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CEREBRAL COMPRESSION

Its Physiological Basis AND Therapeutic Indications

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TO the practitioner who has made no special study of neurology, harrassed as he is by the never-ceasing duties of the daily round, the brain, as well in its physiological and anatomical as in its pathological manifestations, is apt to remain in some degree a *terra incognita*. And yet this ought not to be the case, at least as regards the interpretation of the ordinary cerebral lesions, because the essentials are, after all, relatively few. As a matter of fact, from the standpoint of interpretation, no system of the whole economy gives so uniform or so certain, a response to a given pathological lesion as does the nervous system. The trouble is that we seldom properly learn nervous physiology, which is the chief basis of nervous pathology. Symptoms have been taught us in too much of a catalogue fashion; we have taxed our memory too much and our reason too little. Most of us, for instance, when we graduated knew that a slow bounding pulse was a sign of cerebral compression; how many of us knew that this was due to a stimulation of the vagus and vaso-motor centres by lack of sufficient circulation, and that it was of the deepest prognostic import? Therefore, my object to-day is to resume briefly the results of the more recent work on this subject of compression, work which gives us a very solid basis of knowledge upon which to rest our judgement when we are confronted with a serious clinical condition.

From the anatomical standpoint the cranium is a rigid, closed box, as little capable (to use von Bergmann's expression) of expansion as of contraction, except in the infant. This box is completely filled by the brain with its membranes, the blood and the cerebro-spinal fluid. The cerebral substance, moreover, is incompresible—as incompressible, practically speaking, as water itself. Therefore, if any foreign body, such as a tumour or a blood clot, make room for itself inside the skull, the only elements that can give way are the blood and the cerebro-spinal fluid; and, as a matter of fact, the first result of compression of the brain is the escape of these two elements outside the cranial box, by way of the veins and the spinal canal.

The cerebral functions from the clinical point of view, may be divided into local and general. It has been customary to call "general" or "major" those which reside in the centres of the medulla, because of their being associated with the vital processes of the body, as opposed to the "local" signs which are called forth upon stimulation of localised areas of the brain. It is not my intention to discuss to-day the symptoms resulting from a lesion of any local cerebral territory. I may merely remind you that the Rolandic area limited to the precentral convolution, is concerned with motor responses; that the zone situated just behind the fissure of Rolando, together with the

rest of the parietal lobe has to do mainly with the sensory system in general; the occipital lobe with higher vision; Broca's convolution and the temporo-sphenoidal with the various forms of aphasia; and the pre-frontal lobe with the functions of the higher intellectual and psychic life.

These signs may or may not be present as part of cerebral compression according to whether the latter bear upon a limited area or be more or less generally transmitted. Their finer interpretation belongs rather to the neurologist than to the general practitioner. On the other hand it is much more important for the general practitioner to be conversant with the symptoms of general or major cerebral compression, that is the bulbar symptoms, inasmuch as in this case life itself may be in immediate danger.

I may here perhaps be allowed to recall briefly to your minds the three chief medullary centres situated in the floor of the fourth ventricle. Here we have the respiratory, the vagus or cardio-inhibitory, and the vaso-motor centres. Stimulation of the vagus centre slows the heart; stimulation of the respiratory centre deranges, or may temporarily paralyze respiratory movements; finally, stimulation of the vaso-motor centre raises general blood pressure by constricting the peripheral arteries. These are the few and simple facts which are of fundamental importance.

Now, let us turn for a moment to consider in a general way the clinical conditions which correspond to this division of compression into "local" and "general or bulbar." As examples of local compression we have tumour, cyst, abscess, hæmorrhage from the middle meningeal artery Remember, however, that any one of these local compressions may, if it become great enough, transmit its pressure throughout the skull to such a degree as to become general; that is, as to affect finally the vital centres in the medulla and call forth the "major" symptoms of compression. It is likewise evident that if the local compression be situated primarily in or near the bulb, it will produce these major symptoms first of all.

As examples of general compression, usually without local signs, we have basal hæmorrhage in fracture of the skull, acute hydrocephalus; acute cerebral œdema following concussion, and meningitis. The compression which these exert is general, because as you see it is usually fluid in character and therefore is more uniformily distributed over the cranial cavity and does not press upon any area in particular; or, if so, only over a "silent" area.

