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ON THE MECHANISM OF BRAIN INJURIES.*

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PRELIMINARY CONSIDERATIONS. VARIOUS THEORIES OF CONCUSSION.

THE question as to whether or not it is possible to have fatal injury to the head, in which there is no appreciable gross lesion to the brain substance directly, or through its membranes, so called concussion, has long been discussed by writers on this subject.

By the older authors, the celebrated case, recorded so graphically by Littré (1†) now nearly two centuries ago, was accepted as decisive of the possibility of such a thing, but being almost solitary, it was by some looked upon as the exception which proved the rule. Although the case most quoted, however, Littré's was not the only one of alleged fatal concussion, Sabatier (2), Boyn (3) and Mourier (4), have all left records of similar cases. In a more sceptical age these classic records have been somewhat severely criticised, and the light of a more advanced physiology and pathology, with the information derived from more carefully

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† These figures refer to the corresponding numbers in the Bibliography at the end of the paper.

conducted *post-mortem* examinations, has thrown serious doubt on the accuracy of the observations on which the conclusions were based.

Sir Prescott Hewett (5) leads the attack against the theory of Littré, based on his oft quoted case, and concludes that "it has still to be demonstrated that death can result from mere concussion." Holmes (7), Bryant (8), McEwen (9), and Savory (10) in this country, and Neudorfer (11), Nancrede (12) and Fano (13) abroad, are, in the main, with Hewett; and so far as my experience, based upon *post-mortem* examinations, as well as a large number of experimental observations goes, I must agree with them.

The school which supports the old view is neither so strong, in point of numbers, nor so powerfully armed with statistics as is that of its opponents; but it includes men whose opinion is of sufficient weight to give them the standing of an influential minority. Huguenin (14), Hutchinson (15), as well as some others, believe that death may result from concussion without gross lesion. Mr. Hutchinson admits that lesions exist, but holds that they are to be looked upon "as an index of the violence of the shake, and not as causes of death, nor perhaps even as serious complications;" and Mr. Savory, while denying the occurrence of death without lesions, admits that "the effects must be due to the shock of the violence rather than to the lesions."

This raises the question, What is the ultimate cause of the symptoms in so-called cases of concussion of the brain? Is it, for example, as the old writers supposed, that the brain is thrown into a state of vibration which causes for the time being a suspension of its functions, as was held by Bruns (17), Liston (18) and Miller (19), on more or less purely theoretical grounds; or are the multiple capillary hæmorrhages on which Fano (13), Nèlaton (21) and Rokitansky (22) laid such stress, the important causal pathological factors; or again, must we look to some profound vascular disturbance in the nervous centres as the "*fons et origo mali*?"

The Vibration Theory as stated by the late Professor Miller of Edinburgh, is as follows:—"Under the impulse of a blow or fall the brain must sustain more or less vibration if the cranium

remain entire. It is a pulpy organ, which exactly fills a nearly spherical bony cavity, whose parietes are elastic in a considerable, though very variable, degree; and if these parietes sustain any sudden change of shape, their contents must sustain a corresponding amount of compression, as any alteration in the shape of a spherical cavity must lessen its capacity. Whenever any external force impinges on the cranium with sufficient violence, it must be flattened at the point of impact, and expanded in some other direction; but these changes are, in virtue of the very cause whence they originate, of but momentary duration; the point primarily flattened by the compressing force immediately resumes its original shape, which is necessarily followed by a corresponding return of the expanded portion of the cranium to its previous dimensions. These oscillations may occur several times in rapid succession, their number and extent depending on the elasticity of the cranium, and direction of the force applied. In concussion then, the entire brain sustains a series of vibrations and momentary compressions varying in number and amount in every imaginable degree in different cases."

These views are supported by Bruns, who says that: "the vibrations permeate the brain as a whole, as well as its individual component parts." Gama (23) and Alquié believed that while the brain elements remained unaffected, the brain itself might be knocked from side to side of the cranium; but the results of these observers must be received with due caution, as their investigations were carried out on an artificially constructed apparatus, which could only imperfectly imitate the exceedingly delicate anatomical arrangements of the human brain.

There are several points in the vibration theory, thus enunciated, to which exception must be taken.

In the first place, the brain in its normal state, whether or not it be a "pulpy organ," does not exactly fill the cavity. There is everywhere a slight, and in some places a considerable space between it and the bone, filled by the cerebrospinal fluid, a fact of great pathological as well as anatomical importance.

Nor is the cranium, I submit, by any means a "spherical cavity." It is so far removed from a sphere, that the laws of physics which apply to cavities of that shape do not affect it. It has been shown over and over again that the capacity

of the cranium is in constant proportion to its size, any increase in the quantity of one element, *e.g.*, blood, being compensated for by an escape of another, *e.g.*, cerebro-spinal fluid, and *vice versa*. And it is by an escape of one or other, or both of these fluids that a depression of the parietes is able to diminish the contents of the skull, which, by the mere fact of being depressed, has had its size diminished, and therefore proportionately its capacity.

That this depression is not permitted by any appreciable diminution in size of the brain itself, is shown by the fact that it requires the pressure of a whole atmosphere to reduce a body of the consistence of the brain by $\frac{1}{1000}$ th of its volume, and in very few head injuries is the pressure anything like so great, Duret (24). This theory also leaves out of account the fact that the hemispheres are hollowed out by the ventricles, and that before pressure could directly diminish the volume of the brain substance these cavities would have to be obliterated.

Other anatomical arrangements in the shape of the falx cerebri and tentorium cerebelli, etc., seem to me so to fix the brain in position that any considerable displacement of it would be evidenced by lacerations, due to its impinging on these tight resistant bands.

Taking all these points into consideration, I do not think there is sufficient evidence to show that the vibration theory is tenable.

The next theory, namely, that the symptoms of concussion are to be accounted for by the multitudes of capillary hæmorrhages so often found in the cerebral substance, has as its chief supporters, MM. Fano and Nélaton, and with them in the main Stromeyer (25) agrees, although he differs from them as to when these extravasations take place, the former assigning them to the primary stage, the latter to the stage of reaction. Chassaignac and Sanson are also disposed to look upon these lesions as sufficient to account even for death in fatal cases.

Drs. Bright (20) and Blandin (16) do not go so far. They believe that "in addition to minute extravasations, patches of contusion of the cerebral substance are necessary to account for death, if not for less serious symptoms."

On the other hand Huguenin (14) and McEwen (9) do not believe that these scattered lesions are sufficient to account for the phenomena, and with them M. Duret (24) is at one. He says that he "sought as carefully as possible at the autopsies and found only small lesions quite insufficient to explain so grave phenomena."

Mr. Jonathan Hutchinson argues that these lesions cannot be the cause of the symptoms and of death, because many cases with worse lesions, not only live but have no symptoms.

Unfortunately for this theory multiple hæmorrhages are by no means constant, many cases occurring in which none are to be found. Besides, the fact that the symptoms of concussion, in many cases, so soon pass off, argues against their being due to lesions, which from their very nature, could only disappear after a period, at least much longer than is required for the cessation of all evidences of concussion.

Neither of the theories just considered, namely (1) that of physical mechanical vibration; and (2) multiple petechial hæmorrhages seems to be sufficient to account for the varied and general symptoms of concussion.

Is that of derangement of the cerebral circulation any more acceptable?

When we consider the general character of the symptoms, how widely spread they are, affecting every function and organ in the body, from the prefrontal lobes with their highly specialised actions, to the least important and simplest mechanical muscle, we comprehend how almost impossible it would be for any single focal lesion, however diffuse, to be held responsible for all of these. And if we bear in mind the close resemblance which this group of symptoms bears to other conditions—such as cardiac syncope, reflex syncope from pain, or severe mental shock, where the cause is acknowledged to be a disturbance of the circulation of the brain—we are induced to accept this explanation of the phenomena, as at least a more workable theory than either of the other two. Most modern writers accept this view of the subject.

