INJURIES TO THE SUPERIOR LONGITUDINAL SINUS

BY

G. B. BARKER, M.B., B.Ch., D.P.M.

Late Assistant, E.M.S. Neurosurgical Unit

During the six months following D Day, 1944, a considerable number of cases of head injury were received at an E.M.S. neurosurgical unit. These presented every type of injury, with varied signs and symptoms, but among them were a number of injuries which had involved the superior longitudinal sinus (S.L.S.) to a varying degree. These were an interesting group, and 11 of the 26 cases seen were selected for study as presenting fairly clear-cut examples of the S.L.S. syndrome without the complicating factors of other injuries. As a group these cases did very well, and this study is directed with special reference to their follow-up over the last four years.

Anatomy

The S.L.S. begins in the anterior fossa of the skull, at the crista galli, where it communicates with the veins of the nasal cavity or with the angular veins. It runs longitudinally backwards over the convex surface of the cerebrum, as far back as the level of the internal occipital protuberance, and then curves laterally, usually to the right, to form the transverse sinus. Its anatomy has been investigated by Sargent (1910-11), who describes several features of importance. It receives the superior cerebral veins from the mesial surface and from the upper half of each cerebral cortex. The veins from the lower half drain into the Sylvian system and there is an inconstant but adequate anastomosis between the two systems, so that the occlusion of one set of veins is not necessarily accompanied by blockage of the venous outflow from the area which it drains. The upper set of cortical veins group together to form four main channels—the frontal, pre-central, post-central, and occipital veins.

The S.L.S. is accompanied on each side by three main venous channels or lacunae—the frontal receiving the frontal cortical veins, the parietal receiving the pre- and post-central veins, and the occipital receiving the occipital cortical veins. These lacunae vary in size, but Sargent reports one case in which the parietal lacuna on each side measured 15 cm. in length and 2.5 cm. in breadth at its widest part.

The cortical veins terminate in two main ways. Either they run beneath the lateral lacunae and empty into them by means of openings in their floor, the blood then draining through a number of openings into the S.L.S., or they run completely beneath the floor of the lacunae and open direct into the S.L.S.

Injury

Gordon Holmes and Sargent (1915) reported over 70 cases of injury to the S.L.S. from wounds without infection, and discussed the possible pathology of the symptoms in the light of this anatomical knowledge. The present paper deals with a far smaller group, but one which has been followed up for a considerable period. The data have been accumulated from our own notes and from the field medical cards that accompanied the cases. Work was carried out under pressure and the notes are therefore not always as full as one might wish.

Of the 11 patients five were wounded while under shell or mortar fire, two suffered from bomb blast, one from a bullet wound, and one from some bridging equipment

falling on his head. In two cases the history was unobtainable. Four patients retained consciousness and were able to give good and coherent histories. Loss of consciousness in the others varied from ten minutes to four days, but with the exception of this last case and one other the period of unconsciousness was brief, and retrograde and post-traumatic amnesia was very short.

The injury was described as a "lacerated wound" in six of the 11 cases, and these included three cases in which consciousness was retained. Two cases had protruding brain substance and two had penetrating wounds.

Subsequent x-ray examination revealed the presence of a bony depression in the region of the S.L.S. in seven cases; three others had severe bony injury with in-driven bone fragments, and in only one case was there no evidence of bony injury.

Gordon Holmes and Sargent described three main types of wound which correspond to those from which our own cases suffered: (1) superficial tangential laceration with no evidence of fracture; (2) bony depression with compression of the S.L.S. or of the lacunae, without injury to its walls; and (3) penetrating or in-and-out wounds, with the entrance and exit wounds on opposite sides of the midline and close to it.

Operation

It is not necessary to give detailed reports of each operation, but treatment varied from simple excision of the wound edges to a more elaborate operation involving removal of damaged bone, brain tissue, and foreign bodies. In all cases, except one in which a drain was left *in situ* for two days, the wounds were closed in two layers with closure of the dura. Full courses of penicillin and sulphathiazole were given as a routine.