To-day we leave aside the question of local pressure, which would take us too far afield, and confine ourselves to the consideration of the manner in which general compression affects the vital centres in the medulla. You are all familiar, to take an example, with the fact that a man with a fracture of the skull is apt to lie comatose without localising symptoms, but with a slow, high-tension pulse, and stertorous irregular breathing. These are the effects of pressure exerted generally it is true, but acting in particular on the bulbar centres already mentioned. In what way, now, are these centres stimulated by the compression of the effused blood, a compression which is acting from a distance? It is not, we may say at the outset, by direct pressure, mechanically speaking; it is rather by expressing the blood and by preventing good blood

from reaching the bulbar centres; in other words, it is by anæmia. Only within the last few years have we gained the experimental proof of this fact. The labours of many investigators, and not the least those of England—I need only mention the names of Spencer and Horsley, Roy and Sherrington, Elder and Leonard Hill—have been tending in this direction for many years past; but the credit of proving it to a certainty is due to Dr. Harvey Cushing, of Baltimore, working in the laboratory of Professor Kocher in Berne. His work was briefly as follows:

He produced general cerebral compression in dogs, by inserting a cannula into the occipito-atlantal ligament and pouring in salt solution under a pressure which he could control and register in millimetres of mercury by means of a manometer. Simultaneously he registered the arterial pressure in the femoral, and finally, he placed a glass window in the skull through which he could observe the changes in the cerebral vessels.

It is impossible here to go into the minuter details, but briefly the sequence of events was as follows: and I here paraphase from an article by Cushing, published in the American Journal of Medical Sciences for October, 1902.

"What are now the circulatory changes observed? As the pressure rises the first change is that of a slight dilatation of the veins; the appearance of the smaller radicles, previously invisible, and the development of a distinct difference in color between veins and arteries. At the same time the longitudinal sinus (window in mid line) shows signs of narrowing, beginning usually at the posterior end. These changes are observed long before pressure is great enough to give signs of disturbance of the medullary circulation. Occasionally slight irritative phenomena on the part of the respiration and pulse-rate occur during this early stage, but there is no effect on blood pressure and if the compression is exerted slowly and carefully these may be avoided.

When the pressure has been brought up to near the blood pressure we find the longitudinal sinus collapsed, the brain clearly in a condition of stagnation, or the veins filled and of a deep blue colour.* According to the views of some, such a degree of circulatory stagnation shou'd be sufficient to cause marked symptoms; yet, as the curves show, there need be no change in pulse, respiration, or blood pressure. Further it has been assumed that ultimately, with increasing compression, a complete anæmia of the medulla results, calling out the major symptoms of compression and causing death. This is erroneous. What occurs is this: as soon as the blood pressure is exceeded by the compression pressure, and indeed exactly at that moment, the observer sees through the window that the brain grows pale; i e. the capillaries are emptied, and even the visible arterioles also, while the veins remain full of stagnant blood." This anæmia, however, lasts but a short while Why? Because now the blood pressure rises and rises so as to get again above the compression level. Thus the blood is driven through again, the arteries become visible, and the reddish color returns to the brain. Now if we again increase the compression the same movement on the part of the blood pressure is repeated. In this way Cushing was able to drive blood pressure up to comparatively enormous heights. In one case he pushed

*This condition, by the way, one frequently sees clinically during operations in cases in which cerebral compression is marked.

intracranial pressure up to 276 mm. Hg. and the blood pressure to 290, before this regulatory mechanism failed.

Moreover, it was found that a high compression pressure, if kept below blood pressure, could be borne by the brain for a long time, an hour or more. And clinically we doubt not but that a similar condition of moderate interference with medullary circulation can be borne for days (as in hemiplegia.)

Now we have here evidence of a sort of regulatory or protective mechanism, designed to overcome the effects of cerebral anæmia. It is plain that the vaso-motor centre is the essential factor in raising blood pressure. Therefore the struggle lies between the compressing force and the vaso-motor centre, and, as you see, it is a struggle for life or death. The compression causes anæmia of the bulb, anæmia is a stimulus to the vaso-motor centre; it responds by driving blood pressure higher than the compression pressure. The latter responds, and so it goes on till one or the other becomes exhausted and gives up.

In this way Cushing felt justified in formulating a law to this effect: "An increase of intra-cranial pressure above blood pressure causes a rise of the latter to a point somewhat above that of the former; moreover, this regulatory mechanism is due to the action of the vaso-motor centre and is brought about only by the condition of anæmia."

