examination of sensation revealed, especially on the right side, the disturbances found in pure cortical lesions when the stage of shock or diaschisis has passed; the lightest touches could be appreciated normally, but a certain percentage of purely tactile contacts failed to evoke a response, and this failure bore no definite relation to the intensity of the stimulus. Localization of touch stimuli was, however, not seriously disturbed. There was no diminution to painful stimuli, and no definite subjective difference in pin-pricks between normal and possibly affected parts. The appreciation of position and of passive movement was almost completely lost in both lower limbs and in the right arm, but was little affected in the left arm; and corresponding thereto the discrimination of Weber's compass points was much disturbed in the legs and in the right arm; the two points could be distinguished 1 cm. apart on the left palm, while on the right they could not be recognized at double this distance, or on the soles when separated to 10 cm.

During the time he remained in the base hospital he had slight difficulty in passing urine, and occasionally incontinence; this he explained as due to the fact that he could "only hold his water for five minutes or so," and that it then passed involuntarily if he did not receive a urinal.

He was evacuated to England five weeks after receiving the wound, and had gradually improved during this time. His subsequent history is not at present known.

In other cases we have been able to keep the patients longer under observation and in some instances learn of their subsequent course. From our own observation we are of opinion that when the brain itself has not been at the same time damaged by the missile the symptoms due to obstruction of the venous circulation diminish gradually, and will eventually disappear, almost or entirely; and this impression is borne out by the later histories we have received of patients transferred to England. The degree and rate of improvement may depend as much on the inconstant anatomical arrangement of the veins and the amount of anastomosis between the two lateral venous systems as on the severity of the lesion.

Such relatively rapid improvement in a severe case may be illustrated by

CASE II.

Lance-Corporal S. was wounded by a bullet at short range on December 21st, 1914. He was unconscious for a short time and was afterwards unable to move either leg or his right arm. Speech was, however, unaffected, and he had only slight headache.

There was a sagittal gutter wound of the scalp 6 cm. long, its anterior end slightly to the left of the mid-line and vertically above the tip of the mastoid, and its posterior end on the mid-line, with fracture of the outer table and probably depression of the inner table.

When he entered the base hospital, two days after receiving the wound, his face and tongue were unaffected, but the right arm was completely paralysed. The power of his left arm was unaffected, but there was considerable sensory ataxia in its movements. The lower limbs were also powerless and the right especially was rigid.

All the deep reflexes were exaggerated, stimulation of the soles gave extensor responses, and the abdominal reflexes were absent.

The sense of position and the discrimination of compass points were lost in the right arm and in both lower limbs, but tactile and painful stimuli were normally appreciated everywhere.

He was occasionally incontinent, which he attributed to the fact that often he did not feel when he should pass urine.

Five days after the wound there was some return of power in the right fingers, wrist and elbow, but the movements he could execute were weak. His legs were still rigid and motionless. A week later all movements of the right arm were possible and those of the distal segments almost quite strong, but the limb was very ataxic owing to severe disturbance of sensation in it. His legs were less rigid and he was now able to flex and extend both hips, but no movement of the knees, ankles, or toes was possible. The sense of position, the appreciation of passive movement, the discrimination of compass points, and the localization of tactile stimuli were seriously disturbed in his right arm and in both lower limbs, but light touches, painful stimuli and vibration were normally appreciated. The recognition of form (stereognosis) was also defective, but not quite lost, in the right hand.

Five weeks after being wounded his right arm was slightly weak only at the shoulder, but was still ataxic, and he could now execute all movements at the hips and knees, but they were weaker than normal; the ankle and toe movements were still completely paralysed. The deep reflexes were still much exaggerated, and ankle clonus was present on both sides, as well as the Babinski sign.

