THE MICROSCOPIC EXAMINATION OF THE BRAINS OF TWO MEN DEAD OF COMMOTIO CEREBRI (SHELL SHOCK) WITHOUT VISIBLE EXTERNAL INJURY.¹

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The examination of the brains of two cases of death from shell shock without visible injury and without punctate haemorrhages indicative of gas poisoning is of interest for several reasons. So far as I know, it is the first description that has been given which serves to explain (1) sudden death in shell shock, and (2) the clinical symptoms which persist for some time after the commotion of the brain in non-fatal cases.

I am indebted to Lieutenant-Colonel T. R. Elliott and Professor Arthur Keith for sending me the brains, and to the officers whose names are mentioned for the clinical notes and the notes of the post-mortem examination.

CASE 1.—CLINICAL NOTES.

In this case the man developed, according to a note furnished by Captain J. London, a degree of nervousness on the Somme which he never lost, but was able to control for six months. Later he was in an area which was subjected to an intense bombardment, during which, as far as can be ascertained, no gas shells were used. This lasted about four hours (February 22, 4 p.m. to 8 p.m.). Although he remarked to another man that "he could not stand it much longer," he did not give way until the following day, twelve hours later, when perhaps six shells came over (February 23, 8 a.m.).

He was not buried nor gassed. One shell burst just behind his dug-out—namely, ten feet away—in the morning, but many must have been as near the previous day. Early symptoms were tremors and general

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depression. The later symptoms (February 22) were coarse tremors of
the limbs, crying (February 23), inability to walk or do anything. He
would not answer questions—very like the hysterical manifestations
of melancholia. The pupils were dilated. I was rather busy with some
wounded at the time, and did not make a detailed examination.

A note by Captain Francis A. Duffield, R.A.M.C.(S.R.), states that
the man was admitted to the field ambulance in the evening in a state of
acute mania, shouting "Keep them back, keep them back." He was
quite uncontrollable and quite impossible to examine. He was quieted
with morphine and chloroform, and got better and slept well all night.
In a later note, Lieutenant-Colonel F. J. Crombie, in command of the
field ambulance, stated that the patient had at least two hypodermic
injections of morphine while in the ambulance. Next morning he woke
up apparently well, and suddenly died.

Necropsy.

The following is a note by Captain A. Stokes, R.A.M.C. (Mobile
Laboratory), on the post-mortem examination made on the afternoon of
the day of death.

There were no marks of external violence on the body other than some
small scratches on the chest wall.

Thorax.—The lungs were oedematous, and in the substance of the
lower lobe of the left lung there was a considerable haemorrhage. The
right lung, except for oedema, was normal.

Heart.—Enlarged, and the right side dilated. The muscle was good,
and there were no valvular lesions.

Abdominal Cavity.—Normal. There was no pathological change in
the stomach, oesophagus, intestine, or great intestine. The liver was
normal in size, and was somewhat congested. The spleen was normal.
The kidneys were small, but showed no gross change. The urine con-
tained neither sugar nor albumin.

Skull.—There was a slight bruise on the scalp, in the frontal region.
The brain was extremely congested, and on each side of every superficial
vessel there was an ecchymosis. There were a number of minute punctiform
haemorrhages at the terminations of the smallest vessels on the
surface of the brain. The whole brain was soft but not markedly
edematous. The cerebrospinal fluid appeared to be blood tinged.
There was considerable ecchymosis on each side of the great sinuses
of the skull. There was no large haemorrhage found, and no small
intracerebral petechiae. There was no gross lesion of the viscera, which
would have been a cause of death; but though I have never seen a post-
mortem examination on a man who has died of "shell shock," I consider
the condition of the brain is consistent with that diagnosis.
Examination of the Brains of Two Dead Men

MICROSCOPIC EXAMINATION OF THE BRAIN OF CASE 1.

The brain had been preserved in Kaiserling's fluid, and it was not in very good condition, but seeing that it was placed in this fluid less than twelve hours after death, it is probable that the portions examined yielded satisfactory material for microscopic investigation.

The portions of brain selected were prepared for section by the paraffin method of serial sections. The dyes used to stain the sections were as follows: (1) Hæmatoxylin and eosin; (2) Van Gieson; (3) thionin; (4) polychrome and eosin. The sections were five microns in thickness and were mounted, after staining, in Canada balsam.

