The suggestion that the haemorrhage was intraspinal in origin is supported by several facts: (1) The almost complete absence of cerebral symptoms at onset. (2) The severity and localization of the pain in the lumbar region, the pain being excruciating at times and increased by movement. (3) Rigidity of spine, with a mild degree of opisthotonos. (4) Marked irritability and hyperaesthesia of the spinal roots and nerves. (5) The almost instantaneous relief of symptoms on lumbar puncture.

From whatever cause the haemorrhage arose there is no doubt that on four separate occasions bleeding of an extensive nature occurred, and though the illness was a protracted one, recovery appears to be complete and the sequelae insignificant. It is also of interest to note the close association of the fall of the blood pressure with improvement in the clinical condition.

SPONTANEOUS SUBARACHNOID HAEMORRHAGE

WITH REPORT OF EIGHT CASES

BY

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Leakage of blood into the cerebral subarachnoid space apart from any trauma is being recognized clinically with increasing frequency. The blood escapes from an artery, perhaps sometimes from a vein, into the meshwork of trabeculae which intervenes between the pia mater and the arachnoid membrane: this occurs most often from a vessel lying at the base of the brain. Haemorrhage into the subarachnoid space of the spinal cord has been recorded,^{6,7} but is much rarer. Blood escaping from a vessel within the brain may find its way either through the nervous tissue or through the foramina in the fourth ventricle into the subarachnoid space, but this paper is not concerned with these secondary types.

The underlying pathological lesion falls under several heads, and individuals of all ages may be affected. "Arteriosclerosis," in the form of medial degeneration, associated with high blood pressure and cardiovascular hypertrophy, is probably a common cause of spontaneous subarachnoid haemorrhage at or past middle life. In cases occurring in young adults the blood pressure is usually not raised, and the heart and peripheral arteries generally appear normal. Deficiency of the media has, however, been found in the arteries at the base of the brain, and it has been suggested that this is a congenitally installed defect.¹⁴ Syphilitic arteritis, when the inflammation is severe, leading to necrosis, may end in haemorrhage; when less acute a thickening of the intima occurs and there is no possibility of rupture. Weakening of the arterial wall by an embolus in infective endocarditis may occasionally result in rupture. It is possible that a vessel may give way as a late effect of cerebral embolism when the embolus is sterile; this is indicated by Case II below. Other known causes are: cerebral angioma, blood diseases, acute haemorrhagic infections, sunstroke, eclampsia, haemorrhage from tumours and cysts, and thrombosis of the superior longitudinal sinus.

The clinical picture shows wide variations, depending on the severity and site of the bleeding. In the most severe cases there is a sudden loss of consciousness and such compression of the brain that death supervenes within a few hours. In the mildest cases there will be headache, vomiting, and some pyrexia, followed by gradual recovery. Between these two all intermediate forms occur. The following is an example of a rapidly fatal haemorrhage.

CASE I

A man of 58 was admitted to the London Hospital under Dr. Robert Hutchison on April 22nd, 1929. He had been taken into the hospital seven weeks previously because of severe headaches beginning in the occipital and extending to the frontal region, each lasting about twenty-four hours and recurring weekly. Examination then showed subacute glaucoma of the left eye. The blood pressure was normal, and there was only slight arteriosclerosis.

On April 21st, 1929, the patient had several attacks of faintness of a few minutes' duration. Next day he became completely unconscious, and was admitted to hospital. The temperature was 99° and the pulse rate 60. On the left side the tendon reflexes were brisker, the plantar response was extensor, and there was ptosis of the left eyelid. Consciousness was temporarily recovered at 11 p.m., when he complained of headache. During the night there were three fits, and death occurred at 6.15 a.m. on April 23rd. The blood Wassermann reaction was negative.

Post-mortem Findings

The following is an extract from the records of the Bernhard Baron Institute of Pathology, by the kind permission of Professor H. M. Turnbull.

