RES MEDICA
Journal of the Royal Medical Society

Spontaneous Subarachnoid Haemorrhage: Some Aspects of its Pathogenesis and Management

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Abstract

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1. Rupture of an intracranial aneurysm responsible for 80% of cases.
2. Arteriovenous malformations responsible for 10% of cases.
3. Other diseases (e.g. neoplasm. blood dyscrasia)- responsible for I 0% of cases.

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ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)
Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Summer 1959, 1(4): 10-14
doi:10.2218/resmedica.v1i4.299

Spontaneous Subarachnoid Haemorrhage: Some Aspects of its Pathogenesis and Management

Based on a Dissertation read before the Royal Medical Society on Friday, 17th October 1958.

By F. W. TURNER

The recognition of spontaneous subarachnoid haemorrhage as a clinical entity, distinct from the many other conditions all previously grouped under the comprehensive heading of " Apoplexy " was first made by Collier in 1922. A year or so later Symonds reviewed 124 cases described in the literature and concluded that rupture of an intracranial aneurysm was probably the major cause of this syndrome. His opinion is now widely held, and it seems probable that on a proportionate basis the main aetiological factors are:—

1. Rupture of an intracranial aneurysm— responsible for 80% of cases.
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3. Other diseases (e.g. neoplasm, blood dyscrasia)— responsible for 10% of cases.

The content of this article is largely referable to spontaneous subarachnoid haemorrhage following rupture of an intracranial aneurysm.

The importance of this syndrome is now well recognised, and recourse to the figures of the Registrar General shows that ruptured aneurysms are responsible for about 2% of all deaths from cerebro-vascular disease. While an over-all sex ratio of one to one is generally agreed upon, there is less unity of opinion concerning the age incidence. It seems probable however that the peak incidence occurs in a somewhat older age-group than the widely taught " twenty to forties," and most writers are now agreed that " forty to sixty " is a more accurate estimate — the neurosurgeons tending to see younger patents than the neurologists as a result of some degree of selection of cases referred for surgery.

Since the work of Carmichael in 1950 the pathogenesis of intracranial aneurysms has been better understood. Although still commonly called ruptured " congenital " aneurysms, with the implication of sudden rupture in young persons, they are really acquired lesions occurring at the site of a congenital weakness, and not usually causing symptoms until later life. Histological evidence indicates that they arise as the result of the interaction of two factors:—

1. There is an arteriosclerotic lesion of the intima with degeneration of the elastic lamina;
2. There is a focus of medial aplasia which may be substantially enlarged by superimposed degenerative changes, although the developmental deficiency is the dominant lesion in this layer.

The precise combination of lesions varies greatly from case to case, but both developmental and degenerative factors are concerned in the genesis of

all these aneurysms, and no valid distinction can be made between the so-called congenital and arteriosclerotic types.

It would also appear from evidence obtained at operation and later histological studies, that the rupture of an aneurysm is a two-stage process. There is initially a dissecting process affecting the expanding wall of the aneurysm which results in seepage of blood into the subarachnoid space or into surrounding brain substance. This seepage is however insufficient to prevent further distension of the sac which becomes progressively thinner and may develop small daughter aneurysms. Eventually there is total rupture of the sac with extravasation of much greater quantities of blood; if the aneurysm is applied to brain substance there will be considerable destruction of cerebral tissue already softened by the initial leak of blood. This second episode of haemorrhage which is usually considerably more damaging than the first, commonly occurs between the second and twentieth day of the illness.

The above description is of course a generalisation and there are many cases which do not follow the pattern described. Notable among these are the many cases who apparently never experience a minor episode of bleeding but succumb early to a massive extravasation of blood. This is an assumption and must remain so, for these patients are usually unconscious when first seen and commonly remain unconscious. It is therefore not possible to take any history of premonitory symptoms in these cases.