Cerebrum: Top of Ascending Frontal Convolution; Leg Area.—The veins are congested both in the meninges and in the substance of the grey and white matter. There is subpial hæmorrhage here and there owing to rupture of the dilated congested veins. There are no punctate hæmorrhages observable. The perivascular spaces of the arterioles, capillaries, and venules are dilated, also the peri-neuronal spaces are distinctly seen, some being apparently connected with the perivascular spaces. In some of the sections, empty collapsed vessels can be discerned in places. The general appearance suggests deficiency of blood in the arteries and capillaries, with engorgement of the venous system. A condition very similar to that observed in experimental anæmia in animals produced by ligation of both carotids and vertebrals. There is some degree of chromatolysis of the cells. The Betz cells are the easiest on account of their size to recognize this change, and the accompanying drawing (fig. 1) shows the early chromatolysis of these psychomotor neurones. The pole of the first frontal shows marked congestion of the vessels and some subpial hæmorrhage from dilated congested veins. Many of the arterioles and capillaries are empty and collapsed, and there is the same appearance of dilation of the perivascular sheaths and perineuronal spaces. The pyramidal cells also show early chromatolytic changes.

Internal Capsule.—There is general congestion of veins, and the small vessels appear to be either empty and collapsed, or contain less blood than normal. Some of the vessels show hæmorrhage into the sheath. (Fig. 2.)

Corpus Callosum.—The small vessels are congested and dilated, some have ruptured into the sheath, other small vessels have ruptured into the tissue. There are no typical punctate hæmorrhages
such as are seen in gas poisoning, which are due to hyaline thrombosis of terminal arterioles.

**Pons.**—There is a marked congestion of veins and some of the smaller veins have ruptured, giving rise to hemorrhage into the sheath, blood corpuscles are also seen extravasated in the adjacent nervous tissue. There are small hemorrhages in the white matter (fig. 3). There is dilatation of the perivascular sheaths and perineuronal spaces together with collapsed and empty vessels or partially empty vessels. The hemorrhages, here as elsewhere, appear to be of recent occurrence.Nearly all the cells show some degree of early chromatolytic change.

**Medulla.**—Sections of the medulla at the point of the calamus scriptorius were made, as the upper part of the medulla was rather damaged. In the anterior median fissure a vessel had ruptured, and there were free corpuscles in the lepto-meninges. All the veins on the surface of the medulla were congested. In serial sections the ruptured vessel entering the anterior median fissure and penetrating the median raphe could be followed, and here it was seen to have ruptured into the perivascular space (fig. 4), and blood corpuscles are seen extravasated into the adjacent tissue. The perivascular and perineuronal spaces are seen dilated both in the medulla and pons (fig. 7). The cells of the medulla show only chromatolytic changes as a rule. The cells of the vago-accessorius nucleus (fig. 5) show much more chromatolysis than the adjacent cells of the hypoglossal nucleus (fig. 6). These nuclei are distant about two millimetres from the ruptured vessel in the median raphe.

**Cerebellum.**—Sections stained with thionin and safranin show very unequal staining of the Purkinje cells with the basic dye (fig. 10). This condition is very similar to that described by Crile in the case of "a soldier who had suffered from hunger, thirst, and loss of sleep; had made the extraordinary forced march of 180 miles from Mons to the Marne; in the midst of that great battle was wounded by a shell; lay for hours waiting for help, and died from exhaustion soon after reaching the ambulance."

**Summary of Histological Changes.**

There is a generalized early chromatolytic change in the cells of the central nervous system. This change varies in intensity. The cells most affected are the small cells in which the basophil substance has almost disappeared. In the larger cells the Nissl
granules are smaller and not packed so closely together as normal. The small cells of the medulla and pons are slightly swollen, and the nucleus is large and clear. This change is present in some of the large cells, but it is less evident. This change indicates a relative degree of exhaustion of the kinetoplasm; assuming that the amount of the basophil substance is an index of biochemical neuropotential. The Nissl granules are not present in the neurone during life, but they disappear altogether in a cell that (prior to death of the whole body) has been so injured as to decay and die. Granted this premise, then, it may be assumed that the cells of this man are in a state of commencing nervous exhaustion, some nuclei of cells showing the changes more markedly than others—for example, the cells of the vago-accessorius nucleus.