"There was a collapsed ruptured aneurysm, 1.2 cm. long and 0.7 cm. broad, at the junction of the left carotid, middle cerebral, and posterior communicating arteries. The aneurysm was covered by thin layers of adherent clot, and lay in a haemorrhagic depression in the left uncus, from which a haemorrhagic track led into the left lateral ventricle. The left lateral ventricle contained 4 to 5 drachms of clot and the right lateral and fourth ventricles a smaller amount. Subarachnoid haemorrhage was confined to the immediate neighbourhood of the aneurysm. The arachnoid membrane here was ruptured, and blood had passed into the subdural space. In this space some 6 to 7 drachms covered the left hemisphere, whilst less lay over the right hemisphere and still less in the posterior fossa; a layer of blood 1 mm. thick filled the subdural space of both optic nerves. There was rusty brown pigmentation of the dura in the posterior fossa and of the pia-arachnoid upon the inferior surface of both cerebral hemispheres, being greatest upon the left. An unruptured aneurysm, 1 mm. in diameter, projected from the junction of the right carotid and middle cerebral arteries, with an accessory posterior communicating artery. Microscopic examination of the aneurysm and adjacent arteries showed that the media and internal elastic lamina ended abruptly at the mouths of the aneurysms, whilst the walls of the aneurysms were formed by adventitia lined with a variable thickness of tissue continuous with the intima. The media of the right middle cerebral artery showed at some distance from the aneurysm patches in which muscle fibres were almost entirely absent. There was no evidence of arterial inflammation or degeneration. The aneurysms were therefore considered to be due to rupture of the intima and media at sites of congenital malformation of the media."

A haemorrhage causing initial coma may abate or cease and consciousness be subsequently regained. Symptoms of meningeal irritation, of general cerebral compression, and of local cerebral irritation or paralysis will then probably be observed; these symptoms persist for a variable period; a fresh leakage of blood may suddenly, or perhaps gradually, lead to a fatal conclusion. The following three patients illustrate this type.

CASE II

A man of 38 suddenly fell unconscious on March 17th, 1926, and was admitted in a stuporose condition to the Middlesex Hospital under Dr. E. A. Cockayne. He had had acute rheumatic carditis in 1912, and in 1924 a cerebral embolus causing left hemiplegia. On admission he was mentally confused and disorientated, complained of headache, and was constantly putting his hand to his head. The pupils were unequal, but the cranial nerves were otherwise normal. There was neck rigidity, and Kernig's sign was

present. The arms showed *flexibilitas cerea*, but no loss of power. The tendon reflexes were absent in the legs, and the plantar reflex was flexor. The cerebro-spinal fluid was under increased pressure, and contained a large quantity of blood; culture was sterile. There was evidence of cardiovascular hypertrophy, and the blood pressure was 210/90. Paroxysms of auricular fibrillation occurred while the patient was under observation, and although no diastolic murmur was heard a reduplicated first sound and a systolic murmur at the apex suggested mitral disease. The temperature varied from 100° on admission to 103° just before death. The urine contained some albumin only at first. Four days after admission a lumbar puncture showed less pressure of cerebro-spinal fluid, but the stupor gradually deepened, and death occurred on March 24th, six days after the onset of haemorrhage. Unfortunately there was no necropsy.

CASE III

A woman of 51 was admitted to the Middlesex Hospital under Dr. C. E. Lakin on April 17th, 1928. There was a history of severe headaches and vomiting for years. On the day of admission she was out shopping and feeling quite well, when she was suddenly seized with shaking and trembling, and saw flashes of light. Within a minute consciousness was lost. When examined in hospital she was stuporose, but she rapidly improved, and questions could be answered intelligently. Headache was severe, and for the first two days she vomited. Examination of the nervous system revealed no abnormal signs except some stiffness of the neck. The disks were indistinct, but not beyond the physiological limit. The heart was normal, and the blood pressure 140/75. Lumbar puncture showed a blood-stained sterile fluid; the Wassermann reaction was negative. Blood analysis was normal, and there was no albuminuria. The temperature was intermittent, and reached a maximum of 103°. Eight days after admission the plantar reflex was extensor on the right side; this was followed by a sudden right hemiplegia with absolute aphasia. Lumbar puncture at this time showed an increased quantity of blood in the fluid. The patient's condition remained unchanged for three or four weeks. Then she became slowly weaker, passed gradually into coma, and died on June 11th. There was no necropsy.

