CEREBRAL VASCULAR DISEASE: THE SURGEON'S INTEREST *

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Although I am speaking as a surgeon dealing with cerebral vascular disease, it would be truer to say that the cerebral vascular system deals with the neuro-surgeon. In general we can achieve technically only as much as this system will allow us. Tumours, inflammations and injuries all declare themselves by primary or ultimate interference with cerebral circulation, and our manipulative intervention depends for success upon the extent to which we can correct this circulatory distress. In this short discourse I am concerned, therefore, with cerebral vascular disease in the widest sense; that is, whether the circulation is upset directly by blood-vessel pathology, or secondarily by other disease within the cranium.

There are grave difficulties confronting us in our appreciation of the remote and complex vascular effects of any disease process, and even greater difficulties in judging the need for interference, or the best time for intervention, and also in the choice of methods that will accord with natural circulatory requirements.

Since Cushing's time, the advances in neuro-surgery have mainly depended upon better understanding of the dynamic interrelationships between every component in the cranium. Our approach, however, is still in the main far too anatomical and static. As an investigation angiography has helped us by its simultaneous indication of the site and extent of gross disease, but it fails to reveal the more subtle defects of circulation that are allied to the functional rather than to the structural physiology. Its most serious limitation as a test is that it demonstrates a state of the moment and is therefore unrevealing of the more subtle qualities of reserve and adaptability of the brain as a whole under varying stress conditions.

A further fundamental difficulty in intracranial surgery is that what we assume to be a state of balance within the closed cranium has to be reviewed under circumstances of open operation, where the pressure equilibrium of the blood and cerebrospinal-fluid systems are immediately altered providing a vent to the atmospheric exterior, or the alteration of intervening tissue resistances.

CEREBRAL ANEURYSM

I will take as an example of the effects of this change of habitat the cerebral aneurysmal sac. Most surgeons have, more often than they care to remember, experienced the spontaneous rupture of a sac when it has been stripped naked of its surrounding tissue and fluid support, even before the sac itself has been subjected to handling. A not dissimilar example is the more frequent occurrence of peri-arterial haematoma after carotid arterial puncture in cut-down techniques of angiography as opposed to the percutaneous methods, where the integrity of the surrounding tissues and muscle tone have been maintained.

These experiences have made one feel, especially with aneurysm, that the extra-arterial pressure environment is of paramount importance. In the intact cranium this environment could be expected to be a natural medium for the control of a pressure gradient across the sac wall, so as to prevent its thinning and rupture. This indeed must happen, considering the surprisingly long life of a congenital sac before it ruptures. In the same way as an adverse pressure gradient can be rapidly induced at operation, so in a natural way it can arise within the intact cranium as a result of more insidious aging or of disease which produces softening of tissues by demyelination or the lessening of turgidity of these tissues by dehydration or other means. It is probable that we have under-estimated the importance of those changes which predispose a sac to sudden thinning and rupture upon the application of a minor transient rise of the intrinsic blood pressure.

On the other hand, immediately after rupture the external pressures tend to rise by the formation of surrounding blood clot, cerebral oedema, venous hypertension, and raised pressure of the cerebrospinal fluid. This readjustment by nature serves in many instances to check the extravasation of arterial blood, even while the hiatus in the sac remains potentially patent; otherwise bleeding would be more destructive than it usually is. Were these natural defences to continue long enough, the weak spot in the sac would have time to heal; but should they melt away prematurely, the reversal of the pressure gradient would allow a subsequent bleed to occur.

If these arguments are valid we could devise a more rational perspective of approach to the management of aneurysms. Their application would be more helpful than statistical guides in determining the prognosis of a given case. For instance, it would be better to sustain these natural defences and initially withhold surgical intervention after a first bleed. For the first few days at least we should try to maintain in the closed cranium for as long as possible a reasonable state of hypertension above that of the estimated intrasacular tension (i.e., the normal blood pressure). The longer we can maintain this balance the more likely is the sac to reach a safe stage of healing. If we find on lumbar puncture that the intracranial pressures are dropping too rapidly to normal or sub-normal, we should realize that this is a case very likely to bleed again before the healing of the sac has progressed sufficiently. In this interval, then,

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we should declare for a more direct attack upon the sac without reference to arbitrary timing.

Surgical Practice

It would be fair to review practical current opinion at this point. Provided the sac is accessible in position and capable of isolation and the general intracranial conditions will allow a safe approach, environmental considerations can be ignored in favour of immediate direct surgery.[1] Usually, however, the state of the brain is such that for many days early surgery creates a morbidity as great as the mortality caused by waiting. Norlén and Olivecrona[2] would therefore advocate conservatism even into the 2nd and 3rd week.

