obtained with the usually recommended therapeutic doses. Terramycin comes next in order of effectiveness, followed by streptomycin and aureomycin. Penicillin *in vitro* is as effective as chloramphenicol and terramycin, or even better, on a weight-for-weight basis, but blood concentrations within the effective range are difficult to obtain and maintain.

Deterioration of aureomycin at 37° C. may be responsible for the comparatively low rating of the drug, but clinical impressions in chronic *H. influenzae* infections also suggest the superiority of chloramphenicol.

I am indebted to the Medical Research Council for a grant in aid of expenses.

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The Home Office has recently issued the report of the Commissioners of Prisons for 1952 (Cmd. 8948). It deals with the administration of prisons; statistics of the prison population; the treatment, training, and after-care of prisoners; Borstal training; detention centres; and the health of prisoners. In spite of the serious overcrowding in the prisons, there was no epidemic of infectious disease last year, and even the usual outbreaks of influenza in the early months were conspicuously absent at most establishments. Indeed, the director of medical services notes, as an interesting observation, that during past influenza epidemics, where three men have shared a cell and one has developed the disease, the infection not infrequently has spread to men in neighbouring cells, while others in the original cell may escape it altogether. However, the potential risk to health consequent on overcrowding is constantly in the minds of the prison medical officers. In August, 1952, the first detention centre for boys of 14 to 17 years was opened, and the medical officer reports that the training there has had a beneficial effect on the physique and health of the boys admitted. The idea of the centre is to provide an alternative to prison for young offenders who do not need prolonged Borstal training yet are unlikely to respond to probation.

THE PROGNOSIS AND ROLE OF SURGERY IN SPONTANEOUS INTRACRANIAL HAEMORRHAGE

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A balanced understanding of the diagnosis and treatment at present available for spontaneous intracranial haemorrhage is extremely important, and this paper presents on the one hand our experience and on the other our considered opinion regarding the treatment of this condition. The term "spontaneous intracranial haemorrhage" is used rather than "subarachnoid haemorrhage," as the major portion of the bleeding may be at any site from the subdural space to the ventricle. Apoplexy in the elderly arteriosclerotic, so classical in its clinical manifestations, is not included.

Prognosis

The prognosis in cases treated conservatively has been reviewed many times in the past few years, and there is no doubt that the initial mortality rate is about 50%, rising with recurrent episodes and in older patients. In our assessment our first consideration was a series of 100 consecutive cases managed conservatively by one of us from 1936 until 1951. Conservative care means rest in bed for at least one month, care of the bowels, sedation, and the judicious use of lumbar puncture. In these 100 cases, 53 female and 47 male, there were 43 deaths during the initial period in hospital. Although these 43 deaths occurred at any time up to six weeks after the onset of haemorrhage, one third (14 cases) occurred in the first 24 hours and another third (15 cases) in the second week. Excluding the first day there were only eight deaths in the first week, and six patients died in the second to sixth weeks. Fairly clear evidence of recurrent bleeding was present in half the patients dying during the first and second weeks and in all those dying after the close of the second week. All the 57 survivors have been traced. Eight of them have died from recurrence of intracranial haemorrhage and two others of intercurrent disease. Twenty of them have been left with some permanent neurological disability, although only seven are completely disabled by hemiplegia or intellectual loss. Of the eight late deaths from recurrence only two have been in the disabled group; the other six have occurred in those who had made a complete recovery from the initial haemorrhage. The deaths have occurred after an interval of time up to 12 years from the initial episode. Four died of a single recurrence, but four others of a second, third, or fourth haemorrhage. Before considering the way in which surgical investigation and treatment may improve both the mortality and

the disability rate, we must seek a fairly clear picture of the varying clinical syndromes and pathological findings.

During the first few hours following a haemorrhage, it is clear that certain patients are dying. These are deeply unconscious, with gross changes in the vital signs. Many have a rapidly increasing pulse and respiration rate, and excessive bronchial secretion; often decerebrate rigidity, with the arms and legs held in marked extension and varying areas of the body covered with goose-skin and beads of sweat. They show no response other than a generalized increased rigidity to stimulation. All others must be judged upon the changing signs that occur in the hours immediately after the catastrophe; some are unconscious to varying degree and others never unconscious at all. Headache and neurological disturbance, such as hemiparesis, visual field defects, cranial nerve palsies, or dysphasia, may be present. It is in these hours immediately after the haemorrhage that an assessment of the patient is most important and illuminating. There are those who regain consciousness quickly, who have no evidence of gross neurological disturbance, or rapidly improve. Others are still semiconscious but their condition has remained unchanged over the passage of time, and these are the ones who may recover also. Others, however, begin to show progression of neurological disturbance characterized by increasing drowsiness or coma and pupillary change. It is this group which is developing increasing intracranial pressure and a tentorial pressure cone, the medial portion of the temporal lobe being pushed down between the midbrain and the margin of the tentorium, causing the posterior cerebral artery to press upon the third nerve and bring about the pupillary dilatation and non-reaction to light, as well as disturbing the blood supply and function of the midbrain. Unless urgent surgical treatment is undertaken haemorrhagic infarction of the midbrain will occur with all the signs of impending and inevitable death.