This general rule of an initial small haemorrhage followed after a variable period by a more severe bleed is none the less of extreme importance, for the early recognition of the minor episode for what it is may save many lives. Such recognition is not always easy, and for the busy practitioner it is all too tempting to dismiss a sudden occipital headache, which clears up after a day or two with no residual neurological defect, as due to influenza or sitting in a draught or some such facile explanation. The more obvious cases with neurological deficiencies (e.g. ophthalmoplegic migraine) are less likely to be missed, but if care is taken to test all cases of sudden, severe, unexplained headache for signs of meningism then the diagnosis will usually become obvious. The finding of neck stiffness is particularly important and probably is found in 85% of cases. Absolute confirmation of the diagnosis may be made by the finding of an homogeneously blood-stained cerebrospinal fluid which is under increased pressure. Lindsay in 1950 pointed out that a loss of as little as 3 mls. of blood into the c.s.f. gives approximately 100,000 red cells/cu.mm. of c.s.f., which will appear as heavy blood staining.

The likelihood of a recurrent bleed (or major episode) within three weeks of the ictus has been variously estimated by different authors as between 19 and 45%, but it seems likely that the higher figure is the more probable. This then is the initial period when surgery must be considered, for as previously mentioned the second bleed is likely to be the more serious, and if not resulting in death may well result in neurological deficiencies in the form of hemiplegia, visual or speech disturbances etc., depending to a large extent upon the site of the aneurysm.

Before considering treatment some mention must be made of the prognosis. Many physicians still consider the outlook to be one of gloom and impending death, and may indeed advise their patients so. Such a prognosis is based on ignorance. Walton (1956) collected 1,480 cases and showed that the overall mortality within the first eight weeks of the illness was approximately 45%, the majority of these deaths occurring in the first three week period. Of those surviving the first eight week period about 20% will die of a recurrent bleed at a later date—often within the first six months. This leaves 44% of cases alive and mostly well, although about one third will be to some extent disabled by residual paralysis, epilepsy, headache and often anxiety.

Such a prognosis is based on conservative management. Even better results are obtained by the surgeons, but their results do not give a complete picture for there must inevitably be some selection of cases forwarded for surgery. And besides, in many parts of the world (and even the British Isles) the services of a neurosurgeon are not always readily available.

Treatment

Surgical therapy is not yet universally established, and many cases will continue to be treated conservatively; hence the technique of conservative management is still of considerable importance.

In treating patients conservatively the prime essential is strict bed rest in hospital with careful nursing care. It is probably safest to enforce bed rest until four weeks after headache and neck stiffness have disappeared. Initially the head of the bed should be raised in order to drain blood into the theca. The relief of headache is important and there is still considerable argument as to the best analgesic. Pethidine is advocated by many, and codeine often relieves the milder headaches. The use of morphine is questioned because of a theoretical tendency to raise the intra-cranial pressure, but Walton used morphine in one hundred cases without detriment. Barbiturates are almost invariably unsuccessful.

The headache which occurs in sub-arachnoid haemorrhage is principally the result of the raised intracranial pressure with resultant stretching of blood vessels and meninges. Hence a reduction of pressure helps to relieve headache and may also prevent the development of pressure lesions. The methods available for reducing intracranial pressure are two fold: (1) lumbar puncture, (2) detensifying therapy.

The use of lumbar puncture is also the cause of considerable controversy. Merritt in 1938 advocated twice daily reductions in pressure. Schwartz in 1948 recommended the avoidance of lumbar puncture except for diagnosis. Others such as Sahs and Keil (1943) have suggested that while there is no harm in repeated lumbar puncture neither is there much benefit. The modern view would appear to be that in the average case lumbar puncture should be performed for diagnosis only; but where there is continuing severe headache, restlessness, meningeal irritation or prolonged coma then it may be repeated with slow reduction of pressure to about 100-150 mms. of water. There is no evidence that lumbar puncture causes recurrent bleeding, but there are the ever-present dangers of tentorial herniation or the production of a cerebellar cone. Papilloedema by itself is not a contra-indication since it may be produced by bleeding limited to the sub-arachnoid space.

Detensifying therapy is not of proven value, although it may be effective in decreasing very high intracranial pressures. In many cases the effect is short-lasting and the pressure returns to the previous, or even higher, values. The methods used include the intravenous administration of 50% sucrose, and rectal infusions of 10% magnesium sulphate.

Nutrition and hydration must be maintained. This may prove difficult in the comatose patient since intravenous fluids should be avoided for fear of raising the intracranial pressure or causing pulmonary congestion. Tube feeding may become necessary but most patients recover sufficiently to take food by mouth before the problem becomes urgent. The use of laxatives to prevent straining at stool is important, and the bedside commode may well require less effort than the bed-pan.