The vessels of the pia-arachnoid membranes of the brain are congested, and there are scattered subpial haemorrhages of microscopic size almost everywhere.

In the white matter of the corpus callosum, the internal capsule, the pons, and medulla there are seen congested veins and haemorrhage into the sheaths of these vessels with occasionally extravasation of blood corpuscles into the adjacent tissues.

Case 2.—Clinical Notes.

Captain Duffield reported that information obtained from the medical officer attached to the unit in which the man, a gunner in the Royal Garrison Artillery, was serving, was to the effect that he was sitting in a corrugated iron hut, fifty yards from some boxes of cordite cartridges, when a shell landed and exploded them. The man became unconscious at once, his breathing was stertorous: his body showed no signs of wounds.

On the same day he was removed to a dressing station and thence to a casualty clearing station; in the evening of that day he died. The medical officer there stated that the patient was absolutely unconscious, and could not be roused. His breathing was stertorous and slow; the pupils were equal and reacted to light; knee-jerks were difficult to obtain. He died shortly afterwards, and at the post-mortem examination the brain was removed, placed in spirit, and dispatched.

Macroscopical Appearance of Brain of Case 2.

On the upper surface of the cerebellum, the temporo-sphenoidal, and left orbital lobes there was superficial haemorrhage. On cutting up the pons, oval patches were seen as large as \( \frac{1}{8} \) by \( \frac{1}{4} \) inch; whether
this is simple staining of haemorrhage cannot be determined until a microscopical examination has been made. Portions of the mesencephalon and pons were taken for microscopical examination; the medulla oblongata was not sent.

**Microscopical Examination.**

*Post-parietal.*—Meninges: Marked congestion of all vessels of the surface of the brain with extravasation of blood into the soft membranes. In the grey matter of the cortex the perivascular spaces are dilated throughout, and the capillaries, veins and arteries are for the most part empty. In the white matter no punctate haemorrhages are seen; there is marked dilatation of the perivascular spaces; the capillaries, veins, and arteries are empty. In the cortex there is dilatation of the perineuronal spaces, which in many instances may be seen communicating with the perivascular spaces. (Fig. 8.)

*Ascending Frontal.*—Stained with thionin. The large pyramidal cells show pretty marked chromatolysis without swelling of cell; some of the Betz cells show commencing breaking up of the tigroid bodies; smaller pyramidal cells show undoubted swelling of nucleus and loss of pyramidal shape, very similar to that observed in experimental anemia in animals, with varying degrees of chromatolysis. As a rule, the smaller the cell, the more marked is the change. (Fig. 9.)

*Orbital Lobe.*—On the under surface there is extensive extravasation of blood into the substance of the brain and on the surface, and there is very marked dilatation of the perivascular spaces everywhere. The cortex is in a measure destroyed in one place; there is very marked dilatation of perineuronal as well as perivascular spaces, which intercommunicate.

*Corpus Callosum.*—There is much congestion of vessels, and many have ruptured into the sheath, forming long, irregular branching, hemorrhagic extravasations, but no sign of punctiform haemorrhage.

*Temporo-Sphenoidal Lobe.*—Shows remarkable dilatation of the perivascular spaces, and there is a big globular haemorrhage, and much haemorrhage into the substance of the brain.
Examination of the Brains of Two Dead Men

Opinions of French and German Officers Regarding "Shell-Shock" by Windage.

Many discussions have taken place by French and German neurologists regarding the question of organic changes occurring in the central nervous system as a result of vent du projectile or windage. According to Léri, a true commotion appears only to be produced at a proximal distance of some ten metres from great projectiles. The finding of groups of men dead in the last attitude of life, in closed spaces such as the German "pill-boxes" and concrete dug-outs, and the proven fact that enormous forces of compression and decompression are generated by the detonation of high explosives in great shells, aerial torpedoes, and mines has lent support to the view that mere proximity to the explosion is sufficient to cause organic changes in the brain and spinal cord by the compression and decompression of gases, the result of the explosion, and of the atmospheric air; altogether apart from actual concussion caused by violent contact with solid materials, such as sandbags or the earth forming the walls of a dug-out, which may at the same time cause burial or partial burial, unattended by visible evidence of injury of the body sufficient to account for symptoms of cerebral or spinal concussion. The patient is rendered unconscious and his mind is a blank concerning what happened, in a true case of commotio cerebri; consequently he is unable to say whether he had or had not been concussed by the sand or earth. In the two cases under consideration there was no history of burial.

