THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

MAY, 1925

COMMUNICATIONS

ON THE RELATIONSHIP BETWEEN SUBARACHNOID AND INTRAOCULAR HAEMORRHAGE*

BY

GEORGE RIDDOCH, M.D., and CHARLES GOULDEN, F.R.C.S.

Introduction

THE not infrequent occurrence of haemorrhage into the subarachnoid space within the skull has long been known, but up to relatively recent times mainly as a cause of death in cases in which an incorrect diagnosis had been made during life. It was not associated in the minds of clinicians with any distinctive symptomatology, and in consequence the belief was generally held that its recognition was almost impossible at the bedside and its outcome usually fatal. But after the introduction of lumbar puncture by Quincke over twenty years ago it was found that the presence of blood in the spinal fluid under certain circumstances was a clear indication of bleeding into the subarachnoid space. Since then many important papers have been devoted to the subject, but our knowledge of it nevertheless is by no means complete.

Aetiologically there are three main groups of cases: subarachnoid haemorrhage from: (1) traumatic rupture of meningeal vessels; (2) primary intracerebral haemorrhage, the blood usually reaching the subarachnoid space by bursting through the brain substance; (3) non-traumatic rupture of a meningeal vessel or aneurysm.

* Read at a meeting of the Section of Ophthalmology, Royal Society of Medicine, June 9, 1924.

Subarachnoid haemorrhage from a ruptured aneurysm of one or other of the basal arteries is in some ways the most interesting group and the one to which most attention has been recently directed. Symonds[58], especially, has added much to our knowledge of the clinical picture which is often so distinctive that a diagnosis of primary non-traumatic subarachnoid haemorrhage can frequently be made with reasonable certainty even without the aid of lumbar puncture, but it is clinched when blood is found in the spinal fluid.

Such cases often occur at an age when intracerebral haemorrhage is not very common. According to Ingvar[29] the vascular lesion may be due to inflammatory or degenerative arterial disease, but commonly it is the result of rupture of a so-called *congenital* aneurysm of one of the basal arteries (Turnbull[63], Fearnsides[21], and Symonds[58]). Although a temporary rise of blood-pressure appears often to be the immediate cause of bursting of such an aneurysm, arterial hypertension and arterio-sclerosis is, as in our cases, often strikingly absent.

Bleeding into one or both eyes is a frequent but not constant result of subarachnoid haemorrhage from rupture of an intracranial aneurysm. Four examples of this association have been observed by us and form the basis of this paper. One only (No. I) of the four patients died and came to autopsy, and an eye was removed and examined histologically in order to ascertain the means by which the intraocular haemorrhage occurred.

Illustrative Case.—Subarachnoid haemorrhage from ruptured aneurysm of the right intracranial carotid at the origin of the middle cerebral artery. Haemorrhage into both eyes. Death after thirteen days. Autopsy. Microscopical examination of right eye.

No. 1. L.H., Reg. No. 10584/1924. Charles N., aged 44 years, skin-broker, was admitted to the London Hospital under the care of Mr. Russell Howard on February 14, 1924. His previous health had been good except that he was believed to have had sunstroke in Palestine.

In the evening of February 14, 1924, he was following some friends upstairs in his house when he suddenly fell down. He did not lose consciousness and managed to walk up to his room. A little later his friends heard him fall and found him lying on the floor unconscious. At short intervals he had three fits, in each of which he foamed at the mouth and was convulsed and cyanosed. In the second fit there was opisthotonus.

He was taken to the London Hospital the same evening and on admission was in a state of "cerebral irritation." He was conscious and answered questions more or less intelligently, but resented being disturbed and examined. His pupils were of medium size, equal, and reacted to light and on accommodation. His ocular movements were full. All his tendon-jerks were exaggerated and an extensor plantar response was obtained on both sides. All voluntary movements were carried out well and co-ordination was good. Sensation appeared to be unimpaired. His heart, lungs, and abdomen were clear and the blood-pressure readings were 140/80: temperature, 97°F.; pulse 100, full and strong; respirations, 24.

On February 15, his pulse was slow (52 to 64), but his blood-pressure had not altered. On February 15, his muscles were flaccid, his tendon-jerks were difficult to evoke and his plantar reflexes were normal. On February 19, five days after the onset, his temperature rose to 100°F; pulse 56; respiration 22. On February 20,

he tended to "screw up" his left eye and saw double on looking to the right. His tendon reflexes were absent.

On February 21, temperature 99.2°F.; pulse 72; respirations 22. The diplopia had disappeared, but otherwise his condition had not changed. On February 22, temperature 100°F.; pulse 80; respirations 24.

On February 24, his temperature had dropped to 99°F.; pulse 70. He had difficulty in swallowing, was very drowsy and could be roused only with difficulty. There was weakness of voluntary movement of the left side of his face and his tongue was protruded to the left. His pupils and ocular movements appeared to

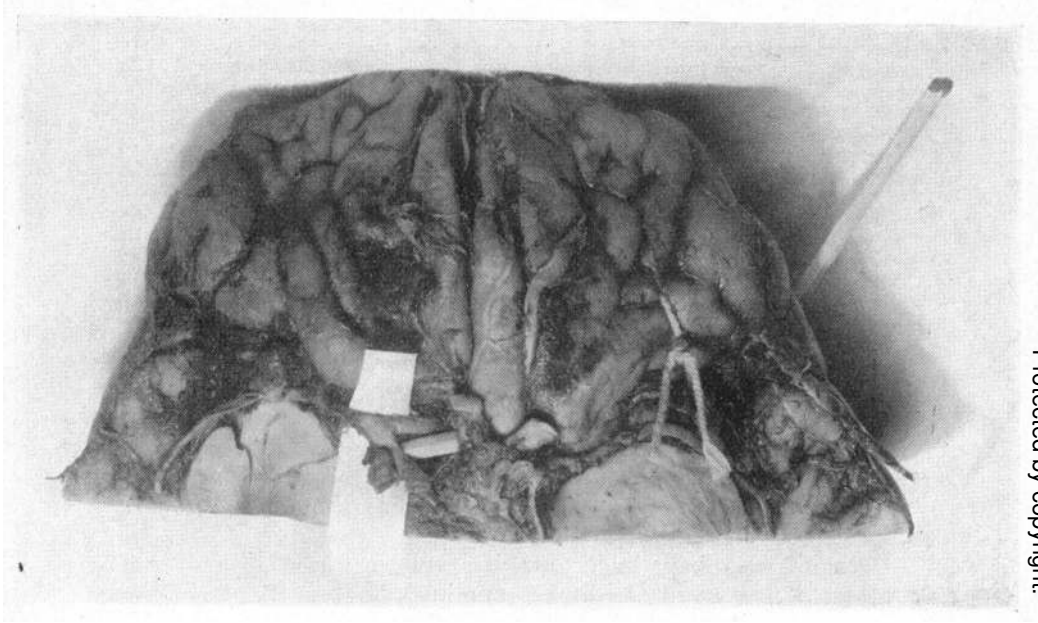

FIG. 1.

To show aneurysm of the right intracranial carotid at the origin of the right middle cerebral in Case I.

be normal. No tendon-jerks could be obtained, and for the first time there was incontinence of urine. Blood-pressure readings were 140/90.

On February 25, his condition had not altered except that the facial palsy was less and his blood-pressure had risen to 170/105.

On February 26, he was examined by one of us and the following note was made: He was stuporous and cyanosed, his breathing stertorous, grunting, and about thirty-four to the minute. His pulse rate was 114 and temperature 102°F. He could not be roused. Both fundi showed haemorrhages in the neighbourhood of the discs which were definitely swollen. In the left fundus there were two large crescentic haemorrhages lying alongside the inferior veins close to the margin of the disc. His pupils were equal and reacted well to light; the reactions on convergence could not be tested because of the patient's mental state.

There was flaccid, left hemiplegia. All tendon-jerks and abdominal reflexes were absent and both plantar responses were extensor. He responded by grimacing to painful stimulation on either side. There was incontinence of urine. His heart's apex beat could not be felt, nor on account of his noisy breathing could his heart's sounds be heard. His urine contained albumen, and there were signs of congestion at the base of each lung.

On lumbar puncture, a small quantity of uniformly pink fluid was drawn off. It was found to contain blood and an excess of lymphocytes. The Wassermann reaction was negative.

Next day, February 27, his temperature rose to 104.2°F.; pulse 132 and respiration 62, and he died on the thirteenth day of his illness.

