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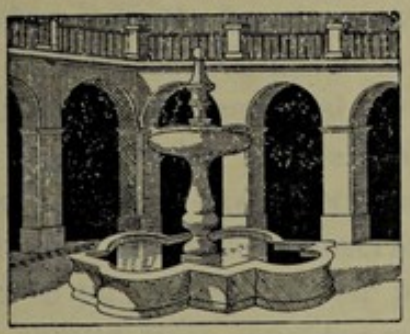

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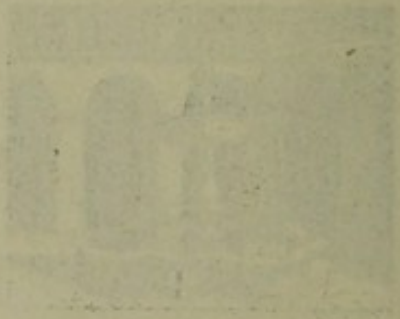

THE VASCULAR ASPECTS OF HEAD INJURIES [1]

HUGH CAIRNS

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The presentation of some observations on the vascular aspects of head injury affords me the welcome opportunity of paying tribute to the classic work of EGAS MONIZ. When in 1927 MONIZ introduced cerebral angiography Portugal provided the medical world with a new method of studying the cerebral circulation. The application of this method to the study of disorders of the cerebral circulation has already disclosed diseases that hitherto were not diagnosed in life, and it has also furnished more precise information about other diseases than was available before. There are indeed reasonable grounds for believing that the application of cerebral angiography is still far from complete, and the cases of head injury which I shall present to you today indicate to some extent the truth of this.

I propose in this lecture to describe some of our observations on disturbances in the cerebral circulation that occur in head injury, and in particular to deal with the injuries to the internal carotid artery and their recognition.

The cases which I shall thus describe are the *exceptional* head injuries, and to put them in their proper perspective it is desirable that we should first consider what part vascular fac-

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(1) Conferência realizada na Faculdade de Medicina de Lisboa em 16 de Junho de 1942.

tors may play in the causation of concussion, that state of unconsciousness which is the basic clinical fact in the great majority of head injuries.

Concussion. For many yeras the view was widely held that the clinical state of concussion was due to a momentary capillary anaemia occasioned by the reduction of cranial capacity at the time of the blow. This in brief was the hypothesis originally put forward by STROHMEYER and elaborated by TROTTER (1914). It is view which has been questioned in England in recent years, on several grounds. On the basis of clinical observation SYMONDS has pointed out that no sharp line of distinction exists between cases showing a short period of unconsciousness followed by spontaneous recovery, and those cases of prolonged stupor or delirium. The groups merge into one another to such an extent that it is reasonable to conclude that they have the same underlying pathological cause, in varying degrees of severity. The duration of unconsciousness and of amnesia often has little influence upon the completeness of the recovery. A patient may be unconscious for over a week and yet recover just as completely as one who has been unconscious for only an hour. This is difficult to explain on a basis of cerebral anaemia. If the prolonged unconsciousness were due to cerebral anaemia one would expect some residual symptoms of a permanent character.

On physical grounds also the hypothesis of cerebral anaemia has been challenged by my assistant Dr. H. HOLBOURN. While it is admitted that in many head injuries, though not in all, a very severe rise of intracranial pressure may exist at the beginning of the impact, it is fairly certain that the amount of available pressure would not be adequate to force out the blood during the time of the blow, which is probably less than 1/100 second. The volume of blood pushed out cannot be greater than the volume of the inward bulge of the skull, and this must in general be far less than the total volume of blood in the brain. Furthermore, at such high speeds and pressures blood and brain behave very much alike. Blood will be almost «solid» under the blow, just as water is when one dives into it and goes too shallow! And even if all the blood disappeared from the brain for 1/100 second it is highly unlikely that its disappearance would affect consciousness; if the anaemia were

prolonged it is unlikely that the loss of consciousness would be as instantaneous as is that of concussion.

On the experimentel side recent work in Oxford by DENNY-BROWN and RITCHIE RUSSELL (1941) has shown that the essential factor in production of concussion is that the head should be subjected to a high rate of change of velocity. When the animal's head was fixed much greater force was required to produce concussion than when the head was free to move under the blow. This experiment is in accord with a common experience in man that when the head is not in motion at the time of injury, as, for example, when it is crushed between the buffers of railway carriages, or when it is pierced by a small, high-velocity missile, unconsciousness often does not occur. These experiments and clinical observations incline us to the belief that the main factor in the production of concussion is not cerebral anaemia, but rather physical damage to the neurones of a reversible character brought about by rotational acceleration (or deceleration) of the brain within the skull.

Damage to the internal carotid artery

With this brief general consideration we must leave the fascinating subject of concussion and turn to these unusual head injuries which produce damage to the internal carotid artery. From recent experiences I have come to the conclusion that damage to the internal carotid is rather more important than was formerly supposed. I have seen examples of four clinical varieties of this condition in blunt head injury.

(1) Traumatic obstruction of the internal carotid artery by thrombosis or embolism producing delayed hemiplegia after head injury.

(2) Traumatic aneurysm of the internal carotid artery into the sphenoidal air sinus.

(3) Traumatic carotico-cavernous aneurysm.

(4) Traumatic rupture of the internal carotid artery with acute subdural haemorrhage.

I — TRAUMATIC OBSTRUCTION OF THE INTERNAL CAROTID ARTERY BY THROMBOSIS OR EMBOLISM PRODUCING DELAYED HEMIPLEGIA

CASE I. L. J. E., a pilot of 23, crash-landed his plane after engine failure on the afternoon of May 16, 1941. He remembers landing and going through a hedge, but was unconscious for probably two or three minutes. The next thing he remembers was being carried away from the burning aircraft and being taken to hospital. On arrival there he recognized the doctor, whom he had known before the accident, and gave a clear account of the accident. Owing to an oedema and lacerations of the neck, his voice was husky, and he had difficulty in swallowing and breathing.

He showed the following wounds: (a) Deep laceration 4 cms. long through right eyebrow; bone exposed. (b) Laceration 2 cms. long at the level of the hyoid bone on right side, deep and bleeding freely. (c) Superficial grazes in the left carotid triangle. (d) Small puncture wound in the left shin.

The marked oedema and the inability to swallow were so striking that a tracheotomy set was prepared (but was not used).

The next day, May 17, he had improved, was conversant and coherent, with no abnormal signs in the central nervous system, until suddenly at 10 o'clock that night— about 30 hours after injury — he was heard to ring the bell in the ward and was found in bed, staring before him, with a paralysis of the right side of his face and of his right arm, and he was unable to speak. He could still move his right leg, but it was weak and showed an extensor plantar response. One hour before this he had got out of bed and walked 10 yards to the lavatory.

The following morning, May 18, he showed a complete motor and sensory aphasia, wit flaccid paralysis in the right upper limb and a severe weakness of the right lower limb with an extensor plantar reflex. He was alert and was not incontinent, but was still having difficulty in swallowing and could not protrude his tongue.

On admission to Hospital on May 19 he was alert and co-operative while being examined, but showed a tendency to relapse into sleep when left alone. The neurological signs were identical with those of the previous day; and in addition there was some impairment of tactile localization on the right side. There was no apraxia. His visual fields appeared full to confrontation. X-ray of the skull was normal.

Lumbar puncture showed normal cerebro spinal fluid under normal pressure, with no block on jugular compression. Blood pressure was 120/60 mm. Hg.; pulse rate 72.

Operation. On May 19, as intracranial haemorrhage was suspected, a burr hole was made over the left temporal region. The subdural space was empty, the brain was soft, of normal colour and under normal tension.

Course. His condition remained substantially unchanged apart from the slow march of hypertonus in arm and leg, with no improvement in the aphasia. A week after the accident further tests showed that there

was diminution and poor localization of pin-prick over the whole of the right half of the body; vibration, fine touch, and postural sense were also impaired. The sensory defect being more marked towards the periphery and the hemiplegia involving face and arm more than leg suggested a vascular lesion affecting the cortex.

Air Encephalography 14 days after the accident showed a normal ventricular system; but further examination 6 weeks after the injury showed dilatation of the left lateral ventricle, indicating that cortical atrophy had taken place on the left side.

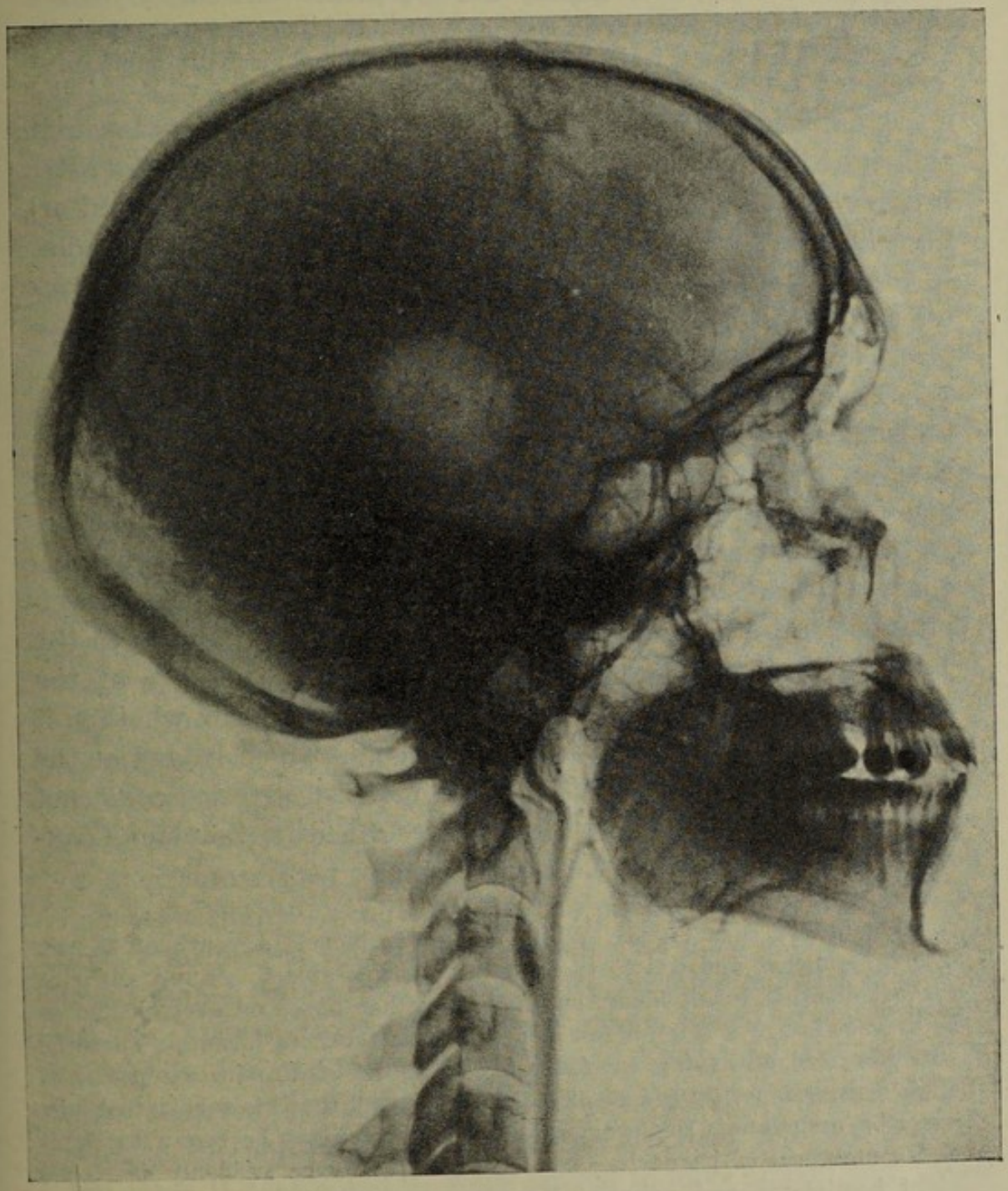


Fig. 1

Electroencephalography showed a constant, severe cerebral dysrhythmia in the left middle Sylvian region.

