THE INFLUENCE OF HIGH ARTERIAL PRESSURES UPON THE BLOOD-FLOW THROUGH THE BRAIN.

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THE conditions controlling the circulation of blood in the brain are peculiar, and offer an intricate physical problem the solution of which has been attempted from an experimental as well as from a purely theoretical standpoint. The fact that the brain is contained within a rigid box that does not permit free expansion of the organ, has led some authors to assume that dilatation of its arteries, however produced, is not followed by an increased flow of blood, as is usual in other organs, but on the contrary, under certain conditions at least, by a lessened flow. Thus, Geigel has held upon theoretical grounds that dilatation of the arteries of the brain is accompanied by a compression of the capillaries, owing to the fact that the expansion of the arteries causes an increase in intracranial pressure that is transmitted to the capillaries. Upon this view, therefore, dilatation of the arteries, whether produced through vaso-dilator nerve fibres or by a rise in general arterial pressure, should be followed by a lessened flow of blood through the brain, and vice versa.

Grashey in his well-known treatise upon the hydro-statics and hydro-dynamics of the cerebral circulation has also admitted a similar possibility. Grashey assumes that intracranial pressure depends upon two conditions, namely, the volume of the cerebro spinal liquid and the amount of arterial pressure. Dilatation of the arteries must lead to an increased intracranial pressure, and this being transmitted through the brain substance acts upon the part of the circulation where the intravascular pressure is least, that is, the cerebral veins. Inasmuch as the pressure within the capillaries is greater than in the veins, an increase of intracranial pressure should affect the veins and not the capillaries, as Geigel assumed. Moreover, Grashey emphasizes a fact that other authors seem sometimes to overlook, namely, that the sinuses of the brain are probably entirely protected from any direct influence of intracranial pressure by their tense and inextensible covering of dura mater. From his theoretical standpoint Grashey concludes that an increase in arterial pressure causes a greater flow
of blood through the brain up to a certain limit only. So soon as intracranial pressure is raised by the expansion of the arteries to such an extent as to cause occlusion of the cerebral veins, the volume of blood circulating through the brain is diminished. There follows under these circumstances what Grashey calls a vibration of the veins. The occlusion of the veins by the intracranial pressure is overcome by the consequent rise of static pressure transmitted through the capillaries, and this in turn is followed by occlusion as the pressure in the patent veins falls, and so on.

A similar view has been held by other authors who have treated the subject from a theoretical standpoint, and by some of those who have investigated the matter experimentally. The differences of opinion seem to be mainly as to the level of arterial pressure at which an obstruction to the blood-flow occurs, whether it falls within the range of normal variations of pressure or is reached only under extraordinary conditions. This admission appears to be of the nature of a theoretical concession to the recognized peculiarities of the cerebral circulation, and it is of interest to inquire how far it is supported by actual experiments. The simple and direct method of solving the problem is to measure the outflow of venous blood from the brain under different conditions of arterial pressure. This method has been employed by a number of authors with results which seem to be uniform and quite opposed to the conclusion obtained from a theoretical consideration alone. The experiments carried out by Gaertner and Wagner, and since practically repeated by Bayliss and Hill, Hill and Nabarro, Reiner and Schnitzler and others, have shown conclusively that the flow of blood through the brain is increased as the general blood pressure is raised. Even maximal blood pressures produced by the action of strychnine or absinthe give only an increased outflow from the cerebral veins with no indication of even a temporary slowing.

While these experiments have shown quite conclusively that a rise of blood pressure, so far as it can be produced by the normal regulating mechanisms of the circulation, fails to produce the condition of a diminished blood-flow as demanded by theory, it is held by some of the authors quoted that if the arterial pressure be still further increased, a point must be reached at which, owing to the compression of the cerebral veins, a permanent or temporary diminution of blood-flow will result. Thus Hill says, "It is, however, possible that a very sudden and abnormally high rise of arterial pressure should so expand
the arteries at the base of the brain as to temporarily express capillary areas and produce anaemia."