Autopsy.—There was no conspicuous flattening of the cerebral convolutions. A considerable amount of red-brown blood and a little blood-clot, altogether measuring a few drachms, was found in the subdural space. In the posterior part of the longitudinal fissure there were two symmetrical areas of subarachnoid haemorrhage. Other small subarachnoid collections of blood were present, scattered over the cerebral convexities. A large amount of red and brown blood-clot lay under the arachnoid in the right Sylvian fissure and a smaller amount in the left Sylvian fissure and anterior part of the median fissure and cisterna basalis. A saccular aneurysm, one centimetre in diameter, projected backwards from the right intracranial carotid at the origin of the right middle cerebral; it was ruptured at its inner pole (Fig. 1). No other aneurysm or arterial abnormality was found. No blood was seen round the optic nerves in the optic foramina, but within the orbits the sheaths of the optic nerves were distended with blood. The right eyeball was opened and multiple haemorrhages were seen round the margin of the optic disc, the two largest lying upon vessels. The left eyeball was not examined.

The lungs were the seat of purulent bronchitis and of broncho-pneumonia. There was no cardiac hypertrophy and only a moderate amount of atheroma. The liver was large, with congestion in the centre of the lobules, and the spleen was flabby.

The left kidney was grossly atrophied and fibrotic, and its pelvis was cystic and projected inwards. The upper part of the ureter was very contracted and apparently impervious. There was compensatory hypertrophy of the right kidney. No abnormality in the renal vessels was found.

The Wassermann reaction in the blood, post-mortem, was negative.

The Clinical Picture of Rupture of a Basal Aneurysm

Before briefly discussing the clinical manifestations of subarachnoid haemorrhage from rupture of a basal aneurysm it might be of interest to mention certain symptoms that may be present before the first seizure.

Patients who have bled into the subarachnoid space from a ruptured basal aneurysm have sometimes had excellent health up to the time of the attack. But careful inquiry into the previous medical history in such cases often elicits the fact that even in the absence of general arterial disease, more or less distressing symptoms have been present for a long time. It is possible that some at least of them, such as headache, are due to recurrent slight leakages.

Paroxysmal *headache*, often severe, has been complained of in several instances and may resemble migraine. Thus the patient in No. IV of our series for years before his first seizure suffered from recurrent headache so intense that he was temporarily incapacitated for work. The headache tended to be brought on by exertion or prolonged stooping, was associated with tinnitus and frequently culminated in vomiting. One of Ingvar's[29] patients was subject to headaches that were for a long time looked upon as migrainous.

Tinnitus, usually continuous with periodic exacerbations, is sometimes referred to in the literature and No. IV was troubled for years with noises in his head that waxed and waned in time

with his pulse. The association of aggravation of the noise with headache in his case has just been mentioned.

The symptoms complained of in No. III are of particular interest. The patient was aged 53 years at the time of his seizure. For as long as he could remember he had been periodically subject to attacks of epistaxis, the bleeding having invariably been preceded by lethargy, frequent yawning, and a sensation of heat on the top of his head. As soon as his nose began to bleed these symptoms disappeared and he felt, as he put it, ten years younger. His father, who died at 89 years of age, soon after he had had a stroke, suffered from recurrent epistaxis for the greater part of his life, and the patient's two children are also nose-bleeders.

The onset of the seizure is always sudden.

From inquiry into the circumstances under which a seizure has developed it is usually clear that the aneurysm had burst at a time when the patient was carrying out some form of activity sufficient temporarily to raise his blood-pressure. In Nos. II and IV the patients were straining to micturate early in the morning when they were suddenly seized with severe headache and rapidly became unconscious. In No. I the attack occurred in the evening, while the patient was walking upstairs with some friends; without any warning he suddenly fell down but did not lose consciousness and was able immediately to rise and ascend the stairs unaided. A little later he was heard to fall in his room and was found lying unconscious on the floor.

The mode of onset in No. III is interesting. It will be remembered that the patient suffered from epistaxis and that the bleeding was always preceded by sleepiness, yawning and sensations of heat on the top of his head. In August last these symptoms appeared and as usual persisted for some time. One evening he went to bed thinking that his nose would soon begin to bleed, but he awoke in the morning with severe occipital headache and rapidly became unconscious. Although this patient's usual blood-pressure was within normal limits there is little doubt that the periodic epistaxis was a measure of safety, for after each attack he felt much better in health. It is highly probable that, on this occasion, if his nose had bled as usual he would have escaped the seizure from subarachnoid haemorrhage that developed.

The coma into which the patient passes may rapidly become profound. All the signs of cerebral and medullary compression appear and he may die within a short time. In such cases the tear in the aneurysmal wall is large and the clinical picture is indistinguishable from that of fatal intracerebral haemorrhage.

More characteristic however is a clinical state which, apart from the onset, resembles that of meningitis. There is an early phase of more or less deep coma, during which the patient may

be convulsed. He then passes into a condition in which there is clouding of consciousness, disorientation and delirium. He talks in an excited rambling fashion, often about his work but may answer questions more or less sensibly. He complains bitterly of headache and pains in the back of his neck. Vomiting sometimes occurs and there is often considerable restlessness, the patient tossing in his bed from one side to the other. His pulse-rate may be normal or a little slower than usual. Later, pains in his limbs appear and he may lie with them curled up resenting interference of any kind. In consequence it is difficult to get him to co-operate in the clinical examination. Signs of local disturbance of neural function are always present but are usually slight. The ophthalmoscopic appearances, when abnormalities are present, will be described later. One or other of the cranial nerves, most commonly the third or sixth, may be affected, probably, as Symonds[58] suggests, as the result of local pressure by, for example, a jet of blood impinging on them. Frequently there are bilateral signs of pyramidal disturbance, such as diminished abdominal reflexes and an extensor plantar response on each side, but usually voluntary power is not seriously impaired. The tendon-jerks may at first be either diminished or abolished. It is impossible, as a rule, to test sensibility in a satisfactory manner, but in none of our cases has it been obviously affected. Not uncommonly there is retention or incontinence of urine but loss of control of bladder function may be less complete.

An almost constant early sign is rigidity of the neck, and later Kernig's sign is often present on both sides. After the first few days, it is usual for the temperature to be moderately raised, this rise being attributed by Froin, probably correctly, to haemolysis.

Such, in brief, is the clinical picture presented during the early stage in a moderately severe case of subarachnoid haemorrhage from a ruptured basal aneurysm. Examples, however, are met with in which the initial disturbance of function is less marked. Although consciousness is probably always affected to some extent, there may be no deep coma or other evidence of gross compression of the brain.

One invariable sign of great importance has yet to be mentioned, namely, blood in the spinal fluid. Froin in 1904, fully described the characteristic changes in the cerebro-spinal fluid drawn off by lumbar puncture. Of these, three only need be mentioned, since they are sufficient to differentiate such a fluid from one that is sanguineous as the result of accidental puncture of a vein: (1) If three successive samples are taken they all show complete admixture of blood and spinal fluid; (2) no coagulum appears when the specimen is allowed to stand; and (3) when the red cells

sink to the bottom the supernatant fluid is coloured yellow or orange brown.

In regard to the course of the complaint, recovery from at least the first seizure is common. After two or three weeks consciousness returns, palsies, when present disappear, and the reflexes become normal. Sometimes slight mental defects, such as unreliable memory for recent events, may persist, but often the general recovery is remarkably complete. When, however, vitreous or

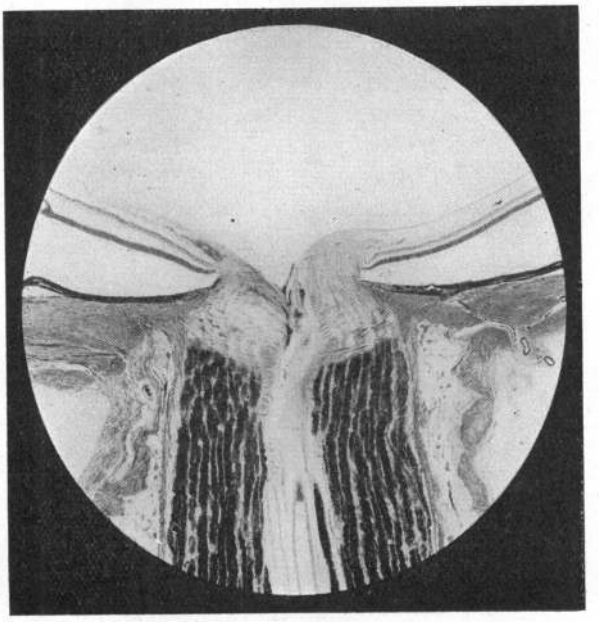

FIG. 2.