Some, such as Savory (10), McEwen (9), Le Grós Clark

(6), and others, define concussion as a form of "shock," which is only modified in so far as the hurt has expended its force on the brain.

Mr. Hutchinson (15) regards "anæmia of the brain" as being "the proximate cause of the phenomena of concussion," but whether this condition is produced by contraction of the arteries, or by diminution of the venous tension leading to congestion, and so an "oxygenless" condition of the cortex, he does not give an opinion.

Huguenin (14) accounts for the anæmia by the blood being squeezed out of the vessels by momentary compression of the brain substance, whose normal function depends on a proper supply of oxygenated blood, and which so suffers from thus being temporarily deprived of it, that its functions are suspended for a period varying in proportion to the severity of the injury as indicated by the amount of vascular disturbance.

It is doubtful if simple mechanical compression of the brain substance is the cause of the anæmia, as we have already seen that a much greater force is necessary to accomplish this than is almost ever applied in simple cases of concussion, and Nothnagel (28) has tried to explain the contraction of the blood-vessels on the assumption that it is the reflex effect of strong irritation of peripheral nerves. He does not explain, however, how this state of contraction can be kept up reflexly for several days, as is sometimes the case in concussion. Jolly (26), Rigel (27), and Frey (29) have failed to reproduce these symptoms by reflex irritation of vaso-motor nerves, although Goltz has produced a reflex paralysis of the vessels by injury to the head, which supports Fischer in his belief that this is the true explanation of the symptoms.

I propose now to consider what clinical and experimental proof we have of the existence of such a vascular disturbance.

INFLUENCE OF CONCUSSION ON THE HEART AND GENERAL CIRCULATION.

The experiments and observations on animals to which I am about to refer are described in detail in another part of

my paper. Unless the injury was intended to kill the animal instantaneously, full chloroform anæsthesia was produced before the experiment was commenced.

In the first case, that of a rabbit before the injury was inflicted, when the animal was in an excited state, the pulse was 200, and only countable with difficulty. After a severe occipital blow had been delivered however, there was marked slowing of the pulse. During the first minute following the blow no cardiac impulse could be felt whatever. During the second minute the action was very irregular—for the first fifteen seconds the pulsations were at the rate of forty to the minute, but this was followed by a rapid succession of small beats which were quite uncountable, but which, if counted, would of course have raised the number of beats per minute considerably. This irregularity, however, was evidently due to the action of respiration, at all events, it was synchronous with long-drawn, deep inspirations, and if we assume the causal relationship between the two, then the heart *per se* was certainly slowed, and that in a marked degree, and whether we make this assumption or not we can safely say that the circulation was seriously interfered with.

In another experiment on a rabbit, before a moderately severe frontal blow had been administered the heart was beating regularly and at the rate of 190 per minute. During the first minute after the blow the heart was acting so tumultuously as to be quite uncountable, and continued to do so for the next two minutes. At the end of four minutes, however, it was again regular, vigorous, and acting at the rate of 130. The animal was deeply under chloroform.

In a third rabbit, from which the cerebro-spinal fluid was rapidly aspirated under chloroform, the heart was from the first slow and feeble.

In a fourth experiment where the pulse before the blow was given was 180, rapid and small, two minutes after, it was down to 120, and described as full and bounding.

M. Duret (24) has demonstrated in an experiment carried out with the object of ascertaining accurately the heart rate following a blow to the head, that there is a

period during which the pulse is very slow, full and bounding, and this period he finds is synchronous with a disturbance and slowing of the respiration.

The experiment was performed on a dog with the following results :—

Before the blow the pulse was	...	...	100 p. min.
1st minute after blow	...	...	14 "
2nd " "	...	...	16 "
3rd " "	...	...	12 "
4th " "	...	...	12 "

This stage is followed by another, during which the pulse, while becoming faster, is of very low tension, even dichrotous ("*avec gruppetti*") and this stage corresponds to an acceleration of respiration.

6th minute after blow pulse was	...	68 p. min.
7th " "	...	68 "
20th " "	...	40 "
30th " "	...	54 "
2 hours " "	...	60 "
4 " " "	...	93 "
8 " " "	...	82 "

This observation seems to agree for the most part with what I have gathered from the results of my experiments, which I may here say were not performed to determine this point particularly, and so are less detailed than they would have been otherwise. The conclusion these observations warrants is, that following a severe blow to the head there is usually a certain degree of slowing of the heart, and in all cases a profound disturbance of the general vascular system, a disturbance which must seriously affect the organs of the central nervous system.

DOES THE BRAIN BECOME ANÆMIC OR HYPERÆMIC?

It is well known from the researches of Vulpian (30), Claude Bernard and others, that a reflex vascular spasm may be produced by irritation of sensory nerves of the body, or by stimulation of the restiform bodies, thus raising the general arterial blood tension, and that this increased tension is due to the contraction of the peripheral vessels

with accumulation of blood in the larger ones, and increased resistance to the heart's action. That such a spasm of the vessels of the brain exists, was demonstrated by Beck, who showed that under such conditions the surface of the brain was pale, and the ordinary pulsations invisible. Duret has confirmed this observation, and adds that the vascular spasm increases in proportion with the amount of violence used. Roy and Sherrington (47), however, state that direct stimulation of the medulla causes congestion of the brain, the vessels passively dilating, but they introduce a fallacy into their experiments by providing for a free escape of the cerebro-spinal fluid, so depriving the vessels of what support they receive from that fluid, and leading to their dilatation.

I hope to be able to show later, that in head injuries there is such an irritation of the restiform bodies as would account for the origin of such a reflex action. Meanwhile let me remark that the result of this alteration in the vascular supply of the brain, whether by spasmodic contraction of the vessels, or by their passive dilatation, will be to deprive the nervous substance, for a time at least, of the copious supply of pure oxygenated blood which is so necessary for the perfect performance of its functions; in other words it produces a temporary anæmia of the brain, just as would be done by bleeding an animal suddenly and almost completely, or by injecting lycopodium seeds into its vessels.

Kussmaul and Tenner (51) made some experiments on the effects of bleeding animals, and in these they produced symptoms which very closely resemble those observed in a number of my experiments, where very severe blows were dealt to the head, such symptoms as tetanism, marked changes in the pupils, localised convulsions, and so on, being very common.

In support of the contention that in my cases the phenomena were due to cerebral anæmia, just as they were in the animals which were bled, I may quote the conclusion come to by these observers, after they had produced similar symptoms by cutting off the blood supply to the brain. They say "that the interruption of the conveyance of arterial

blood to the brain produces tetanic symptoms as surely as hæmorrhage does."

Sir Astley Cooper (52) demonstrated the same point when he tied both carotids and vertebrals in a dog, with the result that the animal "lost sensation, had great respiratory trouble, with dilated pupils, it fell on its side and became violently convulsed. Stupor and general paralysis lasted for two days, and then gradually passed off, the animal recovering completely."

The series of symptoms produced in this animal, and undoubtedly due to cerebral anæmia, corresponds so closely to what I have again and again seen in my experiments, that I feel considerable confidence in believing, especially when supported by the facts brought out by Vulpian and Bernard, that the pathological condition in both is essentially the same, the only difference being in the originating cause of the anæmia.

To sum up then, I think the weight of evidence is in favour of the view that the symptoms of cerebral concussion are due to a profound disturbance of the circulation of the brain, and that the alteration is in the direction of anæmia.