It has been the object of my experiments to determine whether or not this is true. For this purpose I have used the simplest possible method. Upon dogs I have connected the arteries of the brain with reservoirs of blood, or Ringer's solution isotonic with the blood, that could be placed at any desired height, while the outflow from the brain was caught in a large test tube suspended by a spiral spring so that its movement, as it filled with blood, could be registered upon a kymographion. The details of the experiments were as follows: The animal was bled to death under ether. The internal carotids were dissected out and ligated. The vertebral arteries were exposed, and cannulas filled with defibrinated blood or isotonic salt solutions were introduced. These cannulas were connected with the reservoirs of carefully filtered calf's blood or Ringer's solution. In some cases the inflow cannulas were placed in the aorta or subclavians instead of in the vertebrales, and the flow directed into these latter arteries by ligating the other branches. To catch the outflow of blood from the brain, cannulas were placed directly in the superior cerebral veins at their emergence from the skull, or in some cases in the external jugular vein after ligating all communicating branches except the two superior cerebral veins. The internal jugular veins on the two sides were ligated close to the skull. An attempt was also made in some cases to shut off the outflow through the occipital sinuses into the spinal plexuses, but as this involved some danger of altering the conditions of pressure within the skull the attempt was abandoned. In the experiments as made, therefore, two paths of exit were opened to the blood flowing from the brain,—one, which was not measured, into the spinal plexuses, and one through the transverse sinuses and superior cerebral veins. The outflow from the latter was measured. This sufficed for the purposes of the experiment, inasmuch as in the dog this latter path is the one through which most of the blood escapes, and the object of the experiment was simply to determine the effect of very high arterial pressures upon the rate of outflow from the sinuses. The outflow cannulas from the two sides were united to a short tube by means of a Y piece, and this tube opened into the test tube mentioned above. This test tube was provided below with an opening through which it could be emptied rapidly when desired. The tube was swung by a spiral of German silver wire after the manner sug-
gested by Bowditch for plethysmographic records, the spiral being so adjusted that the level of the liquid in the test tube remained constant. The movements of the tube as it filled with blood were recorded upon a kymographion upon which also a time record in seconds was taken simultaneously. As the movement downward of the test tube was constant and could be calibrated beforehand, data were obtained for calculating exactly the volume and the velocity of the outflow from the brain.

In performing an experiment two reservoirs were connected with the inflow cannulas; one of these was placed at a sub-normal level of from 30 to 60 mm. of mercury, while the other was at a height sufficient to cause a pressure of from 300 to 500 mm. Hg. The pinchcocks connected with the lower reservoir were first opened and the rate of flow was determined at this level. This pressure was then changed suddenly to the higher level by opening the pinchcock connected with the upper reservoir and closing the one on the tubing from the lower reservoir. The pressure was then brought back to the original amount by again making connections with the lower reservoir. The experiment was usually repeated a number of times. So long as the pressure was kept low, the rate of outflow remained constant or nearly constant for some time, but exposure to the very high pressures brought about quickly an oedematous condition of the brain which diminished markedly the total outflow or might even suppress it entirely when the arterial pressure was subsequently lowered.

The direct results of the sudden change from sub-normal to supranormal blood pressures were, however, the same in all cases; the venous outflow was increased at once to a proportional amount, and there was never any indication on the record of even a temporary blocking of the flow through the brain. The circulation through the brain under these conditions behaves in fact precisely as it does in the other organs of the body that are not enclosed in rigid cases.

The nature of the results obtained are indicated by the accompanying figures (Figs. 1 and 2) which are reproduced from the curves of two of the experiments. Examples of the actual amounts of outflow as calculated from the records are as follows:

Exp. 1. Small dog — bled to death — blood defibrinated (140 c.c.) and mixed with equal volumes of normal saline and Ringer's solution to make 4 litres. Inflow cannulas placed in the vertebrals, outflow cannulas in the superior cerebral veins just at their emergence from the skull.
High Arterial Pressures upon the Blood-flow.

Arterial pressure of 30 mm. mercury = outflow of 7.02 c.c. per min.
" " " 60 " " " " " " 18.03 " " " " " " 102.66 " " " " " " 10.24 " " " " " " 2d series of observations on the same animal.
Arterial pressure of 60 mm. mercury = outflow of 9.65 c.c. per min.
" " " 60 " " " " " " 8.14 " " " " " " 5.85 " " " " " "

