not in a strict sense nervous diseases at all. Hence a study of pathology corrects specialism. To take but one case: the commonest nervous symptom is an arterial affair. Hemiplegia, in the vast majority of cases, is owing to blocking up of a diseased artery or to rupture of one. Nerve tissue is not here at fault, but suffers. It deliquesces (as in softening), or is smashed up by irruption of blood. A man's nervous tissue does not begin to soften; it is often locally starved because its arterial supply is cut off—it is boycotted. If a man can take care of his arteries he need have but little fear of hemiplegia and apoplexy. His nervous tissues will take care of themselves. If we could conceive a man so badly educated and so curiously minded that he knew nervous symptoms well and nothing else, he would, if he made *post-mortem* examinations, become de-specialized. He would find that he must start anew by studying cardiac, arterial, and renal diseases, syphilis, gout, rheumatism, and so forth. Out of that new study he would get a reasonable basis for prognosis, and for the care of his patients. In a word, he would find that the pathology of most nervous diseases was indirect, and would cease to take a merely nervous view of them."

REFERENCES

- Botterell, E. H., Loughheed, W. M., Scott, J. W., and Vandewater, S. L. (1956). *J. Neurosurg.*, 13, 1.
 Dandy, W. E. (1944). *Intracranial Arterial Aneurysms*. Comstock, New York.
 Fisher, C. M. (1957). Personal communication.
 Foley, W. T., McDevitt, E., Symons, C., and Wright, I. S. (1955). *A.M.A. Arch. intern. Med.*, 95, 497.
 Hamby, W. B. (1952). *Intracranial Aneurysms*. Thomas, Springfield, Illinois.
 Jackson, J. H. (1882). *Brit. med. J.*, 2, 307.
 Kenk, R., and Nall, M. L. (1952). *Physiol. Rev.*, 32, Suppl. No. 1.
 Logue, V. (1956). *Brit. med. J.*, 1, 473.
 Margolis, G., Odum, G. L., Woodhall, B., and Bloor, B. M. (1951). *J. Neurosurg.*, 8, 564.
 Millikan, C. H., and Siekert, R. G. (1955). *Proc. Mayo Clin.*, 30, 61.
 — and Shick, R. M. (1955). *Ibid.*, 30, 578.
 Richardson, J. C., and Hyland, H. H. (1941). *Medicine (Baltimore)*, 20, 1.
 Russell, D. S. (1954). *Proc. roy. Soc. Med.*, 47, 689.
 Symonds, C. P. (1924). *Quart. J. Med.*, 18, 93.
 Walton, J. N. (1956). *Subarachnoid Haemorrhage*. Livingstone, Edinburgh.
 Wright, I. S., and McDevitt, E. (1954). *Ann. intern. Med.*, 41, 682.

THE MEDICAL MANAGEMENT OF SUBARACHNOID HAEMORRHAGE*

BY

JOHN MacDONALD HOLMES, M.D., F.R.C.P.

Physician, Stafford Group of Hospitals; Neurologist,
 Birmingham Regional Hospital Board and Midland
 Centre for Neurosurgery

Subarachnoid haemorrhage is probably the most common and most rapidly lethal neurological emergency encountered in the medical wards of a general hospital, and this is particularly true if the more preferable definition of spontaneous intracranial haemorrhage is used and those cases are included in which intracerebral bleeding also occurs. I prefer this more comprehensive designation because the distinction between primary intracerebral haemorrhage and so-called subarachnoid haemorrhage is often an artificial one and clinically may be very difficult. Nowadays I think it is logical to consider the problem of intracranial haemorrhage as a whole, at any rate from a diagnostic point of view, but I must admit to having been illogical in excluding cases of rapid or sudden death from cerebral haemorrhage in elderly hypertensive or arteriosclerotic patients in the 200 patients I have reviewed. My excuse is that I was more concerned with therapeutic results than with the incidence of causes of haemorrhage.

*Read to the Section of Neurology at the Annual Meeting of the British Medical Association, Newcastle upon Tyne, 1957.