The rapid improvement of his symptoms continued till he was evacuated to England two months after being wounded. His right arm was then quite strong and all movements of both lower limbs were possible, though the distal segments were still slightly weaker than normal. His legs could now bear his weight, but he needed assistance in walking, owing to the marked ataxia of these limbs due to the sensory disturbances, which, though less pronounced, were still considerable.

We have learned from Dr. H. Head, under whose care he came in England, that five and a half months after the infliction of the wound he could perform all movements of both lower limbs, but he could not yet walk alone owing to the pronounced sensory ataxia of his legs.

PATHOLOGICAL CHANGES TO WHICH SYMPTOMS ARE DUE.

Before we discuss the symptoms in this large group of cases it will be advisable to consider the pathological changes to which they are due. We have been able to study these pathological lesions in a certain number of cases by *post-mortem* examination and have obtained microscopical preparations of the affected areas in a few, but the opportunity for a complete histological examination has not yet occurred.

We have already stated that in many of the cases in which the chief symptoms were due to injury of the longitudinal sinus or its tributary veins, associated lesions of the brain existed, and in describing the essential pathological changes it will be obviously necessary to separate such cases from those in which the venous system only has been directly damaged.

The most common type of injury is a gutter or tangential wound at the middle line of the head, which may be either

sagittal, coronal, or oblique. In many cases the skull, though exposed, showed no evidence of fracture of the outer table, either to inspection or on x-ray examination, but the latter generally revealed a depression of the inner table at or near the middle line. In other patients both outer and inner tables were depressed, and if the damaged bone was removed it was seen to present a spoon-shaped depression in the skull, the inner table being more depressed than would be expected from a superficial examination. Such depressions as a rule merely compressed the sinus or its lacunae and rarely injured their walls. Frequently, however, the injury was a perforating wound or an in-and-out wound of the skull, the entrance and exit being on opposite sides of the middle line and close to it, with considerable comminution of the bone between them; here the sinus was generally lacerated, and in certain instances it was completely cut through by the missile, but it was occasionally only compressed by indriven fragments of bone.

On removing the skull-cap *post mortem*, or on operation, a thrombus was usually found in the longitudinal sinus, its character depending on the duration of the case, but as the sinus is divided up by irregular transverse trabeculae and held open by its rigid walls and the support it receives from the dura mater, it is probable that in many cases the thrombus did not occupy its whole lumen. When the injury lies to one side of the middle line the thrombosis may be limited to the lateral lacuna beneath it.

When the dura mater was removed the most striking feature was the condition of those superficial cortical veins which enter the sinus at the position of the wound; these were swollen, firm to touch, and could not be emptied by pressure, and there frequently seemed to be congestion of the neighbouring veins which were not actually thrombosed (Figs. 3 and 4 in special plate). The superior parts of the hemispheres which are drained by these veins were usually swollen and their convolutions flattened by pressure against the inner surface of the skull, and generally firm to touch. On section there was obviously much oedema of the cortex and subcortical white matter, and minute haemorrhages, which were grouped more closely in the neighbourhood of the wound, were found (Fig. 5 in special plate). In a few instances there was an actual softening near the mesial fissure, the disintegrated brain matter being blood-stained, and punctiform haemorrhages were found in the cerebral tissue around it.

In a few of the cases which came to *post-mortem* examination, and in others in which the condition could be observed during operation, there were widespread subdural haemorrhages, which usually formed a thin layer of blood over the convexity, and sometimes extended to the base of the same hemisphere; in other cases cerebrospinal fluid removed by lumbar puncture was either blood-stained or straw-coloured. These superficial haemorrhages undoubtedly produced some rise of intracranial pressure and contributed to the severe headache with which many of these patients suffered, but they seemed to play a subordinate part in the production of the other symptoms. Thin layers of haemorrhage into the soft membranes and even subpial haemorrhages are common.