ON THE EVIDENCE IN FAVOUR OF STIMULATION OF THE CORPORA RESTIFORMES BEING THE CAUSE OF THE VASCULAR DISTURBANCE IN CEREBRAL TRAUMATISM.

While making observations on a series of cases in which rabbits, pigs and birds had been subjected to blows on the head, varying in severity and in the exact region to which they were applied, I was struck with the similarity in the symptoms which immediately followed such injuries.

Let us take the case of a rabbit to which a severe blow had been dealt, as an example.

In the first place, the animal deeply anæsthetised, was thrown into a violent tetanic state, with greatly exaggerated motor phenomena, these lasting only a few seconds, and being followed by other motor symptoms less violent and more localised; such indeed as might be attributed to some lesion of a particular part of the brain. These effects I have

observed in a great many of my experiments, in fact, in all in which the blow has been of even moderate severity.

As time wore on the phenomena of this stage, which Duret has appropriately called the "tetanic stage," were gradually replaced by others of a more serious nature. The rigidity and jerkings of the limbs gave place to paralysis, the animal could no longer be roused, and the respiratory and cardiac functions were so interfered with as to cause death.

The symptoms of the tetanic period are illustrated by the following experiments—in all of which, save those on pigs, the animals were fully anæsthetised before the blow was delivered, so that the element of pain does not require to be considered. The pigs suffered from swine fever and were consequently being killed at the Edinburgh slaughter-house, where I had the opportunity of observing them.

(1) An occipital blow delivered to a rabbit, was immediately followed by spasmodic convulsions of all the muscles of the body; and these by violent seizures which caused the animal to rotate round its long axis.

(2) A severe blow over the frontal region of a young pig caused the animal to fall. It then lay on its back for a few seconds, all its limbs, but especially the hind ones, being violently moved. There was no distinct opisthotonos.

(3) In another pig a blow of moderate severity was dealt over the vertex. With a scream the animal passed into a state of extreme opisthotonos; for an instant it was in a state of extreme rigidity, and then all its limbs became violently agitated.

(4) In this case the blow was delivered over the left parietal region of a rabbit, while the creature was deeply anæsthetised, and rigidity of all the limbs resulted at once.

(5) In this case the blow was over the occiput, the subject being a young domestic cock, and the result was the same; the creature passing into a condition of extreme tetanus, followed for a few seconds by jerking of the limbs.

These examples will suffice to show that in cases of severe head injury in animals of different genera and even classes, and applied to different parts of the head, practically the same results are produced in the first few seconds after the hurt has been sustained.

And if we look at the gross lesions found in these cases after death, we fail to discover a satisfactory explanation of all the phenomena—opisthotonos, pleurosthotonos, agitation of all the limb muscles, sometimes spasms of the facial muscles, the ocular muscles, and even a combination of all these.

But even did we find a hæmorrhage or a laceration in some particular part of the brain, which could have been the cause of symptoms so serious and so widely diffused, we have still to face the difficulty that these symptoms follow so rapidly on the blow that they cannot be attributed to lesions, which, from their nature must of necessity take some little time to be produced.

It seems therefore that this consideration strengthens the view that the phenomena are due to a reflex action, as was first pointed out by Duret.

But for a reflex action we require an efferent stimulation. What then is the area stimulated in these cases? We know from experience that injuries to the scalp, even much more severe than those produced in the cases quoted, do not give rise to such symptoms; and it has been shown that direct stimulation of the nerves of the dura mater likewise fails to produce such a gross reflex manifestation.

We must therefore look to some part of the brain itself for an explanation, some part which has a particularly sensory function, and in which lesions are constant in such cases.

The notes of my observations shew that the area most frequently injured is the bulb, and it has been pointed out by Duret that the most sensitive part of the bulb is the restiform body, by irritating which *mechanically* he has succeeded in producing all the phenomena of this tetanic stage of concussion. He says that "the phenomena of the first stage of concussion find a very satisfactory explanation chiefly in the irritation of the restiform bodies. . . ." and this statement is supported by the fact that in all the cases just referred to there is evident enough source of irritation of these bodies, and that in those in which the symptoms were most marked the lesions are also most prominent.

The next points to be decided are—"How are these parts stimulated? How are the lesions so often found in the region of the bulb produced?"

ON THE ANATOMY AND PHYSIOLOGY OF CEREBRO-SPINAL FLUID.

The physiological importance of the cerebro-spinal fluid has been carefully studied by John Hilton (41), Richet (43), Salathé and others, who have shown that in all probability its function is to permit of the variations in volume of the brain which are known to accompany functional activity, great muscular or respiratory exertion and so on.

Now it is evident that the rigid cranium can take no part in providing for these variations in size, and it is equally evident that they must be provided for, because the delicately constructed cerebral tissue would not be tolerant of such pressure as would otherwise inevitably result, without exhibiting important symptoms. We would therefore conclude, and there is abundant experimental and clinical evidence in support of the view, that the cerebro-spinal fluid is the safety valve of the brain.

Believing that the cerebro-spinal fluid is the all-important factor in the production of the phenomena of concussion, and of the lesions found in severe head injuries, I must briefly consider the anatomical distribution of this fluid in the nerve centres.

A. Everywhere the brain is surrounded by a layer of cerebro-spinal fluid, varying in thickness or depth at different places, constituting what has been called the "water bed of the brain." The main "lakes" or "cisterns" are situated in the region of the base and also along the course of the larger blood vessels, such as the sylvian artery.

B. The brain is hollowed out by a series of cavities—the ventricles—and these are filled with cerebro-spinal fluid.

C. In connection with each blood vessel as it enters the brain substance there are two distinct lymph spaces:—(a) The adventitial lymph space of Robin, between the tunica muscularis and the tunica adventitia, and continuous with the sub-arachnoid space; and (b) the peri-vascular lymph channel of His, between the adventitia and the brain substance, and continuous with the epicerebral space. These spaces, the latter of which continues to surround the blood vessel on to its minutest ramifications are filled with cerebro-spinal fluid, in which the blood vessels are

suspended. This is the anatomical point previously overlooked in this relation.

D. And lastly, the nerve cells themselves are similarly placed, each cell being, as it were, suspended in a small excavation in the brain substance, called a pericellular sac. The communication between the perivascular spaces, and the pericellular sacs depends on the connective tissue cells of the neuroglia, which are distributed along the adventitia of the arterioles, and on to the arterio-venous capillary plexus, and thus establish a connection between the sacs and the spaces.

We are able to trace in successfully prepared sections, in close proximity to every nerve cell a branch of a capillary vessel, and it is to the sides of this loop that the pericellular sac is attached, the nerve cell being apparently suspended in the sac by means of its processes, which, passing through the free space of the sac proceed in all directions into the neuroglia. As these sacs are never terminal the nerve cell is bathed in a constantly renewed lymph bath.

In addition to these perivascular spaces and pericellular sacs, Bevan Lewis (37) describes a terminal set of lymphatic elements which consist "of a system of plasmatic cells with numerous prolongations, which are always in intimate connection with the adventitial lymph space, and which drain the areas between the vascular branches, these we have termed the lymph connective elements."

These four elements, all in direct anatomical continuity with one another, go to make up the cerebro-spinal fluid system of the brain.

Here I may remark that the fluid surrounding the brain is in direct continuity with that around the spinal cord, and that its transit from one cavity to the other is a comparatively easy matter; while the intra-ventricular and sub-arachnoid fluids communicate directly only through a very small aperture in the region of the fourth ventricle—the foramen of Magendie (35).

ON DURET'S THEORY OF CEREBRAL CONCUSSION.