CASE IV

A youth of 17, referred by Dr. Siggs of Canford Cliffs, was playing football on October 8th, 1932. He had an attack of severe headache, and was sick; this he ascribed to heading the ball. He quickly recovered, and was well until November 12th, when he again played football. The ball came to his

Drawing of ruptured aneurysm found at the junction of the right middle cerebral and posterior communicating arteries in Case iv.

feet, but instead of kicking it he was seen to sink to his knees, rapidly becoming unconscious. When he was taken home he was in a state of mental confusion, and complained of headache and pain in the neck. There was photophobia, and he vomited once. I saw him on November 17th, and he was then much better, complaining only of slight headache. He was clear mentally. There were some signs of meningeal irritation—a little pain on full flexion of the neck, and slight Kernig's sign. The temperature and pulse were normal. Examination of the fundi and of the nervous system yielded no further abnormal signs. There was no cardiac lesion, and

the blood pressure was within normal limits. When admitted to the Cornelia Hospital two days later he was completely free from symptoms. The next day, at 8.15 a.m., he suddenly became unconscious, and was dead within an hour and a half.

The cerebro-spinal fluid removed immediately after death was under increased pressure, but was unexpectedly quite clear. It was found, however, to contain 500 red cells per c.mm. (Dr. McNaughton). At necropsy the right hemisphere and base of the brain were found to be covered with a thick layer of blood. At the bifurcation of the right carotid artery in the angle between the middle cerebral and the posterior communicating arteries was an aneurysm about 4 mm. in diameter, which had ruptured (see figure). The brain tissue immediately lateral to the rupture was ploughed up over an area about three-quarters of an inch in diameter.

Yet another variant is noted in Case v. Here it would appear from the symptoms that there was an initial slow leakage, producing the relatively mild symptoms which lasted a fortnight. A sudden increase is then suggested by the patient's passing into coma, from which he slowly recovered, only to succumb quickly to a further attack four weeks after his first symptoms.

CASE V

A young man of 32 was well until July 4th, 1924, when he had pain in the head and back of the neck, and vomited. He went to bed, and remained there for two weeks; there was then a recurrence of vomiting, and he became unconscious. He was immediately admitted to the General Hospital, Birmingham, under Dr. A. P. Thomson. The respiration was slow and deep, the face pale and clammy. The pulse was slow (60), and there was slight pyrexia (99°). He was semiconscious and irritable, so that any attempt at examination was resented. Focal damage to the nervous system was indicated by a right facial palsy and an extensor plantar reflex on this side. Meningeal irritation was shown by well-marked cervical rigidity and the presence of Kernig's sign. The cerebro-spinal fluid did not appear to be under increased pressure, but was uniformly blood-stained; culture was negative. The heart and blood vessels were normal. The blood Wassermann reaction was negative; the urea content was 81 mg. per cent.; the white cells were 15,000 per c.mm. The fundi oculi were normal on admission, but shortly after papilloedema appeared in both eyes, and later became severe; no haemorrhages were noted. The urine contained a haze of albumin. The patient gradually became more conscious, but on August 2nd, eleven days after admission, he suddenly went into coma; the breathing was stertorous; both plantar reflexes were extensor. He never regained consciousness, and died the next day.

Post-mortem Findings

This report is given by the kind permission of Professor G. Haswell Wilson.

There was a firm mass of blood clot adherent to the left carotid artery at the point of its bifurcation. In this region the brain tissue was ploughed up, and blood clot filled the left lateral, third, and fourth ventricles. Microscopical examination of the carotid artery revealed thinning of the wall and dilatation of the vessel at the point of rupture, with no evidence of gross disease at the site of the aneurysm. The cerebral vessels generally were slightly thickened.

Bleeding severe enough to produce total unconsciousness may, however, be recovered from completely and the patient enjoy good health for a considerable period. Always, however, there is the liability to a renewed leak.