Arteriography. A thorotrast arteriogram on the left common carotid, on the 38th day after injury, showed that the external carotid system was completely filled in its whole distribution. The internal carotid was obliterated at a point 2 cms. beyond the bifurcation of the common carotid. The obliteration was a tapering one and apparently quite complete (Fig. 1). The right side was not injected.

Two months after injury there was still gross global dysphasia. The hemiplegia had recovered slightly and the patient was able to walk a little. The sensory defect was also less. At this time it was possible to show that there was a right upper quadrant hemianopic field defect.

Comment. There is thus clear evidence in this case that the right hemiplegia and aphasia which came on 30 hours after injury were associated with a lesion of the internal carotid artery in the neck. The arteriographic appearances are similar to those described by MONIZ (1940, 1941) in his important work on carotid thrombosis But his cases with the possible exception of one case (obs. IV) were not due to trauma.

That there was in Case I, in addition to concussion, an unusual degree of injury to the neck was indicated by the fact that on the first night after the injury oedema of the neck and dysphagia were so striking as to cause the doctor in charge to have a tracheotomy instruments prepared in case of emergency. It seems likely that the onset of hemiplegia was precipitated by the patient's getting out of bed and walking to the lavatory, but whether it was due to complete closure of the internal carotid at the site of injury in the neck or whether it was due to detachment of a small embolus which lodged at the junction of the middle and anterior cerebral arteries could not be determined. The following case described by Zannoni (ZANNONI) gives clear evidence on this point.

Zannoni's case (1933). While riding his bicycle a youth of 23 ran into a hand-cart and fell to the ground. He was able to get up and ride a short distance to his home, but he was rather dazed on arrival, and as he did not improve after a few hours he was taken to hospital. There he was described as having «a slight bruise on the skin, with symptoms of slight concussion». By the evening he was so well that his family took him home by ambulance. However, at 10 p. m. that night he began to vomit and complained of headache. At 3 a. m. next day he suddenly developed right hemiplegia and aphasia. He was at once taken back to hospital, and a diagnosis of middle meningeal haemorrhage on the left side was

made. A left osteoplastic flap was turned, but no extradural or subdural clot was found. His condition deteriorated and he died three days after the accident.

Necropsy confirmed the negative operative findings in the cranium and showed an abrasion on the chin. There was ischaemic softening of the central part of the left cerebral hemisphere. The left Sylvian artery was obstructed by dark, firm, slightly adherent blood clot which extended into and almost completely obstructed the left internal carotid artery. In the neck there was no superficial extravasation, but there was some haemorrhage in the connective tissue around the nerves and vessels on the left side at the level of the thyroid cartilage. The common and external carotid arteries were normal, but the internal carotid artery showed at its proximal end a transverse laceration 8 mm. in length involving the intima and media, and in this part a firmly adherent thrombus was found which reduced considerably the lumen of the vessel.

This case is remarkably similar to Case I and suggests that the hemiplegia in that case was due to embolus. The suddenness of the onset of the hemiplegia supports this contention. It would not be expectd that in a healthy young man gradual obstruction of one internal carotid artery in the neck would produce hemiplegia. In the light of these two cases one is led to wonder whether damage to the carotid arteries in the neck may not occur in other cases of head injury, and pass unnoticed if no embolus is detached. There are certain cases in which, after head injury, Horner's Syndrome occurs without any trace of hemiplegia; and, if these are due to injury of the cervical sympathetic chain, it is unlikely that the adjacent carotid arteries would always escape damage.

The frequency of embolus after indirect injury of the carotid artery by gunshot wound of the neck has been noted by SENCERT (1917). He recounts successive embolisms in such cases and says, «there is no doubt that these emboli originate in a circumscribed thrombosis of the common carotid which had been slighthly contused by the passage of the projectile»; and, «the presence of the thrombus is... only revealed by the small emboli which proceed from it». Similar embolic manifestations appear to have occurred many years after gunshot wound in one of MONIZ cases (MONIZ, 1941, obs. IV) and in a case reported by TÖNNIS (1934). In both these cases emboli entered the ophtalmic as well as the cerebral arteries. Two of my cases (Cases 8 and 9) were proved to have emboli, and these is thus clear evidence that embolus into the main cerebral

vessels is a not infrequent complication after injury of the internal and common carotid arteries.

The following case differs from Case I and from Zannoni's case in that the initial brain injury was a severe one, and the cerebral damage was due to a spreading thrombosis of the distal part of the internal carotid artery from the site of damage caused by fracture of the skull.

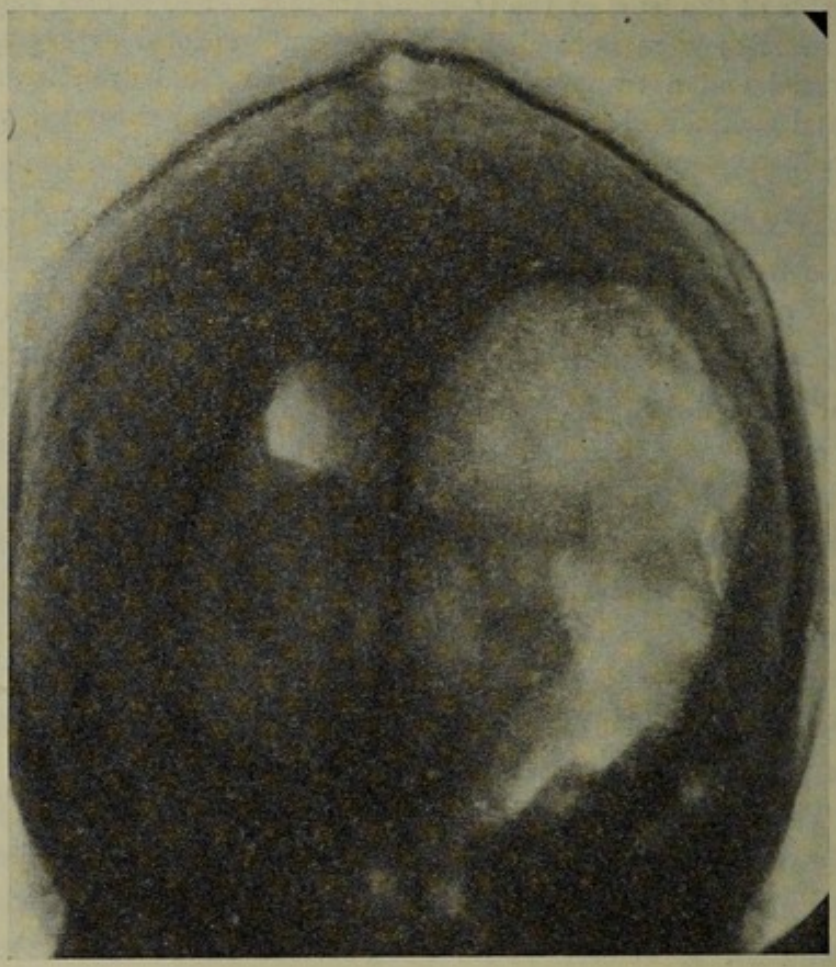


Fig. 2

CASE II. W. M., aged 27, was injured on June 17, 1941, when he was thrown from his motor-cycle. He was admitted to hospital in a condition of extreme gravity, unconscious and extremely restless, but moving all his limbs. There was a deep laceration over his left eyebrow, with some proptosis of the left eye, a severe Colles fracture and a fracture dislocation of the ankle on the left side. The scalp wound was explored, and a linear fissured fracture disclosed; the wound was excised and sutured. The wrist and ankle were reduced and plastered. Lumbar puncture on June 19 showed much blood in the cerebrospinal fluid.

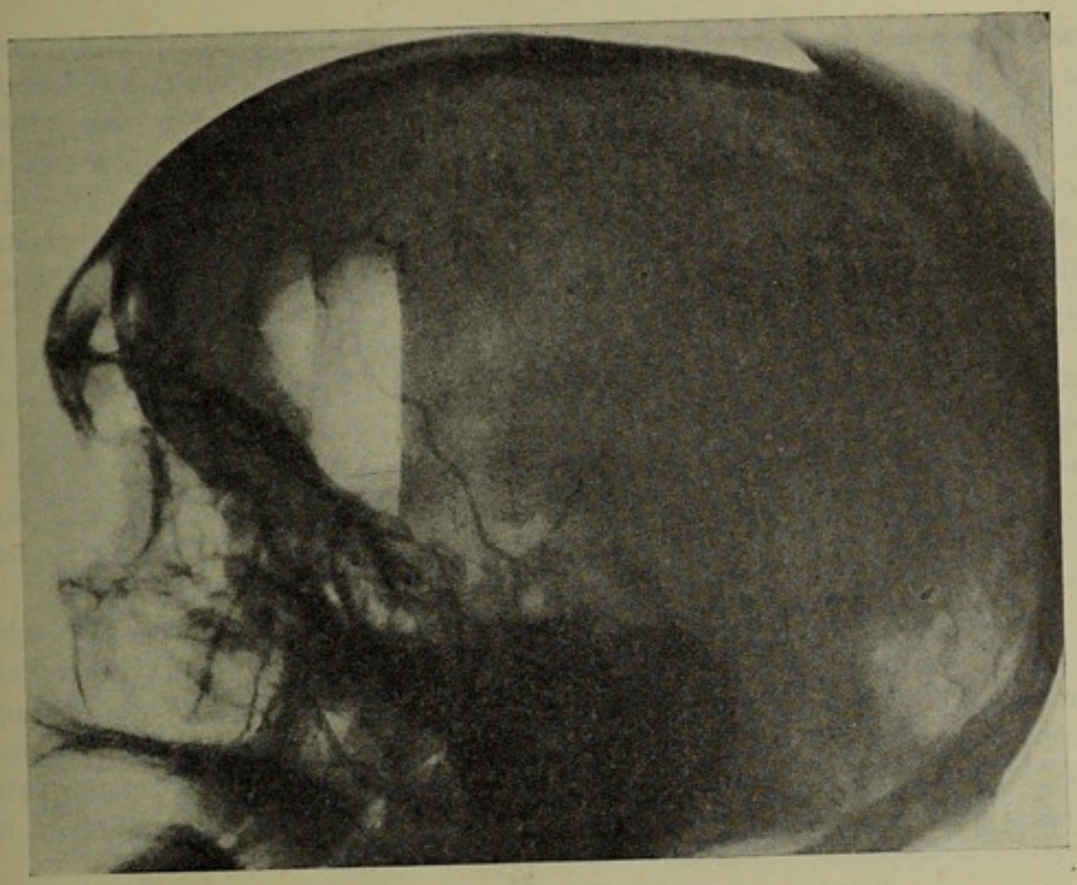


Fig. 3

He continued in an unconscious state until June 23, when he could be roused a little. There is no record of any neurological signs other than a «partial third nerve palsy» on the left side (ptosis, divergent squint, and a sluggish pupillary reaction to light) until June 26, 9 days after injury, when he suddenly developed a flaccid right hemiplegia, with a fixed and dilated left pupil. His pulse rate rose to 140, his temperature to 102.8° and he became once more comatose. The case was regarded as one of cerebral thrombosis.

His pulse and temperature remained high for 8 days, but by June 29 he was «showing glimmerings of understanding, but no speech». His hemiplegia persisted and he began to develop contractures of the right arm and leg. He was throughout incontinent of urine and faeces. Decubitus ulcer ensued, and he became extremely emaciated. His temperature and pulse remained unstable, with peaks of temperature to 101° and an average pulse rate of 110.