The research which led M. Duret to formulate the cerebro-spinal fluid theory of concussion was carried out in the laboratory of M. Tillaux, and the result published in 1878.

In the first of his experiments he forcibly injected into

the cavity of the cranium of a dog, through a small trephine opening, about 300 grammes of water. After severe spasms of the limbs, opisthotonos, and cardiac and respiratory troubles, the animal speedily died, and the brain presented quite a series of very severe lesions, such as a rupture in the floor of the fourth ventricle, tears in the substance of the pons and medulla, hæmorrhages into the restiform bodies, third ventricle, and on to the surface of the brain.

Believing that this enormous destruction of brain tissue might be due to the increased pressure produced in the cranial cavity, and especially in the ventricles, by the large quantity of fluid so rapidly injected, he varied his next experiment by using a coagulable fluid, namely, gelatine, injecting it very forcibly between the dura and the skull. After exhibiting symptoms similar to the last animal, only somewhat more exaggerated, death followed in a few seconds. The lesions corresponded in a general way to those in the previous case, but were on the whole more severe.

These lesions M. Duret believes are all due to the sudden forcible pressing of the fluid of the lateral ventricles backwards through the aqueduct of Sylvius on to the fourth ventricle, and thence to the cord. The fluid in the lateral ventricles being five or six times more than the fourth ventricle can possibly contain, the latter cavity is subjected to such enormous pressure that its walls are torn.

On comparing these results with my own, produced by the less artificial means of administering blows to the surface of the skull, one cannot but observe the remarkable correspondence in the two series, not only as regards the symptoms shown, but also in the nature and distribution of the lesions observed after death.

It is, however, to be observed that in the injection experiments the symptoms and lesions are alike much more serious than in the percussion series—doubtless on account of the greater force employed, and the more direct way in which it is applied to the brain and its contained fluids.

But, however similar the results in the two cases may be, I cannot admit that the means of producing these are so nearly comparable as M. Duret would appear to indicate. In

the one case the force acting on the head is momentary in its duration, and the cranial contents could, but for any lesions produced by the blow, readily resume their normal conditions and continue to act as before. In the other series, however, the material injected necessarily remains, and so profoundly alters the physical and physiological conditions of the nervous centres that a return to their *status quo*, even supposing no lesion had been produced, would be absolutely impossible. In this, therefore, I think Duret's experiments are much better adapted to the elucidation of the symptoms and pathological appearances of *compression* than of *concussion*. At the same time, they have served to draw attention to the mechanism by which concussion symptoms and lesions—for I think we must admit there are lesions in concussion—are produced, and it is to the confirmation of these observations that I desire to direct my attention, by experiments performed more in accordance with the conditions under which our patients receive their injuries, and therefore more likely to lead to a useful clinical appreciation of the at present obscure symptoms, more obscure pathology, and still more obscure remote prognosis in cases of severe head injuries (48).

It is undoubtedly exceedingly difficult to distinguish clinically between Concussion, Compression and Contusion of the brain, and even after the most careful examination of the symptoms in an individual case, even experienced surgeons are unable to form a decided opinion. This is of course due to the fact that these pathological entities seldom exist singly—a blow which is serious enough to produce marked symptoms of concussion, is usually sufficient to cause gross lesion of the membranes, giving rise to hæmorrhage enough to cause symptoms of compression, which take the place of those of concussion as they tend to pass off. Or should the membranes and their vessels escape laceration, the nervous substance itself suffers injury enough to permit of blood being extravasated, and so raising difficulties in the diagnosis. M. Duret in speaking of this difficulty speaks of “doubt, which has not ceased after the autopsy.”

ON THE MECHANISM OF CRANIAL AND CEREBRAL INJURIES.

With these preliminary observations we may proceed to consider how severe blows and other injuries to the head produce their effects.

"Cone of Depression," and "Cone of Bulging."

It has been shown by Félizet (44) that the skull is elastic in a high degree, so much so, that it will rebound from the ground to a considerable extent if dropped from a height.

By smearing the vault of the skull with ink, and then dropping it from a height on to the floor, and comparing the size of the stain left with the area of rounded skull which could be in contact with the flat floor at one moment, he found that the former exceeded the latter in size. This could only be accounted for by the area around the point of contact being momentarily increased by the weight of the skull.

In another series of observations the skull was filled with soft paraffine, and then allowed to fall forcibly on to the ground, and it was found that a large depression was left on the paraffine, exceeding in size the possible area of contact, being distinctly rounded in outline and deeper in the centre than at the margins.

This circle of depression, or as M. Duret more accurately describes it, this "cone of depression," represents the extent to which the cranial parieties are depressed short of being fractured.

In order to account for certain phenomena observed in cerebral traumatism, Duret states that at the opposite pole of the axis of percussion, the vault of the cranium presents a corresponding "cone of bulging." He does not, however, state whether he merely assumes this cone of bulging, or whether he has demonstrated it. If the latter, he does not describe the method by which he has done so, nor does he quote the experiments of others bearing on the point.

Experiments on Production of Cone of Bulging.

As I believe this to be a most important step in his line of argument, I have made a number of experiments to satisfy myself as to the accuracy of his statement.

These I did on the cadaver as soon after death as I could obtain an opportunity, so that the physical conditions might be as nearly similar to those existing during life as possible.

I obtained a quantity of glazier's putty, of such consistence that it could be handled without losing an impression made on it, and having oiled the skin to prevent too close adhesion, spread a pretty thick layer over one or other frontal region. I then marked out what I judged to be the opposite pole of an axis of percussion, and dealt a sharp blow over that spot with a wooden mallet.

In a majority of the cases a distinct impression was left on the putty. I varied the position in which I dealt the blows, with similar results.

In another set of experiments with the same object, I spread a smooth layer of vaseline about a line thick over the forehead and then dealt a smart blow over the opposite pole.

In some, although not in all cases, a clearly marked ring could be seen on the surface of the vaseline, standing out very much as butter stands out from bread when two surfaces have been separated. In one of the cases in which no such ring could be detected, the head had come off the mallet, and the force thereby misspent; in the other negative cases the only explanations I can suggest are (1) The blow not being strong enough to depress the skull at the point of contact, and (2) the blow being too diffuse to form a defined "cone of bulging."

The results, however, seem to confirm what Duret had assumed (or proved), and are, I think, of importance in so far as they help to explain the mechanism of lesions so often found at the opposite pole of the axis of percussion, in head injuries—that is, at the so-called point of *contre-coup*.

In view of these facts, I take it that the mechanism of

an injury to the head producing gross lesions, is, briefly, this :—

A blow is dealt to the cranial wall, and surrounding the point of impact the skull is depressed, forming the “cone of depression,” and from the area into which this cone projects the cerebro-spinal fluid is forcibly expressed. Where does this fluid go? We have seen that under such conditions, at the opposite pole of the axis of percussion, there is formed a “cone of bulging,” which would accommodate all or most of this displaced fluid. What does not find accommodation there will either pass along the perivascular lymph spaces into the brain substance, along the nerve sheaths, or into the subarachnoid space of the cord

The cones of depression and bulging, however, are only of momentary existence, the elasticity of the skull permitting it at once to return to its *status quo*. Therefore, at each point there is formed a vacuum, first at the cone of depression when the bone recoils, and next at the cone of bulging when the fluid recedes. The result of this is, that the blood-vessels of the membranes and brain substance at these points are left for the moment unsupported, and they rupture, a result which is favoured by the fact that the compression to which they have been subjected has partially paralysed them.

For this rupture of vessels suddenly deprived of support derived from fluid under tension we have several surgical analogies—notably where, after withdrawing all the fluid from an over-distended bladder, we have hæmorrhage from the vessels of the lining membrane, or it may be even from the kidney, if the backward pressure has been sufficient to affect that organ.