Our writings and thoughts up to now have been preoccupied with the position of the sac from the point of view of its surgical accessibility, the intrinsic pathology of its wall, and its internal pressures. Perhaps we have not put sufficient emphasis on the extrinsic conditions influencing the environment, and the possibilities of developing an intelligent management of these external factors. Until we have learnt more about the assessment and management of the environment of the aneurysm along these lines, the ideal treatment remains the direct isolation or obliteration of the sac or, failing that, the reinforcement of its wall from outside by the use of muscle, different fibre materials, acrylic resin, and so on. Unfortunately, the inaccessibility of the aneurysm sometimes makes the direct attack impossible. Certainly this is so in many aneurysms in the early stages up to about a week after a 'massive bleed', when high intracranial pressure, cerebral oedema, and the obscuring of cleavage lines by blood pigment persist. The anterior carotid aneurysm in particular, while reasonably accessible, is so often fusiform that isolation is not possible without risking the obliteration of its parent anterior cerebral vessels, and this in particular remains our 'problem child'. Here we often have to resort to the reinforcement of the wall by the materials mentioned above, and in large measure one feels that this manoeuvre is not so satisfactory as we are inclined to think at the time we carry it out. In the majority of instances there is no very good evidence that the sac is in fact reinforced; muscle does not seem to graft naturally upon the exterior of an artery and I do know that quite a few of our cases have subsequently bled fatally from the same site.

It is not feasible to reduce the intrinsic blood pressure of the sac for any length of time. We may do this temporarily by using a hypotensive technique at the time of operation. But this supportive method is, to my mind, fraught with very real dangers to the general cerebral circulation, the complications of which are coming more and more to our notice. Hypothermia is a much more physiological adjunct in this regard. Attempts to reduce the blood pressure for a longer period of time by carotid ligation are also in the main unsatisfactory substitutes for the direct attack. Supposing that the systolic pressure could thereby be reduced to an effective level for a period long enough for the sac to heal, it could not be expected to maintain an adequately low pressure with safety for an indefinite period, and two questions in this regard remain unanswered:

1. Is an adequately low pressure in fact produced and maintained for any sufficiency of time?

2. If it were in fact so provided, would that not mean in itself that there is an inadequate collateral supply to a lesser or larger portion of the brain, so that, even if no symptoms of distress appear initially, the patient's reserve in the face of subsequent aging or disease or stress may be seriously prejudiced?

COLLATERAL CIRCULATION IN THE BRAIN

Having hinted at the global aspects attending this focal vessel disease I pass on to consider another total vascular mechanism, the understanding of which allows us to manage problems involving occlusion of major cerebral arteries.

It has been noted repeatedly that after deliberate or spontaneous occlusion of a major artery, such as the middle cerebral or internal carotid artery, there are often surprisingly few symptoms of brain disturbance in proportion to the distribution of the vessel concerned—sometimes no symptoms at all. It is by no means inevitable that an infarct of any degree should occur in any part of the brain to which the affected vessel is assigned. If it were that the collateral supply depended upon the subsequent opening up of new channels, either to link the proximal to the distal branches of the blocked artery, or from neighbouring arteries to the distal branches, a time lag would occur which would certainly lead to recognizable neuronal death. On the contrary, compensation is usually so rapid and complete as to suggest that the requisite channels are patent and available to the blood flow at the moment of the insult to the major vessel; all that is needed to ensure the maintenance of proper metabolism is to alter the direction of blood flow and impel it with sufficient linear velocity. It might be said that 'we live normally upon our cerebral collateral circulation'; so that we have the almost maximal reserve immediately available in the event of an insult.

A large-scale example of this principle is provided under normal conditions by the existence of a separate flow within the vertebral and carotid systems, although these are linked together by a widely open posterior communicating artery and the circle of Willis.[3] The separate streams do not mix and the blood in the posterior communicating artery has no linear velocity. Any greater call upon the circulation of one or other of these systems is followed by an immediate directional change of flow through the posterior communicating artery, maintained with the full force of the blood pressure. Beever,[4] in 1909, made the postulate that collateral supply depended less upon opening up of branches of an occluded artery than upon the opening of links from adjacent vessels, and recent angiography studies support this contention by showing, in occlusion of the middle cerebral artery, the filling of the distal branch from adjacent collaterals distal to the block.

Use of this mechanism, for the most part unwitting, has stood the surgeon in good stead where he has been forced, for one reason or another, to occlude a major vessel, and has enabled him to operate more physiologically, yet in wider scope. The principle applies in varying degree in the relationship between the vascularity of a cerebral tumour and that of its matrix, survival or death of portions of the tumour being influenced by the effect of surgery or radiation therapy upon the collateral circulation remaining to the tumour. In a reverse sense the principle also applies to arteriovenous malformation where, by virtue of large open redundant collateral channels, blood-robbing takes place from the normal arterial fields, and where our clue to the surgery of these defects would be to interrupt these redundant links (providing

they can be identified and isolated) rather than attempt wholesale occlusion of the normal feeding parent vessels.

