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"Cerebral Circulation"

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"Cerebral Circulation" - Thesis for M.D. 1

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The selection of "Cerebral Circulation" as the subject of a thesis may seem ambitious but the remarks & suggestions offered do not pretend to contain much that is either new or very critical. With the selection made it may appear inconsistent to say that the numerous subjects which presented themselves to our mind, were consecutively dismissed as being beyond us; but here at least the views held are well defined and distinctly opposed, and our object is an attempt, though feeble, at reconciliation rather than to deal in a comprehensive manner with such an admittedly abstruse question.

The man who is engaged in scientific research is, we believe, of all others the most desirous of truth, frequently sacrificing his cherished hopes and beliefs in its pursuit. He does not place himself at the top of creation endeavouring, with prejudiced mind, to reconcile everything to preconceived ideas. He may have theories but they remain as such and are only adhered to so far as the inductive method of observation will permit. We ought then to accept that men actually see, or believe they see, what is described in experimental investigations and endeavour when results are contradictory to find out where fallacies may be introduced. In attempting this our diffidence is much increased by a keen consciousness of the feebleness of our grasp on physics and the other collateral sciences, and we crave indulgence in the many instances in which this must necessarily become so painfully obvious.

2

The following pages may be further prefaced by shortly enumerating the two main points to be considered:- Firstly subsidence of intracranial blood after death (probably effected without its place being taken by any other fluid) with constancy in amount of the intracranial contents during life. Secondly The peculiarities of normal cerebral circulation.

Judging from the appendix of Dr Cappie's book on the "Causation of Sleep" (from which alone most of our data must be drawn) one would think that after all there is but one opinion as to the relation which the cranial contents bear to their immediate environment - that the disagreement has resulted from the ambiguity of terms and ignorance as to the distinction between "red blood" and "serous effusion".

That the cranial cavity is a closed in one, which practically communicates with the external pressure through the blood vessels only, is evident. Dr Kellie says "It does not appear conceivable how any portion of the circulating fluid can ever be withdrawn from within the cranium without being simultaneously occupied by some equivalent, or how anything new or exuberant can be introduced without an equivalent displacement" (Causation of Sleep page 135) and this is supported by experiments on animals and by occurrences and accidents in human life. Dr Burrows, opposing this view, performs experiments which show contrary results and asserts that "the principle of subsidence of fluids after death operates on the parts contained within the cranium as well as on those situated in the thorax or abdomen" and he points to the numerous foramena in the skull as a means by which the atmospheric pressure may be communicated to the interior and thereby allow escape of its contents. The theory that the Cranial contents cannot be materially increased or diminished is one which appeals to our common sense and induces us to look for an explaination of the discrepencies above referred to. Dr Cappie says that subsidence of fluid can only be explained by supposing that the skull of the rabbit cannot resist the atmospheric pressure and by the gravitation of the red blood corpuscles towards the dependent parts of the body, and he thus dismisses the question. (Causation of Sleep, page 164-7)

To quote from Sir Thos. watson (Principles and Practice of Med. Lect. XVI) Dr Burrows' experiments were as follows:-

※ I "Two wellgrown rabbits were killed, the one (A) by opening the jugular vein and carotid artery on one side of the throat; the other (B) by strangulation. Round the throat of the first as soon as it was dead, a ligature was tightly drawn to prevent any further escape of blood from the vessels of the head. The contrast between the two brains in point of vascularity, both on the surface and in the interior was most marked. In the one scarcely the trace of a blood vessel could be seen; in the other every vessel was turgid with blood"

II Two fullgrown rabbits were killed with Prussic acid, and while their hearts were still beating the one (C) was suspended by the ears; the other (D) by the hind legs. They were left suspended twenty four hours; and before they were taken down for examination a tight ligature was placed round the throat of each rabbit to prevent, as effectually as it was possible, any further flow of blood to or from the head, after

※ These quotations, though lengthy, seem necessary.

"they were removed from their respective positions".

"In rabbit D the external parts of the head, the ears, eyeballs etc. were turgid, livid and congested. The muscles and bones of the cranium were of a dark hue and gorged with blood which at some parts appeared extravasated. Upon opening the cranium the membranes and vessels were dark and turgid with livid blood; the superficial veins were prominent, the longitudinal and lateral sinuses were gorged with dark blood and there was staining of the tissues, if not extravasation of blood into the membranes. The substance of the brain was uniformly dark and congested to a remarkable extent".

Dr Kellie's experiments, as given by Cappie (in "Causation of Sleep", page 148) were as follows – "One sheep was bled from the carotid artery alone, another from the jugular vein ..... A dog was bled from both femoral arteries, another from the carotids, and a third from both jugular veins. Then, to afford examples of brain not depleted by previous haemorage, he tied both Carotids in two dogs and allowed them to die, and a third dog was poisoned with Prussic acid. In the majority of cases of bleeding to death the brain did not appear to be seriously depleted; in no case was it all exsanguined like the other parts of the body, but the appearances as indicated by the presence of red blood varied somewhat. In the sheep where the carotids were tied and the jugulars were opened, death did not occur till twenty three minutes after the veins had been wounded, and for a time the blood flowed slowly and by occasional drops only. Here the sinuses of the head were in their usual state; those at the "base of the brain contained less blood than we have hitherto found in them, and the veins on the hemispheres of the brain were less filled; the choroid plexuses were pale and empty; the vessels on the basis of the cerebrum were better filled; those ramifying on the basis cerebelli were minutely injected. There was a slight but very decided effusion within the ventricles."