Exp. 2. Small dog — bled to death from carotid — blood defibrinated (130 c.c.) and mixed with equal volumes of Ringer's solution and normal saline to make 5 litres. Inflow cannulas placed in the vertebrals, outflow cannulas in the external jugulars after ligation all branches except the superior cerebrals.
Arterial pressure of 30 mm. mercury = outflow of 5.26 c.c. per min.
" " " 39 " " " " " " 34.52 " " " " " " 2.22 " " " " " "
2d series of observations on the same animal.
Arterial pressure of 60 mm. mercury = outflow of 1.74 c.c. per min.
" " " 375 " " " " " " 70.2 " " " " " " 1.17 " " " " " "

Exp. 3. Large dog — bled to death from the carotids. For irrigating used freshly defibrinated blood of young calves filtered first through a single layer of muslin and then through four layers of the same. Inflow cannulas in the vertebrals, outflow cannulas in the superior cerebral veins at their emergence from the skull. Mercury manometers were also connected with the torcular and with the sub-dural space at the parietal eminence through trephine holes in the skull.
Arterial pressure of 60 mm. mercury = outflow of 23.4 c.c. per min.
A second determination 5 min. later = 22.23 " " " " " " 231.60 " " " " " "
Arterial pressure of 335 mm. mercury = 231.60 " " " " " "
Rise of pressure in torcular cannula = 36 mm. of mercury.
" " " " cannula in sub-dural space = 3.0 mm. mercury.
Arterial pressure of 60 mm. mercury = outflow of 14.66 c.c. per min.
2d series of observations on the same animal — the flow meanwhile had diminished greatly, owing to leakage.
Arterial pressure 60 mm. mercury = outflow of 1.74 c.c. per min.
" " 460 " " " " 252.72 " " " " " "
Pressure in torcular increased 52 mm. mercury.
" " sub-dural space increased 20 mm. mercury. The want of correspondence between the intracerebral and torcular pressures was evidently due to the brain being forced into the trephine hole in the case of the former, thus blocking off the manometer.

Exp. 4. Small dog — bled to death from carotid. Irrigation liquid was blood of young calves carefully filtered. This blood had been kept over night and had frozen. This fact probably accounts for the unusually rapid diminution in flow as the experiment proceeded, the red corpuscles not passing readily through the capillaries and clogging them. Inflow cannulas in the subclavians, ligatures being so placed as to leave an open path only to the vertebrals; outflow cannulas in the superior cerebral veins at their emergence from the skull.
Arterial pressure of 60 mm. mercury = outflow not measurable with accuracy.

Arterial pressure of 190 mm. mercury = outflow of 28.08 c.c. per min.

Arterial pressure of 320 mm. mercury = outflow of 47.38 c.c. per min.

2d series on the same animal.

Arterial pressure of 60 mm. mercury = outflow not measurable with accuracy.

Arterial pressure of 400 mm. mercury = outflow of 42.12 c.c. per min.

Arterial pressure of 500 mm. mercury = outflow of 50.02 c.c. per min.

Fig. 1. Record of venous outflow from brain under arterial pressures of 60 mm., 380 mm., and 60 mm.

Under 60 mm. Hg. the outflow = 18.13 c.c. per min.

Under 380 mm. Hg. the outflow = 102.66 c.c. per min.

Return to 60 mm. Hg. the outflow = 10.24 c.c. per min.

The time record at the top of the illustration is in seconds.

It is evident from the data given that in all the experiments made, the blood-flow through the brain diminished as the experiment proceeded, and this effect was most marked after the blood vessels had been submitted to very high pressures. The probable explanation of this fact is that the dead capillary walls permitted a rapid filtration of liquid, which rendered the brain edematous. This condition, in-
High Arterial Pressures upon the Blood-flow.

indeed, was apparent to the eye when the brain was exposed after submission to the high intravascular pressures. This variation from normal conditions does not, however, affect the value of the experiments so far as the main point under investigation is concerned. The

![Figure 2: Record of venous outflow from brain under arterial pressures of 60 mm. and 460 mm. (2d experiment.)](image)

Under 60 mm. Hg. the outflow = 1.74 c.c. per min.

" 460 " " " " = 252.72 " " "

The irregularity in the beginning of the curve showing the outflow under 460 mm. arterial pressure, is owing to a stoppage of the drum, as will be seen by consulting the time-record above it.
brain was still in the unopened cranium, and the physical conditions, which have been supposed to cause a compression of the veins and a temporary or permanent slowing of the blood-flow when the arterial pressure is suddenly raised to supra-normal levels, still prevailed. Indeed, the oedematous condition of the brain should have exaggerated this effect instead of counteracting it. Nevertheless, the records show that in all cases where the arterial pressure was suddenly raised to as much as 400 or 500 mm. of mercury, the outflow of blood from the cerebral veins increased promptly, and there was no indication at all of even a temporary blocking of the flow.