CASE VI

A man of 31 had a head injury during the war, for which he was in hospital ten days. He was well till 1924, but after this complained of intermittent headaches. On April 20th, 1928, shortly after a severe headache, he fell unconscious. He was brought to the Middlesex Hospital and admitted under Dr. Cockayne. He was in a stuporose condition, and the pulse rate was slow (64). The abdominal reflexes were absent; otherwise examination of the nervous system was

negative. Consciousness was gradually regained; and three days later the abdominal reflexes were present and equal; early papilloedema was now present in both eyes. The blood pressure was 140/105, falling later to 115/80. The urine contained no albumin. Thirty c.cm. of cerebro-spinal fluid was now drawn off; it was under greatly increased pressure, and contained a large quantity of blood. The blood Wassermann reaction was negative, as was an x-ray examination of the skull. During the next few days the papilloedema became greater, the abdominal reflexes disappeared, and the patient became very drowsy and complained of severe headache. There were independent eye movements, and some stiffness of the neck and the back; Kernig's sign was present. During the next three weeks periods of incoherence alternated with a clear mentality and headache. These symptoms gradually disappeared, the papilloedema subsiding at the same time. The patient was discharged on May 23rd, 1928, feeling very well and without any abnormal signs in the nervous system. Except for occasional occipital headaches he remained well for one year and five months. Later attempts to trace this patient have failed.

The existence of a group of cases in which there is a slow leak from an artery, or perhaps from a vein, can no longer be doubted. Several have been reported by Richardson.¹¹ These represent the most benign form of subarachnoid haemorrhage. There is a slow onset of headache, pain in the neck, vomiting, photophobia and pain behind the eyes, pyrexia, and more or less mental change. There are signs of meningeal irritation and perhaps papilloedema and fundal haemorrhages. Healing of the rupture and recovery of the patient may occur. This recovery is likely to be only temporary, however, and there is a constant risk of a further and probably more serious leakage. On the other hand, a subacute illness which is the result of a small leak may be terminated more or less suddenly by a brisk haemorrhage, as in Case v of this series. Here it was only after two weeks with symptoms of mild grade that coma developed; the post-mortem showed the symptoms to be due to ruptured arterial aneurysm. Fearnside³ described a case in which there were repeated small leaks, each incapacitating the patient for a day or two only; finally there was a large, quickly fatal haemorrhage. In this series there were two examples of a slow leak followed by recovery.

CASE VII

A stout, plethoric woman of 43 was admitted to the London Hospital under Dr. Cecil Wall on October 11th, 1928. There was a history of occipital headache and misty vision coming on five days previously. She had vomited once on the second day of her illness, and had suffered from hallucinations. She had had similar attacks, lasting twenty-four hours, every two or three months for the past two years. On examination she was found to be very drowsy and forgetful, and her answers were incoherent. There was bilateral ptosis, greater on the right side, dissociated ocular movements, and doubtful weakness of the left internal rectus. The pupils were unequal, but reacted to light and accommodation. There was bilateral papilloedema, with scattered retinal haemorrhages and patches of exudate; in the right eye there was also a vitreous haemorrhage. The neck was stiff, and there was some head retraction; sensation, power, and reflexes were normal in all four limbs. The plantar reflex was flexor. Six days after admission lumbar puncture showed a cloudy, pale yellow fluid; a film showed red cells and white cells in the proportions present in blood; culture and Wassermann reaction were negative. An x-ray examination of the skull revealed no abnormality. The blood urea was normal (24 mg. per cent.), as were also the red and white blood cell counts. The urine (catheter specimen) contained a trace of albumin. The blood pressure was 210/110, and there was some cardiac enlargement. For three weeks after admission there was mild fever reaching 100°. There was gradual improvement in the headaches and the vision, and when discharged the patient was feeling well.

