SPONTANEOUS SUBARACHNOID HAEMORRHAGE,
WITH KORSAKOFF'S PSYCHOSIS.

BY

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In the *British Medical Journal* of June 8th, 1929, Professor Arthur J. Hall described two cases of subarachnoid haemorrhage which developed Korsakoff's psychosis. The following case seems worth recording. The patient had recurrent haemorrhages, and from each succeeding haemorrhage the recovery was less complete. He developed Korsakoff's psychosis, and finally died seven days after the first haemorrhage. The diagnosis was confirmed at autopsy.

Clinical History.

The patient was a man aged 57, an old soldier, tall and strongly built, apparently in perfect health, with no history of any illness of importance. On July 29th he was rather unwell, complaining of headache; he vomited once, but recovered. The next evening he went to a whist drive, during which he suddenly collapsed on the floor. When first seen about an hour later he was very restless, throwing his arms and legs about, trying ineffectively to get up and to untie his hands. He made apt but rather curious statements, such as "I'm badly handicapped." He did not answer questions or seem to be aware of his surroundings. He was moving the left side of the body more than the right. The left pupil was rather large, the right small and less active to light. The knee-jerks were brisk, the right less so than the left. Both plantar reflexes were flexor. The heart appeared normal, and the arteries did not seem to be thickened. The urine contained a trace of albumin. During the next hour or two he became quieter and somewhat more rational, and answered questions. A diagnosis of subarachnoid haemorrhage was made.

By next morning the pulse rate had gradually fallen to 58; he was becoming more and more comatose; the respiration was of the Cheyne-Stokes type. The blood pressure was 90/45. He was still moving his limbs, but very slightly. The knee-jerks had become exaggerated; there was double ankle clonus and double extensor plantar response. Lumbar puncture was performed; the fluid was under considerable pressure, and blood-stained throughout. Directly after the lumbar puncture the exaggerated knee-jerks, ankle clonus, and extensor response disappeared. The blood pressure rose to 110/75, and the breathing became regular, but the pulse remained at 58. The left optic disc showed no papilloedema; the right disc was not seen on account of the contracted pupil. Four and a half hours after the lumbar puncture he became conscious, asked where he was, and said he would like some whisky. He was fairly quiet through the night, took fluids, and passed water spontaneously.

Examination of the cerebro-spinal fluid showed:

Red blood corpuscles	...	101,600 per c.mm.
White blood corpuscles	...	396 per c.mm.
Lymphocytes	...	320 per c.mm.
Polymorphs	...	76 per c.mm.
Total protein	...	1,000 mg. per 100 c.cm.
Globulin	...	++
Chlorides	...	740 mg. per 1,000 c.cm.
Sugar	...	Normal
Van den Bergh reaction	...	Delayed reaction 0.2 units
Wassermann reaction	...	Negative
Lange's colloidal gold test	...	0000023211

Next day, August 1st, he showed a definite Korsakoff psychosis. There was complete disorientation as to place and time, fairly good memory for past events, but fabrication of recent occurrences. In the evening the pulse began to fall. Lumbar puncture was again performed, and blood-stained fluid under pressure was withdrawn. The fluid rapidly became more blood-stained; he complained of headache, then suddenly began to breathe stertorously, while clonic movements of the hands and arms took place. The needle was quickly withdrawn. The patient was cold and sweating, the pulse very feeble, and double ankle clonus and extensor response were again present. The left pupil contracted, so that it was now equal to the right. The pulse and respiration gradually improved. He became very restless and destructive. He answered questions with "Yes" and "No," and obeyed orders for a few seconds, but soon began to try to dismember his bed again. He remained in this condition all night. The pulse was full and slow, sometimes falling to 40. The left pupil had dilated, and the ankle clonus and extensor response disappeared.