Such a mechanism would explain the occurrence of lesions, not only at the seat of injury, but at the opposite point of the skull and brain, the so-called lesions of *contre-coup*.

ON “CONTRE-COUP.”

That this term *contre-coup* has become a most useful one clinically there is no doubt, but it is by no means so certain

that it has as satisfactory a pathological significance ; and it is still more doubtful if those by whom it is used always entertain any very definite idea or theory as to what it means.

Mr. Erichsen (45) says that "the explanation . . . is, that the blow starts a wave in the soft cerebral substance, which breaks against the bone on the other side. In very rare cases it is possible to trace the course of this wave by hæmorrhages in the cerebral substance in its track." We have already seen that Gama (23) and Alquié deny that the brain elements can be thrown into vibrations by a force applied to the surface of the skull.

Le Gros Clark (6) denies the admissibility of the term *contre-coup* in the physics of the cranium at all. He points out with some considerable show of reason, that while a series of vibrations set up at a particular point in the circumference of a perfectly spherical glass globe, might find a point of convergence opposite the point of impact, such a result is physically impossible in the skull. When we consider its irregular shape, its varying thickness, the projections externally and internally, the numerous muscular attachments on its outer aspect, the adhesion of the dura mater and its processes on its inner, and the presence of the cerebro-spinal fluid—all of which would either absorb or transmit in different directions the vibrations set up, we see how exceedingly improbable it is that these vibrations would ever find a point of convergence, and especially one situated exactly at the opposite pole of the axis of percussion.

Although we are not here specially concerned with the question of fracture of the skull from *contre-coup*, it bears so close a relation to what we have just considered, that we may glance at it in passing. That it cannot be due to vibrations finding a focal point at the seat of fracture is, I think, beyond doubt, and it is scarcely more likely that the force of the soft resilient brain impinging on the hard, elastic skull could cause fracture of the latter.

In spite of the strong opinion of Mr. Erichsen, who "cannot doubt it," as he has "seen unequivocal instances," I think Mr. Le Gros Clark is nearer the truth when he says

that "a blow on one side of the head could only produce a fracture on the other side, if the head were knocked against an unyielding object," and in a great majority of cases of severe head injury the patient does fall, usually in the direction of the blow, thus providing an explanation such as Clark suggests.

While I believe that most of the so-called fractures by *contre-coup* are produced in this way, I think that in the light of the experiments on the "cone of bulging," others may be explained on the theory that this cone has gone beyond a point from which it could recoil and the bone has given way. After breaking, the skull might resume its previous level and the only evidence be a fissure; or, as in the rare cases quoted by Mr. Walsham (46), the fractured bone raised above the level of the skull constitutes what is called an "elevated fracture." A specimen of this condition is in the Museum of St. Bartholomew's Hospital, No. 879a.

Fracture of the base by a fall on the vertex is often referred to as an example of genuine fracture by *contre-coup*, but as Le Gros Clark points out, this is really a fracture by direct violence, the weight of the body being transmitted through the spinal column, and impinging on the occipital condyles. This view is borne out by the fact that very often the basal fracture is much greater than that of the vault, being therefore presumably the primary lesion, and in addition the fracture of the vault often appears to be spread from the base.

If we accept this theory of *contre-coup* lesions—namely, that they are due to the displacement of the cerebro-spinal fluid by the cone of depression, and its sudden recession from the cone of bulging leading to rupture of blood vessels, and consequent contusion and laceration of brain substance,—it will be evident that these lesions will vary in position according to the point struck.

If the frontal region or vertex be the seat of the cone of depression, it will be at the base of the skull that we must look for the evidences of the cone of bulging. We can scarcely expect, however, that any ordinary violence will cause an outward bulging of the body of the sphenoid bone,

and we have already seen that when it is fractured it is not from violence applied to the vertex, but to the superincumbent weight of the body. The increased quantity of fluid therefore must swell the volume of the cerebro-spinal lakes at the base, and thus the force will be transmitted to the nerve elements themselves. That all fatal cases of concussion are associated with peribulbar lesions was stated by M. Fano in 1853, and with him in a general way Duret agrees. The results of my own experiments, which will be detailed later, have been to confirm these observations.

The late Mr. Vincent, quoted by Spence (47), evidently appreciated this element of the transmission of blows to the base of the brain, because he states as his opinion, that the amount of danger in cases of concussion depends largely on the direction, as well as the degree of the force, being greatest when in a line directed towards the base of the brain.

Mr. Bryant (48) also, by clinical observations, bears out the opinions of the various experimenters named, when he says that "in almost all cases of severe concussion the base of the brain is injured."

EXPERIMENTAL EVIDENCE IN FAVOUR OF CEREBRO-SPINAL FLUID WAVE THEORY.

After a blow to the head, in addition to the wave set up in the cerebro-spinal fluid external to the brain, *i.e.*, in the sub-arachnoid space, there is another and perhaps a more important disturbance, in the intra-ventricular fluid.

When the cortical substance of the hemispheres is pressed upon, it finds in the ventricles and their contents a sort of buffer which enables it to withstand shocks in a manner which would be impossible were each hemisphere a solid mass of brain substance.

Now, let us suppose the head is the subject of a severe blow, say over the frontal bone, a cone of depression is formed, which displaces the sub-arachnoid fluid in this region, and by the mechanism which we have already considered, a laceration of the brain or its membranes may take place. Not only so, but the force passing through the frontal lobe sets the fluid contained in the lateral ventricles into motion

in the direction of the axis of percussion, *i.e.*, towards the base of the brain. Here, however, there is considerable difficulty, because the only point of escapement is through the narrow aqueduct of Sylvius, and through this the fluid rushes, the effect on the aqueduct and its boundaries varying with the force employed, and its direction. When the force is great, the aqueduct may be torn up and lacerated, while in less severe cases it is only stretched. Whichever condition exists, after travelling the narrow aqueduct the fluid impinges with increased force on the wider fourth ventricle, which, as Duret puts it, "forms a cone of bulging ready prepared," with the result that the pressure on this most important locality is enormously increased, not only by the surplus quantity of fluid thrown in, but by the difficulty with which it can escape, there being only the minute central canal of the cord, and the scarcely larger foramen of Magendie to provide for the overflow. Under such circumstances we are not astonished to find primary lacerations of these delicate structures.

We have seen that the lesions on the surface of the hemispheres are in all probability due rather to the aspiration of the receding cerebro-spinal fluid, than to the pressure of the impinging wave, and I believe it is so too, in the ventricles and at the bulb. It is not uncommon to find, as I shall be able to show from my experiments, that the lateral and fourth ventricles are the seat of extensive extravasations, due, I consider, to the rupture of the unsupported vessels of the choroid plexuses during the ebb of the fluid wave.

In order to determine if the absence of the cerebro-spinal fluid would lead to rupture of these vessels and consequent hæmorrhage, I made several experiments by aspirating it.

These I shall proceed to describe.

Experiment 1, Rabbit H.—To determine the effect of rapid aspiration of cerebro-spinal fluid on the vessels of the brain substance, especially those of choroid plexuses.

November 21st, 1890, noon.—The rabbit, an adult male of average size, and in good condition, having been brought thoroughly under chloroform, an opening was made through the occipito-

atloid ligament, and the finest needle of an aspirator passed in. The trocar was then withdrawn, the cannula connected with the vacuum, and the cerebro-spinal fluid *rapidly* aspirated. Between two and three drachms were removed. During the aspiration the animal once had several slight spasms of the facial and limb muscles. These were so slight that at the time they were regarded as probably simply contractions of the panniculus muscle, and that they were due to reflex irritation of the left testiform body, which might have been irritated by the point of the needle, which was turned to that side after the membrane was perforated, and was felt to touch something. The cannula being withdrawn, the puncture was covered with collodion and wool. Antiseptics were used at the operation.