Longitudinal section of optic nerve stained with Weigert-Pal showing the relation of the layers of the optic nerve sheath.

subhyaloid haemorrhage has occurred in one or both eyes, vision seems to be more or less permanently impaired. Recurrent seizures are common, and one of them may prove fatal. One patient (No. IV in our series) has had at least four in the last three and a half years, and is still alive.

Some points in the Anatomy of the Optic Nerve and its Coverings

Within the skull the optic nerve lies in the subarachnoid space covered only with pia mater prolonged upon it from the brain. When it reaches the optic foramen it gains two more coverings, derived also from the meninges of the brain, and all three sheaths accompany the nerve until it reaches the globe of the eye. In this

way the intracanalicular and intraorbital portions of the nerve are covered by three concentric meningeal layers (Fig. 2).

(1) *The Pia Mater.*—This is the internal layer, thin and delicate, which constitutes the true neurilemma. It is a direct continuation of the pia mater, and is covered on its deep surface by a thin layer of neuroglia. Connected with the pial sheath on its inner surface are the septa that pass into the optic nerve, dividing it into two compartments. Blood-vessels pass into the nerve from the pial sheath along the septa.

(2) *The Dural Sheath.*—This is much thicker and more resistant than the inner sheath, having a fibrous structure. It becomes continuous with the dura mater of the brain along the circumference of the optic foramen.

(3) *The arachnoid sheath* is a very thin and delicate structure, and is the middle layer lying between the dura and the pia mater.

The Serous Spaces of the Optic Nerve

(1) *The subdural or arachnoid space* represents the subdural or arachnoid space of the brain. It lies between the dura and arachnoid mater, and is merely a slit-like space lined with endothelium divided into many compartments by a series of fine trabeculae.

(2) *The subarachnoid space* lies between the arachnoid and pia mater; it is continuous with the subarachnoid spaces of the brain, and is crossed in many places by trabeculae connecting the pia and arachnoid. In this way the subarachnoid space of the optic nerve is in direct continuity with the cisternae at the base of the brain, with their enclosed blood-vessels.

The Central Vessels of the Retina

The central artery of the retina, a branch of the ophthalmic, pierces the nerve on its infero-medial surface about 10 mm. from the sclera. It runs for a short distance upon the deep surface of the pial sheath, and gradually reaches the centre of the nerve, accompanied by the central vein, carrying with it a prolongation of the pial sheath.

The central vein has the same relations as the central artery, and opens sometimes into the superior ophthalmic vein, but more often direct into the cavernous sinus.

The Anterior Termination of the Optic Nerve

The nerve, its fibres having lost their myelin sheaths and correspondingly reduced in volume, pierces the sclera by many perforations (the so-called lamina cribrosa), and becomes continuous with the retina.

The following description will show the arrangement of the sheaths of the nerve at this point:

The dural sheath bends outwards at an angle of 110°, and becomes continuous with the outer layers of the sclera so intimately that no line of separation is visible.

The arachnoid sheath attached to the deep surface of the dural sheath, also becomes continuous with the sclera, the subdural space ending in a *cul-de-sac*.

The pial sheath, when it reaches the sclera, also turns outwards like the dural and arachnoid sheaths, becoming continuous with the inner layers of the fibres of the sclera. A few of the fibres, the most internal, enter into relationship with the choroid. The subarachnoid space thus also ends in a *cul-de-sac*, which is situated in the middle of the sclera on a plane posterior to the lamina cribrosa.

Ophthalmoscopic Signs

When considering the ophthalmoscopic signs in cases of haemorrhage into the optic nerve sheath, one must be careful not to confound them with changes that may result from injury to the optic nerve due to fracture of the base of the skull in the neighbourhood of the optic foramen—such an injury may or may not be associated with effusion of blood into the nerve sheath.

The cases discussed in this paper are those in which there is no direct injury to the optic nerve by any such object as a spicule of broken bone, or any damage to the nerve by some sudden strain or crush such as we may imagine is caused when there is fracture in the region of the orbit and optic foramen.

The earlier observers described the ophthalmoscopic signs of haemorrhage into the optic nerve sheath as being similar to those following embolism of the central artery, that is, thread-like arteries, red spot at the macula followed by primary optic atrophy, and later a pigmentary change around the disc which was supposed to be due to staining with blood pigment. In no instance did they carry out a pathological examination, and it is improbable that the appearances they described were due to haemorrhage into the nerve sheath without other injury to the nerve. For haemorrhage into the nerve sheath to result in pressure upon the central vessels of the nerve sufficient to give rise to the appearance of embolism of the artery and sufficiently prolonged to produce optic atrophy, it would be necessary for pressure within the nerve sheath and subarachnoid space of the meninges of the brain to be higher than the general blood-pressure—a state of affairs that is inconsistent with continuance of life. As to the peripapillary staining, it is also extremely unlikely to be due to blood pigment, judging from our experience with haematomas elsewhere in the body. The deep

pigmentation described and figured by Nicod[43] from a case of Rollet[51] suggests pigmentation from other causes, and is most likely of congenital origin. That pigmentation from the blood should occur around the disc is also improbable, as we shall see when discussing the pathological findings in cases of haemorrhage into the nerve sheath. Again, in no case in which the ophthalmoscopic investigation has been followed by pathological examination has the appearance of embolism of the central artery or peripapillary pigmentation been found associated with subarachnoid haemorrhage and haemorrhage into the optic nerve sheath.

In all the cases in which ophthalmoscopic examination has been followed by pathological investigation the appearances are so similar that it is possible to describe a characteristic clinical picture of the fundus that is constant in cases in which blood has spread into the optic nerve sheath from a subarachnoid haemorrhage, whether spontaneous (as in the cases here reported) or due to fracture of the skull.

Papilloedema.—This is the most common abnormality and is mentioned in almost all cases, the only exception being the case reported by Priestley Smith[50] and the three recorded by Elschnig[19 and 20], in the reports of which it is stated that the optic discs were normal.

The papilloedema is usually bilateral, corresponding to haemorrhage into both nerve sheaths. Its rapidity of onset is also remarkable. Uhthoff[64] states that he has seen papilloedema half an hour after haemorrhage into the subarachnoid space and well marked in five hours after the haemorrhage. It is usually slight in amount, but very severe papilloedema has been reported (Rollet[51]) with intense swelling and very small arteries. The veins are always engorged and tortuous.

Retinal Haemorrhages

In the majority of cases there are retinal haemorrhages. They are usually small and are often near the disc, but they may occur at a considerable distance from it, as was observed in one of our cases. They resemble retinal haemorrhages seen in other conditions.

Vitreous Haemorrhages

These are stated to be rare, but, curiously, they were found in four of the cases here reported and were so profuse that an examination of the details of the fundus was impossible in the early stage; and only when the blood had to a large extent disappeared could the whole of the fundus be seen. In one case it was thought that there had been a large pre-retinal haemorrhage

as there was a good deal of pigmentary change over a wide area around and including the macula, and, more peripherally, there was one fairly large haemorrhage along the superior temporal vein that had not been absorbed in four months but had disappeared four weeks later. In the other eye the haemorrhage into the vitreous was so profuse that a view of the fundus was impossible.

Retinal Vessels

Disease of the retinal vessels has not been noted in the cases reported and in our own cases they appeared to be healthy. Although papilloedema and retinal haemorrhages are seen in elderly patients who have haemorrhage into the optic nerve sheath, many of the cases have occurred in quite young or middle-aged individuals, so that the absence of vascular degeneration is not surprising. Apart from the cases of spontaneous haemorrhage into the subarachnoid space, so many cases have occurred of haemorrhage following injury in quite young people, that intraocular haemorrhage cannot always be due to disease of the blood-vessels or abnormality of the blood. In fact, as we shall see later when discussing pathology, the association of subarachnoid and intraocular haemorrhages can be reasonably explained even in the absence of general arterial disease.

Ophthalmic symptoms in the early stage are not mentioned in any of our cases or those already reported, and this is scarcely remarkable, since consciousness is clouded and often completely abolished whether the cause of the subarachnoid haemorrhage be spontaneous or traumatic. When consciousness has been recovered the patients complain of defective sight, which is extreme in those cases in which the vitreous is full of blood.

Pathological Examination of an Eye in Case No. I

The pathological examination was carried out by Professor Turnbull in the Pathological Institute of the London Hospital. This examination was very complete and many hundreds of serial sections of the posterior part of the eye and optic nerve were cut. We express our thanks to him for the trouble he took and for his help in the investigations.