The contribution of the physician to the management of intracranial haemorrhage may be considered under the headings of diagnosis, conservative management, and above all, I think, the selection of cases for surgical treatment.

Diagnosis

The diagnosis of intracranial haemorrhage seldom presents any difficulty and is confirmed in most cases by the finding of blood in the cerebrospinal fluid. Sometimes, however, there may be a slow onset of symptoms of increasing intracranial pressure, with no frank blood-staining of the cerebrospinal fluid, which may show a polymorph or lymphocytic pleocytosis and only at a later stage become xanthochromic. A cerebral tumour or abscess may be simulated in these cases, in which the bleeding is at first entirely intracerebral. There were three such cases in my first series of 100 patients, and the characteristic signs of subarachnoid bleeding, neck rigidity, and Kernig's sign were absent. The onset of headache was not sudden and at first there was little disturbance of consciousness. A diagnostic feature which may sometimes present difficulty to the uninitiated is the finding of a considerable degree of glycosuria in a comatose patient with intracranial haemorrhage. This may sometimes be accompanied by a raised blood-sugar level, but I have never seen any appreciable degree of ketonuria, nor have I seen the massive albuminuria which has been described in some textbooks as an accompaniment of subarachnoid haemorrhage.

Nowadays it is not enough to make a nominal diagnosis of intracranial haemorrhage. The source of bleeding should be located and its cause determined. In most cases this is impossible without carotid angiography. In this country angiography is still not used as much as it should be in general hospitals without a neurosurgical department, and I would make a plea for its wider adoption. The technique is not as difficult to learn as is thought by those who have never practised it, and it is certainly possible to obtain adequate angiograms in a radiological department which is not exclusively devoted to neurological work, although it is admittedly time-consuming.

Conservative Management

The conservative management of intracranial haemorrhage should consist of rest in bed for at least one month; good nursing care and all that it implies; attention to the bowels, bladder, and skin; the preservation of a good airway and the prevention of respiratory infection by the judicious use of antibiotics and sulphonamides; and the prevention of dehydration with preservation of electrolyte balance. Lumbar puncture should be used cautiously to relieve headache, but it may be dangerous if large intracerebral clots are present. There is probably some advantage in treating arterial hypertension with hypotensive drugs such as pentolinium and mecamylamine, or even in lowering the blood pressure in the absence of hypertension. I have used induced hypotension with some apparent benefit in a small series of cases, but the results are not spectacular. Hypothermia may be helpful in cases where there is severe cerebral ischaemia due to arterial spasm, but I have used it in only one fatal case.

It is generally acknowledged that the mortality rate in unselected groups of cases of spontaneous intracranial haemorrhage is in the region of 50%, and, although it is my task to discuss medical management, I do not think that much improvement in this initial mortality of 50% can be expected from the conservative measures I have outlined. I am convinced that the future lies in surgical treatment, and I would suggest that the most important function of physicians, and particularly those who work in hospitals with no neurosurgical department, is in selecting cases suitable for surgery.

Selection of Cases for Surgical Treatment

All patients seen in the early stages, soon after the onset of bleeding, unless they are obviously dying of rapidly advancing mid-brain or medullary compression, should be considered as candidates for surgery, and the investigation and management from the beginning should be with this end in view. It is certain that many patients will have to be rejected as candidates for surgery and for a variety of reasons, such as multiplicity of aneurysms, unsuitability of the site of the aneurysm for direct surgical attack, severe hypertension and cerebral arteriosclerosis, advanced age, inaccessible angiomatous malformations, and any disease process producing poor physical condition. It is also certain, however, that the acceptance or rejection of a patient for surgery cannot be made unless clear topographical and pathological identification of the cause of bleeding is made by means of angiography.

It is the view of many neurosurgeons that angiography carried out at an early stage of the illness is dangerous owing to the risk of aggravating arterial spasm and increasing cerebral damage from ischaemia. I would admit that this risk exists, but I am not convinced that it is as great as is sometimes asserted, and if the condition of the patient is desperate I prefer to perform angiography as soon as possible. Generalizations on debatable points of this kind are impossible, and decisions must be made in each individual case according to the urgency of the situation.