In the dog poisoned with prussic acid the brain was everywhere turgid with blood. The veins and sinuses were loaded and congested and it was quite evident that this brain contained, beyond all doubt or dispute, a much larger quantity of blood than the brains of any of the other animals which had been bled to death. The summary is, that though we cannot by any means of general depletion entirely or nearly empty the vascular system of the brain, as we can the other parts of the body it is yet possible by profuse haemorrhage to drain it of a sensible portion of its "red blood": that the place of its spoliation seems to be supplied both by extra- & intra-vascular serum and that watery effusion within the head is a pretty constant concomitant or consequence of great sanguineous depletion". Dr Kellie completes his experiments by trephining the skull when all the blood is seen to escape - or very little is left behind whilst the brain itself subsides.

Looking at the above it is most apparent how much more thorough the experiments of Dr Burrows were ~~&~~ those of Dr Kellie. The former certainly took care "to exclude every conceivable source of fallacy". If Dr Burrows statements can be trusted what can we wish for more than "the brainsubstance than

"and membranes were pallid - anaemic beyond my expectation - 6
scarcely the trace of a blood vessel could be seen". And again
"vessels dark and turgid - sinuses gorged with blood - the
substance of the brain uniformly dark and congested to a
remarkable extent". The red corpuscles, in the cases of depletion, must have gone somewhere out of the cranium and
the liquor sanguinis left the blood vessels. The question
then arises where and how? Dr Cappie shows impatience
at the results of those experiments, or at least at the conclusions drawn from them ("Causation of Sleep" page 167); and
it seems his assertion that fluid can only subside from the
cranium after death by the skull being unable to resist the
atmospheric pressure is unfortunate; his remark that the
red blood corpuscles may have settled down to the dependent
parts of the body is equally so, for where is the liquor sanguinis?
If the corpuscles had subsided alone the vessels would still
be at least partially full: but says Dr Burrows "the vessels can
scarcely be traced". Has its place then been taken by an increase
in the amount of ventricular fluid? Dr Burrows in his experiments is silent on this point, but considering his attention
to details, we may take for granted he would have stated the
fact had it been so. No increase was apparently observed. And
again in the instance where the carotid arteries of the sheep were
tied and the jugular veins opened, Dr Kellie affirms that "the
sinuses of the head were in their usual state; those at the base
of the brain contained less blood etc. but, there was a slight
though very decided effusion within the ventricles" (Causation of
Sleep, page 150). Now, in this experiment nearly all forward
pressure which would normally be communicated to the brain

through the blood vessels is cut off by their ligature, and the 7
blood flowing from the divided veins must, if there be any effect whatever, produce negative pressure within the cranium. Here an increase of ventricular fluid is associated with diminished pressure! To quote Dr Cappie on this very subject, ("Causation of Sleep", page 189) where he refutes this theory put forward by Mr Durham, he says "in what circumstances do we actually find an accumulation of serous fluid within the cranium? Is it not in those cases in which we can with confidence predicate increased pressure within the vessels or diminished support outside of them"? and so on to the end of page 190, referring to the pressure of inflammation, exposure to cold, hydrocephalus, and the atrophy of old age. "Diminished support outside" must refer to this last condition. In old age pressure must to some extent be kept up so long as circulation is maintained. The tissues of the brain atrophy, tending to produce a vacuum, and their place is taken by fluid passing through the walls of the bloodvessels. Here there is certainly an absolute diminution in support, but it is equally correct to say there is relatively increased pressure; and in this particular instance I fail to see the need for any such distinction. To have effusion there must be increased pressure, absolute or relative. If the Carotid arteries be ligatured and the jugular veins cut there must be negative pressure within the cranium and therefore no effusion. In speaking of the amount of ventricular fluid, which in man varies so much normally, it cannot be considered satisfactory to find "slight" & "decided" within the limits of one short sentence. Something else then must take the place of the blood which subsides from

within the cranium in profuse haemorrhage - But leaving this 8
for a time let us observe the explaination offered by Dr Burrows.

It is asserted by Dr Burrows (Watson's Lectures page 324) that "the principle of subsidence of fluid after death, operates on the parts contained in the cranium as well as upon those situated in the thorax or abdomen" and he supports this by saying that "the numerous foramina do away with the idea of the cranium being a perfect sphere like a glass globe", suggesting that the mechanism here is sufficient to permit of the brain being depleted through the atmospheric pressure. It is conceivable that those foramina may allow of some slight subsidence of fluid, for when the blood was flowing from rabbit A in Dr Burrows experiments "the eye balls were seen to shrink within the sockets" which must have been due to the withdrawal of blood from the soft tissues behind the eyeballs; and if so why not from the tissues in the optic foramen, its place being immediately take by a slight recession of the optic nerve, ophthalmic artery, and connective tissue, due to the atmospheric pressure? The same may be said for the other foramina at the base of the skull though the amount should be measured by drops rather than suppose it could explain the anaemia described. This however, should not be overlooked in explaining the gorged appearance in the contrary experiments.