The sheath of the nerve was tensely filled with blood-clot, which gave the sheath the appearance of a distended vein. The posterior half of the eye was removed with about two centimetres of the nerve attached, and was then placed in formalin. The main piece of nerve was divided half a centimetre behind the globe, so that sections of the eye could be made longitudinally, and of the nerve transversely.

The eye was embedded in celloidin, and the nerve in paraffin. The sections were stained with haematoxylin, eosin and other reagents, to show various points.

Microscopical Examination of Longitudinal Sections of an Eye in No. I

Under a *low* power the optic nerve sheath was seen to be distended with blood-clot, which was most obvious at the anterior extremity of the nerve, where there was an ampulliform dilatation of the sheath. Under a *higher* power it was possible to show that there was no infiltration of the nerve itself with blood, but that the

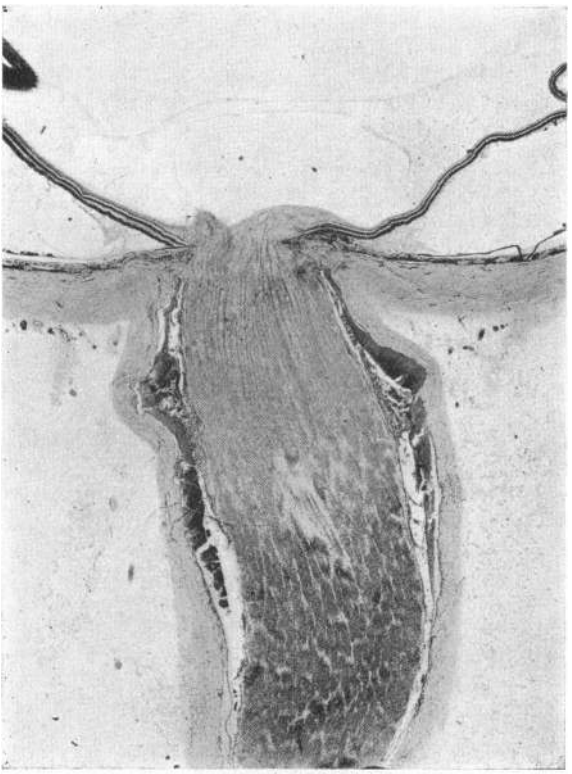

FIG. 3.

Longitudinal section of optic nerve from Case I. The vaginal sheath of the nerve distended with blood. No trace of blood in the nerve itself. Oedema of the optic nerve head.

blood-clot was exactly delimited by the pial sheath. In the sub-arachnoid space individual red blood corpuscles were visible, and some infiltration of the dural sheath by blood corpuscles could be seen. The sclera was not infiltrated with blood, and no corpuscles could be found surrounding the vessels within the nerve (Figs. 3 and 4).

The *papilla* was slightly swollen.

Retinal Haemorrhages could be seen here and there at some considerable distance from the papilla.

The Transverse Section of the Nerve

The subarachnoid space was full of blood. The blood did not pass into the pial sheath, and no sign of haemorrhage could be found within the nerve (Fig. 5).

The *central vessels* within the subarachnoid space were surrounded by blood-clot, and there was infiltration of the adventitial coat. This infiltration was also seen after the vessels

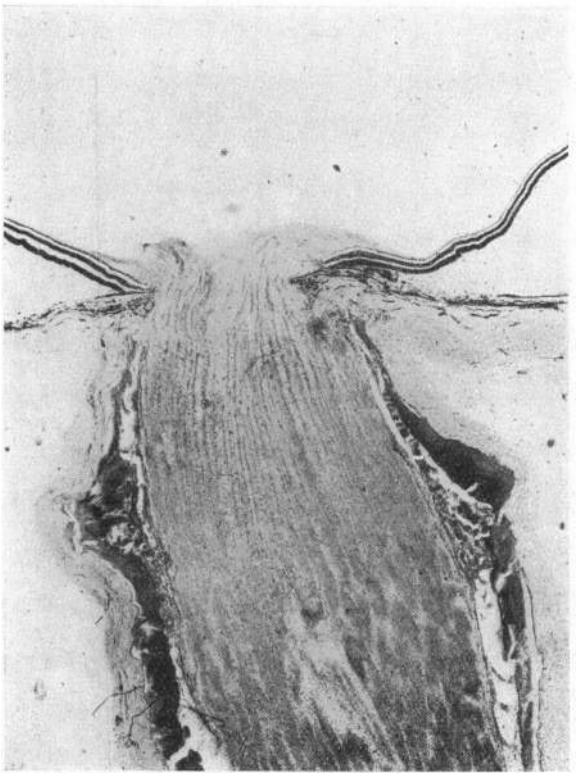

FIG. 4.

Longitudinal section of optic nerve from Case I under a higher power.

had pierced the dural sheath. There was no infiltration of the vessel sheath within the nerve.

It was not possible to show by section an obliteration of the vessels by compression (Fig. 6).

The microphotographs have been taken for us by Sir William Lister, who has also lent Fig. 2 from his collection. We would express our gratitude to him for his kindness.

Pathogenesis of the Intraocular Haemorrhage

How are we to connect the simultaneous appearance of a subarachnoid haemorrhage with haemorrhage into the retina or vitreous, and the development within a short time, of papilloedema?

Histological investigations have demonstrated the absence of any direct continuity between the haemorrhage into the optic

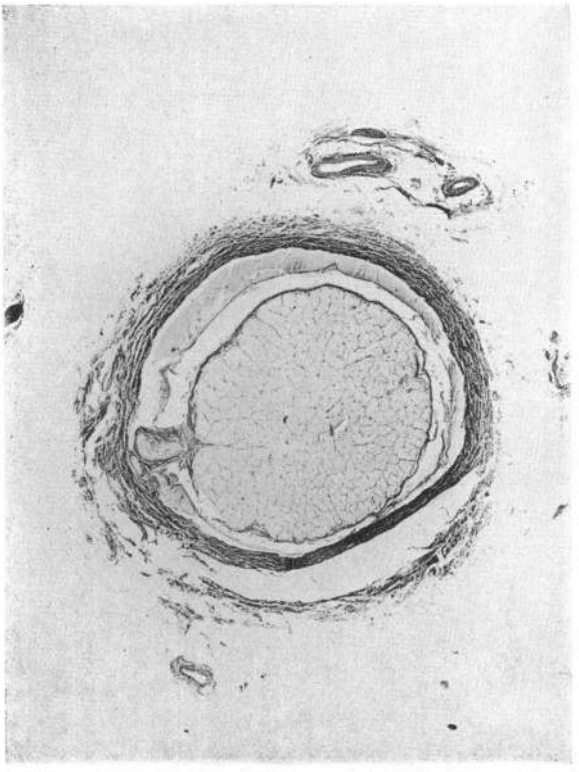

FIG. 5.

Transverse section of optic nerve from Case I before the vessels have entered the nerve. The nerve is surrounded with blood, but none can be seen in the nerve itself.

nerve sheath and the intraocular haemorrhage, a result that was to be anticipated from anatomical considerations. Further experimental investigation has shown that there is no connection between the subarachnoid spaces of the brain and optic nerve on the one hand and the lymphatic spaces within the eye on the other. Schwalbe[53] first showed the connection between the subarachnoid space of the brain and the sheath of the optic nerve, and demonstrated by injections under low pressure that fluid injected

into the subarachnoid space of the brain did not pass further forward than the *cul-de-sac* at the anterior end of the optic nerve sheath. Schmidt-Rimpler[54] injected coloured fluid under pressure into the subarachnoid space of dead animals and was able to make the injection penetrate the nerve itself as far as the papilla, and he described lymphatics piercing the lamina cribrosa. Subsequent investigations by Schwalbe[53] and others have failed to confirm

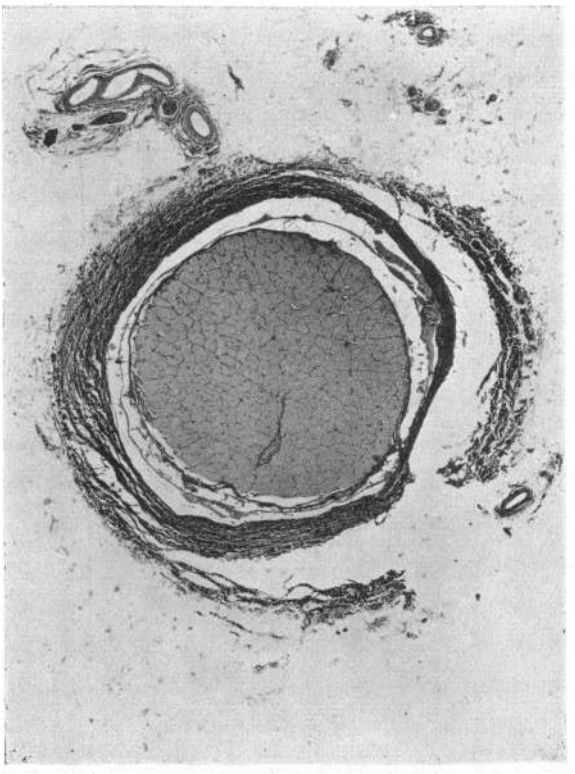

FIG. 6.