Personal Series

I have based my opinions in the first place on a series of 100 consecutive and entirely unselected cases of subarachnoid haemorrhage admitted under my care in a general hospital during the years 1936-51 and followed up completely during that period. I have excluded, perhaps wrongly, cases of rapid or sudden death in elderly or arteriosclerotic patients in which massive intracerebral haemorrhage was the probable lesion. During this period neurosurgical treatment was not available, nor had I begun to use percutaneous angiography. The initial mortality in this first series of 100 cases was 43%, and during the period of follow-up a further eight patients died of intracranial haemorrhage, bringing the total mortality for the period up to 51% and a mortality from recurrences in the survivors of 14%. Two patients died of other lesions.

Table I gives the mortality rates in series of cases treated conservatively. Tables II-IV give a clear idea of what may

TABLE I.—Mortality Rates of Subarachnoid Haemorrhage as Given in Reviews of Cases Treated Conservatively

Authors	No. of Cases	Initial Mortality	Survivors Dying of Recurrence
Taylor and Whitfield (1936)	81	63%	—
Magee (1943)	150	56%	—
Hamby (1948)	130	52%	22%
Hyland (1950)	191	53%	21%
Ask-Upmark and Ingvar (1950)	138	28%	23%
Mount (1951)	752	48%	—
Walton (1952)	312	45%	20%
Wolfe (1953)	93	56%	—
Present series	100	43%	14%

TABLE II.—Duration of Follow-up of 57 Survivors

Period	No. of Cases	Period	No. of Cases	Period	No. of Cases
1-2 years	15	5-6 years	7	11-12 years	1
2-3 "	10	6-7 "	3	13-14 "	5
3-4 "	5	7-8 "	2	14-15 "	4
4-5 "	5				

TABLE III.—Interval Before Death from Late Recurrence During the 15-Year Period of Follow-up

Period	No. of Cases
Less than one year	1
1-2 years	2
2-3 "	1 (after a fourth recurrence)
3-4 "	2 (one after a third recurrence)
4-5 "	1
11 "	1

be expected in a series of cases managed by purely conservative measures in a general hospital.

It seems to me improbable that purely medical management of intracranial haemorrhage will ever be able to give the patient more than about an even chance of recovery in the acute phase of the illness, and since 1952 it has been my practice to consider the possibility of surgery in all cases as soon as they are seen; I have used angiography increasingly often, both in the general hospital and after transfer of the patient to a neurosurgical centre. An analysis of a further series of 100 cases is shown in

TABLE IV.—Interval Between Onset of Haemorrhage and Death in 43 Patients Dying in Hospital

Time	No. of Cases	Clear Evidence of Recurrence of Bleeding
Less than 24 hours	14	—
Up to one week	8	4
1-2 weeks	15	12
2-6 "	6	6

TABLE V.—Lesions Found at Necropsy in 35 Cases

Anterior cerebral and anterior communicating aneurysms	10
Internal carotid aneurysms	2
Middle cerebral aneurysms	12
Posterior communicating aneurysms	4
Angiomatous malformations (1 cerebellar, 1 parietal)	2
Subacute bacterial endocarditis with multiple miliary aneurysms and intracerebral haemorrhage	1
Primary cerebral haemorrhage. No aneurysm found	4
In one case polycystic kidneys and a large aneurysm of the abdominal aorta were found; six small aneurysms were present in the right middle cerebral distribution and one of them had ruptured.	
In another case there were two aneurysms in the left middle cerebral distribution, one of which had ruptured.	
Large intracerebral clots were present in 12 cases, but some clot formation was present in all the cases examined.	

TABLE VI.—Factors Influencing the Prognosis in 100 Cases Managed Conservatively

	43 Fatal Cases	57 Survivors
Arterial hypertension (B.P. over 160/90)	16	11
Coma at onset	24	9
Clear evidence of recurrence of bleeding during period in hospital	22	4
Previous history suggesting subarachnoid haemorrhage	2	0

Twenty of the survivors had permanent neurological disabilities, and of these two died in the first year after the initial haemorrhage of a recurrence. The disabilities were: hemiplegia in 4 patients completely disabled; dysphasia in 4 all able to work; third-nerve palsy in 3 all able to work; sixth-nerve palsy in 2 able to work; visual field defect in 2 able to work; dementia in 3 completely disabled; and epilepsy in 2 able to work.

