BRITISH MEDICAL JOURNAL

LONDON SATURDAY APRIL 5 1958

RECENT DEVELOPMENTS IN CEREBROVASCULAR DISEASES*

BY

RAYMOND D. ADAMS, M.D.

Chief, Neurology Service, Massachusetts General Hospital; Bullard Professor of Neuropathology, Harvard University

In introducing the subject of cerebrovascular diseases by reviewing some of the work that has been published in recent years one must concede at the very outset that the many contributions which have come from all parts of the world have not provided us with a complete understanding of the cause, the means of diagnosis, and treatment of any one of these diseases. Yet one cannot but be impressed with the change in viewpoint which these many studies have engendered, and it is heartening to note the intense interest now being taken in this group of diseases. The "stroke case," formerly relegated to the back wards of the hospital and neglected by internists and neurologists alike, is now being subjected to the scrutiny of medical scientists with promise of substantial gain in our knowledge both of vascular diseases and of the function of the human nervous system.

In the last twenty years a prodigious literature on the anatomy and physiology of the cerebral vessels has been published. The annotated bibliography of Kenk and Nall (1952) on the physiology of the cerebral circulation between 1938 and 1948 contains more than 4,000 titles. Some of these studies are of fundamental importance, for they have taught us that the circulatory system of the human and vertebrate animal brain is made up of a series of "organs or subordinate systems" the functions of which are, on the whole, quite distinct. These include the heart (the propulsive organ), the vertebral and carotid arteries and their branches (the distributing organs), the capillaries (the organs of interchange of substances between the blood and brain), and the veins and sinuses (the collecting organs). The organs of propulsion, distribution, and collection all subserve the functions of capillary exchange. They maintain a remarkably constant cerebral blood flow through the mediation of a series of vascular reflexes, some of which are extracranial (carotid sinus, aortic, etc.) and others intracranial (cerebral vasomotor tone). The topic of the present symposium is the cerebrovascular diseases; these are in fact diseases of these circulatory organs and comprise all the pathological processes which interfere with this process of diffusion, by occlusion of the distributing and collecting vessels or by altering the quality of blood, or which lead to a rupture of a vessel with attrition of the brain tissue by haemorrhage.

Recent pathological studies have placed renewed emphasis on the vascular lesion and its relationship to the anastomotic systems of cerebral vessels. Too much attention in the past has been focused on the brain haemorrhage or infarct. Actually these latter are merely

the secondary or derivative effects of the vascular lesions. It must be remembered that in the final analysis the distinguishing attributes of all these diseases are the vascular changes themselves and not merely the parenchymal lesion which they induce.

The neuropathology of the cerebrovascular lesion itself has been the weakest link in our knowledge of these diseases. Seldom are the aorta, carotid, and vertebral arteries examined with care, and the tendency to search for the haemorrhage or softening by cutting the specimens at the necropsy table, disregarding the nutrient vessels, has not encouraged accurate identification and localization of the vascular abnormality and its relationship to existing anastomotic channels. In recent years, however, vascular pathology has been studied with renewed vigour, and already we have learned that our original formulations were much too simple.

Classification

Recently a small *ad hoc* committee sponsored by the National Institute of Neurology and Blindness drew up the classification shown in the accompanying Table, based on the identification of the vascular lesion. This classification was presented at the recent Princeton Conference for the Study of Cerebrovascular Diseases.

CLASSIFICATION OF CEREBROVASCULAR DISEASES

I. Cerebral Infarction (Pale, Red (Haemorrhagic), and Mixed Types)

A. Thrombosis with atherosclerosis.

B. Embolism: (1) Of cardiac origin: (a) atrial fibrillation and other arrhythmias (with rheumatic, atherosclerotic, hypertensive, congenital heart disease); (b) myocardial infarction with mural thrombus; (c) acute and subacute bacterial endocarditis; (d) heart disease without arrhythmia or mural thrombus. The following are less common: (e) complications of cardiac surgery; (f) non-bacterial thrombotic ("marantic") endocardial vegetations; (g) paradoxical embolism with congenital heart disease. (2) Non-cardiac origin: (a) atherosclerosis of aorta and carotid arteries (mural thrombus, atheromatous material); (b) from sites of cerebral artery thrombosis; (c) thrombus in pulmonary veins. The following are less common: (d) fat; (e) tumour; (f) air; (g) complications of neck and thoracic surgery; (h) miscellaneous, rare types; (i) of undetermined origin.

C. Other conditions causing cerebral infarction: (1) cerebral venous thrombosis; (2) systemic hypotension; (3) complications of arteriography; (4) arteritis (see VI); (5) haematological disease (polycythaemia, sickle-cell disease, thrombotic thrombopenia, etc.); (6) dissecting aortic aneurysm; (7) trauma to carotid artery; (8) anoxia; (9) radioactive or x-ray radiation; (10) with tentorial foramen magnum and subfalcine herniations; (11) miscellaneous, rare types.

D. Cerebral infarction of undetermined cause.

II. Transient Cerebral Ischaemia Without Infarction.—A. Recurrent cerebral ischaemic attacks (previously called vasospasm,

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

usually associated with thrombosis and atherosclerosis). B. Systemic hypotension (i.e., "simple faint," acute blood loss, myocardial infarction, Stokes-Adams syndrome, traumatic and surgical shock, sensitive carotid sinus, severe postural hypotension): (1) With focal neurological deficit, (2) with syncope. C. Migraine.