CASE VIII

A woman of 67 was seen in consultation with Dr. E. K. Le Fleming of Wimborne on June 6th, 1932. There was a history of pain in the neck and the back, and of "falling about" eight weeks previously. About four weeks later she consulted Mr. Dixon of Salisbury because she had felt as though there were a film over her right eye; she was found to have an early cataract. In the course of a general examination her blood pressure was found to be 180. At the end of May an attack of vomiting and diarrhoea led to the suspicion of food poisoning; but continued vomiting, headache, and pain behind the eyes suggested some intracranial lesion. There had been irregular pyrexia for about fourteen days, reaching 101.5°; the pulse remained slow (60-70). She was fully conscious and quite clear mentally. The pupils were equal, rather small, and reacted briskly to light; there was no ocular palsy or nystagmus. The right optic disk was imperfectly seen; the left showed slight oedema. The other cranial nerves were normal. The neck was stiff and tender, and Kernig's sign was present. The blood pressure was 190/90. On lumbar puncture a uniformly bright red fluid under increased pressure was drawn off. Examination showed red blood corpuscles and a few endothelial cells and leucocytes; no organisms were present. The puncture was followed by a rapid improvement in the headaches and a fall in temperature. She made a steady and apparently complete recovery. She is at present (March, 1933) quite well, except for an occasional occipital headache.

POINTS IN DIAGNOSIS

Cerebro-spinal Fluid.—In most cases examination of the cerebro-spinal fluid will give the diagnosis of subarachnoid haemorrhage, or confirm this if it has already been made. The three diagnostic criteria are: uniform admixture with blood, failure to clot on standing, and coloration of the fluid remaining when the red cells have sunk to the bottom of the tube. These three features, which were first explained by Froin in 1904, serve to exclude accidental haemorrhage at the time of puncture. It is important to note that the fluid does not necessarily appear tinted with blood to the naked eye. The concentration of red cells may be so low that the fluid is perfectly clear, and they are only discovered on microscopy, as in Case iv. Here, presumably, the initial haemorrhage was a fairly small one, and clotting must have hindered the passage of blood down the spinal envelope in anything but small quantities. The final severe haemorrhage killed this patient so quickly that one must suppose there was no time for blood to reach the lumbar region. Martin¹⁰ mentions a case of subarachnoid haemorrhage in which the cerebro-spinal fluid withdrawn on the fifth day of the illness was free from blood. Collier¹ refers to slowly leaking aneurysms round which there forms an organizing haematoma, in appearance like liver; in this type the cerebro-spinal fluid does not become blood-stained.

Massive Albuminuria.—Albuminuria tends to appear in any condition which is accompanied by coma, but attention has been called to the exceptionally large quantity of albumin which the urine may contain in subarachnoid haemorrhage. Its occurrence has recently been discussed by Symonds¹³ and Hall,⁶ and the last observer was able in one case to prove that it may occur apart from any demonstrable renal lesion. Of the eight cases here recorded the urine was examined in six. There was never a large quantity of albumin; in five there was a slight or moderate amount, and this was present only for a short time. It must be added that there was never an opportunity to examine a specimen during coma. In the sixth case (Case viii), in which there was a slow haemorrhage and the intracranial pressure could never have reached a very high level, the urine was quite free of albumin.

Mental Changes.—These are almost invariable when the condition has been of such severity that coma has been a part of the clinical picture. Irritability, confusion, and disorientation in time and space are common. Korsakoff's psychosis, characterized by disorientation and pseudo-reminiscences, is occasionally observed,^{5 6 7} but was not noted in this series.

The Fundus Oculi.—Although they are not invariable, changes in the eye grounds are very common and sometimes of great diagnostic value. Three phenomena have been described—namely, papilloedema, retinal haemorrhages, and vitreous haemorrhages. According to Riddoch and Goulden,¹² these appearances can all be attributed to the presence within the optic sheath of blood which, by raising the pressure therein, interferes with the venous return from the eye; and histological examination suggests that the intraocular haemorrhages are never the result of blood passing into the eye from the optic sheath. Among the eight cases in the present series some fundus change was noted in four. In three there was papilloedema only; in the other there was papilloedema and, in addition, retinal and vitreous haemorrhages. In Case iv, although the initial haemorrhage was fairly severe, there were no eye changes. It was possible to account for this at necropsy, when large clots were found round the aneurysm; these had prevented the entry of anything more than a small quantity of blood into the optic sheaths.