In the description of the operation specific mention of the S.L.S. was made in five cases. In each case the impression gained was that the sinus remained patent, which corresponds to the description given by Gordon Holmes and Sargent, whose series of cases was larger and who had the opportunity of performing post-mortem examinations in some cases. At necropsy they usually found a thrombus of the S.L.S., but in view of the presence of trabeculae within its walls they thought that probably the whole lumen was not obstructed. They also described a case in which injury lay to one side of the midline, with thrombosis limited to the lateral lacunae beneath it.

The superficial cortical veins which enter the sinus in the region of the wound were found to be swollen and firm, and could not be emptied by pressure. The area of cortex drained by these veins was swollen and firm, with flattened convolutions.

Examination

Of the 11 cases five had signs involving both lower limbs only, four had involvement of both lower limbs and one upper limb, and two had involvement of all four limbs.

Motor Symptoms.—There was a loss of power in all cases, varying from a slight diminution to complete paralysis. The distribution of the weakness was constant in that in the lower limbs the distal segments were affected to a greater degree than the proximal, and in the upper limbs the opposite was the case. A typical case was that in which there was no movement of either leg below the knee, although power at the hips was such that both legs could be raised from the bed. In another case there was a complete paralysis of both lower limbs, with paralysis of the right upper arm: and in a third case there was a paralysis of both legs, with reduction of power, complete in the left upper limb and affecting all the joints of the right upper limb, particularly the shoulder.

All cases showed an increase in tone varying from a slight degree to a marked spasticity. This rigidity was typical again in that it appeared in each case almost immediately, was maximal at the start, and then showed a progressive improvement *pari passu* with recovery of function. As would be expected in this clinical picture of a spastic paresis, the reflexes in the affected limbs were increased, the plantar responses were extensor in seven cases, and there was ankle clonus in at least four.

Sensory Symptoms.—Only two cases showed no sensory impairment; the others showed, typically, impairment of postural sensibility and loss of tactile discrimination and localization. Subjective numbness and paraesthesia also occurred in two cases. There was loss of postural sensibility in eight cases; in seven of these it was the toes that were affected, in three the fingers, and in one case all the joints of one arm. The duration of the symptoms depended on the degree of injury.

In one case with a depression of the inner table just to the left of the midline for a distance of about 0.5 cm. symptoms consisted of impairment of joint sense in the toes of both feet and some diminution to light touch and pin-prick on the right side below the ankle and on the left on the outer border of the foot. At operation it was found that a small bone fragment had pierced the dura and was embedded in the top of the sensory cortex. It was removed, and eight days later there was gross diminution to light touch over both feet and loss of joint sense in the toes and fingers of both limbs. Joint sense at the knees appeared normal. Four days later sensory symptoms were normal except for slight diminution to light touch over the outer border of the left foot, and after a period of re-education in walking the patient appeared quite normal, with no physical signs. In more severe cases, however, loss of postural sensibility in the toes remained to add to the difficulty experienced in walking.

Lumbar Puncture.—This was performed in six cases, but only as a post-operative measure. The pressure was raised to above normal limits in only two cases, and in these it fell to normal within three and 14 days. This latter case, in fact, had a penetrating wound in the right mid-parietal region with in-driven bone fragments, and was the most severe case of cerebral injury in the series.

Urinary Disturbance.—Only two cases had any urinary trouble; one had a very mild degree of incontinence lasting a few days, and the other had a more prolonged period lasting several weeks.

Epileptic Attacks.—One case had a single focal epileptic attack which lasted for about 15 minutes on the day after injury and consisted mainly of flexion movements limited to the right fingers and arm. None of the cases have had any epileptic attacks for the four years for which they have been followed up.

Follow-up

Ten cases have been successfully followed up over the last four years. The only case lost sight of was one with very mild symptoms following a simple scalp laceration, and he was symptom-free six months after his injury. All the others had bony injury of varying degrees, and only two of these appear to be quite free from symptoms. One wrote in May, 1948, that he was "very well and quite normal in every way." The other has spent some time in prison for petty larceny and is considered physically fit by the authorities.