In general, at the time of the insult to major vessels, conditions may exist which prevent the collateral supply from remaining effective. Compression of the major or minor collateral channels by space-occupying lesions, such as intracerebral clots, cerebral oedema, or excessive intracranial hypertension, may have the effect of delaying or limiting adequate collateral blood flow; and here the surgeon has an active role to play to offset these adverse conditions as soon as possible—a role which extends into more medical conditions such as the encephalitides. Sometimes a very dramatic and sudden improvement occurs in a hemiparesis or an aphasia, for instance, after the reduction of intracranial pressures by single or repeated lumbar punctures or ventricular tappings, an improvement on occasions so immediate that vasospasm is unlikely to be the sole cause of the deficiency. Vasospasm on the whole would appear to be a secondary form of adjustment rather than a primary,[5] although it must be admitted that in operations on aneurysms where the vessels have been directly handled severe arterial vasospasm has been clearly observed, and in turn lessened by the application of local vasodilators such as papaverine. Circulatory improvement following active decompressive measures is probably due to the re-establishment of a favourable balance between the existing arterial blood pressure and the pressure outside the arteries, and the consequent reduction in the resistance to the blood flow.

VENOUS CEREBRAL CIRCULATION

Finally I want to review and discuss briefly a perspective of the venous cerebral circulation. The venous circulation is in every way as important as the arterial. The volume of the venous blood lake in the brain is greater at times than the arterial. This fluid volume becomes an important component of the balance in pressure systems throughout the body as a whole, and is influenced by the regular and irregular pulse waves which reach it from outside the head—from the heart, lungs and even the abdomen, and also from pressure changes in the atmospheric medium in which the body finds itself. These pressure waves are modified by physical factors according to the calibre and nature of the channels of communication that exist between the cranium and these outside systems. The venous 'lake' serves to spread, absorb and further modify these pressure fluxes, doing so in conjunction with the cerebrospinal fluid volume, so helping to regulate the phasing and summation of the primary and reactive pressure waves which tend to pass across, or hammer at, or distend the brain tissue and vessel walls which intervene.[6]

Dynamically speaking, the brain can be looked upon as a complexly involuted membrane situated between the various pulsatile fluid systems. Whereas the force of the arterial pulse is translated into linear velocity within its strong muscular channels, this does not happen to the same extent in the veins, where the force tends to dissipate in all directions, so producing a greater immediate effect upon the enveloping brain tissue. Largely through the venous cerebral blood the elastic brain sponge becomes a passively activated 'pump',[6] which in turn becomes an important mechanism for the promotion of linear flow to the cerebrospinal fluid and to the general blood circulation. In conditions where the dynamic equilibrium is upset by disease, either inside or outside the venous system, the phasing of the pulse waves may become such that the passive cerebral pump is rendered neutral or adynamic. This state of affairs can be seen in extremes of either high or low intracranial pressure and where large portions of the brain become impacted by dislocation under a fixed structure like the falx, as may occur in subdural haematomata. If this neutral adynamic state were to exist for any length of time, stasis of the cerebrospinal fluid and the venous and capillary blood would occur, resulting in varying degrees of cerebral ischaemia. If venous hypertension were also present, cerebral oedema would eventually build up.

Again, active measures can break this vicious cycle by the removal of compressing or dislocating lesions, thereby reconstituting the normal tissue resilience. Further, the free flow of venous and cerebrospinal fluids can be regulated by the provision of appropriate communications between one pressure system and another or by a vent to the environmental atmosphere. Raised intracranial pressure is a relatively infrequent accompaniment of arterial hypertension, but it is an almost inevitable result of venous hypertension. In our everyday surgery we associate the occurrence of high cerebrospinal fluid tension with high venous tension. Lowering the one lowers the other, and by controlling these two factors we can limit or prevent the development of malignant forms of oedema—the greatest obstacle in brain operations. It is for these reasons that good anaesthetic skill has become so vital to the success of the surgeon, especially in the prevention of venous congestion.

CONCLUSION

I hope that, by using these examples, I have brought out perspectives and principles which I personally have found useful. They are dynamic complexities which more often than not cannot be easily appreciated. Where we have failed beyond a reasonable measure to control these dynamic complexities, our surgery has remained limited or unsuccessful. Unfortunately we can never quite reconstruct the reasons for our errors, and even at post-mortem examinations, where dynamic influences have ceased to act, they remain for the most part unrecognized. The broken threads of observation keep bearing upon our judgment and intuitive actions more and more as experience grows, giving rise to conceptions which are hard to command at will and still harder to teach. They all refer fundamentally to the cerebral circulation and force us to conceive this in its completely holistic context. It is here, of course, where practice and theory do not easily meet together and where the essential limitations of the surgeon's craft, basically one of tissue reconstruction, are most manifest. Surgical craft is therefore advancing beyond mere reconstruction of tissues to the regulation of intracranial dynamics as a whole. Since the human is the ultimate subject of this dynamic research and its application, and the neurosurgeon is the mediator bound in all his actions by ethical and sociological considerations, the neurological surgeon has taken his place as the ecologist of the nervous system.

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