Undoubtedly the vast majority of non-fatal cases of shell-shock are more emotional in origin than commotional, and occur especially in subjects of an inborn neurotic or neuropathic temperament; but the two conditions may be associated. Both Léri and Meige emphasize the fact that commotional symptoms are not influenced by psychotherapy. They also point to the fact that in cases where organic changes have occurred the cerebrospinal fluid withdrawn by lumbar puncture exhibits macroscopic or microscopic evidence of blood indicating that haemorrhage had occurred.

In Case 1 Captain Stokes noted at the post-mortem examination that the fluid was blood-stained, and the microscopic findings of ruptured vessels explain this.

Léri states that the subjects of commotion are generally depressed, asthenic, aboulic, and often more or less confused mentally; they present almost constantly, even in light cases, pro-
nounced disturbances of voltaic vertigo. They often suffer with bleeding from the ear, or nasal or vesical haemorrhage. Roussy and l'Hermitte admit that in rare cases "vent du projectile" may cause organic changes.

Robert Bing gives a review of the German opinions upon nervous accidents determined by the near explosion of a projectile. He points out that Vogt and Gaupp, who have occupied themselves with "Granat Kontusion" (bomb contusion), are far from accepting the exclusive psychogenic rôle in the development of this syndrome. Gaupp insists particularly upon the relations which exist between the initial symptoms presented by those patients and the rapid succession of atmospheric compression and decompression which takes place at the moment of the bursting of the projectile. The existence of labyrinthine lesions, almost regularly in this class of case, is in support of this opinion (Schultze and Meyer).

In von Sarbo's numerous publications upon the subject there is a tendency to regard these cases from a uniform point of view. For him the general mass of observations do not permit the diagnosis of organic changes in the usual sense of the word, nor that of psycho-neurosis. He believes microstructural alterations occur, but which are not equivalent to the molecular changes of Charcot. He includes in the microstructural changes meningeal œdema, microscopic haemorrhages, transitory paralysis of vessel walls, and contusion of the nuclei and centres. In the initial period these lesions may give rise to some discrete symptoms of organic disease; later they are manifested by functional physical and psychical symptoms. Bing remarks that the pseudo-neurasthenia of arteriosclerosis supports this view. It is interesting to note that the haemorrhages into the perivascular sheaths of vessels observed in Case 1 resemble in some respects those seen in arteriosclerosis.

Oppenheim's view of traumatic neuroses had few supporters at the Congress at Munich.

Aschaffenburg examined soldiers in Flanders who had been exposed to shell fire in the trenches but had escaped unwounded and were apparently well. The examinations took place in most cases within twenty-four hours after leaving the trenches. Of seventy-four men so examined, sixty-seven showed unmistakable signs of localized organic lesions of the nervous system, although not as a rule of a serious nature. A second examination a week later showed that some, but not all, of these phenomena had disappeared. Here were cases, therefore, in which an organic basis was present but no traumatic neuroses had developed. Aschaffenburg gives the result of his experience in these words:—
Examination of the Brains of Two Dead Men

"In assuming organic changes one of the consequences of shell explosion I do not thereby agree with Oppenheim that the nervous symptoms are to be attributed to these changes. On the contrary it is to be noted that the most exaggerated hysterical cases which develop after exposure to shell firing are the ones which exhibit organic symptoms least of all."

Hypotheses regarding the Lesions of "Commotion."

Two hypotheses have been put forward to explain organic lesions by "commotion."

1. Compression of the gas and atmosphere, so that the cranium and spine are struck, as it were, by a solid body and the vibration is transmitted through the bony structures to the cerebrospinal fluid and thence to the brain and spinal cord, causing a molecular disturbance of the delicate colloidal structures of the neurones particularly those of the nuclei in the floor of the fourth ventricle where the fluid is most abundant, and where it acts as a water cushion upon which the vital-cardio-respiratory centres rest.