Under the microscope the most striking change is the oedema of the affected areas, which is more prominent in the white than in the grey matter. Many of the superficial veins may be thrombosed and the rest are much congested. Minute haemorrhages occur in both the grey and white matter, but chiefly in the proximity of the wound; many are mere perivascular extravasations. Occasionally slight perivascular round cell infiltrations are met with, especially in the neighbourhood of softenings. The walls of the latter consist of disintegrated tissue and large numbers of granule cells.

The nerve cells in the affected areas show pronounced changes; they are generally swollen and in advanced chromatolysis, the Nissl bodies having disappeared, or being represented merely by irregular clumps at the periphery of the cell. Many cells, however, appear almost homogeneous and hyaline, and, in relation to the age of the patient, often contain an excess of pigment when death did not occur soon after the infliction of the wound.

DISTRIBUTION OF MOTOR SYMPTOMS.

When we look at the clinical symptoms we are at first most struck by the unusual distribution and the type of the motor paralysis.

The extent of the palsy naturally varies according to the site, severity, and extent of the lesion; we at present have notes of 20 cases in which all limbs were affected; in 31 both legs and one arm were weak; in 16 only the lower limbs were affected; in 6 the symptoms were mainly hemiplegic, and in 5 one leg alone presented any palsy.

The distribution of the paralysis and its relative severity in different segments of the limbs is, however, peculiar, and differs from that of the cerebral palsies most commonly seen in civil practice. As in Case 1, when the upper limbs are affected, the finger movements either escape, or are weak for only a short time after the injury, and rapidly recover and regain their normal power. The hand movements have never remained long weak except when the sinus condition has been complicated by an

independent injury of the brain. The wrist movements, and especially those of the elbows, are affected more severely and recover less rapidly, while those of the shoulder often suffer when the more distal segments of the limbs escape, and recover much less quickly when the whole limb has been involved.

It is consequently the more proximal segments of the upper limbs which are most seriously paralysed, and the weakness diminishes distalwards. In this respect the paralysis contrasts strongly with that seen in the ordinary hemiplegia due to vascular lesions, in which the distal segments of the upper limbs are almost invariably more severely affected than the proximal, and recover less rapidly. Further, a definite paresis of the face or tongue is extremely uncommon, and is at the most transient, while speech is never affected in the pure sinus injuries. The trunk muscles may be, however, affected, especially those of the back; the patient is then unable to sit up and often cannot roll over in bed.

The distribution of the palsy in the legs is the converse of that of the arms; here it is always the distal movements that suffer more severely, and in slight cases and during the recovery of more severe ones we have

Fig. 2.—a, Sinus laid open as in Fig. 1. b, An elongated lateral lacuna similarly opened. c, Glass rod inserted into the mouth of a large cortical vein, e, which is seen running beneath the lacunar floor. d, Glass rods showing openings from the lacuna into the sinus. (From a specimen in St. Thomas's Hospital Museum.)

repeatedly seen complete paralysis of the toes and ankle only, with the knee movements only relatively weak and those of the hips strong.

This distribution of the paralysis and its relative severity in the different segments of the limbs obviously depends upon the arrangement of the cortical motor centres and on that of the cortical veins. The motor centres for the lower limbs are arranged from above downwards on the apex and lateral surface of the hemisphere in the following order: movements of the toes, ankle, knee, hip; then the small motor centres for the trunk movements intervene, and below them come in succession the motor centres for the shoulder, elbow, wrist, and fingers. On the other hand, the superior cerebral veins which open into the longitudinal sinus drain the central gyri, as a rule, to just below the inferior genu of the fissure of Rolando, which corresponds approximately to the centre for the wrist movements, and if the area they drain be put out of function a complete paralysis of the voluntary movements of the opposite lower limb, shoulder and elbow, with weakness of the wrist movements, and little disturbance of those of the fingers, might be expected.