Transverse section of optic nerve from Case I after the vessels have entered the nerve. Again no blood can be seen in the nerve, neither is there any infiltration of the coats of the vessels with blood.

this, and so the results of Schmidt-Rimpler[54] cannot be accepted. It is to be noted that the injections were made by him *under pressure* of a degree very different to that found in life.

Sicard[56] has used much more delicate procedures, and has definitely proved that these supposed lymphatic connections do not exist. He injected grains of Chinese ink into the subarachnoid space of living animals, and showed by subsequent investigation that the grains never penetrate the pial sheath of the nerve, but

that they traverse the dural sheath so as to reach the neighbouring cellular tissue in great numbers. At the point where the optic nerve pierces the sclera the granules collect in the *cul-de-sac* at the anterior end of the optic nerve sheath, where they may be seen to pierce the dural sheath and enter the retrobulbar tissue. On the other hand none pierced the thickness of the sclera, papilla, retina or choroid. The point was proved by decolorizing the choroidal and retinal tissues with oxygenated water; this process always left them quite free from granules of Chinese ink.

Again, pathological investigations in cases of haemorrhage into the sheath have always proved the pial sheath and optic nerve to be free from blood corpuscles, whereas the dural sheath and the sheaths of the central vessels in the subarachnoid space, during their passage through the dura, and also outside the dura, are often distinctly infiltrated with blood corpuscles.

We may thus dismiss the suggestion that blood finds its way into the eye by piercing the pial sheath of the nerve and in that way passing through the lamina cribrosa into the eye. It must also be borne in mind that retinal haemorrhages in these cases are not necessarily at, or even near, the disc.

It will have been noticed that the ophthalmoscopic appearances conform very much with our ideas of venous engorgement of the retina, which is seen in its most marked instance in cases of thrombosis of the central vein of the retina; namely, papilloedema, enlarged veins and retinal haemorrhages. Since papilloedema has been seen within half an hour of the onset of the subarachnoid haemorrhage (Uhthoff[64]), it is obvious that something has occurred within the optic nerve sheath at least soon after the subarachnoid haemorrhage, and this is most assuredly a distension of the sheath of the nerve with blood, such as we see on pathological examination.

It has been shown by Dupuy-Dutemps[15] and by Paton and Holmes[49] that when the optic nerve sheath is distended in cases of raised intracranial pressure with papilloedema, the lumen of the vein in the subarachnoid space of the nerve is flattened, and thus much narrowed. Although experiments by injecting non-toxic and non-irritating fluids into the subarachnoid space of the brain have failed to give rise to papilloedema, owing to the rapid balancing of the intracranial pressure by dissemination and excretion Leber and Deutschmann produced the exact appearance of papilloedema in an animal by the injection of agar-agar directly into the optic nerve sheath, thereby causing a *prolonged* compression of the central vein of the retina in the subarachnoid space.

In sections of the nerve in these cases of haemorrhage into the nerve sheath oedema of the nerve is found to end abruptly

at the point at which the vein leaves it, so that there is marked oedema between the papilla and the exit of the vein only, behind which point the nerve is healthy.

All these considerations point to the cause of the trouble being interference with the venous return from the retina and optic nerve. We have seen that distension of the subarachnoid space of the optic nerve causes compression of the vein (Dupuy-Dutemps[51], Paton and Holmes[49]), and this is doubtless due, not only to the raised pressure in the space, but also to the traction on the vein in the vaginal space caused by separating the dural and pial sheaths. The obstruction is sudden, thus causing appearances similar to those seen in thrombosis of the central vein, although the results are not so serious as the obstruction is neither so complete nor so prolonged as in thrombosis.

Subarachnoid Haemorrhage without Intraocular Haemorrhage

Although haemorrhage into the subarachnoid space may be followed by bleeding into one or both eyes this complication is by no means invariable as the records of many cases show. (Symonds[58] and others.) Its presence or absence is probably dependent upon several factors, one of which is probably the size of the rupture. But the severity of the haemorrhage cannot be the only determinant, as is evident from a review of the literature. The post-mortem evidence in the following case (Case V) would seem to indicate that the situation of the aneurysm and the presence of previous attacks leading to thickening of the arachnoid between the bleeding point and the optic nerves are also of importance in this connection.

No. II. Case probably of subarachnoid haemorrhage from basal aneurysm in an otherwise healthy man of 46 years. Two attacks within three weeks. Bilateral intraocular haemorrhage and slight pyramidal signs with clouding of consciousness and evidence of meningeal irritation. Almost complete general recovery with considerable improvement of vision.

George K., aged 46 years, Wholesale Clothing Manager.

On June 16, 1923, while passing urine in the early morning he complained of headache and rapidly lost consciousness. The coma was not deep but it persisted and he was very restless with occupational delirium. His temperature was slightly raised.

When he was seen at his home by one of us in the evening he was delirious, talking about his work and continually moved about in the bed. His attention could be held for a short time only and in consequence it was difficult to get him to co-operate in the examination. He was, however, not actively resistive. He appeared to be completely disorientated. In his right eye a haemorrhage in the vitreous entirely obscured the fundus and in the left a small mass of blood was seen. His right pupil failed to react to light and the left reacted only sluggishly. Otherwise the functions of his cranial nerves, so far as they could be tested, appeared to be normal. He was able to move both upper and lower limbs well. There was cervical rigidity and Kernig's sign was positive on both sides. Supinator,

knee- and ankle-jerks were present and equal on the two sides, abdominal reflexes were obtained but both plantar responses were extensor.

When urged to do so he passed urine which was free of albumen, sugar and blood.

On June 24, his condition had not altered. He was still delirious and restless and had to be kept under the influence of morphia. Headache was severe. After this date he began to improve but on July 6, he had a second attack in which he again became comatose with stertorous breathing. After a few days he again gradually passed into his previous restless, delirious state in which clouding of consciousness varied in depth from time to time.

On August 6, he suddenly began to improve. He became completely conscious and orientated and his headaches disappeared. Since then his improvement was steady.

On September 23, he was again examined. Mentally he was perfectly normal; he could think clearly and his memory was good. He did not suffer from headaches but his head felt heavy in the evening and he was still easily tired. There was no tinnitus and he slept well.

His chief complaint was defective vision. Peripheral vision was good but central vision was very poor, worse in the right than in the left eye. It was as if he was looking through a thick mist, but now and again, quite suddenly and for a moment only he was able to see as well as ever. He had been to the seaside and had noticed that when the mist in the front of his eyes had disappeared be could see ships a long distance out at sea.

Examination of his visual fields showed that there was a complete large central scotoma on the right side and a smaller, incomplete scotoma on the left. With the ophthalmoscope only a peripheral rim of retina could be seen. A black mass obscured the rest of each fundus.

His pupils and cranial nerves were normal except that he protruded his tongue slightly to the right.

There was no weakness, ataxia or dystonia of his upper and lower limbs and all his reflexes were normal.

Sensibility to light touch and pin-prick was unimpaired and postural sensibility was perfect.

His heart was not enlarged but the second aortic sound was accentuated and there was a mitral systolic murmur. The blood pressure readings were 200/110.

The right vision was hand movements, the left vision was finger counting. With a magnifying glass he could just read a little of J. 20.

Each vitreous was full of blood, so that there was but a slight fundus reflex. No details were to be seen.

He was examined on April 7, and reported great improvement both in vision and in general health. He had been back at work as manager of a wholesale clothing business since December, and apart from his vision had been very well. The only disturbance of mental functions he had noticed was some impairment of memory for recent events. He slept well, did not become unusually tired nor suffer from headaches.

His vision had improved considerably, more with his right (previously the worse eye) than with his left eye; with his right eye he could read a letter if it was written distinctly. Sometimes a curtain seemed to come in front of his eyes obscuring his vision but if he moved his eyes it would disappear to return again in about half a minute.