2. Compression is followed by a corresponding decompression causing the liberation of bubbles of gas in the blood and tissues leading to embolism.

Probably both the forces of compression and decompression act in producing vascular disturbances in the central nervous system, causing arterio-capillary anæmia and venous congestion and rupture of delicate-walled vessels with microscopic hæmorrhages.

COMMENTARY.

In Case 1, of which I have described the histological changes, it may be observed that there was a condition of mania during life; this maniacal excitement may be correlated with the marked venous congestion of the cortex, the microscopic subpial hæmorrhages, and a certain degree of scattered arterio-capillary collapse and emptiness. This, however, could not be held responsible for the suddenly fatal termination; the hæmorrhage into the sheath of a fair-sized vessel (see fig. 4) in the median raphe of the medulla and the generalized congestive venous stasis, with a condition of exhaustion of the cells of the vago-accessorius nucleus (as shown by the almost complete disappearance of the Nissl granules (see fig. 5) as compared with the cells of the adjacent hypoglossal nucleus), coupled with the condition of the heart found post mortem, may explain the sudden death.
The cerebral anæmia, as shown by collapsed and empty arterioles and capillaries with dilated perivascular and perineuronal spaces (see figs. 7 and 8), similar to the appearances in sections of brains of animals that have been killed within a few days of ligation of both carotid and vertebral arteries. The veins are congested similarly, but the capillary anæmia would explain many of the symptoms of sufferers with true shell shock, namely, headache, giddiness, amnesia—anterograde as well as retrograde—dizzy feelings, lack of power of attention, and fatigue—stupor, inertia, mental confusion, terrifying dreams—symptoms which are generally met with in recent cases.

There is, in both Cases 1 and 2, a general, though as a rule not marked, chromatolytic change indicative of a lack of kinetoplasm in the neurones of variable degree. This may hypothetically, but with reason, be regarded as an expression of a fall in the general store of neuro-potential of the central nervous system. The cells of Purkinje of the cerebellum show especially a complete or partial loss of the basophil substance.

The vascular changes are microscopic and widespread; there are no punctate haemorrhages of the white matter, such as I have described in gas poisoning, and which are due to a hyaline thrombosis of terminal arterioles. The haemorrhages are into the dilated perivascular sheaths (see figs. 2 and 3). In the corpus callosum the networks of capillaries and small vessels show fractures and escape of corpuscles into the tissues. The microscopic changes in the brain confirm in every way the opinion expressed by Captain Stokes when he made the post-mortem examination that he was dealing with a case of shell shock.

In Case 2 the extensive haemorrhage on the under surface of the orbital lobe without visible external injury is of interest. The force of the explosion must have been enormous. What happened to the man when it occurred we do not know. The cortical arterial and capillary vessels were empty, the perivascular sheaths were dilated and filled presumably with cerebrospinal fluid (see fig. 8). The cortical neurones are swollen up, the nuclei are large and clear; the basophil substance is diminished in amount, a condition very like that observed in the cells of the cortex of an animal in which experimental cerebral anæmia had been effected. Owing to the brief clinical and post-mortem notes this case is of much less interest than Case 1.

I am unable to find in the literature at my disposal any description of the microscopic changes in the brains of soldiers dying from *commotio cerebri* without visible external injury.
Examination of the Brains of Two Dead Men

MICROSCOPIC INVESTIGATION OF THE SPINAL CORD IN A CASE OF PROBABLE SPINAL COMMOTION.

1.—Case Notes.

1929 Pte. A. ——, 16th Middlesex. Died July 8, 1916. This man was badly wounded on July 1, 1916, during the early advance. He was brought in from "No Man's Land" on the evening of July 5, 1916, and arrived at the Casualty Clearing Station on the morning of July 6, 1916.