The distribution of the lateral cerebral veins and the amount and the freedom of the anastomosis between the superior and the inferior systems apparently vary greatly, and this will naturally influence the extent of the paralysis, even though there be complete occlusion of the superior system; but if the lesion is slight and the occlusion is incomplete, the circulation through the smaller and more slender veins that drain the apex of the hemisphere and its mesial aspect would be more liable to become blocked than that of the larger and more thick-walled vessels; in this case only the motor centres for the more distal segments of the lower limb may suffer.

Further, while a mesial lesion is liable to produce bilateral symptoms, a one-sided paralysis of the same type may result from an injury to the one side of the middle line, which blocks the circulation through a lateral lacuna or in the veins as they enter the sinus.

When these symptoms which we attribute to venous lesions, and especially weakness of both the lower limbs, result from an injury of the vertex of the skull in the neighbourhood of the upper ends of the fissures of Rolando, they might be attributed to direct damage or to compression of the motor centres which lie under the wound. But, in the first place, the type of the paralysis, and especially the rigidity associated with it, is unlike that which occurs when other parts of the motor cortex are injured or compressed, and in the second, as in Case 1, the paralysis is often too extensive in relation to the severity of the wound to permit the assumption of a direct injury. Thirdly, as the lateral parietal lacunae overlie the more mesially situated motor centres they must obviously be involved by any injury that would directly damage or compress the cortex.

RIGIDITY.

Even more striking than the unusual distribution of the paralysis is the rigidity which is almost always associated with it. It is generally coextensive with the paralysis, and closely related to it in its degree. Thus it is always most pronounced in the lower limbs, and, when the upper are also involved, it is greater at the shoulder than at the elbow, and is rarely present and never pronounced in the wrist or fingers.

The early onset of this rigidity is another striking point; we have seen it well marked within twenty-four hours of the infliction of the wound, and in some instances at least it has been noticed by the patient almost at once. It has

shown no tendency to increase after the patients have reached the base hospitals, that is usually within forty-eight hours, but on the other hand it gradually diminishes *pari passu* with any return of power that may occur. The rigid limbs generally assume very characteristic attitudes; when the arms are affected they lie closely adducted to the sides and rotated inwards, with the elbows flexed and pronated, and in severe cases the wrists and fingers in moderate flexion. Even the trunk muscles may be involved, and then the abdominal wall is unnaturally rigid and respiration is mainly thoracic; in a few cases, indeed, there has been slight difficulty in coughing, and phonation has been monotonous and toneless owing to the poor inspiratory intake.

As a rule the lower extremities lie fully extended at all joints, firmly adducted and rotated inwards, with the feet occasionally crossed; in fact, the attitude is practically identical with that which is so characteristic of a severe cerebral diplegia. In certain cases, however, the knees are partially flexed, but they are always adducted and rotated inwards.

The rigidity is often so great that the resistance to passive movement is extreme; it may, for instance, be quite impossible for a man of moderate strength to separate the knees. If passive movement is possible, it is found that the rigidity involves all groups of muscles and is more or less equal whether the limb is passively flexed or extended. It is also continuous throughout the whole range of any passive movement that is made, and it has never shown any tendency to the "clasp-knife type."

We have also observed that peripheral stimulation, as pricking the sole or palm, increases the rigidity, and the legs may become more rigid when the patient coughs or attempts any strong voluntary movement with his arms.

It is interesting and important that, despite the great rigidity, there seems to be very little tendency for contractures—that is, organic shortening of the muscles—to develop; in one severe case, of which, through the kindness of Dr. Head, we have heard six months after the

infliction of the wound, no contractures have occurred, although there was for a considerable time great rigidity of the legs.

Reflex spasms of the lower limbs have been associated with the rigidity in a certain number of cases; they have been of the flexor type, and in one case at least occurred from the day of the infliction of the wound. They may be so severe as to cause considerable discomfort to the patient, but gradually diminish in frequency and severity as improvement sets in. Reflex withdrawal of the legs can be easily evolved by peripheral stimulation, especially of the soles, but in only one case have we a definite record of an associated contralateral extension of the opposite limb.