When he came under our care on October 11, 1941, he was extremely wasted, incontinent of urine and faeces, and had a large indolent sacral bed-sore. He was apathetic and had an almost complete aphasia of global type. There was a scar 3 cms. long over the outer half of his left supraorbital ridge. X-rays showed a fissured fracture of the left frontal bone involving the frontal sinus; his right pupil was larger than the left, although both reacted well to light and to accommodation. His

smell and his visual fields could not be tested, but there was obviously a gross defect of attention on the right side of the visual fields. On the right side also there was gross hemiplegia with contracture, and diminution of sensibility to pin-prick. The tendon jerks were exaggerated on the right side and both plantar reflexes were extensor. A lumbar puncture showed a pressure of 110 mms., with no block on jugular compression; total protein 15 mgm. per cent., no cells. Wassermann reaction in blood and cerebrospinal fluid was negative.

His pulse was regular at 110, B. P. 140/120, both later falling to 80 and 100/70 respectively. The apical impulse was in the 5th space, 1 inch within the mid-clavicular line, the sounds pure. There were no signs of any pulmonary disease.

Air Encephalography. There was gross dilatation of the left ventricular system, with a moderate dilatation of the right system. The left half of the 3rd ventricle was more dilated than the right (Fig. 2).

Arteriography. The left common carotid was first injected with 12 c. cs. of thorotrast, and an immediate picture showed that the contrast medium had entered only the external carotid system (Fig. 3). The site of the obstruction in the left internal carotid was difficult to determine, but in the light of subsequent findings it is possible to interpret the arteriograms as shown in Fig. 4. The internal carotid is obstructed at its

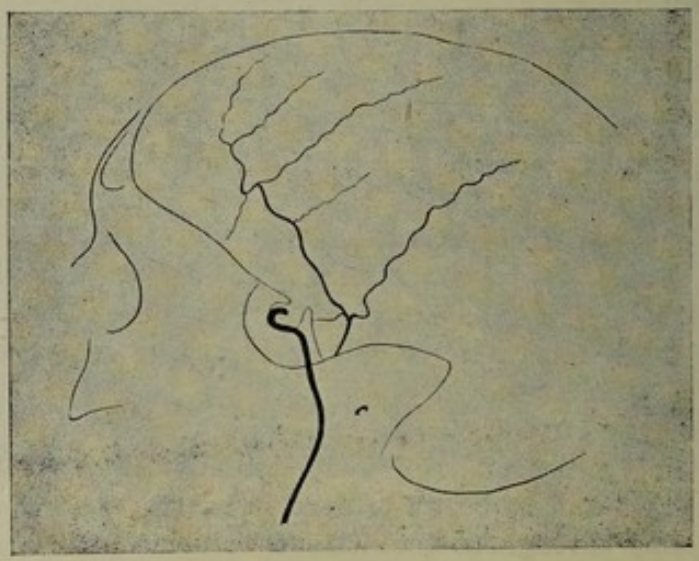


Fig. 4

termination and is also unusual in being only slightly larger than the middle meningeal artery. The left internal and external carotid systems were injected satisfactorily, and the proximal part of the left anterior cerebral artery was seen to be filled from the right side (Fig. 5).

Course. He remained hemiplegic, aphasic, incontinent, and bed-ridden and showed increasing flexor contracture of the right arm and leg. Speech

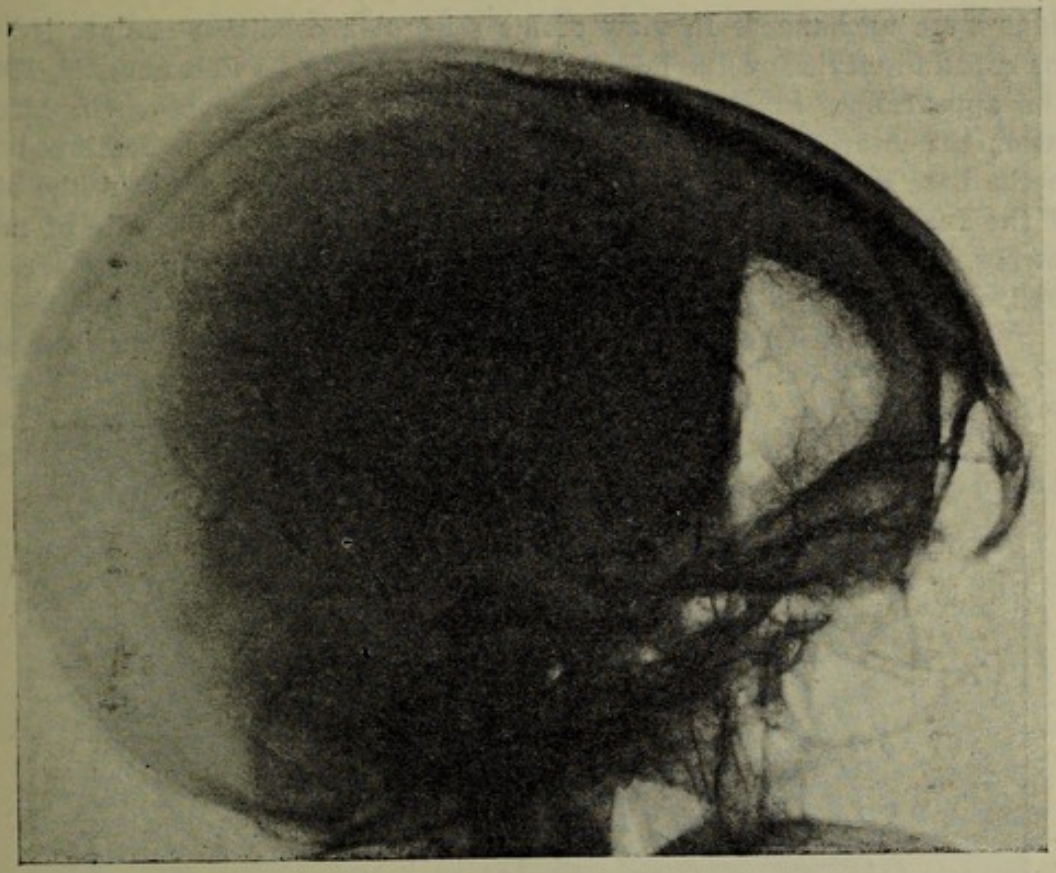


Fig. 5

was virtually limited to «yes» and «no», and his understanding did not go beyond the simplest commands.

Further tests of the visual fields showed fairly definitely that he had a complete right homonymous hemianopia. His motor and sensory

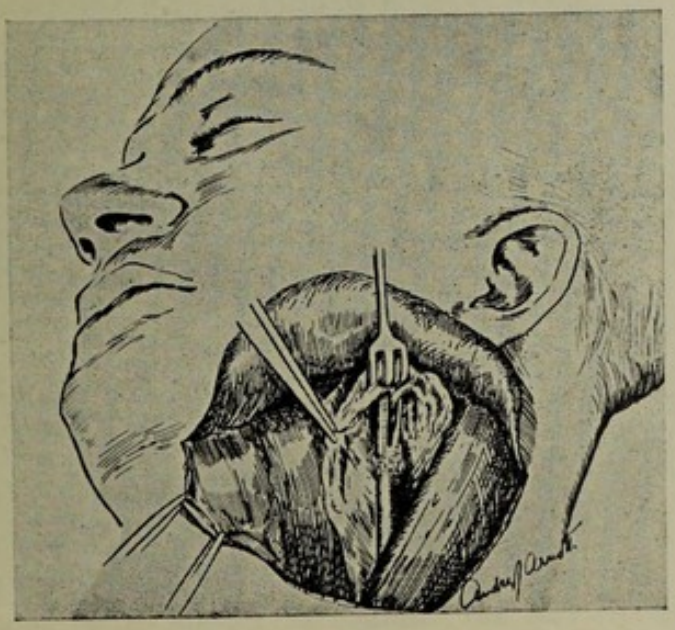


Fig. 6

signs were unchanged. In view of his miserable condition exploration of the carotid arteries on the left side was undertaken on February 23, 1942. The appearances are shown in Fig. 6. One centimetre above the bifurcation the internal carotid tapered fairly sharply, thence it continued up to the base of the skull as a small vessel of even calibre about the size of the facial artery. The vessel pulsated freely distal to the site of narrowing. The patient's condition remained quite satisfactory until March 8, 1942, when he had a series of attacks of unconsciousness and died.

Necropsy showed a linear fracture, 7 cms. long, extending from the

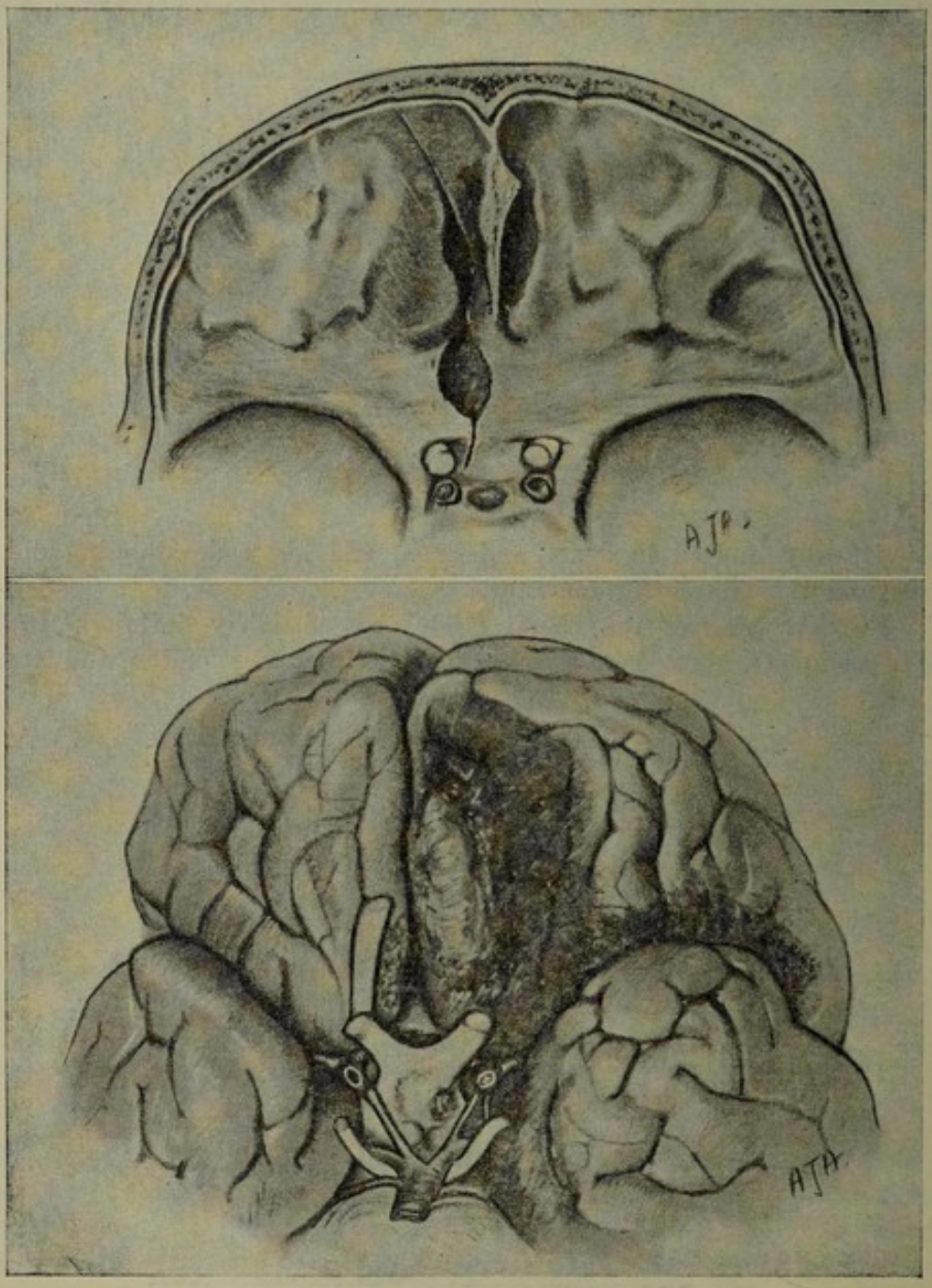


Fig. 7

centre of the anterior border of the left orbital plate postero-medially to cross the left half of the tuberculum sellae, ending in the anterior wall of the sella turcica medial to the left anterior clinoid process (Fig. 7). The inferior surface of the frontal lobe was firmly united to scar tissue in the posterior part of this fracture and to the left cribriform plate, and was diffusely pigmented. The most distal part of the left internal carotid artery was occluded by gelatinous greyisch-white tissue, the remaining cerebral arteries being normal. The right lateral sinus and the adjacent sigmoid sinus were partly occluded over a length of 2 cms. by a small amount of grey, gelatinous, partly organized thrombus. (The superior longitudinal sinus was continuous with the left lateral sinus, which was larger than the right).