Next day Korsakoff's psychosis was well marked. His mind seemed to run on his army days. On being asked what he was doing, when he appeared to be reaching for something,

he replied, "Collecting items." "Items of what?" "Stores." He was very suggestible, and was led into saying that he had been out for a walk that morning, and had met the Archbishop of Canterbury, who asked him how things were going.

At 11 p.m. he suddenly became comatose, with stertorous breathing. There was no apparent cause for this renewed haemorrhage. The breathing became very slow, consisting of deep gasps, which occurred every fifteen to twenty-five seconds. The pulse remained fairly good. The left pupil again contracted, and ankle clonus and extensor responses appeared; both were more marked on the left side. He vomited through his nose, and for a time there was a good deal of rattling in the throat. There was a left divergent squint for a short time, followed later by conjugate deviation to the left. About 12.30 a.m. he became very rigid, the legs extended, and the back arched. An anaesthetic was given to relax the extensor muscles of the back, and lumbar puncture was performed; 40 c.cm. of blood-stained fluid was withdrawn slowly; it was under considerable pressure. The breathing and pulse improved, and the rigidity did not recur.

Next morning, August 3rd, the coma was still deep. Respiration was quiet; the pupils were equally contracted; there was no squint or conjugate deviation; there was marked blepharospasm. The right optic disc showed papilloedema; the left disc was not clearly seen. The blood pressure was 135/90. The temperature, which had remained subnormal, rose to 100.6, and the pulse rose from 48 to 88. There was still a trace of albumin in the urine. There were a few rales at the bases of the lungs. The respiration rate was 24.

In view of the fact that lumbar puncture failed to relieve the coma, it was decided that decompression was justifiable. At 3.30 p.m. a right subtemporal decompression was performed; the dura mater was a little thickened; there was a layer of blood under the arachnoid membrane, and the brain bulged out somewhat. At the end of the operation the respiration and pulse were improved. The coma, however, continued unchanged. About midnight the respiration rate began to rise, and next morning reached 60. Signs of broncho-pneumonia developed, the patient became cyanosed, and died at 9.30 p.m. on August 4th.

Post-mortem Examination.

The brain weighed 2 lb. 2½ oz. The dura mater was thickened. The arachnoid was also thickened, especially over the right parietal region. Blood was diffused all over the surface of the brain between the arachnoid and the pia mater; when the pia-arachnoid was stripped off the brain the convolutions were seen to be a little flattened; the brain substance was not congested, and there were no haemorrhages into it. There was a large clot of blood at the base of the brain in the region of the circle of Willis, around the medulla and pons, between the medulla and cerebellum, and in the right Sylvian fissure. The circle of Willis and its branches showed a considerable amount of thickening; there was one fusiform aneurysm, and one small, thin-walled, saccular aneurysm containing clot; this was possibly the site of the haemorrhage. The fluid in the lateral ventricles was slightly stained with blood; in the wall of the anterior horn of the left lateral ventricle there was a small, yellowish, softened area.

Microscopical section of the brain substance showed no abnormality. Branches of the circle of Willis showed considerable patchy proliferation of the intima, which in many places was thickly infiltrated in its outer layers with round cells. There was no marked thickening or splitting of the elastic laminae. The media was not thickened, but there were areas of round-celled infiltration, which was also well marked in the adventitia.

The heart was large, weighing 17½ oz. There were some fatty patches under the endocardium, especially at the bases of the papillary muscles. The mitral and tricuspid valves were a little thickened. The condition of the coronary arteries was surprisingly good, and the aorta was unusually free from atheroma.

The lungs were congested and oedematous. In the left lower lobe there were patches of broncho-pneumonia, and pus could be squeezed out of the small bronchi. The left kidney showed a depressed area resembling an old infarct. The cortex of both was a little diminished, and the surface slightly granular. To the naked eye there was no evidence of arterio-sclerosis. Microscopical sections showed patchy areas of fibrosis, with atrophy of the epithelium of the tubules; there was very little cellular infiltration; many of the arterioles showed marked reduction, or even obliteration, of their lumen, due to proliferation of the intima, with no cuffing with lymphocytes. Other parts of the kidneys showed no interstitial change, but some cloudy swelling of the tubular epithelium.