When we consider this fact, together with the results obtained by several authors upon the outflow in living animals in which the arterial pressure was raised by action upon the vaso-motor mechanisms, it seems justifiable to conclude that the blood-flow through the brain is always increased by a rise of arterial pressure, no matter how great or how sudden this rise may be, and that a compression of the veins sufficient to block or temporarily impede the blood-flow as a direct result of a sudden rise in pressure in the cerebral arteries is physically impossible. The author can indorse the conclusion drawn by Reiner and Schnitzler from their experiments that a rise of pressure in the cerebro-spinal liquid due to increase of arterial pressure cannot exceed the simultaneous intravenous pressure.

Obviously the authors who have arrived at an opposite conclusion have erred somewhere in the theoretical premises upon which their argument was based. A satisfactory treatment of all the physical factors involved in the statics and dynamics of the blood-flow through the brain is most difficult, and, perhaps, impossible in the present condition of our knowledge; but it seems to the author that in the theoretical considerations of the subject met with in physiological literature, some factors, which explain in large measure the contradiction between the experimental and theoretical conclusions, have been more or less overlooked. In the first place the view distinctly announced by some authors and tacitly assumed by others that arterial expansion causes a compression of the large venous sinuses of the brain seems to the author to be entirely inadmissible. Grashey calls attention to the anatomical facts that make this view improbable. The venous sinuses are covered by layers of the inextensible dura mater tightly stretched in some cases across bony channels. Any one who examines the
condition of these membranous walls in a fresh skull will be impressed with the opinion stated by Grashey, that the resistance to compression at these places must be so great, as compared with the cerebral veins that open into the sinuses, that any local or general increase of intracranial pressure must affect only the smaller veins. It seems to me, in fact, that the walls of the sinuses are practically incompressible, and that their existing structure is a beneficent adaptation to prevent any interference with the venous outflow arising from arterial expansion, since the venous system is thereby protected from compression at the point where its total cross area would be least, and intravascular pressure at its lowest, and where compression might most seriously affect the venous flow.

I have attempted to demonstrate experimentally the practical incompressibility of the large sinuses, but the method that I have used and which was the only one that seemed to be conclusive, developed so many technical difficulties that I have attained so far only partial success. The method was simple in idea, but somewhat difficult of execution. It consisted in first removing the brain entirely by washing out the tissue through the foramen magnum and a trephine hole in the parietal bone. A flexible rubber catheter was then taken, and upon one end was tied a delicate bag, made usually from the outer coats of the intestinc of a frog. The catheter and bag were filled with water from a pressure flask so as to displace all the air, and the catheter was connected by tubing filled with water with a water manometer of barometer tubing. The catheter and bag were then introduced into the transverse sinus through the opening of the superior cerebral vein in the case of the dog, or through the internal jugular when the human skull was used. After the catheter was in position, the pressure in the water manometer connected with it was raised sufficiently to distend the bag and make it lie against the membranous walls of the sinus. Meanwhile the skull had been filled with water, the foramen magnum tightly closed off by a rubber stopper inserted in the sheath of the dura mater, and the trephine hole in the parietal bone connected properly with a mercury manometer and a pressure flask. By means of the latter the pressure within the skull was raised or lowered suddenly to any desired extent, and the effect of this variation in pressure upon the walls of the transverse sinus should have been indicated by the manometer connected with the catheter lying in the sinus.

The experiments of this character made upon human skulls were
not successful owing mainly to the fact that the skulls were obtained from cadavers used in the dissecting room, and the dura mater was so altered as to strip away easily from the bone. In the human skull also there is very great difficulty in getting the catheter through the internal jugular into the transverse sinus owing to the curvature of the channel. In the dog's skull the main difficulty lies in the fact that the transverse sinus throughout most of its extent lies in the bone, only a small portion of it near the torcular having a membranous wall. In other animals, such as the calf and sheep, the transverse sinuses have bony walls throughout their entire course. Another difficulty consists in the fact that the openings of the cerebral veins into the sinuses, particularly into the superior longitudinal sinus, are protected by dura mater so as to remain patent after washing out the brain, and thus make a free channel of communication between the skull cavity and the system of sinuses. This difficulty can be obviated by trephining into the torcular and closing off the sinuses with the exception of the transverse sinus used in the experiment. Still another obstacle is found in the fact that water will filter through the dura mater under high pressures, but the rise of pressure in the sinuses thus produced is gradual and quite different from the sudden rise which would occur if the walls of the sinus were compressed by a quick increase of pressure in the skull cavity.