TREATMENT

Any unnecessary movement or effort on the part of the patient is to be avoided, as this will raise the blood pressure and so be likely to increase or restart the bleeding. Lumbar puncture is a ready method of reducing intracranial pressure, and it takes a prominent place in treatment. The value of the method has, however, been variously assessed. Some doubt the advisability of reducing the intracranial pressure, believing that it determines new or increased bleeding. Ingvar⁹ records one case in which he considers lumbar drainage was responsible for the patient's death within a few hours. On the other hand, Collier¹ has recently expressed the opinion that free and repeated drainage should be performed so long as symptoms of increased intracranial pressure exist. In intracranial haemorrhage death is caused by diminution, through increased pressure, of the blood supply to vital centres, and it is agreed that deepening coma is a clear indication for reduction of this pressure by lumbar puncture. The risk of aggravating the haemorrhage is then outweighed by that of fatal cerebral compression.

The question of whether or not lumbar puncture is to be employed will arise in one other phase of the disease—namely, the period characterized by symptoms of meningeal irritation, such as pains in the head and neck and mental changes. These symptoms will occur after recovery from the coma produced by a severe haemorrhage or may be present from the onset in a mild case without coma. Hall⁷ has sometimes observed them to subside rapidly without lumbar puncture, while Fordham⁴ found the effect of puncture beneficial in some but not in others. He concludes that it should not be repeated unless decided improvement is noted. In the present series of cases the lumbar puncture was undertaken several times for the relief of meningeal symptoms. It was frequently followed by improvement, and never by any immediately unfavourable effect. The cerebro-spinal fluid was slowly removed until the pressure was reduced to normal as judged by the rate of flow. It is better, however, to control the pressure with a manometer, as Ingvar recommends, so that the pressure may not be allowed to fall below 100 mm. Various other methods used in treatment are subsidiary and probably of small

importance. Intracranial pressure may be reduced by giving magnesium sulphate orally or per rectum. Substances designed to promote clotting may be given intravenously, and with the same object anti-meningococcal serum has been given intramuscularly.¹¹ Eskuchen² injected gelatin intrathecally with the object of encouraging coagulation of the blood; the result suggested that the method was risky.

SUMMARY

Eight cases of spontaneous subarachnoid haemorrhage are reported. Various grades of the disease, from the most severe to the mild, are illustrated. Two instances of the latter are recorded in which there is a slow oozing of blood, leading to headache, pyrexia, and symptoms of meningeal irritation. Coma does not occur, and a good recovery follows.

Examination of the cerebro-spinal fluid is the key to the diagnosis, and the fluid is nearly always uniformly tinted, due to the presence of blood. It may, however, be clear to the naked eye, yet red corpuscles can be discovered under the microscope. This was the finding in one case in which the diagnosis was proved at necropsy.

Intraocular changes are valuable in diagnosis, and were observed in half the cases. Clotting of the blood, so that it is prevented from entering the optic sheath, is one cause for their non-appearance.

Periodic headaches are often complained of for years, either before or after the rupture of a cerebral aneurysm, or both. This was so in five of the eight here recorded, one having the characters of migraine.

Treatment consists in absolute rest and in reduction of intracranial pressure when life is threatened and where symptoms of meningeal irritation are severe and persistent.

I am much indebted to those physicians whose names appear in the text for allowing me to publish case notes.

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THE RUBIN TEST AS A THERAPEUTIC AGENT

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As a therapeutic agent the Rubin gas inflation of the upper female genitals stands conspicuously alone. When confronted with a case of sterility due to occlusion of the Fallopian tubes the gynaecologist realizes how limited are the therapeutic agents at his disposal. Surgical intervention to restore patency has been uniformly unsuccessful in accomplishing the desired result. In the Rubin test, however, he has a powerful agent to overcome obstructive sterility. In over 500 consecutive consulting room cases of the gas test there was evidence of obstruction in only 35 per cent. In many of these the obstruction was only of a mild nature that gave way before the high pressure of the first test. In these the