In the rigid cases the tendon-jerks have been much exaggerated from the earliest moment at which they have come under observation, a striking feature, as in ordinary hemiplegia and in cerebral palsies directly due to gunshot injuries of the head these reflexes are frequently absent for some time; but when the limbs are in rigid extension the extensor reflexes—that is, the knee and ankle jerks—are much brisker than the hamstring-jerks, while if in flexion the latter jerk has been the more exaggerated. Similarly, in a case in which the elbows were rigid in flexion, the flexor reflex—that is, the biceps-jerk—was much increased, but the extensor reflex—the triceps-jerk—could not be obtained.

The great toes are usually permanently extended, and a typical extensor response is usually obtained on stimula-

DESCRIPTION OF SPECIAL PLATE.

Fig. 3.—Photograph of the left side of a brain, with the dura mater thrown back over to the right to show its inferior surface and the lateral surface of the left hemisphere. The frontal and parietal lacunae are surrounded by broken lines; the entry of the larger cerebral veins into their under surfaces is clearly seen. Those entering the frontal lacuna, as well as the smaller vein which passes from the precentral sulcus into the parietal lacuna, are thrombosed, but the main post-central veins escape. The dura mater was not torn, but the sinus was compressed between the points A and B by a depressed fracture of the vault. For the sake of clearness the smaller veins which entered the sinus or lacunae have not been preserved in this dissection, or in that from which Fig. 4 is taken.

Fig. 4.—Photograph taken as in Fig. 3. In this case the frontal and parietal lacunae were not definitely separated, and are both enclosed by a broken line. The longitudinal sinus and the lacunae were compressed by a depressed fracture between A and B, and the veins which enter it at this region are completely thrombosed. The swollen oedematous condition of the brain in the region of the thrombosed veins is obvious.

Fig. 5.—A sagittal section of a brain to show the multiple small haemorrhages in the affected region, a subarachnoid haemorrhage on its surface, and oedema of both grey and white matter extending backwards behind the fissure of Rolando.

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tion of the soles, but in a few cases there has been a definite flexor response, although there was unquestionably motor paralysis of the distal segments of the lower limbs. Naturally this is also obtained in another type, to which we shall later refer, in which the inability to move the limbs is due to sensory disturbance only.

Perhaps the most interesting physiological problem presented by a study of these cases is this extreme muscular hypertonus which appears very early after the injury and is closely related to the paralysis of voluntary movement. It is obviously impossible to enter here into the complex problem of the pathogenesis of rigidity, but it is now generally assumed that, as Hughlings Jackson originally taught, the increase of tone is due to removal of the inhibition which higher centres, and in this case the cerebral cortex, normally exert on the lower nervous mechanisms which maintain tone in the muscles. In an ordinary hemiplegia due to a cortical or internal capsular lesion the affected limbs are, apart from some transient early rigidity, flaccid for ten days at least, and lose their tendon reflexes for a shorter period, and we have found the same condition in severe traumatic cerebral lesions. There must be consequently some essential difference in either the site or the nature of the pathological condition which produces paralysis in these cases, which we may group together under the title of the "Longitudinal sinus syndrome." It appears improbable that the level of the injury—that is, the portion of the upper motor neurone that is involved—is the essential factor, as it is only the cell bodies and the upper portions of their axis cylinders which suffer with thrombosis of one of the cortical arteries, and we assume that it is only the cell itself that is temporarily put out of function by a prolonged local epileptic attack, in both of which conditions the palsy is flaccid. This temporary flaccidity, which is later followed by an exaggeration of muscle tone, is attributed to the effect of shock, which depresses for a time the activity of the lower centres that reflexly maintain tone.