During this struggle for life or death the respiratory centre is also affected. It however is not stimulated by lack of blood, it is rather paralyzed. And so we find that breathing is apt to become shallow and slow; in the experiment it frequently ceases entirely until the vaso-motor centre has succeeded in supplying it again with blood. Its stimulus is poor blood containing CO_2, and in the earlier stages of compression where there is considerable venous stagnation we get a deepened respiration, as we see in some cases of cerebral hæmorrhage. The alteration in rhythm called Cheyne-Stokes respiration, is easily explained by these experiments. At those stages in the curves where the blood pressure is below compression pressure, and when, consequently, the bulb is anæmic, the respiration may cease almost entirely, but as blood pressure rises and the respiratory centre receives blood again, breathing recommences, and we get a series of respirations, until the rise of the pressure again deprives the bulb of blood, and respiration again ceases. This may go on for a long time even when the compressing force does not increase, because when compression is at a certain height and is kept there, we find that the vaso-motor centre develops a rhythmic activity which calls forth waves, the so-called Traube-Hering waves. At the bottom of the wave respiration ceases; at the top it is resumed; it is all dependent on the amount of blood the bulb receives. Of course clinically, in cerebral compression we frequently get only modifications of the Cheyne-Stokes type; often the respiration is only slowed, or made less deep or irregular, without any rhythmic alterations. These lesser degrees are nevertheless very important to observe because they confirm the diagnosis of severe compression. The slow pulse, that sign of cerebral compression which is the most relied on clinically, is perhaps the least reliable of the three; its cause is the same, a bulbar anæmia which stimulates the vagus centre

and thereby slows the pulse. There is, however, less regularity in the appearance of this sign than in that of the rise of the blood pressure.

Suppose now that the compressing force be gradually increased, what happens? Ultimately the vaso-motor centre becomes exhausted and has to surrender. With its surrender blood pressure begins to fall; and usually the fall is rapid. The bulb is no longer supplied with sufficient blood; the respiration becomes slower and slower, and finally ceases. The vagus centre is paralyzed, and the slow pulse changes to a rapid one. The end comes, first by arrest of respiration, this being followed for a little while by a rapid, weak pulse, which in its turn finally ceases. But I would remind you that the vaso-motor centre is the key to the situation. It has to fail before the other two fail. It is the rear-guard in a retreating action; and if it is overcome the army is routed and devoted to slaughter.

Now let us briefly apply this knowledge to the clinical conditions. Upon this basis Kocher has divided the ordinary course of an advancing cerebral compression, as for instance a fatal case of apoplexy or of fracture of the skull, into four stages. It is understood that these are by no means arbitrary:

First:—The stage of compensation. The compression is mild and is compensated for by expression of blood and cerebro-spinal fluid. There are practically no symptoms.

Second:—The stage of beginning manifest compression. Here there is present a venous stasis with difficulty in the passage of the blood through the skull. It is characterized by headache, vertigo, restlessness, noises in the ears, disturbed sensorium, with excitement or delirium. Above all it may be seen in the fundus oculi, where the ophthalmoscope shows dilatation and tortuosity of the veins of the papilla; there may be some slowing of the pulse and some rise in the blood pressure.

Third:—The stage of the acme of manifest compression. This is characterized by alternations between total lack of circulation in the brain and a good circulation. It is the stage of active struggle between the vaso-motor centre and the increasing compressing force; the blood pressure is rising constantly. It may last an indefinite time until relief comes or the vaso-motor centre gives up. There are marked respiratory disturbances, especially if the blood pressure is rhythmic, also rhythmic alterations in the size of the pupils, and varieties in the depth of stupor. The slow, high-tension pulse is marked, but usually not rhythmic. If the vaso-motor centre finally gives up we get—

Fourth:—The paralytic stage. Here are alternations between total lack of circulation and an insufficient circulation in the bulb. We get falling blood pressure, irregular cardiac and respiratory efforts, a pulse getting more and more rapid and weaker, deep coma, complete flaccidity of the muscles, wide pupils, broken snoring respiration which grows slower and slower until death ensues. It is a condition of irrecoverable cerebral anæmia.

Gentlemen, I shall have succeeded in my object if I have impressed on you one thing in particular—the predominating influence of the vaso-motor centre and the importance of estimating the blood pressure, which is the outward evidence of the work which that centre is accomplishing.

To base a prognosis is to estimate accurately how near the vaso-motor centre is to exhaustion or how far from it; and for that we must rely chiefly on the degree of blood pressure in the peripheral arteries.