As the hours pass into days so more and more of the patients fall into their obvious categories. We are left with those obviously recovering, those developing evidence of slowly increasing intracranial pressure, and those with varying degrees of unconsciousness or akinetic mutism and disturbance of autonomic function. In any of these patients gross neurological signs other than disturbance of consciousness may be present, but it is when we look to the pathological state that varieties of signs and symptoms fall into their proper perspective and the correct investigation and treatment become clear. It must be understood that at any time there may be a recurrence of haemorrhage of variable severity.

Diagnosis and Diagnostic Procedures

There are certain features in the history and clinical signs which are important, particularly dramatic onset, which in difficult cases may be the only lead to the diagnosis. In those cases in which the bleeding has involved the subarachnoid space there is usually neck-stiffness and a positive Kernig's sign, but the absence of these signs in the early stage does not exclude the presence of a great amount of blood in the subarachnoid space, especially when there is deep unconsciousness. Auscultation of the head, normally so unrewarding, may elicit the presence of a systolic bruit from an angioma. Varieties of neurological signs, and particularly ocular pareses, may be present in many cases, and in some a typical subhyaloid haemorrhage will be observed in the ocular fundus. Ocular pareses when present as isolated signs are likely to be due to an aneurysm in close relation to the nerves, rather than

due to a tentorial pressure cone resulting from an intracranial haematoma, and are therefore valuable localizing signs.

In the early stages, particularly if there has been only a mild or premonitory leak of blood into the subarachnoid space, a variety of lower spinal nerve pains may occur; in one of our patients a diagnosis of appendicitis had been made from the lower abdominal pain, rigidity, and vomiting, whilst another developed an obvious crop of herpetic vesicles around the trunk. The patients in whom a haemorrhage occurs almost exclusively into the substance of the brain may have a sudden onset but a rather slower evolution of a syndrome typified by fairly gross neurological disability and increasing headache, somewhat similar to cerebral abscess. Some cases even more insidious in their progression develop signs of considerably increased intracranial tension with papilloedema and drowsiness, resembling a rapidly developing cerebral tumour syndrome.

Lumbar puncture confirms the diagnosis by the finding of blood or xanthochromic fluid. Where the haemorrhage has been essentially intracerebral, blood is usually absent from the cerebrospinal fluid, but it is surprising how rapidly the fluid may alter, showing a rise in leucocytes and protein content. There are cases of intracerebral haemorrhage which may show a relatively normal cerebrospinal fluid at any early stage, whilst a few days later it may be frankly blood-stained although the patient's clinical condition has remained unchanged or has even improved. This is due to the intracerebral clot discharging itself into some part of the cerebrospinal fluid pathways. In an absolutely uncomplicated subarachnoid haemorrhage the cerebrospinal fluid will have returned to normal in about three weeks. Intracranial tension is usually much raised in cases of intracranial haemorrhage, but in intracerebral clot formation, particularly in the elderly, the pressure may be low, as it is in some cases of chronic subdural haematoma. Although in our experience diagnostic lumbar puncture has never been detrimental, it should be performed with caution, particularly in those cases showing evidence of an early tentorial pressure cone.

Angiography

The essence of investigation of spontaneous intracranial haemorrhage is angiography, and until this has been performed, in the majority of cases, the site and the cause of the haemorrhage are both unknown, and definitive surgical treatment cannot be contemplated. We believe, at this stage in our knowledge, that angiography should be performed on all cases of intracranial haemorrhage unless they have considerable arterial hypertension, which we have regarded as a contraindication. This view may be fallacious, as four of these patients have all died of subsequent haemorrhage within a few months.

The need for angiography at present is to obtain further knowledge, particularly of prognosis as well as the planning of treatment. Our usual practice is to perform angiography by the percutaneous insertion of a needle into the common carotid artery under local analgesia, but in a restless or non-cooperative patient general anaesthesia may be necessary. From 10 to 15 ml. of 35% diodone is injected, which gives a clear radiographic picture of the blood vessels. This must be performed on both sides in all cases, with compression of the opposite carotid artery to prove the adequacy of the collateral circulation through the circle of Willis.