If we look for any possible peculiarity in the nature of the cerebral lesion in these sinus cases we are at once struck by the remarkable absence of evidence of such shock. This is especially seen on investigating the disturbances of sensation which they present; while in the early stage of an ordinary case of cortical hemiplegia the sensory loss is partly due to functional disturbance produced by shock in the subcortical sensory mechanisms, in the cases we are considering here the sensory loss, even in the earliest stage, is almost invariably such as can be attributed wholly to a pure cortical lesion. It must be remembered, too, that the damage to the cortical cells associated with this sinus thrombosis is not complete or irrecoverable; the nature of the histological changes, and the fact that a remarkable degree of recovery of function may occur is evidence of this, but it is improbable that the type of cell change produced by the oedema and ischaemia of the brain can be the explanation of this early persistent rigidity. On the other hand, we are probably correct in assuming that the venous thrombosis produces a pure cortical paralysis unaccompanied by any shock effect on the subcortical centres which subserve muscle tone.

Disturbances of Sensation.

The sensory disturbances in these cases are especially interesting, as they are almost always those of a pure cortical lesion unaccompanied by any shock effect. The appreciation of pain and temperature is unaffected, and there is no definite diminution of tactile sensibility, but a certain number of light contacts are not recognized; there is, however, no threshold alteration, and the proportion of those missed is not directly related to the intensity of the stimulus. On the other hand, the localization of tactile stimuli, the recognition of the position and of passive movements of the limbs, and of form, shape, and size, as well as the discrimination of the compass points, may be seriously disturbed. The slowness of the affection of cutaneous sensibility has been frequently astonishing, as many patients have complained spontaneously of numbness or of having "no feeling" in their legs.

When the wound has been some distance behind the upper ends of the fissures of Rolando, sensory symptoms have been the most prominent feature. One man, in whom there was a superficial coronal wound of the skull

6 cm. behind the mid-point, had no demonstrable weakness of his legs or change in their reflexes, but he complained that both legs were numb, and on examination profound loss of the sense of position and in the discrimination of compass points was found. Owing to this sensory loss he was unable to walk, and on trying to do so only staggered and fell about the room; Romberg's sign was also well marked.

It is, of course, known that when the sense of position and the appreciation of movement are suddenly and completely abolished in a limb, aimless involuntary movements of it may occur spontaneously. This was well illustrated by one case in which an oblique tangential wound crossed the mid-line 7 cm. behind the mid-point. It is possible that the brain was directly damaged by indriven fragments of bone, but more probable that the symptoms were due to venous thrombosis. There was no weakness or rigidity of his legs, though both were very ataxic, and the reflexes were normal, but in both there was pronounced sensory loss of the cortical type. When his legs were uncovered both were jerked about at irregular intervals in a curious aimless and irregular manner. Sometimes the one was raised from the bed and either thrown across or separated from the other; at other times it was quickly drawn up and extended again, or the foot was dorsiflexed or the toes moved about. The patient became conscious of the movements only when one leg touched the other or when it fell to the bed. In their impulsive, aimless, and inco-ordinate character these movements were very similar to those of chorea.

DISCUSSION OF OTHER SYMPTOMS.

In a certain number of cases the functions of the bladder were affected. In the majority of these there was at first some difficulty in passing urine, or even retention necessitating the use of a catheter in one case for as long as five days, but this symptom always disappeared rapidly. Less frequently incontinence occurred, owing to deficient cerebral control, and in a few patients persisted for a considerable time; the bladder apparently emptied itself reflexly when it had filled to a certain point, and some patients, as Case 1, explained that they were able to hold their water for a few moments only after the desire to micturate had come.

As a rule the functions of the cranial nerves were unaffected, but in several patients the ocular movements were disturbed. In one group there was either temporary weakness or paralysis of the associated conjugate movements of the eyes without ptosis or affection of the pupils; one patient, in whom all four limbs were affected, was unable to move his eyes to order in any direction except slightly downwards, but he could follow, though not fully, a finger which was moved to either side or upwards. The visual axes always remained parallel. Within a fortnight, however, all movements had returned, and only upward deviation was at all defective. There was a similar inability to perform all conjugate ocular movements in another patient in whom all four limbs, excepting the fingers, were paralysed, and this persisted till his death on the fifth day after the infliction of the wound. More commonly, however, there was only weakness of the lateral conjugate movements of the eyes to one or both sides, or much effort was needed on the part of the patient to perform them.