Another point to note is the influence of the cerebrospinal fluid, to which we shall refer more fully later on. If fluid be put into a syphon tube it will flow towards which ever end there is the heavier column; and, consequently if an animal be suspended by the ears and

the blood vessels undivided, it is possible that the 9
gravitating blood may alone raise the spinal fluid.
But the case would be altered if the blood vessels were divided
for the column of blood would then be very materially
diminished. These remarks however, seem superfluous
for, as a matter of fact, no increase of ventricular or sub-
arachnoid fluid was observed in Dr Burrows most searching
experiments; and the blood vessels could scarcely be traced,
showing that even the liquor sanguinis had disappeared.

But, taking for granted, in the mean time, that the cerebrospinal fluid is sufficient to permit of the intra-cranial blood subsiding, and that the effect of the atmospheric pressure through the cranial foramina is trifling, what other medium is there by which this may be effected? Cappie, Burrows, Watson, Abercrombie etc, all agree in stating that the cranial contents consist of brain-mass, blood and cerebral fluid; but, in this enumeration there seems to be an important item omitted; and, lest (lest) the idea may appear absurd we will, before mentioning it, make a short quotation from Fosters physiology. Referring to the effect of changes in pressure of the air breathed he says "in sudden diminution death results from the liberation of gases within the blood vessels and the consequent mechanical interference with the circulation. The gas found in the blood vessels on examination after death consists chiefly of nitrogen". It will be apparent from the above that the fourth factor referred to is the gases of the blood, and the one we look to for an explaination of the complete anaemia described by Dr Burrows.

Carbonic acid is the only gas in the blood sufficient in amount. Nitrogen is contained in small quantities, whilst oxygen must practically be absent from the blood of dead animals. Yet, "nitrogen is the chief gas found after death." It must be admitted however, that under diminished pressure Carbonic Acid is given off and that when the pressure is restored it re-enters the blood more rapidly than Nitrogen for the latter is simply held in solution and will therefore be more slowly absorbed by the blood than Carbonic Acid, which in addition possesses an affinity for the sodium, forming Fernetts Compound. And as the presence of Oxy-haemoglobin is supposed to favour dissociation of this Compound so its absence will favour the formation.

Of the 20 cubic inches of gases given off at each expiration, 4 per cent consists of Carbonic acid that is 0.8 cubic inches, most of which comes from the pulmonary blood. We have no means of accurately estimating the amount of blood in the lungs, and much less from what portion of it this is given off, but in Fosters Physiology (page 35) a table shows that 22.76 percent of the whole blood is contained in the heart, lungs and great vessels; and seeing that the latter (especially the veins) usually contain a large proportion of blood after death, it may be supposed that the lungs alone contain only 10 percent. In the same table the blood of the brain and the cord is estimated at 1.24 percent: and deducting 0.24 for the cord, leaves 1.0 for the brain. Then the formula 10 : 1 :: 0.8 (Cubic inches) : CO_2 will give the amount

of Carbonic acid which may be supposed to be given off from 11
the cerebral blood, during each act of respiration, were it
exposed to similar conditions as the pulmonary blood: that
is, about $\frac{1}{12}$ cubic inch of carbonic acid would be given off
from the brain of a rabbit every $\frac{1}{17}$ part of a minute. But
this is a low estimate as "Spehl found in the rabbit, $\frac{1}{8}$
to be the amount of blood contained in the head at a given
moment relatively to the total bulk of the blood in the system."
(B.M.J. 1888, page 503) and Carpenter's physiology (page
529) states that $\frac{1}{5}$ of the whole blood in a healthy man
constitutes the supply to the encephalon. But the Carbonic
acid given off from the cerebral blood, were it exposed
to the air like that of the lungs, is not what is given up in
$\frac{1}{17}$ of a minute but all that accumulates in it, not only
till the heart stops, but till the animal is removed from its
hanging posture: for if the disintegration of vital function
has ceased, that of the dead tissues must still be going
on. By simply holding the breath Becher found the
percentage of Carbonic acid more than doubled - from
3.6 to 7.5 (Foster 308): and so it may be supposed
that the Carbonic acid gas, before and after death, tends
to accumulate and supersaturate the encephalic blood and
and interstitial lymph, ready to escape the moment the
intracranial pressure becomes negative.

The Brain is certainly not exposed to the
influence of atmospheric pressure in the same sense
that other organs of the body are, but we believe that
its environment is not less favourable for its thorough
depletion than if (the animal being decapitated) part of

the skull were removed, as we shall endeavour to show.

If a rabbit be poisoned with prussic acid and suspended by the ears, as soon as the heart stops beating the main compressing force acting on the brain is removed; and the blood generally, as it gravitates towards the dependent parts, must act on that contained within the cranium, exerting its force through the carotid arteries, jugular veins and other vessels, and thus produces a tendency to the formation of a vacuum; and if the carbonic acid be in excess it must be liberated. It escapes by diffusion chiefly, from the pulmonary blood without any tendency to the formation of a vacuum and why not from the brain under these more favourable circumstances? and, being poisoned with prussic acid the blood will remain fluid and thus favour the continuation of the process.

How far then can this theory be applied in explaining the phenomena associated with some of the accidents and occurrences in human life?

