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HEAD INJURIES.*

BY

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It is my privilege to try to put before you certain ideas on head injuries as they occur to me, such as may help you to visualise the conditions that are present in any given case, and assist you in both diagnosis and prognosis. In view of the great frequency of motor-car accidents, and others of an analogous nature, it is absolutely essential in the treatment of the patient that we should be enabled to picture the conditions that are present.

CONCUSSION.

Head injuries of any severity are almost necessarily followed by a period of concussion, the signs and symptoms of which state are well known. It is also, I think, unnecessary to discuss the various theories that have been advanced in explanation of the onset of concussion—all are hypothetical, and some are so fantastic as to merit no mention. It is infinitely preferable, I think, to consider certain outstanding features in all concussion cases, and to try to explain them, thus dealing with concussion from a more or less clinical point of view.

Under ordinary circumstances the brain receives that amount of arterial blood which is required to permit it to carry out its normal functions. I imagine that, in lectures such as this, and in all similar conditions, the brain-cells are hyperactive, encouraged for a special purpose by an extra flushing of arterial blood. Per contra, it can be assumed that, in concussion, in which state there is a sudden abolition of all conscious acts, of all voluntary action, of all knowledge, and of all

memory, that the vascular conditions of the brain are exactly opposite—in other words, that the brain is in a state of *acute arterial anæmia*. When endeavouring to explain, in a simple logical manner, this state of acute anæmia of the brain, I pin my faith in the effects of the injury on the medullary centres, and more especially on the vasomotor centre, the main function of which is to keep up our blood pressure. When this vasomotor centre is violently inhibited, or paralysed, there is a sudden and immediate great fall in blood pressure. The vessels of the brain possess no special vasoconstrictor or vasodilator nerves—they passively follow the changes in the general circulation. Consequently, in the event of inhibition, or paralysis, of the vasomotor centre, with subsequent great fall in general blood pressure, the arteries of the brain are deprived of their normal circulating blood, with consequent immediate loss of power and consciousness.

I would remind you that it is advisable, when estimating the gravity of any head case, to possess some simple form of blood-pressure apparatus, whereby the blood pressure may be estimated from time to time. Next, it is necessary to remember that the diminution in the amount of circulating arterial blood, within the cranial cavity, must be compensated for by a corresponding increase in the amount of venous blood—in other words, the development of acute arterial anæmia is immediately followed by an *acute engorgement of the cerebral venous system*.

If one tries to visualise the brain at this stage one would see a brain in a state of acute venous engorgement, all the veins full and turgid, and in the more severe case the blood almost in a state of venous stasis.

Next, let us turn our attention to the *cerebro-spinal fluid*. This fluid, whatever may be its function, is secreted continuously from the choroid plexuses of the ventricles, and, under normal conditions, it is absorbed into the cerebral venous

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system, mainly per the medium of the arachnoid villi related to the superior longitudinal venous sinus, but also per all the surface cortical veins. The cerebro-spinal fluid exists at a pressure just above that of the cerebro-venous system, and its specific gravity is less than that of the venous blood. The cerebro-spinal fluid, therefore, drains automatically into the cerebral venous system with which it is so closely related—provided that the pressure conditions are normal. But what happens to this process when the cerebral venous system is markedly engorged—as is the case in concussion? The absorptive process is inhibited, and the opposite conditions prevail—the fluid accumulating, first filling up the cisternæ and cerebro-spinal spaces of the brain, overflowing into the subdural space, bogging the surface cortex of the brain, and producing, in general, a state of *acute cerebral oedema*.

So far as we have gone, therefore, we have now to visualise a brain, not only in a state of *acute venous engorgement*, but also *oedematous with cerebro-spinal fluid*. Now, in the event of the injury being of a comparatively trivial nature, there being no exact lesion of the brain substance and no hæmorrhage related to the membranes, the state of concussion or cerebral shock will pass away—the vasomotor centre recovers tone, the blood pressure rises, the brain is again flushed with arterial blood, the venous blood is swept on, the cerebro-spinal fluid again absorbed at a normal rate, and the conditions return eventually to the normal. Between the two stages, the primary abnormal stage of concussion and that of recovery, there may be, and usually is, an intermediate *stage of reaction*—the vasomotor centre, after its primary inhibition, putting forth an excess of energy, raising the blood pressure above the normal, and leading to that post-concussion restless stage, with raised temperature, flushed face, headache, and general irritability—facts observed in practically all cases of concussion. Later on the vasomotor inclines to the normal, and the patient recovers completely.