We have not yet been able to make the histological examinations necessary to determine the cause of this palsy of the conjugate movements, but it seems probable that it is due to a temporary paralysis of the centres for ocular movements in the posterior part of the second frontal convolutions. We have occasionally observed a similar defect in local lesions of this region.

In other cases—8 in all—we found an isolated palsy of one or other oculo-motor nerves, generally of the third, or of the third and fourth cranial nerves. This proportion is very striking when we consider the comparative rarity of ocular palsies in other types of gunshot wounds of the head; it seems probable, therefore, it is related to the lesion we are considering. On the other hand, it might be due to a fracture of the base of the skull, to basal meningitis or haemorrhage, or to the effect of the considerable rise of intracranial pressure which is so often present in these cases. In some cases, however, in which a *post-mortem* examination was made, we could exclude

basal lesions, and in only 2 of the 8 cases was the sixth nerve affected, although it is, of course, known that this nerve is much the most liable to suffer from a pathological increase of intracranial pressure.

Fits were observed in ten of the patients; in two both sides of the body were involved, but they were limited to one side in the others. From the descriptions we received, as well as from our personal observations, the unilateral seizures apparently commenced in the face or hand when there was extensive palsy of the convulsed side, or in the lower limb when the paralysis was limited to its distal segments; that is, the excitation started either in motor centres which were only partially damaged, or in their immediate proximity.

Other complications are relatively rare. Meningitis occurred in some in which the dura mater had been lacerated and the brain damaged directly; and in one a general pyaemia, from which, however, the patient recovered, developed secondarily to a septic wound of the sinus. It is surprising that we have encountered only this one case of general infection considering how commonly pyaemia occurs in connexion with septic bone related to the lateral sinus. A secondary hemiplegia occurred in two cases; in one after an operation in which the longitudinal sinus bled freely and had been plugged with gauze, and in a second thirteen days after the infliction of the wound and ten days after an area of depressed bone compressing the sinus had been removed.

The general symptoms of intracranial pressure have been as a rule pronounced. Most of the patients have suffered considerably from headache, and in some it has been particularly severe. In five cases there was also definite optic neuritis with considerable swelling of the discs, and not merely such congestion and blurring of their edges as is seen in a large proportion of all gunshot wounds of the head. In four of these cases at least we could exclude meningitis and secondary cerebral abscesses, and must consequently attribute the ophthalmoscopic changes to the oedema and swelling of the brain.

TREATMENT.

The treatment of injuries of the longitudinal sinus presents considerable difficulties. When there is a defect in the skull to one side of the middle line, and the brain is lacerated by indriven fragments of bone, the wound should be dealt with as if it lay in other regions of the head, but special care is necessary to avoid and control the serious haemorrhage that is apt to occur from the sinus or its lacunae.

If the symptoms are due, however, only to compression of the sinus or its lacunae the immediate removal of the compressing bone would at first sight appear to be the rational treatment; but experience has shown that the results of surgical interference have been extremely unsatisfactory. Among 39 cases we observed which were operated upon either by ourselves or others, 15 deaths occurred in the base hospitals, while only one among the 37 unoperated upon cases died before transference to England. These figures have not, of course, an absolute value, as it was naturally the most serious cases which were on the whole selected for operation, and in 7 of the fatal ones there was, in addition, some direct injury of the brain. They are, however, sufficient to emphasize the danger of operation.

On the other hand, it must be remembered that the uncomplicated cases show a remarkable tendency to improve, probably owing to the free venous anastomosis permitting a re-establishment of the circulation.