In "hanging", even if very little blood were passed on after "the drop" the gorged appearance of the face would be caused by compression of the superficial veins, associated with the natural contraction and emptying of the arteries after death - and more so if a certain amount of blood escaped from the intracranial vessels. Venous congestion is what one might naturally expect, but I cannot see why we should specially look for a gorged condition of the intra-cranial vessels; for if the carotid arteries be compressed by the cord, then the main blood supply is cut off and the chance of congestion minimised. If on the

other hand, the carotid arteries be patent then what extra 13
blood we might expect to have been forced into the intracranial vessels (if any) whilst the heart was still beating, will tend to drain off through the patent carotid arteries after the heart has ceased?. Watson, in his lectures (page 328) states that the appearances in the brain in such cases vary considerably, and that on the whole there is even less blood than normal or not un-naturally full. This is what we would expect, for if depletion depends on the liberation of gases consequent upon the backward pressure exerted on the intracranial contents through the gravitating column of blood, then the amount of gas liberated must depend on the size of the blood column. If the large vessels be closed by the rope the backward pressure must be little, and the depletion slight in proportion. If the larger channels be left patent, then there will be a greater tendency to form a vacuum; more gases will be liberated and there will be greater depletion. In hanging all the vessels are not cut off: the vertebral sinuses are always patent (Watson's lectures page 325) and therefore the "not un-naturally full" condition is consistent. Each case must necessarily vary directly with the size of the blood column tending to escape. The same may also be said of Kellie's experiments where the vessels were opened. The anaemia was comparatively slight as the blood column necessarily was short. So also in decapitation.

It may now be asked why the gas does not become more obvious and show its presence in the vessels

and tissues: and the answer may be more difficult than 14
we suppose. As far as the arteries and arterioles are concerned it may be said they will contract on it and compress it; but even if so the tendency to form a vacuum is not done away with and it may be supposed that the gas passes on into the inter-cellular spaces and capillaries. And certainly the veins and venous sinuses, which are not contractile, provide ample room for its accomodation. But wherever it may collect its absence on examination is easily explained, for, as the gas is liberated under diminished pressure, it must necessarily be compressed into smaller bulk the moment the atmospheric pressure is allowed to act upon the intra cranial contents by the slightest opening being made into the skull. The lymph and blood which has ~~ha~~ not drained away will re-absorbe part: and should the gas possess the least degree of tension it will escape from the venous sinuses and meningeal veins by those veins which connect the latter with the venous channels in the diploe (Gray's Anat. p. 432), for when the skull-cap is removed those veins must be divided, leaving the sinuses in direct communication with the air. No emphysematous condition of the brain tissues nor distension of its vessels could be expected under those circumstances; and it appears that the phenomena observed by Dr Burrows with regard to depletion are consistent with physics and probably with the facts of physiology as well.

We shall now return to the rabbit suspended by the hind legs; and it may here be remarked that though the pallor just referred to could only be caused by great depletion, yet a comparatively slight addition to the normal amount of blood may produce a gorged appearance.

The engorgement observed in the animals experimented on does not seem to have excited much surprise either in Dr Kellie or Dr Cappie. Both seem to have devoted their energies to explain the absence of "red blood". For instance, referring to the dog poisoned with Prussic acid Dr Kellie says "the brain was every where turgid with blood. The veins and sinuses were loaded and congested; and it was quite evident that this brain contained, beyond all doubt or dispute, a much larger quantity of 'red blood' than the brains of any of the animals which had been bled to death". The words here are almost as strong as those employed by Dr Burrows in describing the congestion of the brain of the rabbit suspended by the hind legs. Dr Kellie only emphasizes the expression "red blood", evidently hinting that it had usurped the place of a certain amount of extra- and intra- vascular serum. No explanation is offered as to how this is effected. "Settling of the red corpuscles" will not afford a solution; neither can we suppose that the extra-vascular serum (not including the cerebrospinal fluid) has diminished for the increased pressure must lead to effusion rather than otherwise, yet some thing must have yielded.

In discussing Mr Durhams theory of Sleep Dr Cappie strongly opposes the idea of the cerebrospinal fluid rising and falling in the spinal column, and more as the transfer from the ventricles to the base of the brain.

His arguments are convincing and we must agree with 16 him concerning what takes place in the living body, but the case may be altered when the cadaver is suspended for twenty-four hours. There is free communication between the subarachnoid fluid of the brain and that of the cord, and the continuity indicates that variations at some time or other may be looked for. When an animal is suspended by the hind legs all the fluid of the body will tend to gravitate towards the head, the most dependent part. The gravitating fluids are the blood and the cerebrospinal fluid - each struggling to supplant the other within the cranium - That these two fluids try to displace the other is evident; for the blood, in its attempt to enter the cranium will tend to expand the brain and and its superficial vessels, whilst the effect of the spinal fluid must be to compress these. Other things being equal it is evident then, that the fluid which exerts the greater pressure will displace the other; and as the amount of blood far exceeds that of the spinal fluid it is not difficult to decide which will give way. But on the other hand there is the fact that the cavity of the spinal column is a closed-in one, and any attempt to force more fluid in must be resented. The probability seems to be that the blood does not enter at the expense of the cerebrospinal fluid.

Only one other explanation can be offered. It must be observed that before removing the rabbit Dr Burrows ties a cord tightly round the neck to prevent any further draining from the head or entrance into it. The animal has hung for twenty-four hours previous to this, and the soft parts about the head and neck must consequently be enormously engorged. The carotid arteries and jugular veins must be distended with blood trying to force its way into the cranium. The ligature must prevent any backward flow, and therefore the pressure must be kept up. An incision is now made through the scalp, and part of the skull removed upon which the pent up blood in the vessels will, from the atmospheric pressure on it, and from the diminished resistance to brain expansion, pour itself into the sinuses, veins and arteries of the encephalon whilst at the same time it is a physical impossibility for any to escape. The blood may even be expected to ooze from the turgid scalp through the veins of the diploe into the meningeal veins & sinuses. This transfer of blood must go on from the moment the slightest entrance is made, and during all the time the skull-cap is being removed. In this way it may be fairly assumed that, on examination, a state of over-fullness may be found without necessitating displacement of the cerebro-spinal fluid.