TABLE VII.—Analysis of a Further Group of 100 Cases Seen in the Period 1952-7

Total No. of cases	100
Total deaths during period in hospital	32
Initial mortality rate for all cases	32%
Cases treated surgically	23 (5 deaths)
Surgical mortality	21.5%
Cases treated with hypotensive drugs	35 (13 deaths)
Mortality rate in group treated with hypotensive drugs	28.5%
Cases treated conservatively as before	42 (14 deaths)
Mortality rate in group treated conservatively	33.3%

TABLE VIII.—Lesions Found in 49 Angiographic Examinations

	Total No.	Surgically Treated	Deaths
Angiomatous malformations	4	3	0
Aneurysms:			
Middle cerebral arteries	9	4	0
Anterior cerebral arteries	3	1	0
Anterior communicating artery	6	4	1
Posterior communicating arteries	8	5	2
Internal carotid arteries	6	2	0
Multiple	3		
No vascular lesion found	6		
Evidence of intracerebral haematoma, but no aneurysm	4	3	2
	49	22	5

One operation was for removal of a spinal angioma which had caused recurrent subarachnoid haemorrhage. No vertebral angiograms were performed in this series.

Table VII. Unfortunately these cases are not strictly comparable with the first series, as they were collected from various hospitals and seen at very variable intervals after the initial bleeding. Of this second group of 100 cases only 32 were unselected and admitted to hospital soon after the onset of symptoms as in the first group. There were 12 deaths, giving a mortality of 55 (41.7%) in 132 cases. Angiography was performed in 49 (Table VIII) of these 100 cases and was attempted but failed in 3 cases. A follow-up has shown that three patients have died of further haemorrhages since leaving hospital. Four patients were untraced. The number treated surgically, 23, is small, but I see no reason for pessimism in the results of operation, which I think are bound to improve with advances in surgical technique and, let me add, more easily accessible neurosurgical aid.

Classification

In classifying my cases I have used four broad groups.

Group 1 includes fatal cases in which death occurs from mid-brain or medullary compression in less than 24 hours following massive intracranial haemorrhage. It soon becomes obvious that these patients are about to die, and it is useless to contemplate treatment of any kind.

Group 2 includes cases in which there is slower development of brain-stem compression, with gradually increasing stupor and neurological signs. Death occurs in this group in from two to three days up to a fortnight after the onset of bleeding, and there may or may not be clear-cut evidence of recurrence of bleeding. This type of case is most important in the consideration of surgical treatment, and I think all should be investigated by angiography as soon as possible. There were 23 cases of this type in the 43 fatal cases in my original group of 100. Also included in this group are those cases of intracerebral haematoma which may simulate a tumour or abscess.

Group 3 comprises cases in which there is an interval of recovery from the initial haemorrhage, followed by a fatal recurrence during the period in hospital. This happened in 6 out of the 43 cases, and it is probable that surgery undertaken during the quiescent period would have prevented some of these fatalities.

Group 4 includes cases of haemorrhage without loss of consciousness or only transient loss at the onset, progressing to complete recovery, or left with some neurological disability. There were 8 deaths in the 57 survivors in this group (14%), and, although many series of cases treated surgically are largely selected from this group, I am doubtful whether surgery is of more advantage than conservative management, unless investigation reveals that the vascular lesion is in a site which is safe for surgical attack.

Conclusion

I am convinced that surgery in suitable cases is the only treatment which has much to offer in reducing the high mortality of subarachnoid haemorrhage. For some years to come, until neurosurgical services are more widely available, it is likely that most cases will be seen in the early stages by the physician, who, in addition to the general medical management of his cases, should attempt to assess their suitability for surgery by the methods I have outlined. I think it is only in this way that an advance will be made, and in my own limited experience I must acknowledge the debt I owe to my neurosurgical colleagues for their close co-operation in the problems I present to them in this field.