Coronal sections of the brain showed complete destruction of the

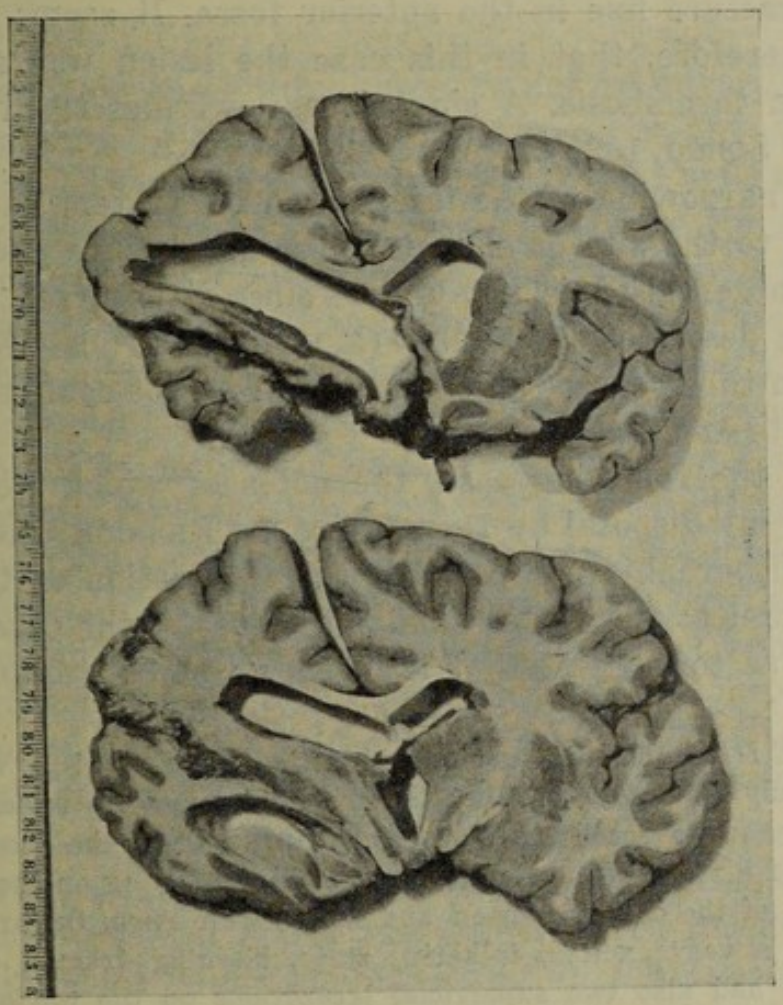


Fig. 8

cortex of the ventral aspect of the left frontal lobe over an area 5 cms. by 4 cms. corresponding to the area of trauma. There was also infarction of the cortex and white matter throughout the left Sylvian region, the normal tissue being replaced by friable yellow material (Fig. 8). The left

basal ganglia and internal capsule were severely atrophied, and descending pyramidal degeneration could be traced throughout the brain stem.

Histological sections showed that the clot in the internal carotid was undergoing recanalization. Careful histological study of the internal carotid artery in the neck showed no trace of damage to the vessel wall.

Comment. This case differs from Case I and Zannoni's case in showing no evidence, after the most through histological examination, of any damage to the walls of the internal carotid artery in the neck. The thrombus in this case was in that part of the internal carotid which lies between the cavernous sinus and the termination of the internal carotid, and this part of the vessel was in close relationship with the posterior end of the fracture line in the anterior fossa. It seems most probable, therefore, that in this case the lesion was a primary traumatic thrombosis. A similar case was described by LÖHR (cited by SORGO, 1939).

The narrowing of the internal carotid artery in the neck, proximal to the site of obstruction is an obscure phenomenon, but the observation is not unique since it occurred in two of the cases described by MONIZ (MONIZ, 1941, obs. IX and X), though in those cases the obstruction was not complete and its precise site was not determined.

In the following case, the notes of which were kindly placed at my disposal by Doctor GEORGE RIDDOCH, there is neither arteriographic nor pathological verification of the lesion, but the clinical story is so similar to those of the cases already reported as to suggest that it belongs to this group.

CASE III. G. E. L., a previously healthy doctor, aged 36, was driving a car on July 22, 1941, when it collided with a lorry. He remembered the bump, but had no recollection of being thrown from the front into the back seat of his car. He evidently came to himself lying on the back seat. He got out of the car by himself and was driven home. On arrival there he was in a very excited, talkative state, bleeding from a cut on his tongue. He had also two superficial lacerations on the top of his head and a bruise in the middle of his forehead. He was bruised also on the legs and complained of pains in different parts of his body. He was taken to a hospital and remained there for a fortnight, during which time he complained of severe headache; he had vomited on the first night. When he got up and returned home the headaches still continued and he complained of dizziness and difficulty in walking straight.

On about August 6 he resumed his work, and at this time noticed

some difficulty in writing. On August 10, nineteen days after the accident, he carried two motor-tyres downstairs from an attic. Almost immediately afterwards he suddenly became unable to speak and he fell to the floor, but was not unconscious. He was found to have complete aphasia and a right hemiplegia which was complete, with the exception that a little voluntary movement was still possible in the right lower limb. Lumbar punctures showed clear cerebrospinal fluid with no trace of blood. Blood pressure at this time was 110/70. He was once more admitted to hospital. He vomited repeatedly during the night of August 10. On August 13 a ventriculogram was done which showed ventricles of normal shape slightly displaced to the right. A right osteoplastic craniotomy was done, but no lesion was found. Soon after this his right leg began to recover its power and there was slight improvement in the right arm, but motor aphasia was still complete four months after the injury. His Wassermann reaction was normal. I have no information as to whether there was a fracture of the skull.

The fact that these three cases, two of them verified, have been encountered within a year suggests that this condition of traumatic obstruction of the internal carotid artery may be more common than has hitherto been supposed. Our attention was only drawn to the syndrome by the swelling of the neck which occurred after injury in Case I, and it was this that led us to do the arteriography which revealed so clearly the diagnosis of the condition. From this experience we were led to suspect the true nature of Case II. Hitherto this syndrome of hemiplegia following at an interval after head injury was evidently usually regarded as being due to intracranial haemorrhage, and it is a significant fact that in two of our three cases and in Zannoni's case operative exploration for intracranial clot was undertaken.

In two of the patients there was clear evidence of injury to the neck and damage to the internal carotid at that level. In another there was injury to the tongue. In the fourth (Case II) the evidence suggested that the internal carotid had been damage at the site of a fracture into the sella turcica, but in addition there was a curious narrowing of the internal carotid in the neck.

While the treatment of the condition once it has occurred must largely be of a conservative character, yet something may be said on the score of prevention. In the first place it is clear that patients showing neck injury in addition to head injury should be moved about with considerable circumspection in the

early days after injury, and until all symptoms have subsided. In three of these patients the serious symptoms indicating complete carotid obstruction have come on soon after unwarranted bodily activity.

The patients so far encountered have all been young and previously healthy individuals, and it is reasonable to suppose that similar patients might be able to establish an adequate collateral circulation if organization of the mural clot is encouraged by rest. MONIZ has shown that such a collateral circulation does become established, but in a number of his cases it had evidently occurred too late to save the area supplied by the midlle cerebral artery.

The question of thrombosis or embolism as the causal factor is a difficult one to decide without further experience. The suddenness of onset of the hemiplegia favours embolus, the more so as some of the cases of thrombosis described by SORGO (1939) hemiplegia did not occur. Presumably in their cases the thrombosis occurred sufficiently gradually to enable the supply of blood to the brain to be maintained by means of the collateral circulation. Certainly in Zannoni's case an embolus occurred; on the other hand, in Case II there was a thrombosis near the site of fracture. Thus the condition can be produced either by thrombosis or by embolism.

On the differential diagnosis of hemiplegia after head injury

At this stage it is convenient to digress from our main theme and consider the differential diagnosis of hemiplegia after head injury. In three of the four cases already discussed in this paper cranial exploration was undertaken on a pre-operative diagnosis of haemorrhage, either extradural, subdural, or intracerebral. In all of these conditions gross hemiplegia is of rare occurrence, and when it does occur it is of gradual onset and is attended by deepening unconsciousness which progresses *pari passu* with it. In three of the four patients with carotid obstruction the onset of hemiplegia was not accompanied by any lowering of consciusness. And the onset is sudden. When a patient develops carotid obstruction while still unconscious from the causal head injury it may be difficult to be certain of the

diagnosis, but if the depth of unconsciousness has been systematically measured from the time of the injury it should be possible as a rule to differentiate by clinical means those cases in which there is deepening of coma — that is to say, the cases in which blood clot is accumulating inside the cranial cavity — from those in which there is no deepening of coma — that is, the cases in which the internal carotid or its main branches have become blocked. Until we know more of these syndromes the final court of appeal will be exploratory burr holes or cerebral angiography. One important reason for pushing the diagnosis as far as possible on clinical grounds is that in most cases the surgeon if he must explore should do so with a limited objective — namely, to exclude the presence of clot in the extradural or subdural plane. When such clots are not found there is always a strong temptation to put needles into the brain. In cases of internal carotid obstruction or of intracerebral haemorrhage such a manoeuvre only adds to the damage, and might undo the attempts of nature to establish an effective collateral circulation.

There is another group of cases which provide a closer resemblance to carotid obstruction than any of the intracranial haematomas. These are cases of traumatic infarction of the fronto-parietal cortex and the following is an example.

CASE IV. Mrs. B., aged 48, a wasted woman, fell out of bed. On the following day she lost her speech and could not move her left arm or leg. On the second day she was admitted to hospital where she was found to have a flaccid left hemiparesis. Her right pupil was dilated and the left was contracted; both reacted to light. She became unconscious and died on the fourth day.

Necropsy showed a carcinoma of the oesophagus which accounted for her wasting. There was a small bruise on the left side of her forehead, but no fracture of the skull. There was no extradural or subdural haemorrhage. A very little subarachnoid haemorrhage, of microscopic rather than macroscopic dimensions, was spread diffusely over the surface of the brain. In the right frontal lobe there was a sharply-defined area of softening and haemorrhage. In this area microscopic examination showed swelling and necrosis of the greater part of the cortex and adjacent white matter, with oedema and degeneration of the surviving areas. Near the surface there were numerous and often confluent petechial haemorrhages. The perforating blood vessels of the area showed fibrinoid necrosis of their walls and they were thrombosed. Around the vessels were leucocytes and sometimes red blood cells.