This is the process, and these are the changes that, as I imagine, occur in all the milder cases of concussion—a transitory loss of consciousness, lasting a few seconds or minutes, and followed by some degree of headache, irritability, &c.—such, for example, as happens when a man is knocked out in a boxing contest, recovering shortly afterwards, and later resuming his normal occupation.

Next, I wish to go to the *opposite extreme*. After a very severe head injury the patient is concussed in a very severe degree, remaining in that state till he dies shortly afterwards. There is no attempt at recovery, no development of reactions or compression symptoms. In all such cases the post-mortem examination reveals the gravest cerebral lesions, gross lacerations of the brain, and various intracranial hæmorrhages.

On comparing these two pictures, the very mild and the very severe, it is at once obvious that the

more prolonged and the more profound the stage of concussion, the more certain is it that the patient is suffering from gross intracranial injuries.

I may remind you at this stage that laceration of the brain is most frequently observed in the *frontal lobes*, the anterior and inferior aspects of that lobe, in the angle between the vertical and horizontal plates of the frontal bone. In a patient, therefore, who presents the profound and prolonged degree of concussion, one may visualise, with a fair degree of accuracy, a severe laceration of the frontal lobe.

THE AVERAGE CASE OF HEAD INJURY.

Now we come to the average case of head injury, concerning which certain questions will arise: (1) Is there present any lesion of the brain or any intracranial hæmorrhage? (2) What is the immediate prognosis? and (3) What can we do to help the patient in the several conditions?

In this average case of head injury the immediate effect of the injury is concussion, varying in degree and intensity, followed by reaction and then presenting, to a varying degree, the symptoms and signs of cerebral compression. In such cases it is obviously highly desirable that we should formulate, as early as possible, some ideas as to the probable outlook, and it is essential above all things that we should be in a position to know whether the brain is injured or not—for on that question hinges the prognosis, both immediate and remote, as also the treatment for that particular case.

During the past two years every case of head injury admitted to hospital under my care has been submitted to *lumbar puncture*, soon after their admission, when still unconscious—with the object of determining whether or not there is *blood in the cerebro-spinal fluid*. The importance of this procedure is obvious for, in the event of brain laceration or intracranial hæmorrhage, blood is effused into the subarachnoid space, becoming intimately mixed with the cerebro-spinal fluid, to be verified on lumbar puncture. It is essential, of course, that the puncture should be carried out with that degree of skill necessary to avoid injury to the rachidian vessels. In the event of brain laceration, the cerebro-spinal fluid will be deeply tinged with blood within a few minutes of the injury—in some cases almost pure blood may be withdrawn. In the slighter cases the fluid contained in the test-tube may be allowed to stand, the corpuscles settling to the bottom, but leaving the supernatant fluid slightly brown.

So far, therefore, we have considered two points: The depth and duration of the concussion stage; and the presence or absence of blood in the cerebro-spinal fluid.

Before passing on to the third point in our diagnosis as to the conditions present, I would like to add that, although I wish to make my points as definite as possible, yet to almost every rule there are exceptions. For example, in respect of the statement that blood will be intimately mixed

with the cerebro-spinal fluid in the event of intracranial bleeding, this is not absolutely correct. In the event of bleeding from the middle meningeal artery, the hæmorrhage is situated outside the dura mater, between it and the bone. In such cases the bleeding cannot be detected by lumbar puncture. We do not, however, often encounter a case of middle meningeal hæmorrhage that is not complicated by associated laceration of the brain, in which cases blood will be intimately mixed with cerebro-spinal fluid, and in which case our lumbar puncture investigation will at least lead us in part along the right road.