Thus while only two patients can be considered quite fit, seven of the 11 are able to earn their own living in some kind of work. One has a light job, five days a week, as a battery attendant. He is quite active, but one foot requires a

calliper splint and the other, he writes, "gets stuck occasionally." He also suffers from headaches and is rather forgetful. He had a scalp laceration in the right parietal region running over the vertex and there was a small in-driven metal foreign body that was removed without trouble. His condition now appears to be stationary.

Another man has his own small business as a book-seller. He had a severe injury with a midline laceration and a depressed comminuted fracture with penetration of the dura. His recovery was slow but steady; he can now walk unaided, but finds it difficult to tackle inclines owing to some spasticity of his foot. He also complains that if he puts his left foot down suddenly he gets a sharp pain as if he had trodden on a spike.

Another man has a sedentary job as an electrical-instrument maker, and yet another has a like job with the Ministry of Supply. The former stated in January, 1947, that he could get about alone, but that he needed a stick in wet or slippery weather. He could move his toes freely up and down, but had not got normal easy movement in his ankle or knee.

Of the four who are still incapacitated two had penetrating wounds and two had severe depressed comminuted fractures. They differ from the others only in degree, their disability being due to an inability to walk unaided owing to weakness and spasticity of the lower limbs. One of these cases (Case 6) may here be summarized, as it well illustrates the march of events.

Report of Typical Case

Aug. 14, 1944.—Patient wounded by mortar bomb. Deep laceration of scalp, no obvious fracture. Comatose.

Aug. 15.—Conscious; dysphasic; responds to name and to pin-prick. Right arm, complete spastic paralysis. Legs: complete spastic paralysis, left more spastic than right; ankle clonus; extensor plantars.

Aug. 16.—Conscious; dysphasic. Right arm, spastic paralysis. Legs: complete spastic paralysis; extensor plantars. X-ray report: "An oval depressed fracture with much comminution of the depressed portion lying up alongside the midline in the mid-parietal region on the left side. From the lower part of the 5-cm. depression a fissured fracture runs downward on, I think, the left side of the skull to terminate in the roof of the orbit. Not much separation of the edges." **Operation:**—Removal of damaged bone together with fragments that had penetrated the dura and brain. Clot and necrotic brain sucked away and wound closed.

Sept. 6.—Very mild nominal dysphasia. Gives a clear account of his injury. Right arm: tone flaccid, with some finger movements and rotation of the elbow only. Posture sense impaired in the fingers. Legs: power good on the left except for dorsiflexion of the foot, which cannot be performed; on the right he can draw his leg up but cannot move the toes and cannot raise his leg off the bed; tone flaccid; ankle clonus; plantars equivocal.

Sept. 23.—Alert. Movements of right fingers, elbow, and shoulder returning. Movement improved in right leg.

Sept. 1, 1945.—Walking, but a tendency to fall.

Feb. 12, 1948.—Right arm: floppier than left; full range of active and passive movements, but these are slower and weaker than normal. Gripping of right hand is slow, and opening of hand is associated with flexion of wrist. No discrete finger movements possible. Falling away of outstretched right fingers and wrist. Legs: both legs "floppy," right more than left; he cannot evert his right foot and weakness of eversion of the left. Plantars extensor. Bilateral knee and ankle clonus. Loss of position sense in toes and inner three fingers of right hand and diminution of tactile discrimination at the ulnar border of right hand and wrist. He is reported as being a co-operative man who is learning to use his weak hand well and hopes to get placed eventually in a carpenter's shop.

In this case it was a full year before the patient was able to walk. The fact that he made such a good recovery was largely due to his own determination, reflected in his desire to perform work that required the use of his injured hand.

Discussion

A study of this series reveals that there is little to be added to the critical study of a series of similar cases made by Gordon Holmes and Sargent after the 1914-18 war.

The syndrome consists of a spastic paresis of characteristic distribution affecting one or more limbs and associated with cortical sensory loss in the same region. This clinical picture is the result of injuries in the region of the superior longitudinal sinus, and is remarkable particularly for the rapid, in fact, almost instantaneous, onset of the spasticity which accompanies the paralysis. This is especially emphasized in the present group of cases, for, owing to the rapidity with which cases were evacuated in this war, they were often in the hands of a neurosurgical team a few hours after injury. In addition, the injuries themselves were not of great severity in some cases, and consciousness was retained and a clear account of the injury and its effects could be obtained.