After aspiration the animal lay on its right side evidently in a perfectly unconscious condition. Pulse feeble and slow, breathing very shallow and rapid. No other sign of life was given. Loud sounds made close to the ears did not startle the creature.

Notes.—This moribund appearance has continued for an hour. General condition now improving. Pulse better, respiration normal. Both pupils dilated equally. The muscles of the right side of the neck are tonically contracted, forcibly bending the head to that side. All the muscles of the limbs seem to share in this tonic rigidity, though to a less extent than those of the neck. On the other hand, the muscles of the trunk appear to be paralysed, or at least in a state of paresis, because the animal is quite unable to move its body from the ground, although it can move about. In doing so, it exhibits an amount of inco-ordination of the limb movements. It now gives evidence of seeing, and of hearing loud sounds. Died soon after.

Autopsy.—As in life head forcibly turned to the right and dorsiflexed.

Brain.—Hemispheres look pink and congested; vessels on left side, both above and below, distinctly more engorged and prominent than those on right side. No hæmorrhage on surface.

Cerebellum congested in same way as cerebrum. Just over the cerebellum, in the space between the posterior ends of the two hemispheres is a small blood clot about size of split pea.

Interior of Brain:—On opening up hemispheres large clot found filling up the left lateral ventricle, and resting on upper aspect of basal ganglia and corpora quadrigemina of that side. The small clot seen above the cerebellum is found to be the termination of this one. Similar clot but much smaller found in right lateral ventricle.

Bulb.—Over left side of medulla in region of superior olive and restiform body is a small laceration, probably caused by the needle in aspirating. Near lower end of medulla, towards middle line and extending to left side is a hæmorrhage into the substance of the part. It extends along the medulla for about one-eighth of an inch, is irregularly rounded in shape, and has a piece of apparently normal brain tissue in the centre.

The aqueduct of Sylvius is dilated and exhibits one or two small hæmorrhages round it, the largest being in the left side of the corpora quadrigemina.

Cord is very pale and anæmic.

Remarks.—This experiment appears to bear out the theory that the sudden withdrawal of the cerebro-spinal fluid, and the consequent leaving of the vessels unsupported, leads to rupture of the latter and extravasation of blood into the brain substance (*e.g.*, the medullary hæmorrhage) and into the ventricular cavities. It also shows that the rushing of fluid through the aqueduct of Sylvius not only leads to dilatation of that passage but also to hæmorrhage into the comparatively dense tissues which surround it. Of course it may be suggested that the hæmorrhages were due to the aspirating force acting on the blood vessels after all the fluid had been withdrawn. I tried to guard against this fallacy by limiting the power of the vacuum, only partially exhausting the bottle. Besides, one's clinical experience in the use of the aspirator does not give much support to this view.

The symptoms due to the actual withdrawal of the fluid were neither numerous nor important, the animal of course being deeply anæsthetised. I have already said that the slight spasms, if such they could be called, were probably reflex.

The coma, rigidity of the limbs, with deviation of the head to one side, and slight variation in these irritative indications, are to be accounted for by the hæmorrhage into the left lateral ventricle, though why this hæmorrhage should have been more abundant on one side is not so easy to understand, unless, indeed, it be that the turning of the needle point to that side determined the direction of the flow. This we would not expect, because the needle should

have been in the inferior cerebellar lake, and the fluid to reach it would have to traverse the foramen of Magendie, where any inequality in the streams from the right and left ventricles would be equalised. The fluid of course may have escaped from the lateral opening of Key and Retzius. However, working in such a limited space and with such small quantities of fluid, the sources of error are so numerous, that we must not draw too wide conclusions from a single case.

To the hæmorrhages in the corpora quadrigemina we may assign but few symptoms. Abnormalities in the action of the ocular muscles often depend on lesions of these bodies, but I think the general comatose condition of the animal is amply sufficient to account for the phenomena in this case.

The peculiar inco-ordination of movement is explicable on one of two theories. We know that in certain cases of tumour of these bodies this is a prominent symptom, but Dr. Gowers (49) has pointed out that it is probably due to the pressure on the middle lobe of the cerebellum, rather than to any influence on the corpora quadrigemina. Here we had both hæmorrhage into the corpora quadrigemina and pressure, though slight, on the middle lobe of the cerebellum by the posterior end of the intra-ventricular clot. Probably both played a part.

The condition of the pulse and respiration most likely depended on the large hæmorrhage into the substance of the medulla, caused I believe by the recession of the fluid supporting the blood vessels.

The excessive congestion of the vessels of the left hemisphere, doubtless was due to the increased difficulty in the return of the venous blood, on account of the large clot blocking the vessels of the choroid plexus, and secondarily all the vascular arrangements of that side.

So far as it goes, I think this experiment is satisfactory in showing that by rapidly withdrawing the cerebro-spinal fluid, hæmorrhage can be produced in the cavities and substance of the brain.

The next points to determine are, what is the effect of

slow aspiration of the fluid; and what is the result of a severe blow on the head when the cavity has been deprived of its cerebro-spinal fluid.

The following experiment fortunately, throws light on both of these points.

Experiment 2, Rabbit I.—To determine the rôle of the cerebro-spinal fluid in the production of lesions from external injuries to the head.

November 21st, 1890, 1 p.m.—The subject of the experiment was an adult male Belgian hare, well fed and not very timid. While deeply under chloroform, an opening was made in the skull by means of a small trephine over the left prefrontal lobe, and through this the cerebro-spinal fluid was slowly aspirated, to prevent a too sudden withdrawal leading to rupture of the vessels, as occurred in Rabbit H. Only about two drams of fluid were obtained and this was slightly blood stained.

During the withdrawal the animal spasmodically jerked its limbs, in the same way as, but to a less extent than the animal last operated on.

While still under the influence of chloroform, a severe blow was dealt over the frontal region in a direction downwards and backwards, in a direct line towards the base of the skull.

The animal was thrown into a state of extreme opisthotonos with tonic contractions of all the limbs, lasting for a few seconds. This passed off, leaving only a distinct paralysis of the right fore limb. It was lying on its side. Some slight symptoms of compression gradually supervened but did not become serious. But for some drowsiness and deafness the animal seemed to recover completely, and was ultimately killed by chloroform inhalation.

Autopsy.—*Scalp.*—The incision in the scalp had healed by first intention. There was no ecchymosis into the scalp in the region of the blow. *Skull.*—The trephine opening was found to be over the left frontal lobe about the level of the anterior edge of the orbit. It was filled by a small mass of purulent looking material. No fracture of vault, and no contusion of pericranium. At the base there was a transverse fracture right through the body of the sphenoid bone, which permitted a bristle to be easily passed through into the pharynx.

Membranes.—Between the dura and the bone on the right side, there was a thin clot about the size of a split pea, situated on the upper aspect of the orbital plate. This clot was partly decolorised.

Hemispheres.—Just beneath the seat of the trephine opening, *i.e.*, over the left olfactory lobe, and inside the dura was a small clot, presumably due to the needle having injured the brain. The whole upper aspect was normal, and the lower paler than normal. No evidence whatever of blood clot either old or recent.

Cerebellum.—Normal.

Interior of Brain and Ventricles.—Normal. In the aqueduct of Sylvius was what appeared to be a small decolorised clot.