In experiments of this sort then, Dr Burrows statement that both engorgement and depletion (subsidence of fluid) do take place, according to circumstances, is not surprising. Munro may fill his glass globe with water and Dr Cappie a skull closely covered by the scalp etc. and not a drop may escape; but if either be filled with venous blood similar results can not be expected.

But though it may be correct that fluids sub-

side

in these experiments, it must be remembered that in their 18
chief bearing they are performed on dead animals, and, with regard to the living, any theory founded upon them must be accepted with caution. Because the brain of an animal can be drained of its blood after death, under extreme circumstances, are we to infer that any thing analogous may take place in the living? Consistent with life how can there be backward pressure produced in the carotids to any degree whatever, much less to cause the eye balls to shrink, the spinal fluid to be raised or the gases of the blood to escape - the latter being inevitably accompanied by "gas embolism" and death. In cases of fainting from diminished supply of blood to the head it is probable that this calamity is prevented by the recumbent posture which nature inforces, the intra-cranial pressure being thereby kept up. The "Edinburgh dogma" then if applied to the living exists, but to the dead it may possibly be considered demolished. Any further remarks as to its bearing on cerebral pathology and the movement of the cerebrospinal fluid will be introduced in the consideration of the normal cerebral circulation.

If we leave out of consideration the supposed power of the tissues in attracting blood, which is doubtful (Lects by Prof. Rutherford) the general circulation of the body is carried on by the heart propelling the blood into the large arteries, the latter by their recoil after systole, driving it on through the arteriols and capillaries into the veins. And though the Cerebral circulation is stated to be so peculiar, it would be

strange indeed if it varied in the slightest in these points. 19
Accordingly we shall endeavour to show that, though subject to restrictions it is essentially the same as that in other parts of the body. In tracing the circulation as above, we find that the heart throws the blood into the cerebral vessels during systole only as elsewhere and not continuously. The large cerebral arteries must then cause the blood to circulate through the arterioles and capillaries and empty it into the veins. But here the difficulty arises. The veins must be filled gradually: there must be a continuous flow into ~~of~~ them, and we would naturally suppose a continuous flow out of them. But how is this consistent with the sudden propulsion of blood into the cranium (its contents being a constant quantity) during systole only? It is beyond reason to suppose that the blood is forced directly through the cerebral arteries into the veins, causing venous pulsation. This only occurs in less highly organised structures when in a condition that might be considered pathological, with extreme relaxation of the arterioles. But again, another apparent contradiction is the fact that the jugular veins do pulsate. Dr Broadbent says "the movement of the blood in the internal jugulars has been shown to be pulsatile." (B.M.J. 1887, page 764). On the same page he states "it can not be supposed that the brain substance is compressed into smaller compass, at each pulsation of its vessels". He further goes on to show the impossibility of the cerebrospinal fluid being able to afford accomodation by its movement; but still there is no explanation offered of the phenomenon of venous pulsation.

"There are two discrepitant views" says Dr Mickle (B.M.J. 1888 p. 505) as to the mode in which provision is made for the variations in cerebral circulation. One, supported by Majendie, Carpenter etc. is that the movement of the cerebrospinal fluid is sufficient to account for these variations: The other, held by Foster, Cappie and Frank, is that the venous system inside the cranium explains it. The "variations permitted in health (B.M.J. 1888- page 504) must include those occurring during normal circulation, and consequently those who believe that it depends on the to-and-fro movement of the cerebrospinal fluid have to admit that the same amount must be displaced as that which enters with each cardiac contraction: and to effect this, accomodation must be provided in the spinal canal for the same amount. That is, in 1/3 of a second about an ounce of fluid is forced out of the spinal canal (vide infra). The time thus allowed for the transmission of blood from inside the canal is simply incredible however large the veins of its meninges and of the bodies of the vertebrae may be.

The amount of blood sent to the head at each cardiac contraction is not definitely known but seeing that the heart throws out six ounces each time and that in the healthy man the head contains 1/5 of the whole mass of blood (Carpenter's physiology, page 529) it must receive, as shown by a simple calculation ($1 : \frac{1}{5} :: 6 : \overline{X} = \overline{X} = 6 \times \frac{1}{5} = 1\frac{1}{5}$ ozs) about one ounce each time the heart contracts Now, "the cerebrospinal fluid varies from two to ten ounces" (Gray's Anat. Ed. VII, page 449) and two ounces being apparently the smallest amount consistent with health, only one ounce therefor is left, "yet the chief" use of this fluid "is probably to afford mechanical protection to the nervous centres and to prevent the effects of concussion from without" (Gray's Anat. Ed. VIII, p. 472)

But even granting it possible for the fluid to be forced into the spinal canal, there is no means by which it could be raised suddenly so as to account for jugular pulsation: pulsation would indeed be inconsistent, because the fluid being forced out by the sudden expansion of the arteries it must necessarily return slowly during diastole whilst the latter are emptied of their contents and the venous flow must therefore be continuous: yet, pulsation has been demonstrated. If venous pulsation could be produced at all by the returning cerebrospinal fluid it must in point of time be diastolic and be the result of pressure applied directly to the venous system inside the cranium.