Having made such a demand for angiography, it is only right that its possible dangers should be carefully considered. In only one case in which angiography has been performed for the diagnosis of spontaneous intracranial haemorrhage has there been any detrimental result, and this was a mild decrease in the conscious level of a patient who died several weeks later with infarction in the region of the hypothalamus and the anterior portion of the corpus callosum, the angiographic pictures having been thought to be quite normal. In 200 consecutive carotid angiographies performed for a variety of reasons, there has been one death, due to a thrombosis of the internal carotid artery

which was the site of a previous partial occlusion. Seven other patients, all suffering from organic brain disease, have shown some transient increase of physical signs following the investigation. Air studies of the ventricles are necessary only in confirming the presence of an intracranial clot, though ventriculography at a later stage may show a cyst which was a previous clot cavity in communication with the ventricle. The method should be by ventriculography rather than air encephalography, so that the intracranial pressures are disturbed as little as possible to avoid a recurrence of bleeding. An electroencephalogram is a useful adjunct to other investigations in cases of intracranial clot, and also in proving the tolerance of carotid artery occlusion.

Some Outstanding Facts

Using these methods of investigation in 50 consecutive cases referred to us for a variety of reasons and at variable lengths of time after the initial haemorrhage, certain outstanding facts emerge from their comparison with the 100 cases previously referred to in this paper. The first point is that the causes of haemorrhage demonstrated in the living differ from those found in the dead. In all reviews of the natural history of spontaneous intracranial haemorrhage based upon necropsy findings, aneurysm is the greatest cause, but angiographic investigations in the living show a much higher incidence of angiomas. Even after severe haemorrhages, and in some cases where large intracerebral clots exist, no cause can be demonstrated either on angiographic investigation or at operation. This is not surprising, since haemorrhage can occur from minute angiomas of capillary type, and this has been the cause of large intracerebral haemorrhages in two of our cases.

It is also true that aneurysms which are not visible on angiography, because of their small size or the almost complete occlusion of their cavity by clot, may be the cause of severe haemorrhage. In this respect one patient of ours, having a left-sided aneurysm which had given rise to bleeding and a right-sided hemiparesis, died in hospital as a result of haemorrhage from an aneurysm of the right internal carotid bifurcation which could not be seen on angiography four days previously. At necropsy this aneurysm was found to be completely full of clot, and a slit in the arterial wall beside the aneurysm had caused this torrential haemorrhage. The usual finding, however, will either be a typical small aneurysm, an angioma, or, more rarely, a very large saccular aneurysm which may have reached most remarkable dimensions. It is in these large aneurysms that relatively small haemorrhages seem to occur into the aneurysmal wall, producing cranial nerve paralysis by direct involvement of the nerves and large amounts of clot within the sac.

The following table of figures compares the post-mortem findings in the deaths occurring in the 100 cases treated conservatively with the angiographic and operative findings in those treated surgically, and demonstrates the different aetiological factors in the living and the dead.

Cases of Spontaneous Intracranial Haemorrhage

Findings at 35 Necropsies	100 Cases Treated Conservatively (43% Initial Mortality)	50 Cases Investigated and Treated Surgically (12% Immediate Mortality)	
		Living	Dead
Aneurysms ..	29 (85%)	29 (58%)	5 (10%)
Angiomas ..	2 (6%)	8 (16%)	0 (0%)
No cause found ..	4 (11%)	11 (22%)	1 (2%)

Angiography may demonstrate the cause of the haemorrhage and also show distortion of the cerebral vessels indicative of intracranial clots.

In several cases we have been surprised to find at operation that upward displacement of the middle cerebral vessels shown on angiography, and suggesting a clot in the temporal lobe, has proved to be due to long-continued local oedema. The findings on investigation may demonstrate the basic pathology, but the vascular anomalies alone cannot always explain neurological disturbances of widespread nature which may occur. Although the angiographic

picture of the major arterial tree may appear complete, terminal branches may be affected by either thrombosis or spasm leading to these additional gross and widespread signs. In 3 of the 50 cases surgically investigated no obvious abnormality was found on angiography or ventriculography apart from ventricular dilatation of unknown cause. These patients have all demonstrated a condition of prolonged akinetic mutism and varying autonomic disturbances, and have eventually died. In the two cases in which post-mortem examinations were performed there were small areas of infarction in the medial surfaces of the frontal lobes and hypothalamus, with a tiny aneurysm of the anterior cerebral artery.