Fundi:

April 7, 1924. R.V. 6/12 J. 1. L.V. 6/18 J. 2. R. fundus well seen, the main mass of the vitreous haemorrhage being below. The optic disc and vessels appeared healthy. In the region of the macula the retina was mottled and in places white, glistening dots were seen. No retinal haemorrhages were seen.

The left optic disc and vessels were healthy and nothing abnormal was seen in the fundus which was less easily examined than the right fundus owing to there being much more opacity in the vitreous.

His pupils were moderately dilated, equal, regular, central and reacted well to light and on accommodation. Examination of motor, reflex and sensory function revealed no abnormality. His blood pressure was 190/120 and his urine apart from specific gravity was normal.

On June 10, he reported that his health had been excellent. His memory was still defective and his sight had not altered but there had been no headaches or

tinnitus and he had been sleeping well. The Wassermann reaction in his blood was negative.

No. III. Case probably of subarachnoid haemorrhage from basal aneurysm. Patient, aged 53 years, subject for years to recurrent epistaxis. Father and two sons of patient liable to nose-bleeding. One attack of unconsciousness with bilateral intraocular haemorrhage.

George T., aged 53 years, saddler. For as long as he can remember he had been subject to attacks of epistaxis, the bleeding on each occasion going on for about an hour. The attacks used to occur twice a year but more recently the interval between them had been about a year. Premonitory symptoms lasting for two or three weeks invariably preceded the onset of nose bleeding. They consisted of sleepiness, frequent yawning and sensations of heat in the top of his head. The epistaxis always gave him relief; as he said: "I then feel ten years younger."

His father, who had died at the age of 89 years soon after he had had a stroke, suffered from the same complaint. The patient's two sons, the only children, are similarly afflicted. His mother died at 56 years of age of chronic nephritis.

His wife was healthy. She had had two miscarriages.

As a boy the patient suffered from severe headaches, but after scarlet fever at the age of 12 years they disappeared. At 19 years of age he had gonorrhoea and was operated upon for stricture at 33 years of age. Radical cure for hydrocele had been carried out; prior to two years ago he was troubled with frequent micturition. He has taken alcohol in moderation and has been a heavy smoker.

Early in August, 1923, lethargy, sensations of heat in his head and yawning, the usual precursors of an attack of epistaxis, appeared but his nose did not bleed. These symptoms became more pronounced and on August 15, he woke in the morning with severe occipital headache. He told his wife to call in the doctor and remembers nothing more of what happened for three weeks. During this time he was evidently conscious enough at intervals to talk intelligently and to answer questions. He complained of feeling sick but did not vomit. His doctor reported that on the 15th, the day of his seizure, he was very restless, incoherent and giddy, but there was no paralysis of his face or limbs. His right pupil was dilated and fixed and he was blind. On the 16th, his condition had not altered except that he had become incontinent and his tongue was foul. There was no fever and he was able to take fluids. On the 17th, he was moved to Eltham Hospital, where he remained for five weeks, during which time he slowly improved. He "came to himself" three weeks after the onset of his illness of which he had no recollection, and realized that the sight of his right eye was very poor. There was no headache and he felt well.

Since then there has been no improvement in vision.

On November 2, 1923, he came to the Royal London Ophthalmic Hospital and was seen by one of us. In his right eye, vision was misty, there was no red reflex, and the vitreous was full of blood. In his left eye, vision was 6/18 and there were two haemorrhages, one on the superior temporal vein about half the diameter of the disc above the disc, and another smaller one to the inner side of the macula.

On December 4, he was examined by both of us at the London Hospital. He was an intelligent, healthy-looking man. Speech and articulation were normal. He was completely deaf to a watch in his right ear, air conduction being abolished whilst bone conduction was fair. In his left ear he could hear a watch at half an inch and air conduction was better than bone conduction. With Weber's test he referred the vibrations to his right ear.

Vision: Right eye—P.L. Left eye—6/18, 1.5D. sph. J. 2. Field full.

The right vitreous was full of blood-clot and the red reflex was absent. In his left eye the two haemorrhages were still present but smaller than before.

The functions of the other cranial nerves were normal. There was no weakness, ataxia, wasting or dystonia of his upper or lower limbs. His gait was normal. Sensibility to light touch, and pin-prick and postural sensibility were perfect. Supinator, knee- and ankle-jerks were brisk and equal on the two sides; the plantar responses were flexor. There was slight frequency of micturition.

His lungs were emphysematous. His heart's apex beat was not palpable; there were no murmurs. Blood-pressure readings were 150/98. Urine: 1020, pale, no albumen or sugar.

On June 10, he reported that he had had several slight attacks of epistaxis in the previous few weeks. His neck felt stiff as it had been since his seizure and occasionally there was a sensation of tightness in the occipital region. He was always conscious of his head. His sight had not materially altered.

The mist in front of his right eye at times disappeared entirely when he kept his head steady and then his sight was as good as ever, but as soon as he moved his head the mist reappeared. In consequence his sight in his right eye was always dim when he walked about.

The Wassermann reaction in his blood was negative.

This patient was seen again on March 20, 1925, when the Right Vision with an appropriate glass was 6/12 and the Left Vision 6/9. The optic discs were of good colour, and there were no signs of retinal haemorrhage. In the Right Eye there were a few large vitreous floaters.

No. IV. Subarachnoid and intraocular haemorrhage probably from a basal aneurysm in an otherwise healthy man of 43 years, who for years had suffered from severe headaches. History of several attacks, one severe, from which he recovered with visual defect.

S.V., aged 43 years, chemist For years he had been subject to periodic severe headaches often associated with vomiting. In each attack the pain was so severe that he had to remain in bed while it lasted, usually for a few days but sometimes for more than a week. In the more severe attacks he was said to have been semi-conscious and disorientated.

For long he had been partially deaf and troubled with hissing noises in his head that varied in intensity in time with his pulse.

Otherwise his health has been good. He denied venereal disease and had indulged very moderately in tobacco and alcohol.

On the morning of November 11, 1921, he got out of bed to pass urine. When straining to start micturition he was suddenly seized with severe pain in his head, rapidly became unconscious and had a series of convulsions. There was repeated vomiting. On the following afternoon he was seen by one of us and his family doctor, and was then semi-conscious, disorientated, delirious and restless, complaining at times of severe pain in his head. He resented interference and could be examined with difficulty, his temperature was normal and his pulse slow. He lay on one or other side with his head retracted and his lower limbs semiflexed, and an attempt to flex his neck or extend his knees with his hips flexed obviously increased his pain. He did not know where he was, and in answer to questions complained of severe headache and expressed himself forcibly as wishing to be left alone.

His pupils were moderately dilated and reacted to light. In his right eye there was a large vitreous haemorrhage that obscured the disc and the central part of the retina: in his left eye the veins were engorged, the disc hazy in outline, but not definitely swollen and no haemorrhages were seen. There was a slight divergent squint.

Facial movements appeared to be unaffected; he moved his upper and lower limbs fairly well, but it was impossible to get him to carry out movements on request. Supinator, knee- and ankle-jerks were absent as also were the abdominal reflexes, and an extensor plantar response was obtained on both sides. He responded well to pin-pricks, but sensibility could not be properly tested. He was incontinent. His heart, lungs and abdomen were clear, his arteries not thickened and his blood-pressure not raised. No albumen or sugar was found in his urine and the specific gravity was normal.

He was removed to a nursing home and was seen in consultation with Dr. Farquhar Buzzard, who recognized the condition as due to subarachnoid haemorrhage from a basal aneurysm. On lumbar puncture the spinal fluid was found to be uniformly blood-stained but was otherwise normal.

Mild pyrexia appeared after a few days, but apart from this the patient slowly but steadily improved. In March, 1922, he was able to go home.

On September 4, 1922, he came for re-examination. It appeared that three weeks after his discharge from the nursing home he had another but slighter attack when discussing his business affairs with his brother. The hissing noise in his ears

suddenly became interrupted and half an hour later severe headache commenced. It lasted for ten days but he neither lost consciousness nor vomited, and no symptoms referable to his limbs developed. But during this time the visual defect in his right eye increased.

Since then he had had no severe attack, but four days out of seven he woke in the morning with pain in the mid-frontal region that spread to the right temple and sometimes to the left. With the headache there was tenderness and stiffness in the back of his neck. These symptoms usually disappeared in six or seven hours if he kept quiet. He said that if on waking he felt excited he knew he was "in for a bad time."