Bulb and Spinal Cord.—In the upper part of the spinal canal there had been some effusion outside the membranes, but it had been small in quantity, and by the time of death was nearly all absorbed.

Eyes.—There was no extravasation into the sheath of either optic nerve, and no subconjunctival ecchymosis.

Ears.—*Right*: Large red clot filling up middle ear. Membrane ruptured. *Left*: Partly decolorised clot in middle ear. Canals normal. Membrane ruptured.

Remarks.—On comparing this experiment with the last, we see that although in both the cerebro-spinal fluid was aspirated, the results differ considerably, and I attribute this to the varying rapidity with which the fluid was withdrawn. In rabbit H the fluid supporting the blood-vessels was rapidly withdrawn, while in this case it was slowly removed by gentle aspiration, thus giving the vessels time to adapt themselves so as to compensate for their loss of support. This, I take it, bears out the theory that in concussion the hæmorrhages found in such widely separated parts of the brain are due to this rupture of vessels suddenly deprived of the support of the cerebro-spinal fluid.

That the rôle of the cerebro-spinal fluid is an all-important one in concussion is further borne out by the result of the blow given to the head after removal of the fluid. If we look at cases of rabbits, pigs and birds, in every way identical, save in this one particular, that the fluid has been withdrawn, we find that while in them abundant lesions in the shape of hæmorrhages on the surface of the hemispheres, bulb and cord, into the substance of the parts, and into the ventricles occur, it is only very rarely that we have fracture of the strong base of the skull. Here, however, the conditions are entirely

reversed, because while we can only find two hæmorrhages on the surface attributable to the blow, and both of these external to the dura, and the ear lesions, there is a complete fracture of the basi-sphenoid. Let me point out that this process lies directly in the line of the axis of percussion, and would therefore be at the seat of the "cone of bulging." The cerebro-spinal fluid not being available to protect it, the force has spent itself on the bone.

In spite of the absence of the fluid however, there were some hæmorrhages resulting from the blow, *e.g.*, hæmorrhages into both middle ears, with none into internal auditory meatus. These can be explained on the theory that they were secondary to the fracture of the base. It is to be noted that the other extravasations are all outside the dura, and therefore more likely to be due to fracture than to cerebro-spinal disturbance. The bleeding from the mouth and nose also were doubtless secondary to the basal fracture.

The evidences of deafness exhibited all along, are of course quite intelligible in the light of the *post-mortem* appearances.

The absence of extravasation into the sheaths of the optic and auditory nerves, is due to the want of the cerebro-spinal fluid. At the moment of percussion these perineural spaces were not dilated, and so their vessels were not put into the dangerous condition which leads to rupture.

As a contrast experiment to the above, let us look at that performed on Rabbit C. In this experiment the blow was administered on the occipital region, but in every other respect, save that the cerebro-spinal fluid was not withdrawn, the two cases are similar.

Experiment 3, Rabbit C.—To compare results of severe blow to head with cerebro-spinal fluid in situ, with those of previous experiments where it had been aspirated.

November 30th, 1890, 10.45 a.m.—The subject was an adult grey rabbit, not in very good condition, but quite active.

Before any blow was administered the animal sat quietly, its pupils were equal, pulse small, rapid, 180; respiration also rapid, 90.

Anæsthesia having been induced by chloroform, a severe blow, intended to be lethal, was dealt over the middle line in the

occipital region, with the knob of a Kaffir stick. Immediately the creature fell on its right side, all the limbs were spasmodically convulsed for a few seconds. The animal then rolled over and over around its long axis five or six times, and came to rest on its left side. While in this position all the limbs were making violent movements similar in character to those made by an animal galloping, only very much exaggerated. For the first few seconds the fore paws were rigidly extended, but they soon took on the same actions as the hind ones. The head was dorsiflexed, and the tail extended in a straight line with the body. The pupils were equally contracted. This condition lasted for two minutes, then the limbs assumed different positions; front limbs were rigidly extended, and if flexed passively they immediately assumed their former position. The right hind limb was in the same attitude, the rigidity, however, being less marked, while the left hind leg was flexed; the head remained dorsiflexed; pulse full, bounding 120; respiration 124, shallow, very irregular and intermittent, mainly costal and noisy, whistling through the nares.

The pupils subsequently dilated equally, and the limbs exhibited regular and rythmical movements as of walking. The heart became weaker, and the respiration more difficult, and the animal was killed by chloroform inhalation.

Autopsy.—*Scalp*: Large hæmatoma over occiput and extending down as far as level of third cervical vertebra. *Skull*: Depressed and comminuted fracture of the right occipital bone; no basal fracture.

Membranes.—Only slight hæmorrhage from fracture between dura and bone.

Hemispheres.—Under the dura over the left cerebral lobe is a large extravasation, consisting of several large clots with thinner clots between; a smaller clot over the right lobe.

Cerebellum.—Over the middle lobe of the cerebellum is a blood clot of considerable thickness. It extends on to both lateral lobes, and is found to be continuous with an intraventricular extravasation described below.

Interior of Hemispheres and Ventricles.—The left lateral ventricle is filled completely by a large clot which is found to be continuous with that lying on the posterior aspect of the occipital lobe, and on the cerebellum.

This same clot extends into the third ventricle. A clot of considerable size fills the cavity of the fourth ventricle, and on being removed exposes several smaller extravasations into the

substance of the floor of that cavity. The part of the cerebellum forming the roof of the fourth ventricle is pulpy and infiltrated with blood.

The Sylvian aqueduct is enormously dilated, almost sufficient to admit a toothpick. It is filled with clot. There is a small hæmorrhage into the substance of the anterior corpus quadrigeminum of left side.

No hæmorrhage into basal ganglia.

Bulb and Cord.—Various hæmorrhages on base of brain. There is a tear in the substance of the medulla on right side, but no visible hæmorrhages into substance beyond those mentioned. Some extravasation into upper part of spinal cord.

Eyes.—Well marked extravasation of blood into optic nerve sheaths of both eyes, extending backwards from the eyeballs for about half an inch.

Ears.—*Right*: Some extravasation into the first part of the auditory nerve sheath, in the internal auditory meatus. Canals and internal ear normal. *Left*: Numerous well marked points of ecchymosis into the membranous semi-circular canals, and also hæmorrhages into the middle and internal ear.

REMARKS.—We may compare in a general way the two experiments on rabbits I and C. In the former the cerebro-spinal fluid was aspirated before the blow was dealt, in the latter it was left, and the result of that blow is of the greatest possible interest, because, while in rabbit I the brain tissue practically escaped serious harm, in rabbit C the gross lesions found in the substance of the brain were so numerous, and so widely spread as to be spoken of without exaggeration as universal. On the other hand, in the case where the fluid was absent the base of the skull was fractured, while in the other it was uninjured. When we consider that the intensity of the blow in these two cases was as near as may be equal, the results of the experiments lend great support to the cerebro-spinal fluid theory of cerebral trauma.

If we now compare the result of experiment on rabbit C with that on rabbit H, in which the cerebro-spinal fluid was rapidly withdrawn, we find that in the two the symptoms during life and the *post-mortem* appearances, so closely correspond, that we must consider that in both, the lesions are produced by the same mechanism, a mechanism differ-

ing in the two cases only in the means by which it is started. With so many gross lesions any one of which might produce symptoms, it is useless to attempt either to assign a symptom to each lesion or to each symptom a lesion. Rather must we look upon the injuries to the brain tissue as an index of the severity of the blow, and not as the definable causes of the phenomena.

The presence of blood extravasations into (a) the sheath of the optic nerves; (b) the sheath of the auditory nerves; (c) the aqueduct of Sylvius; (d) the lateral, third and fourth ventricles, is of importance for my argument as showing the rôle of the cerebro-spinal fluid in the production of lesions in cases of concussion.