The possible advantages of hypotension and hypothermia are interesting Hypotension might be considered of use in the prevention of recurrent bleeds, but it is probable that such an advantage is more than outweighed by the danger of anoxic damage to cerebral tissue. It must be remembered that vascular spasm with the production of small infarcts is one of the commonest complications of sub-arachnoid haemorrhage, and the production of pro-

longed hypotension would undoubtedly increase this risk. Hypothermia by reducing the rate of cerebral metabolism would seem to offer greater advantages.

As in all patients confined to bed for long periods, great care must be taken to prevent renal and pulmonary complications and should they occur prompt therapy must be instituted. Passive physiotherapy should be given to prevent deep venous thrombosis, for should it become established treatment with anticoagulants is obviously not advisable.

The role of surgical treatment has grown apace. In 1934 Ayer wrote that: " Sub-arachnoid haemorrhage has little interest from a standpoint of active surgical procedure." Sixteen years later Falconer is to be found advocating that: "All cases should be investigated and treated surgically." Two factors have been of the greatest importance in this radical change of opinion. Firstly the development of cerebral arteriography by Moniz in 1927 has enabled the accurate localisation of many aneurysms. In 1957 Gillingham showed that with careful bilateral carotid and vertebral angiograms it was possible to demonstrate the lesion in 65% of cases of sub-arachnoid haemorrhage. Nonetheless in about 35% of cases no abnormality is demonstrable and it must be presumed that either the responsible lesion is too small to be outlined, or else it has undergone thrombosis with the " apparent spontaneous healing " described by Falconer. Fortunately however negative angiography indicates a good prognosis. The use of angiography, together with a reduction in operative risk following the introduction of controlled hypotension and hypothermia, has led to a great improvement in surgical results.

The details of surgical techniques may be read elsewhere. Suffice it to say that two methods are available: —

1. Carotid Artery Ligation.
2. Direct Intracranial Attack.

Both methods were pioneered by Dott in the early nineteen-thirties and many of the refinements of technique have come from the Edinburgh School. Direct intracranial attack is probably preferable with clipping or ligation of the neck of the sac; where an adequate collateral circulation is available clipping of the proximal limb of the artery of supply is probably a more reliable technique.

The results of surgical treatment will depend upon the site of the lesion, the time since bleeding occurred, the technique used and of course the condition of the patient. Gillingham in a personal series of eighty patients had an overall mortality of 21%. Like many others he found that the operative mortality was three times as great in those patients treated during the first three weeks after bleeding began, as in those where treatment was delayed. However in a later series of forty-seven patients managed by carefully judged expectant and operative treatment he obtained an overall mortality of 10.9%, although the operative mortality remained at his previous figure of just over 20%.

A direct comparison of medical and surgical results comes down heavily in favour of the surgeons, although it must be remembered that most surgeons carefully select their cases—in fact one surgeon (Poppen, 1951) going so far as to admit that ". . . death occurred in patients who were not carefully chosen." And even those surgeons who do not select their cases see only those patients who are referred to them by their medical colleagues; in other words, those cases which have survived the G.P. and the Physician.

To refer back to Walton's series of 1,480 cases treated medically. He found an overall mortality of 45% in the first eight weeks; these then are the cases where surgery might improve on medical treatment. Of these, approximately 15% died within the first twenty-four hours and at present

lie outside the scope of surgery. This reduces the percentage available for surgery to 38% of the original hundred. When these are examined angiographically a further 30% of this group will be found unsuitable for operation (e.g. negative angiogram, multiple lesions, inaccessible lesions) and so we obtain a final figure of 27% of all cases where surgery can offer improvement over conservative management. But there remains an operative mortality of about 20% and this further reduces the figure to 22%. Thus in only 22% of all cases of sub-arachnoid haemorrhage due to intracranial aneurysm can surgery at present offer any real advantage over medical treatment; it is therefore obvious that there is still an important place for careful conservative management.

In conclusion it must be emphasised that while refinements of surgical technique will continue to improve the prognosis, such improvements can only be used to their full advantage in the light of improved diagnosis and assessment and it is imperative that the early minor episode of bleeding should be recognised in those cases where it occurs and the patient referred at once to the neurosurgeon.