Comment. This is an important type of focal brain damage far away from the site of direct violence. The sharp rotation of the head which in our view is the main cause of damage in head injuries produces distortion of a shearing type which is maximal at the surface of the brain. In this case, over a circumscribed area of the right frontal cortex the perforating vessels are all affected by fibrinoid necrosis and obstruction of their lumina. The appearances are consistent with the perforating vessels having been wrenched out of the cortex.

I have seen other cases which probably fall into this group.

CASE V. W. A. C. (St. H. 1439) had a motor-cycle accident in April, 1940. He had a post-traumatic amnesia of 14 days. After recovering consciousness he had aphasia and partial weakness of the right arm and leg which recovered completely within 4 weeks of the accident. However, he remained somewhat dazed and forgetful and 3 months after the injury he began to have focal fits which began with clonic movements of the right middle and ring fingers and were followed by a temporary right

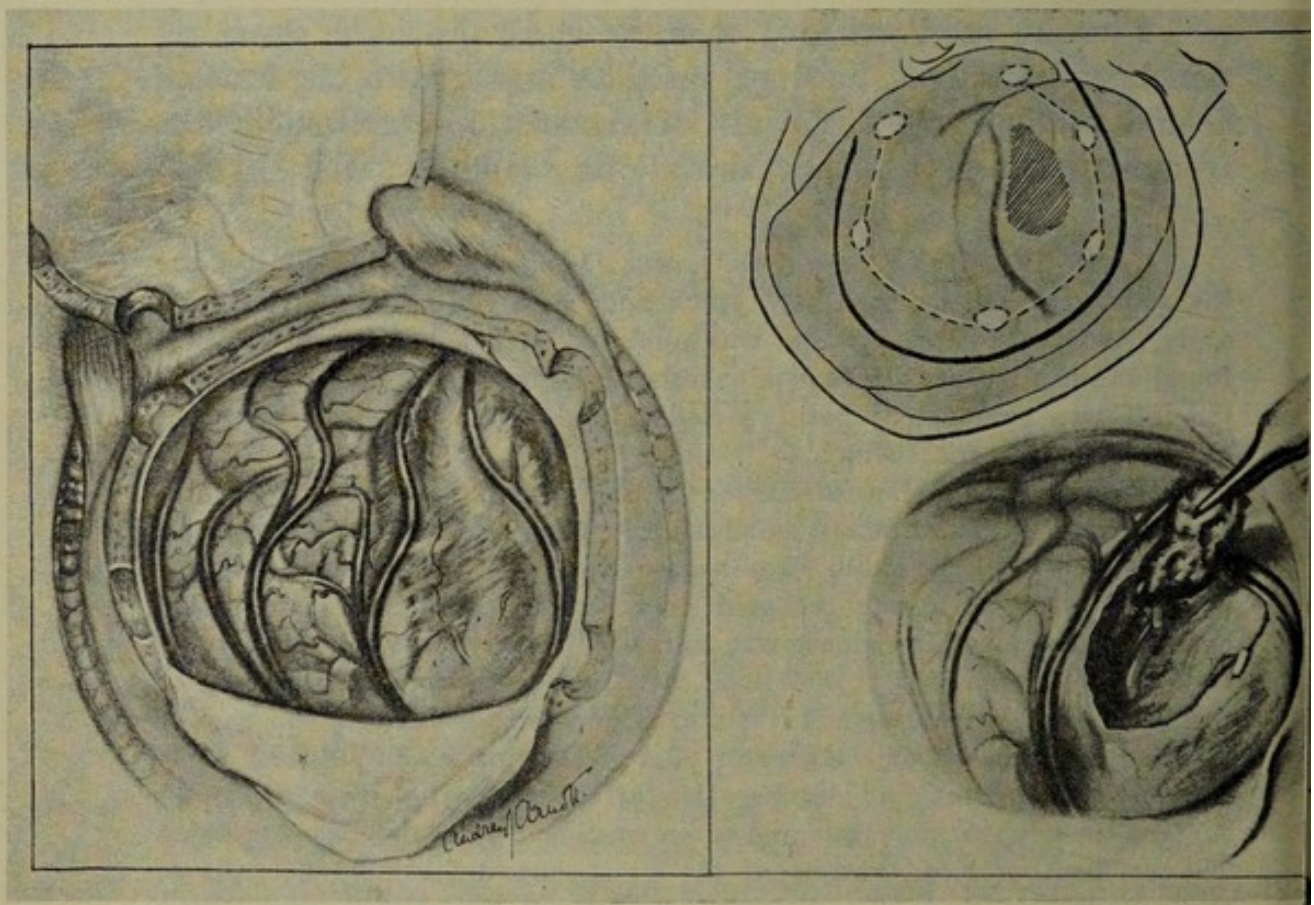


Fig. 9

hemiplegia and aphasia. Between August, 1940, and June, 1941, when he came under my care, he had 20 fits in all.

Examination showed slight right facial weakness, but no other neurological sign. Cisternal encephalography showed diffuse dilatation of the body and frontal horn of the left lateral ventricle.

At operation on July 17, 1941, a left osteoplastic flap was turned. No fracture was seen on either surface of the left parietal bone, and there was no sign of an old tear of the dura; but on the outer surface of the dura, alongside the middle meningeal artery in the lateral part of its course, there was a thin granular film of greyish-white material which may have been related to the old injury. In the hand and face area of the motor cortex there was a subarachnoid cystic area enclosing a mass of soft opaque yellow necrotic material, which measured 1.5 by 1.0 by 1.0 cm. (Fig. 9). This material was removed and the cyst emptied, leaving a cavity 2.5 by 2 by 2 cms. in the motor cortex.

He recovered satisfactorily from the operation, and during the ensuing nine months he had only two fits.

Comment. This I assume to be a later stage of the lesion seen at necropsy in Case 4. In this case, as in the previous one, there was no evidence of direct injury over the frontal cortex. I have seen several other cases of this type in which there was partial hemiplegia, and I suggest that in these cases we have a variety of head injury which may be recognizable as a distinct clinico-pathological entity. As a rule there was no fracture of the skull. In all of them there was prolonged unconsciousness after the accident and hemiparesis was observed during this period. It was not as a rule complete. In the early stages the corresponding lateral ventricle was normal or slightly narrowed as compared with the opposite one; later there was dilatation of its anterior horn and body.

The incompleteness of the hemiplegia in these cases should usually serve to distinguish them from the hemiplegia of carotid obstruction, though further experience may well show that the hemiplegia from carotid obstruction may at times be partial. And when a patient is deeply unconscious a hemiparesis may seem for a time to be a complete or nearly complete hemiplegia.

Pending more experiences with arteriorgraphy a tentative rule might be made that (1) cases of hemiparesis with prolonged unconsciousness (and usually without fracture) are more likely to be due to frontal lobe infarction; (2) cases of hemiplegia without unconsciousness are most likely due to carotid obstruction and embolus; (3) cases of hemiplegia with unconsciousness

and with fracture of one or both orbital roofs, perhaps also with damage to the optic nerve on the side opposite to the hemiplegia, are most probably due to carotid obstruction.

But this type of working rule is not very satisfactory. When there is need to speak of *differential* diagnosis it usually means that none of the conditions under consideration is clearly understood. Accurate differential diagnosis can probably not be obtained without arteriography.

II — TRAUMATIC ANEURYSM OF THE INTERNAL CAROTID ARTERY INTO THE SPHENOIDAL AIR SINUS

CASE VI. Head injury. Fractures of orbital plates of frontal bone and of right maxilla. Injury to right olfactory and optic nerves. Severe recurrent epistaxis. Aneurysm of right internal carotid artery displayed by arteriography. Ligation of right internal carotid artery. Recovery.

History. R. N. G. F., aged 32, had a motor-cycle accident on October 22, 1941. He received numerous lacerations about the face, fractures of the mandible, of the floor and roof of the right orbit, and a fracture of the roof of the left orbit involving the left frontal sinus. He had a total amnesia of four days' duration. When he recovered consciousness he found that the sight of his right eye was grossly defective, but thereafter it improved slightly and he saw double in all directions. He had also some impairment of hearing of his right ear and some pain in the right occipital region.

Examination. When he came under our care on November 17, 1941, his mental state was normal. He showed complete anosmia on the right side and partial loss of smell on the left side. Vision was J. 1 and 6/5 on the left, and on the right was reduced to J. 16 and 6/24 and was limited to the upper nasal quadrant of the field. The right optic disc was atrophied and the left was normal. The movements of the right eyeball were particularly impaired in an upward and inward direction. There was relative anaesthesia in the distribution of the right infraorbital nerve. He coul not open his mouth to quite the normal extent, and his bite was disturbed. Hearing was considerably reduced in the right ear. There was some weakness of the left deltoid, infra spinatus and biceps, due to a partial lesion of the right brachial plexus; it eventually made a good spontaneous recovery.

Subsequent Course. Early in December he began to get up. In the last week of December he was more active, and was well, apart from occasional small amounts of epistaxis which continued.

On January 7, 1942, eleven weeks after the accident, he had sudden severe epistaxis from both nostrils, approximately 1,000 c. c. s. of bright arterial blood being lost in seven minutes. The bleeding ceased spontaneously, leaving him in a collapsed state, with blood pressure of 86/58

and pulse rate of 118. He was restored with morphia and warmth, and was kept severely at rest.

On January 8 red blood count was 2,800,000; haemoglobin 60 per cent.

On January 9 there was a very slight epistaxis.

On January 10, quite suddenly, there was severe bleeding from the mouth and nose. Pressure on the right common carotid in the neck failed to control it. Pressure on the left common carotid was soon followed by cessation of bleeding, but by this time he was collapsed. Blood pressure was 60/42. He was given a blood transfusion and this brought moderate improvement.

Diagnosis. The severity and character of the epistaxis left little doubt that the bleeding was coming from one internal carotid artery, but there was no definite evidence as to which artery was involved. The damage to cranial nerves was virtually confined to the right side, and the fact that the right optic nerve was involved was in this connexion particularly significant. On the other hand, the fractures of the anterior fossa were radiologically more evident on the left side and there was also the observation that severe epistaxis had stopped at the time when pressure was applied to the left common carotid. This was probably of no great significance, since at that particular moment the patient was collapsed. The balance of evidence, it was felt, was in favour of the view that the bleeding came from the right side, but, up to this stage, repeated auscultation had failed to reveal any murmur over the temples or orbits, and the patient himself was not aware of any murmur. There was never at any time any protrusion of either eyeball. However, on January 11, the day after the second severe epistaxis, a soft murmur was heard all over the head, and this was stopped by compression of the right common carotid and was not affected by compression of the left.

Operation. In view of this, we decided to ligate the right common carotid, but when on January 12 the operation was to begin the murmur could no longer be heard. Accordingly arteriography of the right internal carotid artery was performed, and this showed an aneurysm of the right internal carotid artery projecting into the sphenoidal sinus (Fig. 10). The right internal carotid artery was ligated.