His sight has not altered. He still complains of a muddy brown film in front of his right eye and a large partial central scotoma was found while the peripheral field was full. Vision with his left eye was excellent. Ophthalmoscopically, the right optic disc was clear but perhaps a little paler than the left: the macula could not be seen because of a vitreous opacity. The left fundus appeared to be normal. Examination of the rest of his nervous system revealed no abnormality except that the left lower abdominal reflex was relatively diminished. Mentally he was acute and declared that he felt perfectly well between attacks.

His heart was clear and his arteries not thickened, the blood-pressure readings being 150/110.

No. V. Subarachnoid haemorrhage in a woman, aged 42 years, without bleeding into the eyes. History of periodic severe headaches. Blood in spinal fluid. Death on the eleventh day of the illness. Post-mortem there was no sign of arterio-sclerosis of the cerebral vessels but a small ruptured saccular aneurysm was found at the origin of the posterior cerebrals from the basilar. The arachnoid over the cisterna basilis appeared to be thickened.

Miss S., aged 42 years. In childhood she had had an attack of acute anterior poliomyelitis and as the result of it her right leg below the knee had wasted and was weak. For years she had suffered from periodic severe headaches but otherwise her health had been excellent.

Her family history is of interest. Her father who died of tuberculosis had been married twice. By his first wife there were three children all of whom died of tuberculous meningitis. By his second wife, who is alive and well, he had five children, four sons and one daughter, the patient. One son died, aged over 40 years, in an apoplectiform seizure, and he is said to have taken alcohol to excess. Another son also died, but in early adult life after an apoplectiform seizure and a short illness similar to the patient's sister. The other two sons are alive and well except that they are subject to severe headaches as their sister was for many years before her illness about to be recorded.

One of us was asked to see her on June 29, 1924. Ten days before the patient had gone to the bathroom in the morning for her bath. Some days later when able to tell what had happened she said that she had felt an unpleasant taste in her mouth and examined her tongue in the mirror. She remembered nothing more and was found lying on the bathroom floor unconscious.

Her family declared that she had been unusually irritable for two or three weeks before and had complained of one of her severe headaches.

Dr. Stedman was at once called in and he found her completely unconscious with widely dilated inactive pupils and complete flaccidity except for doubtful slight rigidity of her left upper limb. She vomited repeatedly. Within half an hour she regained consciousness but remained in a confused state for most of the day. She complained of severe vertical and occipital headaches and continued to vomit at intervals. In the evening she had completely recovered consciousness. Her neck was tender and her pulse rate 72.

The severe headache persisted and four days after the onset her temperature was found to be 100.6°F., pulse 42, and respirations 26.

On the sixth day she was again unconscious with pin-point pupils. Lumbar puncture was carried out and Dr. Greenfield's report of the fluid was as follows: The sample of fluid received into three tubes were evenly tinged with blood. On setting a fine coagulum formed and the clear fluid was coloured canary-yellow, and

although no haemoglobin absorption spectrum was obtained it gave a definite bile pigment reaction due to altered haemoglobin. The blood admixture was estimated by counting the red cells as 1 in 400. The leucocytes were roughly in the same proportion as in normal blood.

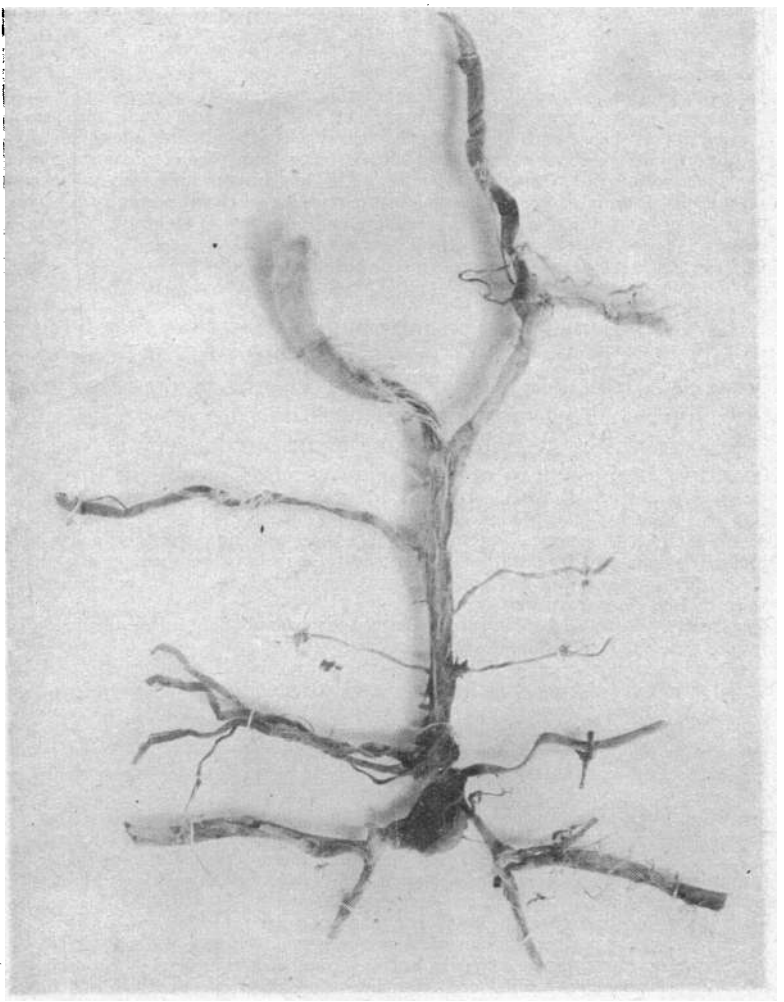

FIG. 7.

To show aneurysm projecting from the point of origin of the posterior cerebral arteries from the basilar in Case V.

The withdrawal of spinal fluid was beneficial and consciousness gradually returned. For the next three days she was relatively clear mentally during the day but complained of defective vision and of pain in her head, neck, back, and limbs. There was definite cervical rigidity. In the night she tended to ramble and sometimes tried to get out of bed.

On the evening of the ninth day she became confused, disorientated and irritable and it was difficult to keep her in bed. She was incontinent and hardly sensible enough to open her lips for food. Her temperature was still raised to 100.2° F., pulse 58, respirations 19.

On the tenth day when examined by one of us she was restless, tossing herself about in bed and rambling incoherently. Consciousness was clouded and it was impossible to get her to answer questions satisfactorily. There was stiffness of her neck and tenderness on gently stretching her sciatic nerves. The optic discs were clear, no haemorrhages were seen in the fundus or vitreous of either eye, and the retinal vessels appeared to be normal. Her pupils were small, equal and reacted to light; the reaction on convergence could not be tested. Ocular movements were full and there was no strabismus. Voluntary movements of her limbs and face were carried out but it was impossible to test them properly. The muscles below her right knee were wasted, the result of old poliomyelitis. Her right ankle-jerk and plantar reflex were absent and the left plantar response was extensor, but the other reflexes were present and equal. She responded to pin-pricks on both sides. Her heart, lungs, and abdomen were clear, her radial arteries not thickened and the blood-pressure readings 150/90. No abnormality was found in her urine. About 15 c.c. of spinal fluid, coloured light brown and under slight pressure, were drawn off by lumbar puncture.

She did not recover consciousness and died on the eleventh day of her illness.

A post-mortem examination of her brain was carried out by Dr. Greenfield, who reported as follows:

There was no subcutaneous effusion nor fracture of the cranium. The dura mater was under considerable tension and on removing it the convolutions of the brain were flattened. There was much clotted blood in the cisterna basalis, cisterna magna and pontine cisterna, and it extended out along the Sylvian fissures and over the surface of the cerebral hemispheres along the lines of the larger veins. There was no subdural haemorrhage nor was there any venous thrombosis. The cut cortex was of the normal pale colour.

The brain was washed and the arteries of the base examined carefully, but in the fresh condition no disease could be seen in them. The arterial walls were thin and delicate and there was nowhere the least sign of arterio-sclerosis. The basilar artery appeared to be smaller and thinner than normal. After fixation the arteries were re-examined on July 7. The internal carotid arteries and their branches were perfectly healthy. At the point of origin of the posterior cerebral arteries from the basilar, slightly to the right and towards the upper (deep) surface of the fork there was a small saccular aneurysm about the size of a small pea, with an oblong hole in it which would admit a fine knitting needle (Fig. 7). In the fixed condition it measured about 2 by 1 mm. The arachnoid over the cisterna basalis appeared to be thickened. Blood was not obviously present in the optic nerve sheaths.

There was a moderate degree of internal hydrocephalus.

This examination afforded evidence that death was due to the rupture of a saccular aneurysm which was probably of congenital origin, and to the hydrocephalus resulting from the accumulation of clotted blood in the cisterna magna and cisterna basalis.