One man, for example, stated that a mortar bomb fell five yards (4.57 metres) away from him on the other side of a wall and that he was struck by fragments. He fell to his knees and could not get up. He was flown to England and was in a neurosurgical unit within eight hours. On examination he showed a very typical picture with no movement of either leg below the knee, although he could raise both legs off the bed. There was hyperaesthesia to light touch and pin-prick on the left leg below the knee and on the dorsum of the right foot. Reflexes were brisk and equal and there was ankle clonus with indefinite plantar responses. Loss of position sense occurred in the toes of both feet. X-ray examination revealed a metal foreign body situated just above the midline at the midpoint between theinion and nasion. Beneath there was a depression of the inner table for a distance of about 3 cm., the most marked depression measuring about 4 mm. The depressed bone was rongueured away, leaving a bone defect down the centre of which was running the longitudinal sinus, which appeared patent. He made a good recovery from the operation, and twelve days later the first flicker of returning power in the right toes was noted. His progress was slow, but a month later he was able to walk with assistance. His right foot, recovered by the end of six months, but his left foot made no progress and was eventually fitted with a calliper. Two years later he was working and reported only occasional periods when his right toes "got stuck."

Another man sustained a tangential scalp wound while attacking a house. He fell down, but did not lose consciousness, and was able to crawl to cover. His complaint at that time was that his legs "felt heavy." The fingers of his right hand were also affected, and he complained later of transient attacks of blindness. The scalp wound ran from the left post-parietal region across the vertex to the right parietal region with depression of both tables extending across the mid-parietal region, the maximal depression being directly over the longitudinal sinus. This man made a steady recovery to full health.

These two cases illustrate the following points: (1) a relatively mild injury with no loss of consciousness; (2) the immediate onset of weakness and spasticity in the lower limbs; (3) the presence of bony depression in the immediate region of the longitudinal sinus; and (4) the slow and steady recovery over a long period, although in the second of these cases the depressed fracture was not elevated.

According to Gordon Holmes and Sargent the most striking pathological feature of these injuries is the firm swollen cortical veins which cannot be emptied by pressure, and the oedema of the area of cortex that they drain. Microscopically, there is oedema of the affected area, which is more prominent in the white than in the grey matter.

Minute haemorrhages occur in the grey and white matter chiefly in proximity to the wound. The nerve cells in the affected areas show pronounced changes and are greatly swollen and in advanced chromatolysis.

It is thought that this clinical picture results from the situation of the longitudinal sinus near the site of the injury. The sinus undergoes some degree of thrombosis which causes the oedema and produces the associated pathological changes in the cerebral cortex.

Thrombosis is the natural response of a large vessel of this type to trauma in its vicinity, and it is possible that it occurs independently of changes in the cortex. For although the sinus was only specifically mentioned at operation in five of these cases, the impression gained was that it remained patent owing to the presence of trabeculae within its lumen.

Lumbar puncture was not performed before operation, but the pressure was not consistently high when it was done post-operatively. Headache was not a common symptom, and in fact only one man complained of it specifically. Neither did any case show changes in the optic fundi compatible with a persistently raised C.S.F. pressure, although this would depend to some extent on the site of the thrombus within the sinus.

Wounds of varying severity in this situation cause similar symptoms, differing from each other only in degree. For example, Case 1 sustained a scalp wound with no bony injury. Symptoms were limited to a slight loss of power and some subjective numbness in the left foot which passed off in five days. In the cases previously cited the symptoms persisted over years.

None of these cases showed the initial period of flaccidity which usually occurs in cases of cerebral damage, whether due to trauma or to vascular catastrophe, and which is thought to be due to shock. This persists normally in such cases for a period of about 10 days, and only then does spasticity begin to appear. Indeed, the very feature of these cases is the immediate appearance of spasticity.