Lastly, the view is untenable because, first, the veins in the spinal canal must have sufficient to do to provide for the blood supply of the cord alone; second, we should expect that the arterial pressure in canal would counteract the pressure exerted on its fluid from above; and third, if cerebral circulation is only to be explained by pressure through the medium of the cerebrospinal fluid on veins, why look for those veins in the spinal canal when they are to be found within the cranium itself?

In the face of the anatomical difficulties so clearly put forward by Dr Cappie ("Causation of Sleep", page 181) it is difficult to understand how a logical mind like that of the late Dr Moxon could so fully accept this cerebrospinal-fluid theory. He says "there is a wider and freer passage for it than "for the blood which passes through the capillary arteries of the brain, and, if the circulation be feeble, the heart may drive the blood less forcibly to the textures of the brain than the venous pressure of the canal drives the cerebrospinal fluid into its cavities, so that when the head is raised the venous blood falls away more rapidly from the skull and the cerebrospinal fluid outstrips the arterial blood in the race to supply its place - and the ventricles may tend to fill more quickly with water than the substance of the brain with blood and so the person feels giddy" (B.M.J. 1881. page 492). This explaination of momentary giddiness, the result of raising the head, involves almost as free movement as that which we have just been disputing: but the facts at our command, as far as we are able to use them, lead us to the conclusion that no variation in quantity does take place except by the more gradual process of secretion and absorption.

The cavity of the cranium then is a closed-in one whose contents are a constant quantity: the amount of cerebrospinal fluid inside the cranium does not vary during the period of a single cardiac cycle: as the blood enters suddenly so must its exit be sudden: and therefore, in trying to explain the events which take place inside the cranium during a single cardiac cycle, it will be allowable to make use of an illustration which involves the above principles and then endeavour to see whether there be any analogy.

A glass globe then, is filled with a substance corresponding to the brain mass and fitted up with an hydraulic apparatus. Outside the globe there is the propelling apparatus sending fluid by a tube through the wall of the globe opening into what may be called the "proximal sinus". From the latter smaller tubes penetrate the contents of the globe, these collecting to form larger tubes which again open into a "distal sinus". To start with, the proximal sinus is empty and the distal one full. If now the pump be made to force fluid onwards the first effect will be to distend the proximal sinus. But the pressure exerted must be communicated through the contents of the globe to the distal sinus forcing its contents out just as if there was a large and direct communication by another tube between the two sinuses, the result being that as fluid is forced into the proximal sinus exactly the same amount is forced out of the distal. There cannot be any fallacy here so long as the compressibility is insured and the exit free. Here too, the exit must be pulsatile and in point of time correspond with the entrance. When the proximal sinus has been filled at the close of the stroke of the pump the distal must be empty or partially so to a corresponding extent. When the pump has ceased acting then the proximal sinus from its elasticity diminishes in size and gradually forces its contents on through the smaller intervening tubes, the distal sinus filling as the proximal empties. Here there is the transfer of fluid from one part to another of the contents of the globe without there being necessarily any undue disturbance. 23

The attempt to apply the above illustration to explain the intra-cranial circulation may appear difficult or even absurd, but if the ingress of blood is not provided for by the movement of the cerebrospinal fluid, the involvement of the principles of some such scheme must be admitted.

The skull is practically a closed-in box or globe; its contents must be constant in amount; additional fluid is forced in and accomodation must be provided.

To the superficial observer at least there is some analogy in both. The pump or propelling apparatus is of course represented by the heart; the "proximal sinus" by the large arteries chiefly at the base of the brain; the "smaller tubes" by the capillaries of the brain; and lastly the "distal sinus" by the venous system within the cranium communicating with the exterior through the jugular foramena. The expression "venous system" is here used as a general one for the meningeal veins, the venous sinuses of the skull or both combined, as at present it is more convenient to leave it undecided which takes the more important part or both.

It is obvious that immediately before the cardiac systole the large cerebral arteries forming the circle of Willis etc. are empty or at least are in that condition in which they contain their least amount of blood during the period of any single cardiac cycle. The capillaries must also contain their usual constant (the flow through them being uniform) amount of blood, and the venous system must be at its fullest. The heart now contracts and distends the large arteries; more blood has been forced into the closed-in cranial cavity: what has yielded? The only answer is that the venous system has been partially emptied by direct pressure on it communicated through the cranial contents from the distended cerebral arteries. But these arteries are now charged with potential energy (as they are elsewhere in the body) and during diastole they continue to contract and transfer their contents gradually into the capillaries, the contents of the capillaries being pushed on into the venous system, the venous system (having been emptied by the cardiac systole) being filled in proportion as the arteries are emptied and simultaneously with that event. But can this transfer of blood from one part of the intra cranial contents to another be shown to be effected without undue disturbance to the brain substance? Considering the difficulties experienced in trying to grasp the relation which the brain mass, cerebral fluid, and blood vessels bear to each other it will not be surprising if we go far astray, but it may be within reach to indicate a few of the more obvious points bearing on the above. Two questions may be put – first, is the venous system compressible? And second, what are the means causing and permitting such compression? To answer these separately would involve much repetition, and besides, all that we have to say may conveniently be put together.