THE TEMPERATURE CHART.

And now we come to a *third* point, the temperature chart. In every case of head injury of any severity, the initial temperature of the patient is markedly subnormal, in the region of 96°. The changes in temperature that occur subsequently are so constant, according to the severity of the lesion, that I would assert that the prognosis in any given case can be made by a glance at the temperature chart alone—in the majority of cases without even seeing the patient.

In one type of case, subsequent to the initial shock stage with its subnormal temperature, the temperature steadily rises to 104°, 105° or higher. Such cases almost inevitably terminate fatally within 24 hours or so, and autopsy reveals extensive injury to the brain substance and intracranial hæmorrhages, subarachnoid or subdural. In another type of case the shock stage obstinately persists, in spite of all restoratives, till death ensues, again within 24 to 48 hours. Extensive brain lacerations and hæmorrhages are nearly always present.

I cannot put forward any convincing explanation as to why, in these two types of case, when the lesions are equally serious, there is in the one case the rise in temperature, in the other the persistence of the shock stage. The fact remains that, whether the temperature steadily and progressively rises, or when it remains obstinately subnormal, a fatal result is almost certain within 24 to 48 hours.

And now we come again to the average case. From the initial shock stage, with its subnormal temperature, the temperature rises to a moderate degree, 101° to 103°, then marking time, hanging fire, so to speak, till further changes ensue. A subsequent persistent rise indicates a fatal result, a definite fall allows of every hope of recovery, so far as the immediate result is concerned. The remote effects are quite another matter, with which we are not concerned this afternoon.

As previously stated, the greatest reliance can be placed on the temperature chart. The points to which I have alluded are of the greatest possible assistance, guiding us as to the eventual result in any given case.

We have now another factor added to our list, as aids to prognosis and treatment: the depth and

duration of the concussion stage; the presence or absence of blood in the cerebro-spinal fluid; and the temperature chart. One of these alone may be sufficient, in our estimation of the gravity of the case. The three together are more than helpful.

TREATMENT.

Up to now the treatment of the case has not been considered, and rightly so, because our treatment is entirely based on a correct estimation of the conditions present, based on the points already alluded to.

Treatment must be considered from two points of view: (1) where there is an obvious or suspected fracture of the vault of the skull; and (2) where the patient presents general symptoms of cerebral compression, with or without fracture of the base of the skull. I would venture to point out to you that when a patient is said to have died from fracture of the base of the skull, that the material part of the case is entirely omitted. People do not die from fracture of the base of the skull—they die because the basal lesion is complicated by a far graver state of affairs—laceration of the brain and other intracranial lesions. And it is up to us to determine, so far as we can, the presence of the brain lesion and its presumed degree of severity. This question can be cleared up in the great majority of cases by the methods already indicated.

In all these cases of head injury, excluding such measures as may be required for the cleansing of a scalp wound, and for the control of bleeding from scalp vessels, operative treatment is absolutely contra-indicated during the persistence of the shock stage. This state must be combated first by the adoption of suitable remedies—rest, warmth, and the administration of small doses of morphia. Morphia will not disguise the signs and symptoms that may develop subsequently, and it does act as a damper to the shock impulses transferred to the medullary centres. At this early stage it is necessary to avoid those more active methods of combating shock—infusion and transfusion; the diagnosis is the first consideration, as to whether the patient is suffering from intracranial hæmorrhage or not.

In further consideration of the question of treatment, the patient must be watched most carefully, the temperature, blood pressure, and pulse-rate being noted hourly, and lumbar puncture carried out in order to clinch the diagnosis. In the event of the obstinate persistence of the shock stage, as evidenced by the subnormal temperature and general signs and symptoms of concussion, all question of operative interference is abandoned, at any rate for the time being.