It is naturally impossible to open, in the human, a blood vessel and take the blood pressure as in physiological experiments. To fill this lacuna various instruments have been devised for the recording of blood pressure clinically. The most generally useful of these I show you here. It is the Riva Rocci, modified by Cook, of Baltimore. As you see it is composed of an upright tube graduated in millimeters for the recording of pressure. Attached to this is a rubber tube connected by a T-piece with this hollow band of rubber which is bound round the patient's arm. The other end is attached to a rubber bulb which blows air into the armlet and so gradually obliterates the patient's artery, at the same time driving the Hg. up in the glass tube. The pulse being once obliterated, one lets the air gradually escape. The pressure on the artery and on the Hg. decreases equally. At the moment when one first feels the return of the pulse, the degree of pressure on the glass tube in mm. of Hg. is read off. Normally, it is from 110 to 130 mm. In cases of severe cerebral compression I have found it as high as 280; it may go above 300. This indicates the enormous power of the vaso-motor centre.

It must be remembered that the arteries of the brain have no vaso-constrictor nerves; the vaso-constrictor action and the rise in arterial tension are confined to the arteries of the rest of the body, and it has been proved that the chief gain is got by the constriction of the splanchnic arterioles; less blood going there provides more for the brain.

The use then of this instrument is simply this; it tells us approximately to how great a degree the vaso-motor centre is being compressed; the height of blood pressure corresponds directly with the degree of compression. The interpretation of course is a matter of clinical experience. In a general way if the instrument records over 200 Hg. mm. the compression is decidedly dangerous; I have seen a case go on to death with the pressure no higher than 180. Naturally this is rather a matter of surgical judgment than of rule of thumb, and must be based upon a consideration of the case as a whole. Absolute figures are always a delusion and a snare if one trust to them blindly. Of course surgeons in the past have not been quite without recourse upon this point. They have roughly estimated the pulse tension with the finger. But this has been proved to be very unreliable as compared with this instrument. I am decidedly of the opinion that this instrument should be regularly used in all head cases in hospitals at intervals of half an hour or less in critical cases. Not only so, but it should be used by the general practitioner, just as he uses the larvngoscope or otoscope, or any other special instrument. Its cost is small, about $8.00, and its use is simple. Lives may be saved by it. If, for instance, the pressure at a first visit is 140 mm. Hg., at a second 160, at a third 180 or 200, action is urgently indicated.

Such is in brief the physiological basis upon which we must judge our cases; the bulbar signs are those which indicate danger to life, because in the bulb are situated the vital centres. Therefore, it is that they are of commanding importance in prog-

nosis. Although it is plainly the cases of acute traumatic compression with basal hæmorrhage that most closely resemble the experimental conditions, yet one frequently gets the same bulbar signs in the terminal stages of brain tumour, and they give then a very dark prognosis and warn to immediate operation if at all possible.

[At this point, Dr. Archibald onstrated a number of charts illustrative of the blood-pressure reaction in cases of cerebral compression. The first, reproduced from Kocher's volume in Nothnagel's System, showed a tracing of one of Harvey Cushing's experiments, and illustrated the effectual response of the vaso-motor centre in driving up blood-pressure to overcome the rise of compression pressure, which latter was not pushed to a fatal height; also the Traube - Hering blood - pressure waves with their graphic explanation of rhythmic or Cheyne-Stokes breathing; and the gradual descent of blood pressure upon release of the compressing force.

The second * illustrated the mode of death in acute cerebral compression by failure of the vaso-motor centre. A jockey thrown from his horse at 5 p. m., was brought to the Royal Victoria hospital quite unconscious. No definite localizing signs. At 8.30 p. m., pulse 40; at 8.40 blood-pressure 190 mm Hg; at 9, pulse 57; at 9.05 pulse 64; at 9.15, blood-pressure 190; at 9.30, pulse about 74. At this point 15 cc of blood-tinged fluid were withdrawn by lumbar puncture. This seemed to change the whole aspect of affairs; he quickly grew cyanosed, respiration became Cheyne-Stokes, the pulse grew rapid and small, and the blood-pressure quickly sank; this all within 5 or 10 minutes. Artificial respiration kept the heart beating for a half-hour longer, but to no avail. At post-mortem a large intracerebral clot was found. Here evidently, from 8.30 o'clock on, the vagus was becoming paralysed as shown by the increasing rate of the pulse; blood-pressure had probably been higher than 190 and was on the brink of giving out. The lumbar-puncture by removing some of the support from the spinal canal, presumably let the bulb be squeezed down into the foramen magnum and against its rim, and thus by acute pressure on the bulbar centres, gave the finishing touch to the beginning vaso-motor paralysis with consequent rapid fall of blood-pressure, which in its turn induced complete vagus and respiratory paralysis. Note the implied warning against withdrawing any material amount of spinal fluid.

A third chart showed the drop of blood-pressure from 130 mm Hg to 88 during the evacuation of a large cyst at the base of the brain extending into the cerebellar fossa in a child of 6 years of age. (personal case, Royal Victoria Hospital). The relief of the cerebral compression exercised by the cyst was very clearly shown in the blood-pressure readings.