The venous sinuses are defined as incompressible and inextensible channels, tunnelled out in the dura-mater of the brain (Essays in Medical Science, page 66) and it does not seem necessary to dispute this. When the top of the skull is removed they certainly can be flattened by pressure, numerous veins being there by made visible by blood pouring out (Ellis pract. anat. page 10) but suppose we could exert pressure on them from within, the skull being intact, then the tight falx cerebri etc. stretching from the anterior to the posterior parts of the skull, would ~~necessarily~~ certainly resist such pressure, and no doubt successfully all that could be exerted through the cranial contents. But even though compressible they are certainly not sufficiently distensible to accomodate a great increase of venous blood; and besides, the veins may be considered sufficient for our purpose. The latter too are numerous, equally distributed and thin-walled, and therefore more fully under the influence of wide spread pressure and more easily distended.

Though the pressure within the cranium may correctly be said to be the same at all parts of its contents, yet it cannot be asserted that the effects are the same. The parts which are soft will yield more and become altered in shape to a greater extent than those that are firmer, if there be any difference in consistence. More especially will this be the case with the fluid parts - the blood contained in the vessels and part of the cerebrospinal fluid. Now, the blood vessels and the subarachnoid fluid, being placed between the unyielding bone on the one hand and the comparatively resisting brain on the other, will reasonably be subject to a greater amount of displacement than any of the other parts of the intracranial contents - more so than the ventricular fluid which must receive some support from the brain substance. The blood vessels and subarachnoid fluid are therefore placed in that part of the cranial cavity where the effects of varying pressure will be most marked - where the vessels can be most speedily emptied and, the pressure being relieved, most speedily refilled.

Suppose the blood vessels be made to penetrate the brain, artery and vein together as in many instances elsewhere. Here the continuity of its structure would be considerably pressure on them from within

marred. This tunnelling of the brain substance however,
would be the least of the evils, for with each contraction of the heart the brain would be rendered liable to a simultaneous separation and compression of its substance which we believe, being widespread, might seriously interfere with its function. Even if expansion were allowed by the cerebro-spinal fluid having as free outlet as some suppose, still this injurious effect would not be lessened to any great extent for there would still be the tendency to separation and laceration of its elements. Not only this, but the circulation could not possibly be carried on satisfactorily for the expanding artery would at once compress the vein and lead to interrupted circulation incompatible with the maintainance of normal cerebral function.

Again, suppose the arteries placed mainly outside the brain, and the veins, having emerged from the brain substance, opening directly into the sinuses which are incompressible; then, as the cerebrospinal fluid cannot escape some thing else must yeild when the arteries are distended with blood forced in by the heart. Here there is evidently nothing that can yeild only the veins penetrating the brain mass, and to effect compression of those would entail alteration of the brain in shape or rather a total diminution in bulk, which is out of reason.

From these considerations it is evident that the large blood vessels and veins of the brain are placed in the only possible position for them to be in, consistent with normal circulation in an organ situated in a closed-in cavity. In all other positions there would be imperfect circulation and disordered function.

Varying pressure (intra cranial) is demonstrated 28 in every healthy child, and more so in acute febrile affections where the rise and fall of the fontonelles show that it varies directly with the propelling force of the heart. It is said "the brain would pulsate if it could" (Meckle, B.M.J. 1888) but the term pulsate is objectionable for, according to the definition which Broadbent gives, pulsation ~~[illegible]~~ involves a change of shape in the wall of the artery from the flattened to the circular form (B.M.J. 26-3-1887) and therefore if the brain pulsate we must assume that the vessels penetrating it increase in size and expand the brain at each cardiac systole: but this cannot be admitted for the following reasons:-

The "cortical" branches as well as the "central" (going to the ganglia of the brain) arising from the circle of Willis are not large arteries but minute twigs all but capillary in size and the flow of blood through them must be continuous during the complete cardiac cycle and not pulsatile. And further, the fact that the cortical branches, seen dipping into the substance of the brain from the pia mater, are not actually capillaries but minute arteriole, excludes entirely the possibility of pulsation for, according to physiologists, an important function of the arteriols is to act as stopcocks for the prevention of pulsation. Under the microscope the blood is seen to pass "in a continuous stream from the small arteriols through the capillaries into the veins". Therefore, unless the cerebral circulation in this respect differs from that in the body generally, or is in a continuous pathological condition, pulsation of the intra-cerebral vessels is an impossibility and systolic expansion of the brain as well. Gradual expansion during functional activity, or expansion and contraction associated with sleep, it is needless to attempt denying: this is stated as a simple fact by Lauder Brunton in his "Therapeutics". But besides, if pulsation occurred in those minute vessels the circulation would be obstructed as previously indicated.

It would seem then, that the compressing force exerted through the heart must mainly take effect, not on the brain itself, but on the more yielding structures outside, namely on the veins through the medium of the subarachnoid fluid.

However redicululous this view may appear to superior intelligence, this much at least may be said in it's favour that it is not so "round-about" as the theory put forward by so many authorities, that the blood pressure during systole is communicated to the cerebro-spinal fluid, forcing the latter into the spinal canal and there displacing venous blood for its accomodation. In this it is admitted there is pressure exerted on the cerebro-spinal fluid but its effect on the cerebral veins is ignored. As before stated, why should we fall back on the veins in the spinal canal when the cerebral veins are at hand, ready to be acted upon? Pressure on the former can not explain jugular pulsation: on the latter it does.