Treatment

With all the results of investigations at our disposal four distinct lines of treatment become obvious: non-intervention, to allow natural recovery; evacuation of large intracranial clots; proximal ligation of vessels; and direct attacks upon aneurysm or angioma. Sympathetic interruption is still open to question, but we believe, from the extreme narrowness of vessels often seen on angiography in these cases, that spasm is likely to be present. The aim must be to save the patient's life, make his future more secure, and improve his chances of recovery from severe neurological disability, but in so doing never exposing him to a greater risk. Our investigations have shown such a diversity of gross vascular anomalies, with amazing recovery by natural means alone, that we are sure no one should be exposed to a great risk for an uncertain purpose. Many of these natural recoveries have been so good that they would have delighted the heart of any surgeon who had operated and thought his act responsible for the recovery.

Evacuation of intracranial clots usually means the evacuation of intracerebral ones; the majority of the clot, however, may be in the subdural space, particularly in children. The removal of these clots is not technically difficult; in the acute case it is life-saving, and in the more chronic one it reduces the likelihood of severe persistent disability. These intracerebral clots have certain interesting features: in any particular area the enlargement of clot from the haemorrhage is always along certain apparent well-defined tracks, with the ventricular wall forming the inner boundary. In the very chronic cases the clot is still solid, with a reactive glial wall forming around it, stained yellowish-brown from blood-pigment absorption. Within such a clot cavity will lie the cause of the haemorrhage, and in our experience this is always in one of the walls and not in the centre of the cavity. It is through such clot cavities that aneurysms and angiomas may easily be exposed and local treatment decided upon. In the 35 post-mortem examinations from the 100 cases treated conservatively, 34% had large intracerebral clot formations, and there is no doubt that these clots were the cause of fatality in most of these cases. In the 50 cases surgically managed, 16, or 32%, have had intracerebral clots. All those from whom clots were evacuated survived, but mistaken surgical judgment in three cases resulted in death of the patient with large cerebral clots existing. Cases with oedema simulating clot benefit a great deal from decompression; therefore the distinction is less important than would be theoretically apparent.

We believe that, whenever possible, proximal arterial occlusion should be the first consideration in any case, and that in many it will remain the ideal or only method applicable. Proof of tolerance of carotid occlusion must always be sought by angiography and trial compression before it is utilized as a permanent treatment. The form of proximal arterial occlusion is best considered according to the type of lesion and its situation. Let us first consider varieties of intracranial aneurysm in this respect. For those upon the internal carotid artery or at its bifurcation there is no doubt that ligation of the common carotid artery, followed at an interval by that of the internal itself, is of great value in reducing the intra-arterial pressure at the site of the aneurysm and leading to thrombosis of the aneurysmal cavity. We hope shortly to show by recordings

of the intra-arterial pressure above the point of arterial occlusion that adequacy of anastomotic circulation can be assessed accurately, and certainty or uncertainty of tolerance predicted. For aneurysms upon the anterior cerebral artery, proximal ligation of the anterior cerebral artery itself just beyond its origin is advised. This also applies to the posterior cerebral artery. For the posterior communicating artery temporary carotid ligation should be employed, with direct attack upon the aneurysm itself in chosen cases. This also applies to aneurysms of the middle cerebral artery, though we are still uncertain of the best treatment of aneurysms affecting this essential vessel.

The angiomas of large size, particularly when they are basal or medial in position or bilateral in extent, are often amenable only to treatment by arterial interruption. Here, as in the case of aneurysms, each and every case must be judged on its own merits, its individual anatomy, and its tolerance to arterial interruption. In this respect individual anatomy often causes great surprise: in one of our cases having a large bilateral angioma in the medial surfaces of the frontal lobes angiography showed that the blood supply to the angioma passed through the internal carotid arteries, and that the normal circulation of the brain was via the external carotid and vertebral arteries. This patient tolerated ligation of both internal carotid arteries, but digital compression of both common carotid arteries immediately proved them to be well named as the arteries of sleep, for the patient immediately became unconscious, only to recover within seconds of release of the compression.

It is important to appreciate the individuality of the cerebral circulation and the need for careful proof of tolerance of its interruption. In particular, ligation should not be performed whilst the collateral circulation may be in jeopardy from high intracranial pressure due to intracranial clots, which must be removed by operation before arterial interruption is undertaken. In this respect sympathetic interruption and carbon dioxide inhalation to produce vasodilatation of the cerebral vessels may be useful to improve the cerebral collateral circulation.