Progress. During the first twenty-four hours after operation he gradually developed considerable weakness of a flaccid type in the left upper limb, most marked in the distal joints and indeed virtually complete

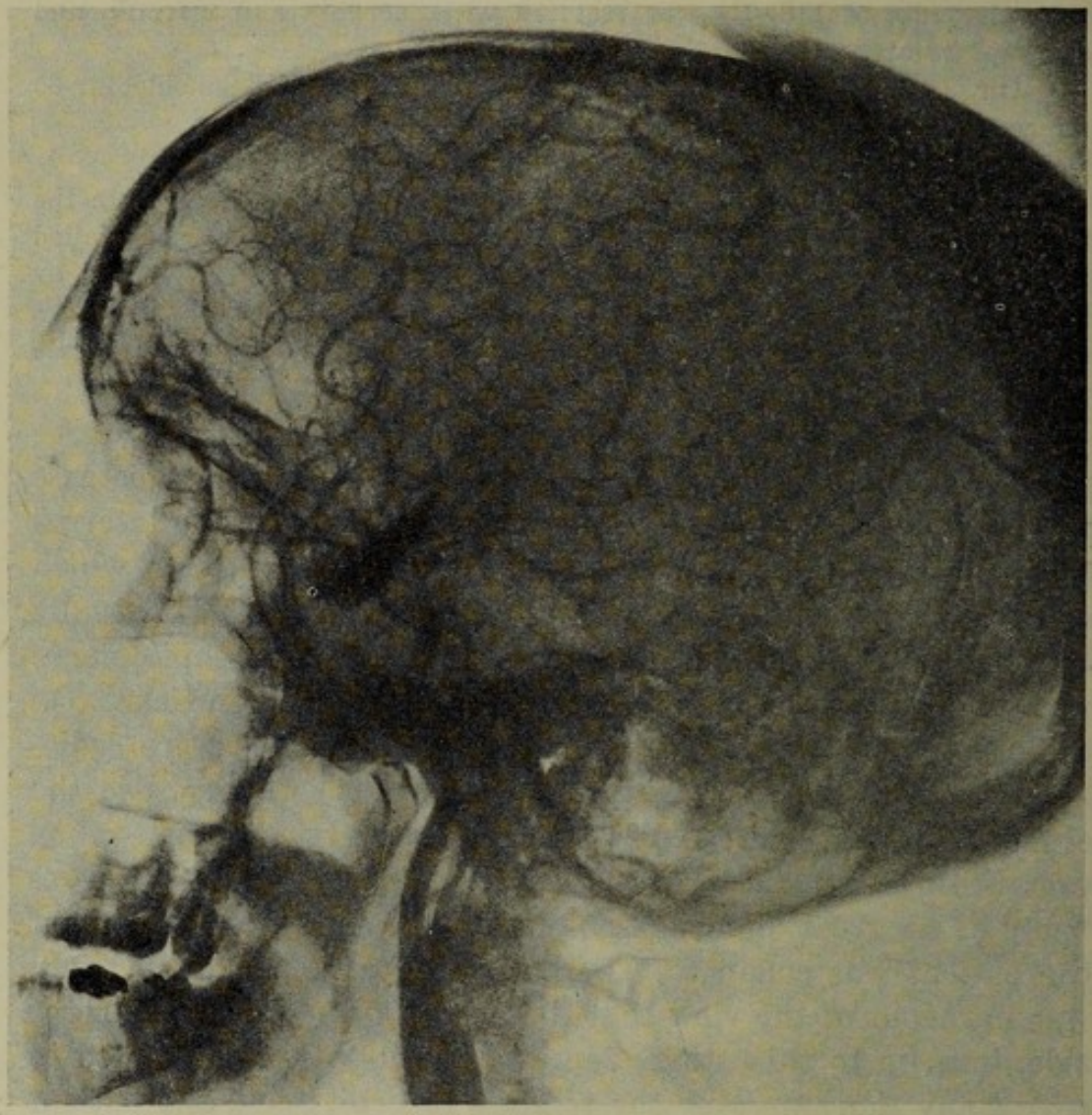


Fig. 10

in the fingers, and there was impairment of all modalities of sensation in the left hand. By January 16 these signs were beginning to improve and during the next two weeks they gradually cleared up completely. The patient returned to work on May 6, 1942, without further symptoms.

Comment. In this case it was clear enough that the severe epistaxis came from the internal carotid artery. Speaking generally, a sudden gush of arterial blood from the nose and throat in amounts of 500 to 1,000 c. c. can only come from a really large artery, and the internal carotid artery is the only vessel that is in most cases likely to be incriminated. Its closest relationship with the nasopharynx is at its terminal part just as it leaves the cavernous sinus. Here its anteromedial aspect is

immediately in contact with the bony roof of the sphenoidal air sinus. The difficulty that arose in Case III was to decide from which internal carotid artery the bleeding came, and though the balance of evidence favoured the right side, from the fact that the cranial nerves (including the optic nerve) were severely involved on that side and almost unaffected on the other side, yet there was no absolute certainty. In the absence of a murmur the only clinical test that could be made was to compress each common carotid separately while severe epistaxis was in progress, and this, as was shown, turned out to be an unsatisfactory test. In such cases as this, then, it is clear that arteriography should wherever possible be employed, as affording a satisfactory and precise method of diagnosis.

In the following case the train of events was similar to those of Case III, but the treatment through a misunderstanding had a most unfortunate outcome.

CASE VII. Traumatic aneurysm of the right internal carotid artery. Severe epistaxis. Multiple arterial ligations on both sides of the neck. Death.

History. N. S., an airman aged 19, (R. I. 914) was involved in a motor-cycle accident on July 10, 1938. He was severely concussed, and his total amnesia was about a week. He had a haematoma of the right eye, a lacerated wound in the right temples, and he was suspected of having a fracture of the base of the skull. On recovering consciousness he was found to be almost blind in the right eye, vision being limited to appreciation of hand movements. He was discharged from hospital on August 19. On October 4, twelve weeks after the accident, he had copious bleeding from the nose which was controlled by plugging the right nostril. He was readmitted to hospital. Further epistaxis occurred on November 2, November 5, December 2, and December 18. On December 21 his red cell count was down to 3,500,000 and haemoglobin content was 60 per cent., and at this time he complained that he could not see anything with his hitherto sound left eye. Examination of the eyes at this time showed total blindness, optic atrophy, on the right side and a normal disc on the left.

On January 6, 1939, he had two sharp attacks of epistaxis, and the nose was plugged again. By this time he had become so anaemic that blood transfusion was given. On January 10 lumbar puncture showed normal cerebrospinal fluid. There were on this day several small, brisk haemorrhages from the right nostril; on January 14 a very severe haemorrhage of 1 ½ to 2 pints poured out of the nose and mouth, and his pulse rate rose to 140.

Auscultation on this day revealed a sharp, high-pitched systolic murmur over the front of the head and face, and this was stopped by compression of the right common carotid and intensified by compression

of the left common carotid. There was complete anosmia, no perception of light, the condition of the optic discs as described above, both pupils fixed to light. On the right side the external ocular movements were all grossly impaired, except for outward movement, which was about half its normal range; on the left side movements of the eyeball were normal. There was ptosis of the right upper eyelid and very slight proptosis of both eyeballs. (The precise time of onset of the oculomotor nerve palsies on the right side is uncertain, but as the man had gone back to work before his first epistaxis occurred it is likely that they were not present at that time. And observations during the days preceding January 14 suggested that they were at that time progressive in character.)

First operation. On January 14 the right common carotid artery was tied under local anaesthesia and the bruit ceased immediately. A further transfusion was given on the same day, and another on January 20.

He remained well, but without any recovery of eyesight, until January 22, when his nose bled again, and further epistaxis took place on January 26 and January 28.

Second operation. January 30. The right common carotid was found to be pulsating above the ligature and was accordingly tied again.

Third operation. As bleeding continued the neck was explored again on February 2. No pulsation was found in the common carotid above the ligature. The right external carotid was tied.

Subsequent Course. February 20. Some oozing of blood from the right nostril. Bruit audible again. External ocular movements were now completely normal, but there was no recovery of sight, and the left optic disc was now, like the right, pale.

February 21. After slight headache for several hours, severe epistaxis of 1 ½ pints. The patient became pulseless and unconscious, but rallied and was given 1,500 c. c. of blood by slow drip.

February 25. A further severe epistaxis. Given 900 c. c. of blood.

February 27. On this day the patient came under my care. In spite of a good blood count he had a waxy, pale appearance. X-rays showed almost complete destruction of the anterior part of the floor of the sella turcica. At times a faint murmur was heard over the right orbit and it was not influenced by compression of the right common carotid artery below the site of ligation. No pulsation could be felt in the right facial artery, whereas on the left side there was good pulsation. These signs were taken, wrongly as later events showed, to indicate that the carotid ligations on the right side of the neck were complete.

In the following weeks a series of arterial ligations, intracranial and extracranial, were undertaken to stop the bleeding, but the relief after each was only temporary, and the patient died of epistaxis.

Post mortem showed a saccular aneurysm 2 cms. diameter in the sphenoidal air sinus (Fig. 11). In the right internal carotid in the bend alongside the anterior clinoid process and just proximal to the level of the ophthalmic artery was a hole 0.2 c. m. diameter which led into the aneurysm. The greater part of the floor of the sella turcica was eroded. Apart from presumptive evidence of fracture in this region no other

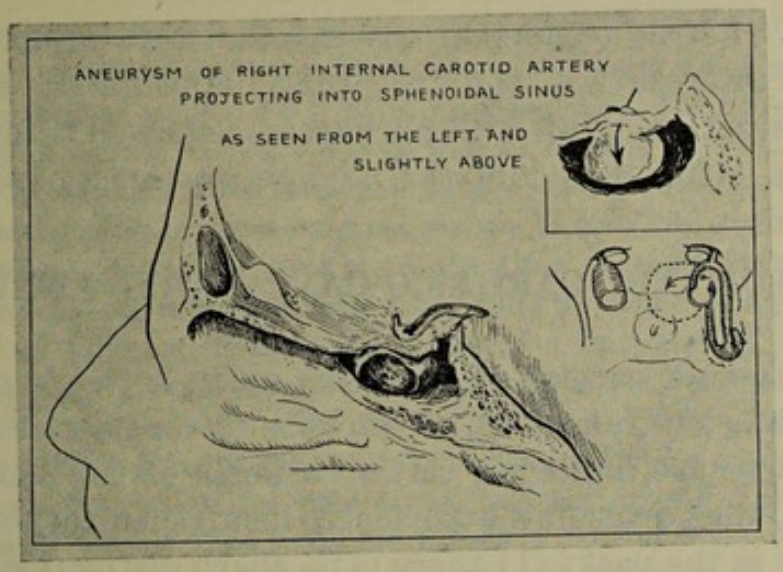


Fig. 11

fracture of the base of the skull was found. Examination of the vessels in the neck showed that the original ligature of the right common carotid artery had been incomplete.

Comment. The primary cause of failure in this case was incomplete ligation of the right common carotid. This was doubtless done deliberately in the first instance; for, as Schorstein has shown, there is considerable danger of infarction of part of the corresponding cerebral hemisphere attendant on complete ligation of the common or internal carotid. In consequence, many surgeons have come to practise incomplete or subtotal occlusion of the vessel; this proves to be a safer procedure and also is effective in a considerable proportion of the cases HALSTED (cited by DANDY) considered that partial occlusion of an artery would eventually bring about total occlusion of its lumen. While such treatment may be effective in carotico-cavernous aneurysm where the leakage of blood occurs from artery to vein, the needs are quite different in carotid aneurysms bleeding profusely into the nose. The haemorrhage must be stopped at all costs, and as the experience of Case 7 shows this cannot be done by incomplete ligation.

The literature on traumatic carotid aneurysms bleeding into the nose is scanty, and I have only been able to find one similar case, reported by BIRLEY (1928).

In Birley's case there was severe, exsanguinating epistaxis some months after a severe head injury, and it was stopped by

ligation of the right common and external carotid arteries. Of particular interest was the fact that, immediately after the injury, the patient while still unconscious showed a flaccid left hemiplegia. After 48 hours the hemiplegia rapidly cleared up.