Again, if the temperature steadily rises, with acute development of compression symptoms, I feel sure that the surgeon should abstain from any operative interference. In my experience no benefit results from such measures. The only time where, so far as I can see, the surgeon can

help the medullary centres in their fight against the increasing intracranial pressure is when, as in type three of the temperature chart changes, the temperature has risen to about 102° or 103° and there halts, where the blood pressure is raised to 180° or 200° , where the pulse-rate is slowed to 50 or less, the pulse being full and perhaps slightly intermittent, and the respiration is noisy but not yet stertorous. This is the critical stage of the case, and the surgeon should watch the progress with the utmost vigilance, and where, in the event of persistence of compression symptoms and in the absence of any real sign of improvement, he should carry out such measures as are indicated. These are (1) a direct attack in the presence of an osseous lesion of the vault, and (2) an indirect decompression operation in all other cases.

Such operations should be carried out, whenever possible, under local anæsthesia, or without any anæsthetic, the operation being preceded by the administration of a dose of morphia. A decompression operation has for its main object the provision of such decompression as will compensate for the increased intradural pressure.

Any operation should be conducted with the least possible disturbance of the patient, and the patient returned to bed as soon as is possible. From this it would appear that, judging from my own experience, every head case requires the most careful supervision, and operative measures should only be carried out during that stage of compression to which I have alluded. It is absolutely futile to decompress a patient in the later stages of compression, when the medullary centres are failing, when the blood pressure is falling, when the pulse-rate is rapidly increasing, when the respiration becomes deeply stertorous, even Cheyne-Stokes. In such cases nothing can be done to save the patient's life.

Before concluding I should like to say a few words about the operations that may be carried out. In the event of a direct attack on a vault fracture the injured area must be fully exposed, preceded by the excision of the scalp wound itself. Trephining may or may not be necessary, but all depressed fragments of bone should be elevated or removed and the dura mater widely exposed. Under normal conditions this membrane is sufficiently translucent to allow of the inspection of the underlying conditions. If very tense, and showing obvious subjacent blood extravasation, the dura should be incised and the blood allowed to escape. This course should, however, only be adopted under the conditions specified; the risk of secondary infection of the meninges has always to be considered.

In respect of decompression operations carried out for the attempted relief of general compression symptoms, the following points require consideration. The decompression is conducted so as to allow of that outward bulging of the brain which would compensate for the degree of increased intracranial pressure. It is obvious, therefore, that such

decompression operations should always be conducted over a silent area of the brain, never over the Rolandic region. A wide removal of bone over the temporo-sphenoidal lobe, on the right side of the head so as to avoid any possible inclusion, in the subsequent protrusion, of Broca's speech centre, fulfills all the desired requirements. The aperture should be generous in size, not less than four inches in the antero-posterior direction and two inches in the vertical, lying over and running parallel to the temporo-sphenoidal lobe. The dura mater must be incised freely, and the brain permitted to bulge as much as it desires. The scalp flap is then accurately approximated. The intradural pressure is thus relieved, and it is hoped that the decompression will tide the patient over the acute compression period.

The surgeon, when dealing with these cases of head injury, will meet with many disappointments, but the successes obtained will compensate for his failures and encourage him in the future.

SPECTRAL "HEART DISEASE"

AND RESPIRATORY ARRHYTHMIA.*

BY

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PRESENT-DAY psychologists, as I believe, still define an illusion as a false perception, something seen or felt by a person who wrongly interprets it—and a delusion as an erroneous judgment.

By the term "spectral" in the title of my lecture on this occasion, I wish to include both of these ideas as applied to supposed "heart disease." My main theme, however, is illusory "heart disease." In that phrase I wish to imply a fear of heart disease based, it is true, on what are popularly considered heart symptoms, but on such as have little, if any, pathological importance.

As I imagine it, the fear of "heart disease" in the popular mind is derived from the idea that persons with heart disease often die suddenly, or "drop down dead" as the phrase goes. The proper treatment for "spectres" in general is, we know, to face them and, if it may be, lay them. This end, I take it, we shall best further by trying to educate the subjects of such "heart symptoms," and, at the same time, arouse their medical advisers rightly to estimate importance, and rightly to estimate probability in this relation.

Some years ago a friend of mine, at that time holding a commission in the R.A.M.C., came to

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