In one experiment of this kind made upon the dog's skull — and in which subsequent dissection showed that the bag upon the end of the catheter lay properly in the membranous portion of the sinus, it was found that an increase in pressure within the skull up to 500 mm. of mercury caused not the slightest change in the level of the manometer connected with the catheter. This experiment seemed to show quite positively that the membranous walls of the transverse sinus cannot be compressed by intracranial pressures as high as 500 mm. of mercury. Unfortunately corroborative results could not be obtained upon the more favorable human skull owing to lack of appropriate material. But to the author's mind at least the anatomical structure of the sinuses and the physical characteristics of their walls are sufficiently conclusive in showing that normal or supra-normal variations in intracranial pressure cannot affect the calibre of these channels, particularly when we remember that the system of cerebral veins opening into the sinuses offers a relatively very low resistance to compression. As between the cerebral veins and the cerebral capillaries the lower internal pressure prevailing
within the former would seem to ensure that a general rise of intracranial pressure must affect the veins first.

With every increase in arterial pressure there is a tendency to the expansion of the cerebral arteries, but this expansion is only possible when a corresponding amount of liquid is forced out of the brain, the substance of the brain itself being practically incompressible. A clearer understanding of the conditions in the brain has led most authors to believe that room for a sudden expansion of the arteries is made in one of two ways, either by forcing out the cerebro-spinal liquid into the spinal cavity, or by a corresponding compression of the cerebral veins. With regard to the former of these two possibilities exact evidence is lacking of the extent to which it can compensate for arterial expansion, but the facts in our possession would seem to indicate that it plays a minor part. Hill has called attention to the fact that normally the quantity of liquid in the sub-dural and sub-arachnoid spaces is very small, and that under moderate expansion this is expressed into the spinal canal, where room is made for it by expansion of the vertebral ligaments and the possibilities of leakage through the sheaths of the spinal nerves. With a further increase of arterial pressure expansion becomes impossible unless there is a corresponding compression on the venous side, and, as we have seen, this compression should affect first the smaller cerebral veins.

That compression of these veins happens even before the cerebro-spinal liquid is entirely expressed seems to be proved by the occurrence of a pulse in the cerebral veins coincident with the arterial pulse. This venous pulse has been noticed by a number of authors. I have also observed and recorded it a number of times both in the outflow from the superior cerebral veins and from the torcular Herophili. It would seem that this pulse can be accounted for in one of two ways only. It is caused by a wave of pressure transmitted through the capillaries, or it results from a compression of the veins following upon arterial expansion. In the former case there should be a measurable interval between the time of appearance of the pulse in the circle of Willis and in the efferent veins; in the latter case the pulse should be practically simultaneous in the arteries and veins. To determine this point I have made simultaneous records of the pulse in the arteries and veins. The arterial pulse was measured in the circle of Willis by means of a Hürthle spring manometer connected with the head end of the internal carotid; the venous pulse was recorded by a delicate manometer constructed on the principle of
a Hürtthle membrane manometer and connected with the torcular Herophili. This connection was made by means of a brass tube screwed into a trephine hole made into the torcular and filled with sodium carbonate solution.

A specimen of the record thus obtained is given in the accompanying illustration.

![Simultaneous record of the pulse wave in the torcular and in the circle of Willis (through the internal carotid). The upper curve records the pulse in the torcular, the lower that in the circle of Willis.](image)

It was found that the two pulse waves occurred nearly simultaneously, and that in cases in which any difference in time was detectable the venous pulse slightly preceded the arterial pulse, the maximum difference in extreme cases being as much as 0.01 of a second. This difference is sufficiently accounted for by the stretch of elastic artery (internal carotid) extending from the circle of Willis to the point in the neck where the arterial manometer was inserted. These experiments seem to indicate that the venous pulse is not transmitted through the capillaries, but is due to a positive wave of pressure transmitted through the brain substance by the expanding arteries. This being the case it follows a fortiori that a greater general expansion of the arteries of the brain following upon a large rise of arterial pressure should be accompanied by a corresponding compression of the veins. This is the view that Grashey and others have taken, and that has led them to the apparently logical conclusion that a large rise of arterial pressure should result in a compression of the veins sufficient to occlude them temporarily and thus diminish the volume of the blood-flow. The experiments recorded in this paper, however, show that this conclusion is not justifiable. The venous outflow is not impeded even temporarily by a sudden rise of arterial pressure as great as 500 mm. of mercury. It remains for us to explain therefore this apparent discrepancy.