Case 11 is interesting in this respect. This man sustained a penetrating injury close to the midline in the left parietal region. He presented a rather mixed picture of right hemiplegia, with a spastic paresis of the lower limbs. He had a right focal epileptic attack on the day following injury confined to his hand and arm. Operation involved removal of damaged brain tissue and of in-driven bone fragments in the left parietal region. He made a good recovery from a severe operation, and two days afterwards showed the following signs: complete flaccid paralysis of the right arm, and a flaccid paralysis of both legs, complete on the right and almost complete on the left. He showed, in addition, loss of joint sense of the ankles and toes and an extensor plantar response.

Eleven days after operation the following signs were observed: slight spasticity of the right upper limb with some movement now possible at the elbow and slight dorsiflexion and palmar flexion at the wrist. Grip good, but some difficulty in extension of the fourth and fifth fingers. In the lower limbs tone was increased on both sides, right slightly more than left. Power was improving in the left leg, which could be held off the bed and flexed at the knee; dorsiflexion and plantar flexion were just present and he could move the toes slightly on this side. On the right, paralysis persisted except for slight movement at the hip. Ankle clonus was ill-sustained and the right plantar was extensor. Three years later he was able to get about with the aid of sticks in wet and slippery weather and was earning his living. He considered that improvement was still continuing.

This case showed a stage of flaccidity occurring after operation, but eleven days later the picture of spasticity had again become apparent. It does seem, therefore, that the site of the injury in close proximity to the superior longitudinal sinus is responsible for this clinical picture; but

this does not explain why spasticity occurs at once, and indeed its occurrence is so immediate that it must be related directly to the injury rather than to the secondary pathological changes following a sinus thrombosis. In cases where operative procedure was necessarily severe a stage of shock and flaccid paresis did appear, but ten days or so after operation spasticity had again appeared, often together with the first signs of recovery.

Recovery begins early and continues for a considerable time—several years at least. Its degree depends upon the degree of injury, and if this is severe spasticity and weakness remain and recovery depends upon how much this can be overcome by re-education in walking and the use of appliances. The main difficulty that these patients experience is the negotiation of uneven surfaces and inclines that require the presence of unimpaired postural sensibility in the toes and the ability to dorsiflex and plantar-flex the foot accurately. In the early stages of recovery the gait may be likened to that of a patient suffering from Little's disease, but most cases appear to progress enough to afford adequate movement at the hips and knees, and the residual disability is confined in these cases to the feet and ankles.

Furthermore, although the series is small, the presence of other symptoms often associated with cranial injury do not seem to be of common occurrence—that is, such symptoms as headache, dizziness, or epileptiform attacks.

Summary

A series of cases of injury in the region of the superior longitudinal sinus is presented.

The clinical picture is described and the pathology briefly discussed.

Particular attention has been paid to the clinical progress of the cases and their condition up to four years after injury.

The very early occurrence of spasticity after the injury is also emphasized.

I should like to thank Mr. Wylie McKissock for permission to study and follow up these cases, and Dr. J. E. S. Lloyd for permission to publish this paper. I should also like to thank Miss Montgomery for her help in contacting the cases.

BIBLIOGRAPHY

- Bailey, O. T., and Hass, G. M. (1937). *Brain*, **60**, 293.
 Beevor, C. E. (1907). *Ibid.*, **30**, 403.
 Burger, H. (1924). *Acta oto-laryng.*, *Stockh.*, **6**, 4.
 Foerster, O. (1936). *Brain*, **59**, 135.
 Holmes, G., and Sargent, P. (1915). *British Medical Journal*, **2**, 493.
 Langworthy, H. G. (1916). *Ann. Otol., etc.*, *St. Louis*, **25**, 554.
 Sargent, P. (1910-11). *J. Anat.*, *Lond.*, **45**, 69.
 Symonds, C. P. (1937). *Brain*, **60**, 531.
 Walshe, F. M. R. (1914-15). *Ibid.*, **37**, 269.
 — (1919). *Ibid.*, **42**, 1.