There was a superficial graze (probably caused by shrapnel) over the spine of the left scapula, and a small "in and out" wound over the right gluteal region. This wound was about two inches long, and superficial. It was clean, and the muscles were not involved. He had had tetanus antitoxin (quantity unknown). His mental condition was fairly clear, although somewhat masked by his halting speech and extreme somnolence. He was, of course, much fatigued, and had suffered from lack of food. He had complete paralysis of the legs and abdominal muscles and the left side of the face. There was marked equal loss of power in both arms. Complete anesthsia from the level of the umbilicus downwards, atony of the bladder with overflow incontinence, and loss of control of the rectum were present.

The pulse varied between 80 and 90 per minute, but was weak in tension. There was no albumin in the urine. He merely became weaker; eventually coma preceded death on July 8, 1916, without any additional symptoms having presented themselves.

Post mortem: complete examination was made, and nothing to account for death was found.

(Signed) William Moodie,
Captain R.A.M.C.
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MICROSCOPIC EXAMINATION OF PORTIONS OF THE SPINAL CORD.

A portion of the spinal cord extending from the eighth dorsal to the fourth lumbar segment was sent to me for examination by my former assistant, Captain Moodie. The material arrived in good condition in formol solution. Portions were blocked in paraffin, and sections of five microns were cut and stained by Van Gieson, Nissl, and Leishman stains; the last named yielded the best results.

The eighth, tenth, twelfth dorsal, first and second lumbar segments were examined; similar appearances, although the changes varied in intensity and degree, were observed in all the sections. Briefly they were as follows: On the surface of the spinal cord blood corpuscles were seen adhering—evidence that the cerebrospinal fluid had contained blood during life. The veins upon the
The surface of the spinal cord were everywhere congested; the arteries and capillaries as a rule were empty. In places the veins could be seen ruptured, and in some sections intraradicular hemorrhage was observed. In the substance of the spinal cord itself were numerous minute hemorrhages, varying in size from a pin's head, and visible to the naked eye, to a pin's point, invisible except by aid of the microscope.

The hemorrhages are seen especially in situations where the surrounding tissue offers least support; consequently they are found in the grey matter of the anterior horns, but especially at the base of the posterior horn near the central canal (vide figs. 11 and 12).

Frequently small veins are observable both in the grey and white matter which have ruptured, and numbers of the escaped red corpuscles are seen in the perivascular sheath.

There are distinct changes in the anterior cornual cells of varying intensity. There is perivascular chromatolysis, and not infrequently there is some swelling of the cell and eccentrically placed nucleus (fig. 13). These changes do not seem to bear a direct relationship to the hemorrhages; it is probable that the finding of these wide-spread capillary and venous ruptures with blood extravasation is important in showing the violence of the commotion to which the delicate fibrils, forming the neuronic synapses in the grey matter, have been subjected. Mechanical compression by the escaped blood corpuscles probably plays only a minor part in producing the loss of function. Had an examination of the cervical cord, of the bulb, and of the pons been made, no doubt similar changes would have been found to account for the symptoms noted. The anaesthesia below the level of the umbilicus likewise may be accounted for by the damage to the grey matter especially noted at the base of the posterior horns.

From the situation of the wounds caused by shrapnel (pieces of the shell?) it is probable the main effect of the commotion was upon the lower part of the spinal cord.

Bearing upon this question of commotion I will refer to an interesting article by A. Mairet and G. Durante, on the "Commotional Syndrome," which was published in the Presse Médicale, June 15, 1917. They have experimented upon rabbits by means of powerful explosives in order to try and find out what happens to soldiers in the trenches.

A charge of melinite or chédite placed at 1.50 metres, then at 1 metre, was successively raised from 125 grammes to 1 kilogramme.
Examination of the Brains of Two Dead Men

Of twelve animals used five died spontaneously, respectively in five minutes, one hour, one day, eight days and thirteen days after. The others after a momentary unconsciousness with acceleration of respiration and temporary excitement, sometimes rapidly recovered and were killed, with the result that no signs of local lesions were present. Histological examination in all the animals that died showed early lesions consisting of more or less extensive islands of pulmonary apoplexy, caused by rupture of alveolar capillaries. In most cases hæmorrhages and suffusions of blood were found on the surface of the spinal cord, also in the roots between their emergence from the cord and at their conjugation; also limited ruptures of small vessels in the grey matter of the cortex and of the bulb, causing a blood effusion into the perivascular lymphatic sheath, were found.