The evidence is not sufficient to justify a certain diagnosis, but in spite of the negative Wassermann reaction and the undoubted arterio-sclerotic changes in the kidneys it seems probable that the changes in the membranes and vessels of the brain were due to syphilis, and that the

haemorrhage was due to the rupture of a small aneurysm of syphilitic origin on one of the branches of the circle of Willis.

The sudden appearance of pupillary and pyramidal tract signs with each fresh haemorrhage, and their rapid disappearance after the relief of pressure by lumbar puncture, was an interesting feature of the case.

I have to thank Dr. J. L. Livingstone and Mr. J. B. Hunter for their permission to record this case.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

RUPTURED LIVER: SUTURE.

RUPTURE of the liver does not commonly come to operation, as, in the first place, it is not very frequently seen; and in the second place, when it does occur, it is either so severe and complicated by other injuries as to be rapidly fatal, or else it is a small subcapsular tear not requiring operative intervention. An interesting point about the case reported below was the interval between the accident and the onset of abdominal pain, during which the patient felt comparatively well. There was also surprisingly little shock consequent on such a severe injury.

A builder, aged 30, was admitted to hospital on March 9th after a cycling accident, in which he had a head-on collision with a car. He was struck in the abdomen (he thought, by the mudguard of the car), and immediately afterwards got up unaided and felt no pain. Ten minutes later, however, he was seized with severe abdominal pain, unaccompanied by faintness, nausea, or vomiting.

On admission the pulse was 64, temperature normal, and there were no signs of shock; there was some rigidity and tenderness in the right upper quadrant of the abdomen. The patient had vomited once since the accident. The pulse proceeded to rise slowly but steadily, and rupture of the liver was diagnosed. When I saw him, twenty-four hours after the accident, the pulse was 100, rigidity and tenderness on the right side were marked; there were no signs of free fluid in the abdomen, but the patient looked strained, and was still complaining of pain.

Laparotomy was performed through a right paramedian incision, enlarged by transverse division of the right rectus. There was a considerable amount of free blood in the peritoneum, and a rent was found in the right lobe of the liver, extending from the edge for four inches on the upper surface and six inches on the lower, steadily oozing blood. The tear was very difficult to get at, being in the posterior axillary line, but six or seven interrupted catgut stitches were inserted, bringing the edges into apposition. No other visceral injuries were present. The wound was closed in the usual manner. Nine days later a biliary fistula appeared, and persisted for four weeks, but ultimately healed, and the man was discharged fit.

I am indebted to the staff of the Farnham Infirmary for the opportunity to operate on this case.

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A PATHOLOGICAL MONOCYTE.

THE leucoblast was described by Pappenheim as being a large cell, with a nucleus resembling that of a myelocyte; the basophil cytoplasm is often rich in azur granules, coarser and darker than any ever seen in the lymphocyte or monocyte. Its presence in the blood is always pathological; it is invariably associated with serious disturbance of the marrow, and all the evidence points to its originating in the marrow. Since it is numerous only in myelæmia, Pappenheim assumed that it belonged to the neutrophil series. In the leucocytosis of pneumonia a few myelocytes appear, and, in a severe case, even one or two premyelocytes, but no leucoblast is seen. For this and other reasons, he regarded the leucoblast as being more immature than the premyelocyte.

In pernicious anaemia, however, leucoblasts may be present when there are few myelocytes in the blood and no premyelocytes. Pappenheim supposed, therefore, that in certain pathological conditions this very immature cell was forced into the blood; that (to account for the absence of premyelocytes) onward development then ceased, and that it retrogressed into a large mononuclear, the so-called leucoblastic monocyte. Thus he believed that in the marrow

his leucoblast preceded the premyelocyte in the direct line of neutrophil descent, but that, pathologically, it entered the blood to become a monocyte.