Under the National Insurance (Overlapping Benefits) Provisional Regulations, 1948, certain reductions are made in the rate of benefit payable to in-patients who are maintained free of charge in hospital. These provisions have thrown into relief the question of the extent to which hospitals should provide their patients with the non-medical necessities of life and with minor luxuries. There are certain articles which the Minister considers should be provided as a matter of course for all patients who do not prefer to provide their own—such things as soap, tooth paste or powder, razor blades, sanitary towels or their equivalent, the services of a barber, a reasonable supply of notepaper on request, and a selection of newspapers and magazines in the wards (though not an individual issue). Other articles may be supplied to patients on loan—e.g., towels, hair-brushes, and combs, and, where necessary, night and day clothing. It is not easy to distinguish between essentials of this kind and minor luxuries, or, in other words, between what should be provided at the cost of the Exchequer and what may be provided from the committee's or board's free money; and the Minister leaves committees and boards a certain discretion in determining this issue. He considers, however, that hospitals should not provide as a charge on Exchequer funds such things as cosmetics, permanent waving, postage stamps, and tobacco or sweets. Where patients remain in hospital for a long time it is reasonable that the range of articles to be provided without charge and at Exchequer cost should be somewhat extended.

A CASE OF BILATERAL PHAEOCHROMOCYTOMA

BY

T. M. REID, F.R.C.S.Ed.

Honorary Surgeon

AND

R. SALM, M.D., L.R.C.P.&S.Ed.

Pathologist

Camborne-Redruth Miners' and General Hospital

Phaeochromocytomata are rare enough to warrant the recording of an additional case.

Case Report

The patient, a married woman aged 40, was admitted in October, 1946, complaining of generalized throbbing headaches for the past ten years. These were present by day and by night, but had increased in severity during the past two years. Sometimes after exertion or on stooping the headaches became very severe, spreading from the temporal and infraorbital regions to the vertex and occiput. These attacks lasted for two to three minutes, but the patient emphasized that she was never completely free from headaches. On moderate exertion she soon became breathless and suffered from palpitations. Weakness and loss of weight had increased, particularly during the three weeks before her admission. Her past history had been uneventful. Her family history was very interesting.

Her father died at 46 of Addison's disease, though its cause (atrophy, tuberculous infection, chromaffinoma) is debatable. Of six other siblings, one brother died at 28 of diabetes, while one sister died at 29 of an obscure bone disease. She was apparently healthy up to the age of 14, when she started to "shrink." At the time of her death her body was about the size of a child of 4. The condition was regarded as that of an "achondroplastic dwarf."

Physical Examination.—The patient looked very much older than her age. Pigmentations were not present on skin or mucous membranes. There were visible arterial pulsations in the anterior cervical triangles and in the suprasternal notch, and the apex beat was forcible. The pulse was regular, but there was a tachycardia of 140. The blood pressure was raised to 227/128. The blood urea was 65 mg. per 100 ml. Hyaline casts were constantly found in the urinary sediment, and a moderately large tumour which did not move with respiration could be felt in the right lower abdomen. Both fundi of the eyes showed sparse, scattered, superficial retinal haemorrhages of moderate size in the paracentral areas. One vein below the left optic disk appeared to be compressed by the overlying artery.

X-ray Examination.—The ascending pyelogram showed that the right kidney corresponded to the tumour felt by abdominal palpation and was found to be rotated forward, outward, and displaced downwards. Both kidneys were secreting. The left kidney was normal in appearance and position. Above the right kidney there was a dense shadow, and a new growth of the right adrenal gland was thought to be most likely.

A presumptive diagnosis of chromaffinoma of the right adrenal gland was made and operation was decided upon. Under N₂O-ether anaesthesia the right lumbar region was explored and a large adrenal tumour removed. During the operation it was noted that both pupils became maximally dilated as soon as the tumour was handled. Post-operatively the patient was immediately treated for shock, adrenaline was given, but in spite of this she did not rally and death occurred within 24 hours.

At necropsy the left adrenal gland appeared to be also grossly enlarged, weighing 40 g. In parts the usual shape of an adrenal gland was still recognizable, but a large round tumour mass occupied the central portion of the gland. On sectioning, normal yellow cortical tissue was only sporadically recognizable. For the greater part the whole organ and round mass consisted of soft yellowish or dark brown jelly-like material. The right