REFERENCES

- Ask-Upmark, E., and Ingvar, D. (1950). *Acta med. scand.*, 138, 15.
Hamby, W. B. (1948). *J. Amer. med. Ass.*, 136, 522.
Hyland, H. H. (1950). *Arch. Neurol. Psychiat.* (Chicago), 63, 61.
Magee, C. G. (1943). *Lancet*, 2, 497.
Mount, L. A. (1951). *J. Amer. med. Ass.*, 146, 693.
Taylor, A. B., and Whitfield, A. G. W. (1936). *Quart. J. Med.*, 5, 461.
Walton, J. N. (1952). *Brit. med. J.*, 2, 802.
Wolfe, H. R. I. (1953). *Brit. J. Surg.*, 40, 319.

SURGICAL TREATMENT OF SPONTANEOUS INTRACRANIAL HAEMORRHAGE*

BY

MURRAY A. FALCONER, M.Ch., F.R.C.S.
F.R.A.C.S.

Director, Guy's-Maudsley Neurosurgical Unit, London

During the past decade considerable advances have been made in the surgical treatment of spontaneous intracranial haemorrhage. Thus many neurosurgeons, including Mount (1951), Hamby (1952), Small, Holmes, and Connolly (1953), Norlén and Olivecrona (1953), Logue (1956), McKissock and Walsh (1956), Gillingham (1957), as well as myself (Falconer, 1950, 1951, and 1956a) have endeavoured to show that surgery can appreciably lessen the mortality of bleeding intracranial aneurysms. However, I do not propose to compare the different series which have been published, but rather to formulate a general policy for dealing with these cases.

Spontaneous intracranial haemorrhage (excluding chronic subdural haematoma) usually takes one of two main forms; intracerebral haemorrhage and subarachnoid haemorrhage. The distinction between the two, however, is an arbitrary one, for the two conditions share the same causes (although in differing proportions), and they often occur together. Primary intracerebral haemorrhage is by far the commoner, and is characterized by signs of focal cerebral damage, such as hemiplegia or hemianopia. It occurs chiefly in patients of over 50 suffering from advanced arterial degeneration, and in this group it is usually quickly fatal: indeed, it ranks as one of the great killing diseases (Wilson, 1940). Such cases with their rapid evolution are outside the scope of surgery. A minority of cases, however, which include patients of all age groups, survive the first two or three days, and in these the bleeding may come from an aneurysm, arteriovenous malformation, cryptic hamartoma, or even a leakage from a small atheromatous artery. Some such cases can be benefited by surgery (Falconer, 1952; Beck, 1953).

Subarachnoid Haemorrhage

Subarachnoid haemorrhage is less common, and typically is without localizing signs, although in many instances signs due to associated intracerebral bleeding, such as a mild hemiparesis, a hemianopia, or a frontal-lobe syndrome, or compression of the third cranial nerve by an aneurysm, may also be present. Contrary to past teaching that this is a disease of young adults, primary subarachnoid haemorrhage reaches its peak incidence in the 40-60 age group (Falconer, 1954; Walton, 1956), an incidence which is only a decade lower than that of primary intracerebral haemorrhage. Several statistical studies (Richardson and Hyland, 1941; Magee, 1943; Hamby, 1948; Walton, 1956) have shown that about 30% of patients treated conservatively in hospital will die shortly after their initial haemorrhage, and that a further 30% will die of recurrent haemorrhage within the next six to eight weeks. These recurrences reach their peak between the second and fourth weeks after the initial bleed. Only 40% of patients survive to leave hospital, and half of these are disabled. Ultimately 20% of those who leave hospital will die of recurrent haemorrhage within the next few years (Hyland, 1950; Walton, 1952). Thus subarachnoid haemorrhage, as seen in general hospital practice, has a forbidding mortality, and it is this which has stimulated surgical treatment.

*Read to the Section of Neurology at the Annual Meeting of the British Medical Association, Newcastle upon Tyne, 1957.