More rarely perivascular suffusion of the radiate vessels of the medulla oblongata and of small vessels behind the ependyma were observed. The nerve cells were healthy. Vascular changes were found in the anterior horn and spinal ganglia only in two rabbits, and hæmorrhages in the kidney were found in one animal.

The hæmorrhages especially occur from vessels which are badly supported by surrounding tissues, the blood then escapes into the perivascular lymph sheath which does not offer any support. The hæmorrhages are minute and are diffused, and this fact speaks in favour of a sudden rupture of the wall caused by the decompression which suddenly follows on the wave of compression.

These changes observed by Mairet and Durante are very similar to those which I have described in the cases examined.

It will be noted that in Case 1 there was pulmonary hæmorrhage found at the autopsy.

COMMENTARY.

We do not know what happened to this man, but the shrapnel wounds and the condition of paraplegia, together with the histological microscopic findings in the spinal cord, strongly support the view that a large shell burst near by, wounding him and causing spinal commotion but without injury of the spine. He may have been blown up in the air and thrown violently on the ground, but this seems unlikely, as the notes state that his mental condition was unimpaired and there was no visible injury of the spine; consequently the most plausible explanation of the cause of the pathological condition of the spinal cord is commotion. No cause for
death could be found except shock. It is a pity that the medulla oblongata and the upper part of the spinal cord with the phrenic nucleus were not sent for examination.

Several cases of spinal concussion without visible signs of injury have been under my care and have so far recovered that they could be discharged from the hospital, and I will briefly relate one case in which I diagnosed spinal commotion and haemorrhage.

Pte. C., 8th Seaforths, aged 20, was admitted January 5, 1917, with two years' service, six months under fire. On December 22 he was buried in a dug-out by the explosion of an 8·6 shell which struck the back of the dug-out. He was standing up at the time and he remained in the upright position, never lost consciousness, was got out in a few minutes. He was sent to Havre and then to the Maudsley Hospital. Catheterized three days at Havre. On admission he had incontinence of urine and faces.

Sensory Symptoms.

Except slight hyperesthesia of epigastric region; no sensory disturbances were detected.

There was no evidence of bruising nor any tender spot over the spine.

There was no evidence of paralysis of face or tongue.

There was marked weakness of arm muscles. Right more marked than left. Could grip slightly; he was able to lift arm above head; he was able to turn over in bed. Very slight power of movement in legs, the movement of the knees better than ankles. No muscular wasting, no marked flabbiness. Patellar and ankle clonus on both sides. Plantar extensor on both sides. Wrist tap and triceps jerk obtained easily.

Pupils normal. No ocular paralysis or nystagmus.

Hearing and sight unaffected, also taste and smell.

He had no signs of emotional disturbance; he had no headache. He did not dream, and invariably replied "All right" when asked how he felt. In about a month he recovered power over his bladder and the bowels opened naturally. Movements in arms and legs also increased, and he was able to sit on the edge of the bed and put his feet on the ground. Two months after admission he was able to stand and walk with the assistance of two men. The right hand grip was still weak but the left improved. Three months after admission he was able to walk with the aid of a stick and was sent to a convalescent hospital, where he made further progress.

There were no visible signs of injury in this case, but here we see that the whole wall of the dug-out was blown in and buried him; the force of the explosive was communicated to the spinal column by the solid earth. This man suffered spinal concussion
Examination of the Brains of Two Dead Men

and shock; but the persistence of the plantar extensor response, ankle clonus and patellar clonus, and the loss of voluntary power pointed to damage of the upper motor neurones, and degeneration of the pyramidal tracts. The absence of sensory disturbance might be thought to be against microscopic hemorrhages, such as have been found in the histological investigation. We know however by experiments on animals that the path for sensation is not localized in the same way as that for voluntary movement, and that hemorrhages might occur at the base of the anterior horn destroying the terminal fibrils of the pyramidal tract fibres at their synapsis with the anterior horn cells without closing the sensory path. The shock effect would contribute largely to the loss of power of voluntary movement in the limbs, and the control of the bladder and bowel. The recovery which was made shows that shock, as well as organic changes in the spinal cord, was accountable for the symptoms.