But there are numerous difficulties to be to be encountered here which we can do little more than hint at - difficulties in connection with the quantity

of the subarachnoid fluid, its distribution on the cerebral
surface, and its behaviour under special circumstances. Anatomists and surgeons overlook these details in a way which is only equalled by the contempt of the physiologist for cerebral circulation as a whole.

In "Ellis pract. Anat." and the more recent editions of "Gray" the quantity is not referred to, but if the above theory be tenable, it must be at least equal in amount to the quantity of blood expelled at each cardiac contraction. If less or absent, the result must be a predisposition to, or an actually diseased condition, though no doubt the circulation would be carried on by some compensating effort on the part of nature. If absent, as in hypertrophy of the brain, then it cannot be admitted that the large arteries at the base of the brain alter much in caliber during the cardiac cycle. The continuous flow of blood cannot be kept up by their contraction but by the general tension in the vessels outside the skull; and under such circumstances there would be no jugular pulsation. Within certain limits the circulation would be normal, but a pathological state would be more readily induced than with a due amount of fluid.

As to distribution, it is most plentiful at the base of the brain (Ellis' pract. Anat. page 179) which is consistent, for if the arteries at the base of the brain receive as much blood as is forced out of the veins on the cerebral surface, as we have suggested, then there is as much fluid displaced from the limited part at the base as is forced on to the more extensive surface, and consequently the subarachnoid space at the base must

be larger. The contracted state of the arteries after death would,
to some extent resemble the condition of affairs immediately before systole: the fluid would be more plentiful then at the base than immediately after systole.

But in cases of trephining the results are contrary to what we should expect. If pressure be exerted on the subarachnoid fluid, why does it not escape when the dura mater with the arachnoid membrane has been opened into? But the question comes with equal force if we accept the other view that the cerebrospinal fluid is forced into the spinal canal. In operation on the brain I have not been able to find any reference, in textbooks, to the escape of fluid and the natural conclusion is that it does not occur, or very slightly. If this is so, the explaination may be, that when the bone is removed the atmospheric pressure possibly may allow the brain to subside in the cranium so that the arteries may have room to expand without raising the fluid. On the other hand it is said that the substance of the brain often bulges out into the opening. In fracture of the base however, there is an escape of fluid though not pulsatile – its non-pulsatile character being due probably to the minuteness of the fissure combined with its distance from the external meatus.

Lastly, that the pressure exerted through the arteries is sufficient to empty the veins is obvious, for the force which expands the arteries must be enough to compress the veins to which it is equally communicated through the medium of the subarachnoid fluid.

It would be presumptuous to speculate on the nature of convulsions, but when so many different views are held of a particular disease, the mind naturally leans towards some one or other; and that which seems to us most reasonable is Dr Wilks belief that it is an affection of the whole encephalon, and that the exciting cause is in most instances high arterial pressure. (B. M. J. 1887, page 766. Broadbent)

If the cerebral veins are normally subjected to pressure what is there more natural than to suppose that this physiological process may, like so many others, be carried to an extreme and become pathological. The brain circulation must be more readily obstructed if compression of the veins is an essential feature in its maintainance than if it were carried on by the movement of the cerebrospinal fluid, for the latter would ~~not~~ need to be more or less expelled from the cranium before the venous outlet could be obstructed.

The statement that "the fits are the disease .... and that underlying them there is no essential disease which can be conceived to exist independent of them" (Hilton Fagge page 679) leaves the mind unsatisfied. That it is simply "the dissipation of accumulated energy" (page 689) does not seem probable, for how then can the "Status Epilepticus" continue for hours without intermission and end in death purely from energy stored up? and that, under conditions when it cannot be replaced. To interrupted attacks it might be applicable.

To the "centre" theory we can only state that no centre has yet been found, the moderate stimulation of which ever produces convulsions; whilst, on the other hand there is scarcely a centre which, if severely stimulated, does not produce convulsion. Neither is there any special centre or set of centres associated with the loss of consciousness, the essential phenomenon in Epilepsy. The "loss of consciousness" points to a general affection of the gray matter; and the "status Epilepticus" indicates the existence of an irritant or exciting cause continuously applied: and both these may be accounted for by high arterial generally, either compressing the cortex (Broadbent) or interfering with its blood supply. A predisposition to convulsions is insisted on, and no doubt rightly, but what is the predisposition in the previously healthy man who, after depressed fracture of the skull, lives and becomes epileptic? And what is the exciting cause? The depressed bone is not the exciting cause for it is always present, without variation, yet the fits are not continuous. Does it not rather appear that the depressed bone is the predisposing cause which favours alteration in the general cerebral circulation, the exciting cause being high arterial tension in the body generally, whatever induced by.

I have seen only one case of convulsions. J. G. aged 12, had Scarlet fever, followed by nephritis. Attended by my principle. Convulsions suddenly set in one day and on my arrival the boy had been unconscious for one hour but the convulsive movements had almost ceased and in ½ an hour there were signs of returning sensibility. The pulse was extremely soft though rapid and I felt that this case at least did not bear out the pressure theory. A week later I again saw him & the tension was then as high as it had previously been low. Was the low tension then, the result of natures attempt to throw off the attack, and an indication for a special line of treatment? The Nitrite and Bromide of sodium were prescribed.