Granting fully that expansion of the arteries causes at once a rise of intracranial pressure sufficient to compress the veins, the fact that the blood-flow is not thereby momentarily impeded may be explained
by two considerations. In the first place in any vascular region the
total cross area of the veins exceeds that of the arteries, so that an ex-
pansion of the arteries, if accompanied by a resulting symmetrical
compression of the veins, would be distributed over a larger cross
section and result in a relatively smaller diminution in calibre of the
single veins. This consideration in the case of the brain is rendered
more important, perhaps, by the fact that the venous system at its
termination, namely, in the sinuses, is protected from external com-
pression. The narrowing of the veins occurs, therefore, in the
relatively wide area of the small veins, and although the total com-
pression of the veins must equal the total expansion of the arterial
stems, the narrowing of the single venous channels is slight, appar-
ently too slight to cause a perceptible obstruction to the blood flow.
In the second place the curve of extension of the elastic arterial walls
under increasing pressures within follows the general law of extensi-
bility for muscular tissue, showing a gradually decreasing extension
for increasing increments of pressure. As the arterial pressure
increases, therefore, the expansion of the arterial walls becomes
rapidly less, the arteries approach nearer and nearer to the condition
of rigid tubes, and the compression of the veins is correspondingly
less important. These considerations suffice to explain why an
actual occlusion of the veins, such as Grashey assumed, cannot
possibly occur, and they explain also, perhaps, why not even a
perceptible momentary blocking of the venous flow is obtained
under normal and experimental conditions.

As to the permanent effects of arterial expansion upon the resist-
ance to the flow in the veins, several authors have demonstrated quite
clearly that even though the compression of the veins were sufficient
to bring about a marked resistance to the blood-flow, this effect could
only be temporary, inasmuch as the rise of static pressure within the
capillaries and veins must always be more than sufficient to again
expand the veins. Hill has expressed this view in the following
language: "The veins and capillaries, therefore, again become
patent, because arterial pressure transmitted directly is obviously
greater than arterial pressure minus the tension of the arterial wall
transmitted through the brain substance." This author, however,
admits that "if the cerebral arteries suddenly expand beyond a
certain limit, a process of temporary self-strangulation of these
vessels (veins and capillaries) takes place. The circulation itself for
a short time stops, and the symptoms of acute cerebral anæmia are
produced." This conclusion, as I understand it, is based entirely upon theoretical grounds, and my experiments show that it is not correct, if it is assumed that the arterial expansion results from a rise of intravascular pressure.

The general conclusion, which seems to me to follow from the experiments given in this paper, is that a rise of pressure, however great, in the cerebral arteries does not cause directly any impediment to the blood-flow either temporarily or permanently. The circulation in the brain behaves in this respect precisely as it does in the other organs of the body; the greater the arterial pressure the more abundant is the flow of blood, and temporary anæmia cannot be produced in this way.

If it is found experimentally that a sudden great rise of arterial pressure causes an injurious effect upon the brain, this result cannot be attributed to the direct production of a temporary cerebral anæmia. We can imagine that under these conditions the functional activity of the brain might be interfered with for one of two reasons. It is possible, in the first place, that the resulting rise of intracranial pressure might affect the brain tissues directly; or, secondly, a rapid accumulation of lymph might take place within the brain, which, for a time, would impede the blood-flow, particularly after arterial pressure had again fallen. There is not much probability, however, that this latter effect could occur. Bergmann found that injecting defibrinated blood at high pressures (800-1200 mm. Hg.) into the cerebral vessels of a horse caused no distinct increase in the lymph flow, and several observers have shown that in other organs arterial hyperæmia alone is not necessarily followed by an increased formation of lymph.

REFERENCES.