The exposure of aneurysms is usually not in itself technically difficult, although it may prove an anxiety to the surgeon and his assistants. The fear of torrential haemorrhage always exists, even though this can be controlled to a large extent by hypotensive drugs and arterial compression. There is really no part of the circle of Willis above the tentorium which cannot be exposed surgically, but, when an aneurysm is found, often only one side of it can be seen so that a number of vessels taking origin from the aneurysmal wall may remain unseen. Carotid interruption will reduce the pressure in aneurysms even of the posterior communicating artery; at operation we have seen fairly large vigorously pulsating aneurysms of this artery cease pulsating, both visually and to a palpating finger, when the carotid artery has been occluded in the neck. If at this stage the aneurysmal wall is opened, blood flows from the aneurysm in a steady stream, which undoubtedly represents the diastolic pressure. The aneurysmal cavity can now be packed with muscle, bleeding controlled, and the cavity completely obliterated. We believe that packing of the aneurysm in appropriate cases is the best treatment, and, failing this, wrapping of the aneurysm in muscle or gauze. The wrapping of an aneurysm is possible only when it is completely exposed, and we have found it to be of considerable value with the small dangerous aneurysms of the middle cerebral artery. Clipping the neck of an aneurysm has not been possible in our hands, and we doubt if by this method the danger area of the aneurysm—namely, the neck and its junction with its parent artery—is rendered safe. It is unfortunate that obliteration of the aneurysmal cavity is no proof that there will not be a torrential haemorrhage from rupture of the artery close to the neck of the aneurysm, which itself is full of organized laminated clot. We must therefore follow up with great care our cases treated surgically, to see if their future is better than that of those treated conservatively.

The removal of a cerebral angioma may be very simple, particularly if it is a small one lying within the wall of a

clot cavity or cyst cavity resulting from a previous haemorrhage. On the other hand, a large ramifying angioma may be extremely difficult to remove and the patient's life jeopardized by the attempt. We must remember, therefore, that the natural history of the angiomas is not so menacing to the patient's life as that of aneurysms. These angiomas must be very carefully assessed and the patient's disability considered before removal is decided upon. Certainly it has not been our experience that epileptic attacks can be diminished by the removal of these vascular anomalies.

Surgical treatment as outlined above will undoubtedly reduce the number of deaths due to intracranial haemorrhage of varying cause and severity, particularly those deaths occurring several days after the initial haemorrhage. It is unlikely that the mortality rate over the first 24 hours will be altered very much. It will be remembered that 34% of the cases examined at necropsy in the conservatively treated group had large intracerebral clot formations, and it is true to say that a large percentage of this group could have been saved by surgery. Well-planned proximal ligation will reduce the pressure within the aneurysm and its parent vessels and probably reduce the incidence of recurrent haemorrhage. The incidence of recurrent haemorrhage will also be reduced in those cases in which aneurysms or angiomas are completely removed.

The reduction in the mortality rate inevitably increases the number of patients surviving with a disability, and, whereas in the conservatively treated group 35% of the survivors had residual defects, 52% of the survivors in the surgical group were left with some form of neurological disability. In only 12%, however, was this severe enough to prevent gainful employment, and it was in these six patients that the three late deaths occurred. Disabilities among the survivors from spontaneous intracranial haemorrhages make the need for physiotherapy, speech therapy, and nursing care greater. The provision of facilities for investigation, treatment, and aftercare of these patients is an increasing problem in the neurosurgical departments of the country.

Conclusion

Spontaneous intracranial haemorrhage is a dangerous and treacherous occurrence. The initial catastrophe may be overwhelming, and death may take place in a few minutes or hours; but, should a victim of such a haemorrhage survive beyond this, other dangers no less severe lie before him. Provided the first 24 hours are passed few deaths occur in the first week, whilst in the second week the number of deaths equals that of the first 24 hours, unless surgical investigation and treatment are undertaken. These later deaths are due mainly to large intracranial clots or recurrent haemorrhages. The surgical aim, therefore, should be to investigate these patients in the first week after the initial haemorrhage, so that intracranial clots may be removed or other operative procedures performed to prevent recurrence of haemorrhage. Investigation of spontaneous intracranial haemorrhage is of great importance, for there is no doubt that surgical intervention can improve the individual patient's expectancy of life and reduce his neurological disabilities.

Summary

One hundred cases of spontaneous intracranial haemorrhage treated conservatively have been analysed and compared with 50 cases investigated by angiography and treated surgically. It has been shown from these figures that angiography should be done in the first week following the haemorrhage, so that removal of intracranial clots and the treatment of vascular anomalies by proximal ligation or direct attack may be undertaken in order to prevent recurrence of haemorrhage. It has been stressed that caution is needed not only in treatment but in assessing its curative value. There is no doubt that great help can be given to individual patients and the mortality rate considerably decreased.