The examination of the spinal cord of the fatal case described indicated to my mind that the lesions were not so severe and gross that he could not have recovered had it been possible to bring him to hospital sooner. His paraplegic condition, in my judgment, was largely due to commotional shock more than actual organic change. The microscopic changes discovered in the grey matter are the visible evidences of the severity of the shock to the spinal cord in its most sensitive and delicate structure, viz., the fibrillary synapses through which are transmitted volitional impulses and sensory impulses from superficial and deep structures.

The biochemical oxidation processes incidental to the transmission of nervous impulses from one system of neurones to another, take place at the synapses, possibly as Professor Marinesco suggests, under the influence of an oxidase ferment.

The intercalary neurones, second type of Golgi, whose axons never leave the grey matter, are always interposed between the neurones of the first type. Thus in the path of voluntary movement the axons of the psychomotor neurones of the motor area of the brain break up into a brush of fibrils at the base of the posterior horn where they are connected with intercalary neurones, which again are connected with the dendrons of the spinal motor neurones, the axis cylinders of which terminate in the voluntary muscles. We have seen that the most vulnerable part of the cord to commotion is the base of the posterior horn. At first there is a flaccid paralysis because the whole sensory reflex arc is knocked out; but as this shock effect passes off, the less vulnerable sensory
reflex path is again opened up, but the inhibitory influence of the more vulnerable psychomotor path on spinal reflex action having been lessened, if not abolished, the plantar reflex becomes extensor, ankle clonus is obtained and the deep reflexes are exaggerated.

BIBLIOGRAPHY.

DESCRIPTION OF FIGURES.

Fig. 1.—Betz cells of leg area. There is commencing chromatolysis of varying degree. The Nissl granules are not so closely packed together as in normal cells. The nucleus is larger and clearer than normal. Magnification 350.

Fig. 2.—A small vessel cut longitudinally in the internal capsule. The vessel is filled with blood corpuscles; the perivascular sheath is seen dilated and filled with red blood corpuscles. Magnification 225.

Fig. 3.—Hemorrhages into the white matter of the pons. Magnification 90.

Fig. 4.—Hemorrhage into the sheath of a vessel in the median raphe of the medulla. Magnification 170.

Fig. 5.—Cells of the vago-accessorius nucleus at the level of the calamus scriptorius. Observe the marked chromatolysis and eccentric position of the nucleus. Compare the same with fig. 6. Magnification 400.

Fig. 6.—Cells of the adjacent hypoglossal nucleus, showing early slight chromatolysis. Magnification 400.

Fig. 7.—An arteriole breaking up into capillaries with dilated perivascular space. This space is in communication with the perineuronal space around the nerve cells. Magnification 300.

Fig. 8.—Section of cortex, Case 2. Dilated perivascular space around collapsed arteriole and capillaries. Dilated perineuronal spaces. Magnification 375.

Fig. 9.—Cortical cells from Case 2, showing swelling and chromatolysis of cytoplasm and clear swollen nuclei. Magnification 400.

Fig. 10.—Section of cerebellum, Case 1, stained with polychrome and eosin. Note the Purkinje cells are not all similarly stained. Two are stained faintly with the basic dye; the remaining ones are stained with the acid dye indicative of a chemical change. Magnification 270.

Fig. 11.—Medium sized anterior horn cells in first lumbar segment; a microscopic hemorrhage is seen near, the Nissl granules have almost disappeared in the cells, and the staining is diffused and uniform without the displacement of the nucleus. Magnification 390.

Fig. 12.—Hemorrhage, the size of a small pin's head, at the base of the posterior horn; the tissues around are fractured and retracted, but this may be in part due to the action of the fixing fluid. Magnification 185.

Fig. 13.—Two large anterior cornual cells from the third lumbar segment showing fairly well marked perivascular chromatolysis; the nucleus in one is eccentric and the nucleolus cannot be seen. Magnification 360.
To illustrate "The Microscopic Examination of the Brains of two men dead of Commotio Cerebri (Shell Shock) without visible external injury" (Figs. 9 and 10), and "Commotion of the Spinal Cord" (Figs. 11, 12, 13), by F. W. Mott, M.D., LL.D., F.R.S., F.R.C.P., Major R.A.M.C.(T.).