A fourth chart showed this fact much more strikingly. It was taken from one of Harvey Cushing's cases (*American Journal Medical Sciences*, June, 1903), a patient suffering from apoplexy with threatening paralysis of the vital centres as seen in Cheyne-Stokes breathing, a slow pulse, and a blood-pressure of over 300 mm Hg. Trephining with evacuation of the intra-cerebral clot caused within 20 minutes a fall of blood-pressure of over 200 mm Hg, from 380 down to normal. This evidenced complete relief of the cerebral compression,

A personal case.

a relief maintained for two days, when unfortunately, the patient died of pneumonia.

Finally a fifth chart served to show the diagnostic value of blood-pressure examinations in distinguishing between concussion and compression. A man suffering from concussion (presumably) was admitted to the Royal Victoria Hospital about 1 p. m. His blood-pressure was 95; at 8 p. m., it was 92; at midnight, 98; 3 a. m., 102; 8 a. m., 122; and at this normal figure it remained. It is characteristic of most cases of concussion to show a subnormal blood-pressure; but if compession from intracranial bleeding be superadded, this will show itself in a rise of blood-pressure above normal. In this case, the slow recovery of blood-pressure and its remaining at the normal level were strong evidence that the brain was not suffering material compression and that the case was one of pure concussion.] To discuss at all fully the operative indications that may rationally be deduced from these physiological premises, would carry us too far. I fear I have already overstepped my time—I shall therefore add but a few words.

The treatment of compression in the light of these considerations must be to relieve the compresion—not to lower a high blood pressure, which as we have seen is Nature's attempt to overcome the obstruction; therefore bleeding and depressing drugs are in general contraindicated; while operation, either to remove the compressing body or to give more room is indicated. On the one hand we have the radical operation, on the other the palliative or "decompressive."

In intracranial hæmorrhage from injury, there is of course no doubt as to operation if the bleeding come from the middle meningeal artery. But with regard to those cases in which the bleeding is inside the dura mater from pial vessels, I believe we must enlarge the limits of the indications for operation and interfere to relieve dangerous pressure even where gross localising signs are absent. I think it is important that in hospitals the neurologist should be called in consultation early for traumatic cases, as well as for tumour cases. He may perceive the finer localizing signs which the surgeon may miss. Or else the surgeon must acquire a good neurological knowledge himself, which of course is the desideratum.

I believe we shall soon be operating to evacuate the clot from the cerebral substance in dangerous cases of apoplexy. I believe the obstetrician should call the surgeon in consultation for the intracranial hæmorrhages of the new born, where convulsions and a bulging tense fontanelle indicate high intracranial tension. As to tumour, the general practitioner and the internist must learn to recognise them early, and not wait until the optic neuritis has gone on to blindness, and the vomiting and headache have reduced the patient to a sadly weak condition before they call in the surgeon. In these cases surgeons are getting brilliant results, both from radical operation and in the relief of symptoms from the palliative decompressive operation, consisting in the removal of bone sufficient to relieve tension without invading the brain itself. This last for cases where diagnosis of the seat of the disease is impossible. It must be remembered that we can now localise the seat of a tumour in 50 per cent. more cases than

we could ten years ago, and that results have improved 50 per cent.

There are many conditions which are still considered inoperable, which in my opinion will soon belong, partly at least, to surgical territory. Such are, for instance, suppurative meningitis, in which a number of cures are reported from trephining with drainage. True, I can hardly believe these cures were accomplished by any drainage of the subdural space which to me seems scarcely possible, but rather to the removal of bone and the consequent relief of pressure, which latter in some cases of meningitis is the true cause of death. Even tuberculous meningitis I have some hope for from decompression, though this is a more unlikely event. The treatment of hydrocephalus by drainage of the ventricular fluid into the subdural space, will, I believe, ultimately be rewarded by some success, although results are as yet discouraging.

The field is constantly widening for surgical intervention; and this is especially justified in the otherwise hopeless cases. As Celsus said long ago: "Better a doubtful hope than sure despair." If only the internist, especially the neurologist, and also the general practitioner, will learn to give up their traditional pessimism with regard to cerebral cases, and will work hand in hand with the surgeon, I am convinced that great results are possible of attainment. Witness the work of the surgeons, especially Sir Victor Horsley, in the great London Hospital for Nervous Diseases, at Queen's Square, where one sees extraordinary results obtained.

Hope, therefore, is the note upon which I wish to close. Let me thank you in conclusion most heartily for the honour and privilege of speaking before you.