If operation is necessary, it is advisable to remove bone all round the depressed portion, and only then elevate this; for if haemorrhage occurs the surgeon is then in a more favourable position to control it. As a rule, some bleeding from either the sinus or its lacunae occurs when the fragments of bone are removed, but it can generally be arrested by placing a piece of pericranial tissue or muscle on the laceration, keeping it in position for a short time by moderate pressure, and then carefully replacing the scalp flap over it. The method is certainly preferable to arresting the haemorrhage by a gauze plug, as it is not so liable to produce further thrombosis. When the sinus is completely divided or much lacerated a plug may be, however, necessary.

When there are serious symptoms of intracranial pressure a subtemporal decompression may be necessary;

it was performed in a few of our patients. It is, of course, a safer operation than a large opening in the neighbourhood of the wound, as the latter exposes the patient to the risk of intracranial infection from the septic scalp.

We have, however, found lumbar puncture, repeated frequently if necessary, sufficient to relieve the pressure symptoms in several cases.

It might naturally be expected that an injury which damages the sinus, or the lacunae and their tributary veins, might be complicated by the occurrence of a subdural haemorrhage, especially as the somewhat similar injuries in the newly born which produce cerebral diplegia have been shown by Harvey Cushing¹ to be frequently associated with such haemorrhage; and one would on these grounds be tempted to perform a subtemporal decompression in cases which show evidence of a high degree of intracranial pressure, especially when prolonged or increasing. Both operative experience and *post-mortem* examinations, however, have made it clear that such haemorrhage is rare. When it does occur it takes the form of a thin film of blood spreading over the cortex down to the base of the brain—a condition in which surgical interference is unnecessary and undesirable. In only one case in our series was there a haemorrhage of such magnitude as to need operative relief, and in this case the progress of the symptoms gave definite information before the operation was undertaken.

REFERENCE.

¹ *Amer. Journ. of the Med. Sciences*, October, 1905.

THE TREATMENT OF GUNSHOT WOUNDS OF THE HEAD, WITH SPECIAL REFERENCE TO APPARENTLY MINOR INJURIES.

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The number of patients who arrive at the base hospitals with gunshot wounds of the scalp is large. As their injuries are apparently superficial and their symptoms few or none, they frequently come down as "sitting" cases, and on arrival there is a tendency to overlook the fact that among them there is a fairly high percentage of cases with definite injury to the skull or to the skull and brain.

The following analysis of the lesions found at operation in 140 cases demonstrates that any scalp wound should be viewed with grave suspicion and the necessary measures to determine the presence or absence of a fracture of the skull taken.

Scalp wounds only	82
Fracture of outer table only	19
Fracture of inner table only	1
Fracture of both tables, dura uninjured	18
Fracture of both tables, dura lacerated	1
Fracture with laceration of dura and brain..	19
	140

Excluding scalp wounds only, the percentage of head injuries = 41.5; excluding scalp wounds and fractures of outer table only, 27.8.

A careful examination of the patient should be made, and any disturbance of cerebral function or lesion of cranial nerves observed. The optic discs should be examined. The anamnesis is important. If unconsciousness is caused at the time of injury by the force transmitted by the bullet or piece of shell which is concentrated on a very small area of the skull, the chances are considerable that at least fracture, and possibly injury of the brain, has been caused. The latter is more probable if the unconsciousness has been deep or prolonged. Transient local paralysis, or the occurrence of local spasm or epilepsy does not necessarily indicate gross trauma of the brain, but in such cases at least depressed fracture or localized haemorrhage may have occurred. Persistent headache should be viewed with suspicion.

Owing to the difference in velocity of the missile, and therefore to the difference in the momentum imparted to the tissues displaced when the skull is struck, an injury to the skull or brain is usually far less severe when caused by a non-penetrating shrapnel bullet or piece of shell than when caused by a non-penetrating rifle bullet which strikes the skull tangentially. The injury is the same in kind in both cases.