These facts afford ample evidence of the marrow origin of the leucoblast, but not for its neutrophil descent. On the contrary, in the glandular fever of children (*British Medical Journal*, 1927, vol. i, p. 1056), it may enter the circulation owing to the specific action of the toxin causing monocytosis. This fact and the appearance of the cell in the stained film suggest that it is a monocyte. Pappenheim admitted that it was often difficult to distinguish the leucoblast from a monocyte; if it is really such a cell it is always pathological, and the simplest explanation is that it is an immature monocyte (premonocyte) derived from a lymphoid marrow cell (myelomonoblast). This would explain its emergence in malignant disease. In internal cancer (liver, lung, kidney) there is usually a considerable leucocytosis of neutrophilic cells with a few myelocytes, and monocytosis with a few pneumonocytes. Because the usual stains do not show these cells I abandoned them ten years ago in favour of the following procedure, which I consider the simplest, cheapest, and best blood-staining method for general purposes.

Manson's methylene blue contains 2 per cent. of the dye in a 5 per cent. solution of borax in distilled water; the water should be boiling when the borax is added, but have been removed from the flame. This stock solution may be made by any druggist, and will keep for some years. It is rendered polychrome in small quantities as required, by boiling about 10 c.cm. in a clean test tube gently over a small flame for one minute. Fresh solutions require this boiling to be repeated three times, at intervals of a few hours; older solutions are already slightly polychrome, and one boiling may be sufficient, after which the polychrome fluid is stored by itself in a small bottle, preferably pipette stoppered, and will keep good for months. A similar bottle contains 1 per cent. eosin solution in distilled water.

Into one ounce of distilled water I put four drops of the eosin and two drops of the polychrome solution, and mix. Films are fixed in methyl alcohol or Jenner's stain for three minutes or longer, and are then stained for half an hour to two hours, washing briefly in distilled water. It is convenient to have two small wide-mouthed bottles with glass stoppers. One contains about 15 c.cm. of fixative, and slides are dropped in to be fixed by tilting the bottle sideways. It may be used for months, as there is no waste. The other bottle holds the staining fluid, and the fixed films are transferred to it with forceps. It is not necessary to cleanse the staining bottle every time, but only to renew the staining fluid immediately before each examination.

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IDIOPATHIC HAEMORRHAGE IN THE NEWBORN.

THE value of injecting parental blood in cases of melaena neonatorum seems to be insufficiently recognized, and the following clinical details of such a case may therefore be worth publication.

A multipara gave birth to a girl without instrumental intervention, the presentation being a vertex L.O.A. After an easy labour, lasting only a few hours, the baby was born, apparently perfectly healthy. The baby became restless thirty hours later, and was found to have passed a tarry black stool; this was about three ounces in bulk, and was covered with patches of bright red blood. A diagnosis of melaena neonatorum was accordingly made, and 5 c.cm. of fresh blood, removed from a vein in the mother's arm, were injected immediately into the baby's buttock at 8 a.m. Subsequent haemorrhages occurred at 12.30 p.m. and 5.15 p.m. on the same day. At 7 p.m. a further 5 c.cm. of the mother's blood was injected, followed by 1 c.cm. of colloidal calcium into the thigh muscles, the calcium being repeated daily for a week. By the end of the first day the baby was plainly anaemic, but only one other bleeding occurred—on the day following the first injection—and that was only a relatively small loss. Complete recovery has ensued, and the child is now apparently in excellent health. There was no history of haemophilia or purpura in the family.

We are prompted to publish this case because none of our textbooks gave any help, either in diagnosis or treatment, when we referred to them after having instituted treatment, except *Nelson's Loose-Leaf Medicine*. Our treatment at first was based on memories of a case reported in the *British Medical Journal* (January 12th, 1929, p. 68).

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