I have thus shown by experiment that sudden aspiration of cerebro-spinal fluid leads to extravasation of blood; that slow aspiration does not do so; that a blow on the skull after the fluid has been so removed causes much less destructive lesions than when that fluid has been left in its normal position.

THE RÔLE OF CEREBRO-SPINAL FLUID IN PRODUCING HÆMORRHAGE INTO NERVE SHEATHS.

I have noticed in several cases that, as a result of severe blows, there were found extravasations of blood into the sheaths of different cranial nerves, and this fact I think throws considerable light upon the mechanism of head injuries. It is an anatomical fact of some importance that the cranial nerves have a reflection of the arachnoid membrane accompanying them as far as their foramina of exit from the skull.

Axel Key and Retzius (32) have shown by their injection experiments that the coloured liquid can be made to penetrate into the lymphatic channels which Ranvier (50) has shewn to exist around the fasciculi of nerves, and Schwalbe (33) has demonstrated that these intra-nervous channels communicate with serous or lymphatic spaces of the organs of sense. He states that, "these intra-nervous, serous canals communicate with certain spaces in the organs of

special sense, which we have described by the name of serous spaces or lymphatics. Besides, by the internal auditory canal the serous canals of the auditory nerve are in communication with the spaces filled with peri-lymph contained between the osseous and membranous labyrinth; by the optic foramen the serous spaces of the lamina fusca which accompany the vessels of the choroid, and communicate with the anterior chamber of the eye, are in relation with the serous canals of the optic nerve, and with the central lake at the level of the chiasma; an analogous arrangement exists in connection with the olfactory nerves."

In this relation also, Duret announces this general law, that "wherever we have a nervous element and a blood vessel nourishing it, around the vessel is disposed a serous space destined to receive in the case of excessive tension in the latter the aqueous part of the blood which it contains."

This being so, it is evident that the wave set up in the cerebro-spinal fluid by a blow impinging on the skull, will be transmitted along these peri-vascular channels, and cause distension of them, which will result in rupture and extravasation when the fluid recedes.

It appeared to me that if this fluid could escape from the nerve sheath, and thus obviate its distension, such hæmorrhages would not occur, and to test this point I performed experiment 4.

Experiment 4, Rabbit F.—To determine the rôle of cerebro-spinal fluid in producing hæmorrhage into nerve-sheaths, especially those of optic nerves; and other points.

Subject, an adult male Belgian hare in good condition. Both pupils equal and of medium size before experiment. The animal was put fully under the influence of chloroform, and then the left eye was enucleated, the optic nerve being cut as cleanly, and as far from the eyeball as possible with curved scissors. The orbit was washed clear of all blood, and hæmorrhage stopped by plugging. The animal, still deeply anæsthetised, a severe blow intended to be lethal was delivered on the occiput, having a direction from behind forwards, as nearly as possible in the axis of the optic nerves.

Death was almost instantaneous, no other movement than a

slight twitching of the right hind leg taking place after the blow. The right pupil was much contracted after the injury was inflicted.

Autopsy.—*Scalp* : Slight ecchymosis under the skin over the occipital bone in middle line. No wound of scalp. *Cranium* : No fracture of vault or base.

Membranes.—No hæmorrhage external to dura. Under the dura over the right cerebral lobe was an extensive but very thin clot. None on left side.

Hemispheres.—Extensive hæmorrhages into the lateral ventricles on both sides.

Cerebellum.—Much injured, covered with blood clot, and on right side more or less pulpy, blood clot and brain tissue being mixed up.

Bulb.—Laceration of wall, and extravasation of blood around the aqueduct of Sylvius, and into substance of corpora quadrigemina. Large blood clot on front of pons and medulla pressing on the superficial origins of the vagi, spinal accessory, and hypoglossal nerves. The floor of fourth ventricle exhibited numerous small petechial hæmorrhages, scattered over it in all its areas. The left restiform body was covered by a large thick blood clot; a smaller one existed further down on same side.

Cord.—Extensive hæmorrhage extended down the cord as far as the upper lumbar region, especially on left side, but also marked on right.

Eyes.—*Right* : Pupil normal after death. There was a considerable extravasation of blood into the back of right orbit, a clot about size of a threepenny piece pressing on the branches of third nerve. Sheath of optic nerve filled with blood clot. *Left* : The optic nerve had been severed about one-eighth of an inch from the eyeball, and there was no blood around the stump. There was no hæmorrhage along its sheath whatever.

Ears.—No hæmorrhage or escape of cerebro-spinal fluid from either ear. Both membranes intact. *Right* : Some blood in the sheath of the nerve. A few spots in the semicircular canals. None in cochlea or middle ear. *Left* : No hæmorrhage along course of auditory nerve in internal meatus. Semicircular canals filled by a large extravasation.

Jaws.—*Right* : At the inferior dental foramen by which the branch of the third division of the fifth nerve enters the bone was a fairly large clot of blood in the sheath of the nerve. *Left* : One small hæmorrhage close to foramen, and another, a short distance higher up the nerve

Remarks.—There can be little doubt that the theory enunciated as to the origin of these hæmorrhages into the sheath of the optic nerve, was the correct one, because in the left eye the conditions were such that the cerebro-spinal fluid rushing along the sheath could escape without dilating it, and no hæmorrhage resulted; while on the right side the distension was followed by a copious hæmorrhage.

Not only in the sheaths of the optic nerves have we this recession of cerebro-spinal fluid, but also in the auditory nerves, fifth nerves, and probably in all the others the same thing happens, as is evidenced by the hæmorrhages found in these various positions.

Referring to the extravasations into the internal ears and along the course of the auditory nerves, one can easily understand how, if the tympanic membranes had been ruptured or previously perforated, a symptom supposed to be characteristic and diagnostic of fracture of the base of the skull, namely bleeding from the ears and “welling” of cerebro-spinal fluid, might have been present while no such fracture existed.

The lesions of the left side of the brain are much less marked than those of the right side, and it appears to me not improbable that this is due to a certain amount of the fluid finding a point of escape through the severed optic nerve sheath of that side, so diminishing the size of the wave set up by the blow.

SUMMARY.

Such are the grounds on which I am led to support and confirm this theory of the important part played by the cerebro-spinal fluid in the mechanism of cerebral concussion and other forms of cerebral trauma.

The conclusions I have formed are briefly these:—

(1) That the group of phenomena, commonly spoken of as “concussion of the brain,” is the result of a temporary anæmia of that organ.

(2) That this anæmia is the reflex result of stimulation of the restiform bodies, and perhaps other important centres in the region of the bulb.

(3) That these parts are stimulated by the wave of cerebro-spinal fluid, which rushes through the aqueduct of Sylvius, the foramen of Magendie, and from the subarachnoid space of the brain to that of the cord when a severe blow is dealt over the skull.

(4) That in accordance with the laws of hydrostatics this cerebro-spinal fluid wave will disturb the equilibrium of the ultimate nerve cells throughout the central nervous system.

(5) That the hæmorrhages found throughout the brain substance and on its surface are to be ascribed to the recession of the cerebro-spinal fluid, which naturally supports the blood vessels of the cerebrum.

(6) That the petechial hæmorrhages found in cases of so-called concussion are not the proximate cause of the symptoms of that condition. They are rather to be looked upon as an index of the force producing the injury, than as the cause of the resulting phenomena.

In another paper (48) I have described the microscopic appearances found in these and other cases of cerebral trauma, most of which, I feel sure, may be satisfactorily explained on the same theory as are the grosser lesions to which I have alone referred in the foregoing paper.

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