Conclusions

1. Five cases of subarachnoid haemorrhage from ruptured basal aneurysm are described. In two cases only was the clinical diagnosis corroborated by post-mortem investigation. In another the diagnosis was strongly supported by the presence of blood in the spinal fluid. In the other two cases the diagnosis rested solely upon the history of the illness and the clinical signs present.

2. In no case was there evidence of cardio-vascular, renal disease, or syphilitic infection.

3. Four patients showed intraocular haemorrhage, retinal, subhyaloid or vitreous. The visual defect tended to improve, but recovery was never complete.

4. Histological investigation of an eye strongly suggests that the cause of the intraocular haemorrhage in such cases is obstruction of the central vein of the retina at the point where it leaves the optic nerve and enters the dural sheath. As the result of this obstruction the intraocular venous pressure is raised so that one or more vessels rupture. The mechanism of production of the haemorrhage is therefore in our opinion the same as that for papilloedema due to increased intracranial pressure.

5. The main factors that determine the occurrence of intraocular haemorrhage in cases of bleeding into the cerebral subarachnoid space from ruptured aneurysm are the severity of the leak, the distance of the aneurysm from the optic nerves and the presence or absence of arachnoid adhesions in the cisterna basalis.

BIBLIOGRAPHY

This bibliography includes references to cases of subarachnoid haemorrhage of traumatic origin associated with intraocular haemorrhage although this group has not been dealt with in this paper.

1. **Abadie.**—" Traité des Maladies des Yeux." II, p. 88, 1877.
2. **Battle.**—*Lancet*, II, pp 1, 57, 107, July 5, 1890.
3. **Bergmann.** - *Volkm. Klinisch. Vortraege*, No. 190, p. 154, 1881.
4. **Berlin.**—Graefe-Saemisch. Handbuch, VI, p. 558, 1880.
5. **Bernede.**—" L'Amaurose traumatique." *Thèse de Paris*, 1883.
6. **Bouchut.**—Communication à l'Academie des Sciences, July, 1875.
7. **Bougault.**—*Thèse de Paris*, 1913.
8. **Bouveret.**—*Rev. de Méd.*, XV, p. 536, 1895.
9. **Bristowe.**—*Trans. Ophthal. Soc. U.K.*, VII, p. 363, 1886.
10. **Bunge.**—*Münch. Med. Wochenschr.*, LII, p. 1296, 1905.
11. **Cantonnet.**—*Rev. de Chir.*, XI, p. 169, 1909.
12. **Chaillous.**—*Soc. d'Ophtal.*, XXIV, p. 305, 1907.
13. **Doubler and Marlow.**—*Arch. of Ophthal.*, XLVI, p. 531, 1917.
14. **Dufour et Gonin.**—*Encycl. franç. d'Ophtal.*, VII, p. 371, 1908.
15. **Dupuy-Dutemps.**—*Thèse de Paris*, 1900.
16. **Idem.**—*Arch. d'Ophtal.*, XXVII, p. 697, 1907.
17. **Idem.**—*Annal. d Ocul.*, CLI, p. 161, 1914.
18. **Durand.**—*Bull. de l'Assoc. franç. pour l'advanc. des Sciences*, Grenoble, p. 1217, 1904.
19. **Elschnig.**—*Arch. f. Ophthal.*. XLI, pp. 2, 281, 1895.
20. **Idem.**—*Ibid.*, XLVIII, pp. 2, 461, 1899.
21. **Fearnsides.**—*Brain*, XXXIX, p. 224, 1916.
22. **Flemming.**—*Lancet*, p. 429, 1903.
23. **Freud.**—*Wien. Med. Wochenschr.*, No. 9, p. 243, 1884.
24. **Ginsburg.**—*Westnik. Ophthal.*, XVI, p. 243, 1899.
25. **Gonin.**—*Annal d'Ocul.*, CXXIX, p. 89, 1903.
26. **Graf.**—*Deut. Zeit. f. Chir.*, LXVIII, p. 464, 1903.
27. **Haselberg.**—*Charité Ann.*, XXVII, p. 1094, 1903.
28. **Hutchinson.**—*Roy. Lond. Ophthal. Hosp. Rep.*, VI, p. 43, 1869.
29. **Ingvar.**—*Jour. Icon. de la Salpétrière*, XXVIII, p. 313, 1916-18.

30. **Kabsch.**—*Thèse de Würzburg*, 1891.
31. **Knapp.**—*Arch. f. Ophthal.*, XIV, p. 255, 1868.
32. **Lang.**—*Trans. Ophthal. Soc. U.K.*, XXIX, p. 156, 1909.
33. **Lawford and Edmunds.**—*Trans. Ophthal. Soc., U.K.*, VII, p. 208, 1886.
34. **Idem.**—*St. Thomas's Hosp. Rep.*, XI, p. 171. 1886.
35. **Leber.**—Graefe-Saemisch Handbuch, V, p. 907, 1877.
36. **Leber u. Deutschmann.**—*Arch. f. Ophthal.*, XXVII, i, p. 272, 1881.
37. **Mackenzie.**—Text-book, 4th edit., p. 1052.
38. **Magnus.**—*Die Sehnervenblütung*, Leipzig, 1874.
39. **Manx.**—*Deut. Arch. f. Klin. Med.*, IX, p. 339, 1872.
40. **Mardellis.**—*Thèse de Lyons*, 1900.
41. **Michel.**—*Arch. f. Ophthal.*, XXIII, pp. 2, 216, 1877.
42. **Nettleship.**—*Ophthal. Rev.*, XIV, p. 97, 1895.
43. **Nicod.**—*Thèse de Lyons*, 1906.
44. **Oishi.**—*Arch. f Augenheilk.*, LXI, p, 17, 1908.
45. **Panas.**—"Traité des Maladies des Yeux." I, p. 698. 1894.
46. **Idem.**—*Arch. d'Ophtal.*, XXII, p. 624, 1895.
47. **Parinaud.**—*Annal. d'Ocul.*, LXXXII, p. 26, 1879.
48. **Idem.**—*Ibid*, CXIV, p. 5, 1895.
49. **Paton and Holmes.**—*Trans. Ophthal. Soc. U.K.*, XXXI, p. 117, 1911.
50. **Priestley-Smith.**—*Trans. Ophthal. Soc. U.K.*, IV, p. 271, 1884.
51. **Rollet.**—*Rev. Gén. d'Ophtal.*, XXVII. p. 49, 1908.
52. **Schnaudigel.**—*Arch. f. Ophthal.*, XLVIII, pp 3, 460, 1899.
53. **Schwalbe.**—"Lehrbuch der Anatomie des Auges," Erlangen, 1887.
54. **Schmidt-Rimpler.**—*Arch. f. Augenheilk.*, XII, 1883.
55. **de Schweinitz and Holloway.**—*Trans. Amer. Ophthal. Soc.*, XIII, p. 120, 1912.
56. **Sicard.**—Quoted by Dupuy-Dutemps.
57. **Silcock.**—*Trans. Ophthal. Soc. U.K.*, IV., p. 274, 1884.
58. **Symonds.**—*Quart. Jl. Med.*, XVIII, p. 93, 1924.
59. **Talko.**—*Klin. Monatsbl. f. Augenheilk.*, XI, p. 341. 1873.
60. **Thenevet.**—*Thèse de Lyons*, 1894.
61. **Terson.**—*Annal. d'Ocul.*, CXLVII, p. 410, 1912.
62. **Tiegel.**—*Thèse de Breslau*, 1904.
63. **Turnbull.**—*Brain*, XII, p. 50, 1918.
64. **Uhthoff.**—*Berichte Ophthal. Gesellsch., Heidelberg*, p. 143, 1901.
65 **Wilbrand u. Saenger.**—*Neurol. des Auges.* III, p. 766.
66. **Williams.**—*Brit. Med. Jl.*, I, p. 157, 1882.
67. **Zacher.**—*Neurol. Centralbl.*, II, p. 125, 1883.

CENTRIPETAL FAN SCOTOMA IN GLAUCOMA

BY

DAVID PRIESTLEY SMITH, M.B.

BIRMINGHAM

THE accompanying maps of the field of vision, with the exception of Fig. 25, belong to cases of chronic glaucoma. They are selected as having one noticeable feature in common, namely, a scotoma tapering from the periphery of the field towards the fixation point, and not connected with—nor leading towards—Mariotte's Blind Spot. Fig. 3 is a striking specimen of the type. Fig. 22 is an exception in that it includes the region of the Blind Spot. Some of them were shown at a meeting of the Midland