III — TRAUMATIC CAROTICO-CAVERNOUS ANEURYSMS

This type of carotid aneurysm is much better known than the preceding one, and I do not propose to consider it systematically. There are, however, certain aspects of the condition to which, to judge from my experience, insufficient attention has been given in the past: (1) there is no sharp distinction between this and the preceding group; (2) there is a risk of embolism into the tributary cerebral arteries after carotid ligation.

CASE VIII. Head Injury. Left carotico-cavernous aneurysm. Ligation of left internal carotid artery. Improvement. Sudden death from cerebral embolus and epistaxis.

History. While riding a motor-cycle in daylight on December 3, 1941, R. D. M., aged 17 (St. H. 2815) crashed into the back of a car which pulled up suddenly. He had severe bruising of his left temple and the left side of his face, a fracture of the left side of the frontal portion of the frontal bone, going down to the frontal sinus and the roof of the orbit, a fracture of the left squamous temporal bone, and diastasis of the lambdoid sutures. There was severe epistaxis and bleeding from the left ear. Within half an hour his left eyeball was observed to be proptosed, with gross chemosis of its conjunctiva and its pupil fixed to light. Cerebrospinal fluid drained from his nose for the first 48 hours. He was unconscious for 3 days, with periods of restlessness and delirium for a further 5 days, and his amnesia was subsequently found to extend from 7 minutes before the accident to 10 days after it. However, by the fourth day he appeared rational for long periods, and at this time he began to complain of a noise in the head; on auscultation a loud murmur was heard over the greater part of the head, with its maximal intensity in the left temporal region. By December 21 he was able to get up.

Examination. He came under our care first on January 13, 1942, complaining only of noises in the head, blindness of the left eye, and general weakness. There was a loud systolic murmur in the head which was stopped by pressure on the left common carotid and not altered, so far as auscultation could determine, by pressure on the right common carotid, though the patient himself said that it was then reduced to half its previous intensity. Pressure on the left common carotid made the patient feel queer. Sense of smell was normal. Vision was normal on the right side; the left eye was completely blind and the left optic disc was pale. There was partial ptosis on the left side. The left eyeball was

proptosed, with engorgement and chemosis of the conjunctiva; its movements were normal except for limitation in an upward direction. The pupillary reactions corresponded with the visual loss. The central nervous system was otherwise normal. His mental state was normal, except for slight lack of insight. Blood pressure 122/84; pulse rate 88; and neither of these values was affected by compression of the left common carotid.

Treatment. As a preliminary to operation the left common carotid was compressed on five occasions for gradually lengthening periods of from 7 to 30 minutes, without ill effects. On January 21, under local anaesthesia the internal carotid was gently closed by two thick silk ligatures in a single hitch until the bruit ceased. Fifteen minutes later the bruit returned faintly, and digital obstruction of the exposed common and external carotid arteries, also of the internal carotid, did not abolish it. The ligation of the vessel was completed.

Course. After operation the proptosis and chemosis of the left eye diminished. The murmur still persisted faintly. No motor signs developed in the right limbs or disturbance of speech. The patient got up 10 days later. On February 3, while drying himself after a hot bath, he suddenly felt unsteady on his feet and then noticed that his right upper limb became helpless and objects looked misty. He lost consciousness for two or three minutes and was laid on his bed. In bed he felt better, but his right upper and lower limbs began to twitch and continued to do so with diminishing intensity and frequency for half an hour. From this attack he made a complete recovery, and he was discharged from hospital on March 1, at which time the murmur was still faintly audible. On arrival home he seemed perfectly well, but on the following evening while playing cards his nose suddenly began to bleed, and within a few minutes the haemorrhage became severe. Within 15 minutes he was unconscious. He was taken to hospital, but was dead on arrival there 35 minutes after the bleeding began.

Necropsy. Through the kindness of Dr. J. R. Stead the brain and part of the base of the skull were procured for further study. There was a fracture of the left frontal bone extending down on to the roof of the left orbit, but not into the optic foramen.

Projecting down from the left side of the roof of the sphenoidal sinus there was an aneurysmal false sac, 2.0 cm. by 1.4 cm. by 1.0 cm., covered on its inferior surface by a thin layer, 0.2 cm. thick, of shaggy, friable brown and yellowish-brown clot. This aneurysmal sac was connected with the anterior part of the left cavernous sinus through a hole in the bony roof of the sphenoidal sinus, and indeed the posterior third of the wall of the sac was composed of the bulging and thinned dura of the wall of the cavernous sinus. The interior of the sac communicated by wide openings with the interior of the cavernous sinus.

The distal part of the cavernous portion of the left internal carotid artery showed two holes. One was a pin-head opening in its postero-lateral wall 0.4 cm. proximal to the line at which the cavernous portion perforated the dura to become the intracranial portion of the artery. This opening led directly into the aneurysmal false sac. The second hole was

in the medial and inferior wall of the most distal part of the cavernous portion of the artery in the concavity of its last bend, and measured 0.25 cm. in diameter; it opened both into the sac and into the cavernous sinus.

Proximal to the larger hole the interior of the internal carotid artery had a glistening wall, but distal to this hole the arterial wall had a finely granular appearance and its lumen was narrowed throughout the whole 1.1 cm. of its intracranial part.

The orbital surface of the left frontal lobe was adherent to the roof of the left orbit and showed an area of superficial laceration 6 cm. from before back and up to 3.4 cm. from side to side; and there was also superficial laceration of the posterior 1.3 cms. of the right gyrus rectus (Fig. 12).

The Circle of Willis had a normal distribution; but the anterior

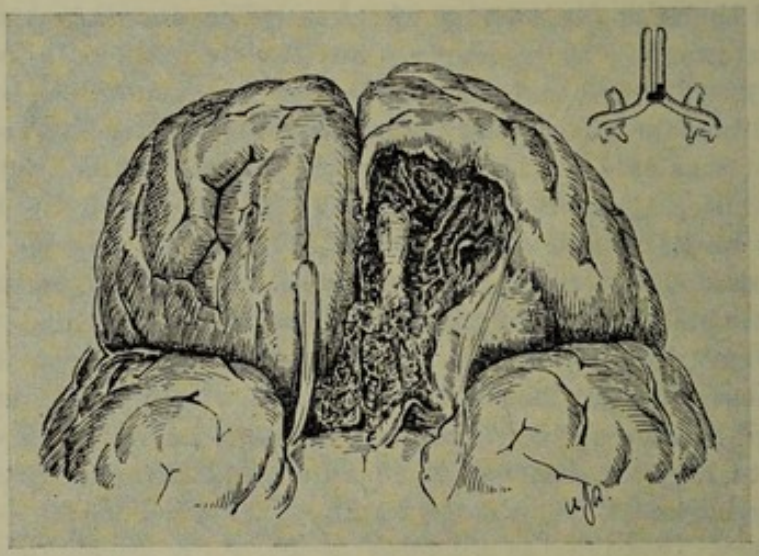


Fig. 12

communicating artery and the adjacent 0.5 cm. of the left anterior cerebral artery were occluded by soft grey clot (Fig. 12) which protruded slightly into the lumen of the adjacent right anterior cerebral artery, wich was of slightly greater calibre than its fellow.

Comment. Until the brain was carefully examined the presence of embolus was quite unsuspected. In the absence of any softening in the area of brain supplied by the anterior cerebral arteries it may be concluded that the obstruction produced by the embolus occurred shortly before death and that it so altered the blood flow as to raise the pressure in the aneurysmal sac, with resultant fatal epistaxis.

The case was an unusual one of carotico-cavernous aneurysm because of the extension of the sac into the sphenoidal sinus. In the presence of obvious signs of carotico-cavernous aneurysm this state of affairs could only have been appreciated by means of arteriography. The demonstration of a sac in the sphenoidal sinus should call for complete arterial ligation. In addition, in such cases the onset of fleeting attacks of unconsciousness, such as that which occurred two wecks after ligation of the carotid in the neck, might well raise the question whether, in order to avoid serious cerebral embolus the carotid should not be ligated inside the cranium, after suitable measures have been taken to prove that ligation in the neck is complete.

CASE IX. Head injury. Left carotico-cavernous aneurysm. Ligation of left internal carotid. Cerebral embolism. Death.

History. C. S., a male aged 40 (No. 22) received a blow on the left temple during a fight on November 25, 1927. He remembered the blow, and remembered walking a little way from the scene of the fight, but then he became unconscious. He recovered consciousness within a few minutes, but for 15 minutes after that he was unable to speak properly and could only make stuttering noises. For 30 minutes after the accident his right arm was numb, but then it recovered completely.

Next day he noticed a rhytmic blowing noise in his left ear which continued without change up to the time of his admission to hospital. Two weeks after the injury he began to see double and his right eye became more prominent. He had occasional shooting pains in the left temple. Three weeks after the accident he gave up work on account of double vision.

Sixteen years before he had received a blow on the left ear, which afterwards bled, and from that time his left ear had been slightly deaf.

On admission to the London Hospital (January 25, 1928) he was a healthy-looking man with pulsating exophthalmos of his left eye (Fig. 13). The conjunctiva was injected. His left lower lid drooped slightly. His pupils were equal and reacted normally. Movements of the left eyeball were greatly limited in all directions and there was double vision in all directions. There was a soft, systolic murmur over the left eyeball, most of the head, and down the left side of the neck, synchronous with carotid pulsation in the neck, which stopped when the left common carotid was compressed. The right retina was normal; on the left side the retinal veins were greatly engorged, but did not pulsate; and they did not alter when the left carotid was compressed for two minutes, although the conjunctival engorgement became noticeably less during the same period. Visual acuity was 6/9 in each eye.

There was slight left facial weakness of the lower neurone type. Hearing was reduced to whispers at 2 inches on the left side.

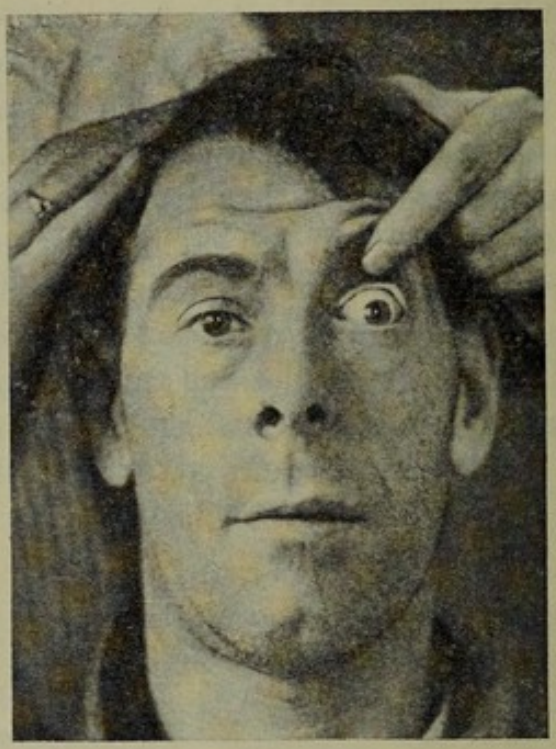


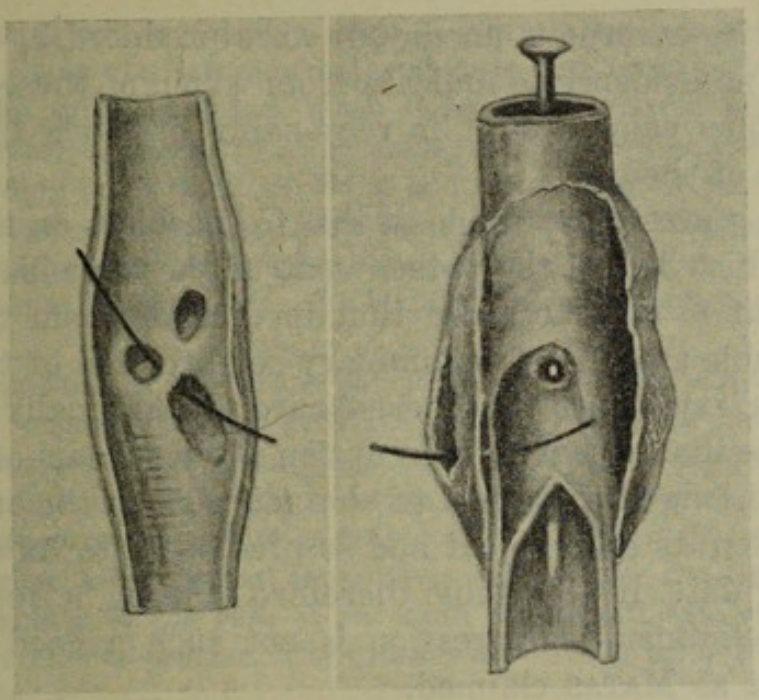
Fig. 13

X-ray of the skull showed no fracture. B. P. was 155/90; pulse rate 70, and regular.

Course. A few days after admission he began a course of carotid compression on the left side of the neck. At first he could only tolerate compression for 7 minutes because of feeling faint, short of breath and giddy, but he gradually worked up to 65 minutes three times a day. Every 10 minutes he would have to change the compressing finger owing to fatigue, and during 65 minutes, compression he would let about 20 beats through, as judged by the bruit. During this period of compression treatment his eye became less prominent and ocular movements improved.

February 23. Operation. The left internal carotid was tied under ether anaesthesia. Eight hours later the man had not regained consciousness. The ligature was undone, but he never recovered consciousness, and he died seventeen hours after the first operation.

Necropsy showed no blood clot in the left internal carotid artery in the neck, and the site of ligature could not be recognized. In the internal carotid 1 cm. above the bifurcation there was a minute fracture in an atheromatous plaque which might have been due to the ligature. Higher up in the neck, about 3 cms. below the foramen lacerum medium, there was an old dissecting aneurysm (Fig. 14-a). In the centre of the cavernous part of the left internal carotid on its inferior surface there was a hole (0.3 cms. diameter) in the wall of the artery which communicated with the cavernous sinus, and this hole was plugged with recent blood clot. The adjacent adventitia of the artery was dissected



a b

Fig. 14

away from the media, and it also had a hole (0.1 cm. diameter) which communicated with the cavernous sinus. The cavernous sinus showed on its inferior and medial aspects some dilated saccules, but contained no clot.

The most distal part of the internal carotid was occupied by a plug of tough yellow organized clot which, however, was not attached to the vessel wall and could be expressed from the cut end of the artery.

The other main cerebral vessels were normal.

There was slight swelling and pallor of the left cerebral hemisphere in the area supplied by the middle cerebral artery, and sections showed slight softening of this area, with foci of haemorrhage in the cortex and basal ganglia.

Comment. In this case death 90 days after the injury was evidently due to embolism lodging in the middle cerebral artery, and this probably took place at the time of operation. The fact that aphasia and weakness of the right hand occurred immediately after the injury is fairly strong evidence that the carotid was damaged by the injury to such an extent that the circulation of the left hemisphere was for a time imperfect. Doubtless spasm after injury would play some part. On this basis, and in the absence of history of any earlier injury, we may suppose that both the dissecting aneurysm in the neck and

the carotico-cavernous aneurysm were produced by the injury. There is no evidence to indicate from which of these aneurysms the embolus came, but it is reasonably probable that it came from one of them.

It is scarcely profitable at this to speculate on the circumstances which led to the detachment of the embolus in Cases 8 and 9, but it may be noted that in each of them there was a considerable amount of preliminary compression of the common carotid artery before the internal carotid was finally tied, and it is possible that repeated interruption of the stream of blood may tend to dislodge a fragment of clot adherent to the arterial wall, or to loosen its attachment and so predispose to its detachment at a later date. It is possible therefore that, as SCHORSTEIN suggests, preliminary compression is not such a good method of treatment as MATAS claimed.

Cases VIII and IX may be regarded as atypical examples of carotico-cavernous aneurysm inasmuch as they both died from cerebral embolus, the one 17 hours and the other 40 days after carotid ligation. In both cases it is probable that the clot came from the site of the aneurysm. As already noted, cerebral and retinal embolisms have occurred years after a gunshot wound of the neck (TÖNNIS (1934) MONIZ (1941).

IV — INJURY TO THE INTERNAL CAROTID ARTERY IN THE SUBDURAL SPACE

Case X. Mrs. K., aged 60 fell down some cellar steps in the dark and was found unconscious within 15 minutes. She had a cut on the left eye and bruising of the scalp just above the left ear and on the left shoulder. One hour later she was admitted to hospital: she could be roused to make restless movements, and when she did so she vomited. Her pupils were small and equal and reacted to light. Her other reflexes were present and both plantar reflexes were flexor in type. Pulse rate was 56; Systolic blood pressure, 150.

Three hours after the injury she was more difficult to rouse and her pulse rate had slowed to 36. Her pupils were still small, but no longer reacted to light.

Within an hour she could no longer be roused. Her face was pale, her breathing slow and periodic; all her limbs were flaccid and the tendon jerks on the right side were depressed. The right plantar reflex was absent; the left, extensor. Her left pupil was dilated and the right was small.

Operation was undertaken about 5 hours after injury. A burr hole was made below the left parietal eminence and large solid clots were found in the subdural space. When these were removed bright arterial blood continued to escape in large amounts. The burr hole was enlarged, but no bleeding point could be found and the surface of the brain looked normal, though sunken away from the dura. As the bleeding showed no sign of stopping, the left common carotid was tied, after a preliminary test, and this appeared to stop the bleeding. Blood transfusion of 300 c. c. was given.

During the next 12 hours she improved to some extent, becoming less deeply unconscious and moving both legs and her left arm. Her left pupil became small again for a time, but then it dilated once more. She died 24 hours after operation, without having regained consciousness.

A post-mortem examination was done by Dr. Gardner, who set aside certain specimens for me, but these never reached me. Dr. Gardner found a linear fracture of the base of the skull on the left side leading up to the anterior clinoid process. The brain was covered by a film of blood.

Comment. It is unfortunate that no examination of the carotid artery was made. The indirect evidence suggests that the internal carotid artery was torn in its most distal part, but, in the absence of post-mortem evidence, the possibility that the patient's fall might have been caused by rupture of an aneurysm of the internal carotid cannot be definitely excluded.

CONCLUSION

The evidence so far available for enabling us to suspect internal carotid damage at an early stage after closed head injury is not substantial, but these cases give some hope that, with further experience and the use of cerebral angiography, the clinical picture of the conditions will be eventually defined.

The most important symptoms so far elicited are those which indicate an immediate and transient anaemia of the cerebral hemisphere supplied by the damaged artery. Thus, in Case 9 aphasia and «numbness» of the right arm were present for 30 minutes after the injury; in Birley's case flaccid hemiplegia was observed for the first 48 hours, even though the patient was unconscious. So many of the patients are at first unconscious, when mild degrees of hemiplegia may be easily overlooked, that temporary unilateral cerebral anaemia may be more common than is at present supposed. However, there is reasonable certainty that it does not always occur, at least in those

cases such as Case I and Zannoni's case, in which the proximal part of the internal carotid artery is damaged in the neck. In these cases swelling of the neck may occur, and any case of head injury complicated by such swelling should always be suspect, to the extent that rigid rest in bed is prescribed.

The injuries of the distal part of the internal carotid artery appear to occur in two groups, either as an isolated lesion which results in a carotico-cavernous aneurysm, or as part of a severe frontal injury. In this second group there is usually damage to the frontal or ethmoidal sinuses, fracture of the roof of the orbit, damage to the adjacent optic nerve and to the olfactory tracts, and severe epistaxis. Though, as DAVIS (1939) has shown, the bleeding may come from a damaged anterior ethmoidal artery, any patient with severe arterial epistaxis after head injury should be suspected of injury to the internal carotid artery; and the suspicion becomes stronger in cases in which severe epistaxis is associated with damage to the corresponding optic nerve.

The importance of early recognition of these cases lies in the need to do everything possible to prevent embolus or completely obstructing thrombosis, and to be on the alert at a later stage to take appropriate steps to deal with severe epistaxis. These cases show that there is a strong tendency to embolus after injury of the internal carotid artery, and perhaps also to progressive thrombosis. The embolus appears to be precipitated by early movement, and it is reasonable to expect that the risk would be diminished by enforced rest in bed in the early stages.

Where, by reason of epistaxis and damage to the structures in the anterior fossa adjacent to the carotid artery, aneurysm projecting into the sphenoidal air sinus is suspected, arteriopraphy should be carried out before severe recurrent bleeding occurs; and, once the diagnosis is firmly established, and before the patient has been rendered anaemic by blood loss, the appropriate arterial ligation should be performed.

ABSTRACT

Evidence is brought forward to show that the syndrome of cerebral concussion is not due to cerebral anaemia, but to rotational aceleration, or deceleration, of the brain within the skull.

Ten cases are described of injury to the internal carotid artery in association with blunt head injury. Four varieties of injury are described:

(1) Traumatic obstruction of the internal carotid artery by thrombosis or embolism, producing delayed hemiplegia after head injury. This syndrome has to be distinguished from the various types of traumatic intracranial haemorhage and from traumatic infartion of the frontal lobe, a condition which is described in detail.

(2) Traumatic aneurysm of the internal carotid artery into the sphenoidal sinus.

(3) Traumatic carotico-cavernous aneurysm.

(4) Traumatic rupture of the internal carotid artery, producing subdural haemorhage.

The precise diagnosis of the conditions depends to a large extent upon cerebral arteriography. Early clinical signs of injury to the internal carotid artery are in some cases swelling of the neck, in other cases temporary hemiplegia in the first stage after the injury. Later, epistaxis may occur and, when associated with an injury that has damaged the corresponding optic nerve, is strongly suggestive of aneurysm of the internal carotid artery.

After injury to the internal carotid artery there is considerable risk of cerebral embolus and this may occur even years after the initial injury. It may also occur after therapeutic carotid ligation in the neck.

RESUMO

O A. apresenta várias provas de que o sindroma de comoção cerebral não é devido a anemia cerebral, mas às variações da aceleração rotatória do cérebro dentro do crânio.

Apresenta dez casos de lesões da carótida interna, associados a traumatismos não penetrantes do crânio das quais descreve quatro variedades:

1) Obstrução traumática da carótida interna por trombose ou embolia provocando hemiplegia retardada após traumatismo craniano. Êste sindroma tem de ser separado dos outros tipos de hemorragia intracraniana post-traumática e do enfarto traumático do lobo frontal, lesão que é descrita em pormenor.

2) Aneurisma traumático da carótida interna desenvolvido para dentro do seio esfenoidal.

3) Aneurisma carótido-cavernoso traumático.

4) Rotura traumática da carótida interna provocando hemorragia subdural.

O diagnóstico exacto destas várias lesões depende principalmente do uso da arteriografia cerebral. Os sinais clínicos precoces de lesão traumática da carótida interna são, nalguns casos, edema da região cervical e em outros hemiplegia temporária logo a seguir ao traumatismo. Mais tarde podem manifestar-se violentas epistaxis, que, quando associadas a um traumatismo que lesou o nervo óptico correspondente, são fortemente sugestivas de aneurisma da carótida interna.

Depois do traumatismo da carótida interna há sempre grande risco de embolias cerebrais, que podem dar-se mesmo vários anos após a lesão inicial, o que também pode suceder após laqueações terapêuticas da artéria no pescoço.

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