III. *Intracranial Haemorrhage* (including intracerebral, subarachnoid, ventricular, rarely subdural).—A. "Hypertensive intracerebral haemorrhage." B. Ruptured saccular aneurysm (if unruptured see IV (a)). C. Angioma (if unruptured see IV (b)). D. Trauma. E. Haemorrhagic disorders (leukaemia, aplastic anaemia, thrombopenic purpura, liver disease, complication of anticoagulant therapy, etc.). F. Of undetermined cause (normal blood pressure and no angioma). The following are less common or of lesser diagnostic importance: G. Haemorrhage into primary and secondary brain tumours. H. Septic embolism, mycotic aneurysm. I. With haemorrhagic infarction, arterial or venous (see under I and VII). J. Secondary brain-stem haemorrhage (temporal lobe herniation). K. Hypertensive encephalopathy. L. Idiopathic brain purpura. M. With inflammatory disease of arteries and veins (see under VI and VII). N. Miscellaneous, rare types.

IV. *Vascular Malformations and Developmental Abnormalities*.—A. Aneurysm—saccular, fusiform, globular, diffuse (if ruptured see III (b)). B. Angioma (including familial telangiectasis, trigeminal encephalo-angiomas (Sturge-Weber-Dmitri), retinal-pontine haemangiomas) (if ruptured see III (c)). C. Absence, hypoplasia, or other abnormality of vessels (including variations in pattern of circle of Willis).

V. *Inflammatory Disease of Arteries*.—A. Infections and infestations: (1) meningovascular syphilis; (2) septic embolism; (3) arteritis secondary to pyogenic and tuberculous meningitis; (4) rare types (i.e., malaria, trichinosis, typhus, schistosomiasis mansoni, etc.). B. Diseases of undetermined cause: (1) lupus erythematosus; (2) arteritis with rheumatic fever; (3) polyarteritis nodosa (necrotizing and granulomatous forms); (4) cranial arteritis (temporal); (5) idiopathic granulomatous arteritis of aorta and of its major branches.

VI. *Vascular Diseases Without Changes in the Brain*.—A. Atherosclerosis. B. Hypertensive arteriosclerosis and arteriolosclerosis. C. Hyaline arteriosclerosis and arteriolosclerosis. D. Calcification and ferruginization of vessels. E. Capillary sclerosis, etc.

VII. *Hypertensive Encephalopathy*.—A. Malignant hypertension (essential, chronic renal disease, phaeochromocytoma, etc.). B. Acute glomerulonephritis. C. Eclampsia.

VIII. *Dural Sinus and Cerebral Vein Thrombus*.—A. Secondary to infection of ear, paranasal sinus, face, or other cranial structures. B. With meningitis and subdural empyema. C. Debilitating states (marantic). D. Post-operative. E. Post-operative. F. Haematological disease (polycythaemia, sickle-cell disease). G. Cardiac failure and congestive heart disease. H. Miscellaneous, rare types. I. Of undetermined cause.

IX. *Strokes of Undetermined Origin*.

This classification has several merits. (1) It provides a general idea of the number and scope of the cerebrovascular diseases. One can see at a glance that there are nearly 40 diseases that affect the vascular system of the brain and that our subject is much more complex than is that of cardiovascular diseases. (2) It offers an approach which follows the steps taken by both the clinician and the pathologist in the study of any given case of cerebrovascular disease, for they proceed always from the identification of a stroke—that is, a focal circulatory disturbance in brain function—to the identification of an infarct or haemorrhage, and, finally, to a determination of its mechanism. (3) It reveals at a glance the major gaps in our knowledge—the ischaemic attacks with established mechanism, and the strokes of undetermined type.

These diseases are conveniently grouped under nine headings. However, it is the impression of specialists in this field that most of them are in the end attributable to hypertension and atherosclerosis. In a sense the cerebrovascular diseases are but complications of these two basic processes. Expressed in another way, the foremost diseases of the human nervous system, from the standpoint of frequency and gravity, lie partly outside the orbit of neurology. Their ultimate solution will be accomplished by whosoever learns the secret of atheromatosis and elevated blood pressure.

Intracranial Haemorrhage

There have been a number of recent advances in our understanding of intracranial haemorrhage. First, it has been discovered that several different diseases may lead to this condition. From many excellent pathological and clinical articles, the most comprehensive of which are those of Symonds (1924), Richardson and Hyland (1941), Dandy (1944), Hamby (1952), and Walton (1956), we learn that the saccular aneurysm may give rise to subarachnoid, subdural, meningo-cerebral, or meningo-cerebro-ventricular haemorrhage. Furthermore, it often induces ischaemia in the territory of the ruptured aneurysm, through a mechanism as yet unknown. These aneurysms account for about 25% of the brain haemorrhages which come to necropsy in a general hospital, always, in our experience, in association with meningeal haemorrhage. The development of arteriography has been the most important advance in this field, for it is of great value in diagnosis. Nevertheless it fails to visualize the lesion underlying many cases of non-hypertensive, non-traumatic intracranial haemorrhage. Walton's (1956) series provides the clearest view of the natural course of the disease, stressing the high mortality in the first days and weeks after rupture, and the increasingly favourable prognosis if the patient survives the first few weeks.

Surgical therapy, ligation of the vessel, and clipping the neck of the aneurysm appear to be the therapeutic methods of greatest promise, for even though healing takes place the weak vessel always remains a threat to life. Many feel that the ruptured aneurysm will eventually be recognized as a surgical disease. Unfortunately there are at present no comparable series of cases proved by arteriography and treated by non-surgical and surgical methods. The studies of Botterell *et al.* (1956), Logue (1956), and others, however, suggest that the surgical results in aneurysms of certain localities are infinitely better than have ever been obtained by non-surgical methods. Techniques for inducing hypotension and hypothermia, which are being widely adopted, have undoubtedly made the surgical approach to some of these lesions feasible for the first time.

The demonstration of the importance of haemangiomas and vascular malformations as a cause of hitherto unexplained brain haemorrhages has been another noteworthy development (Margolis *et al.*, 1951; Russell, 1954). Many of these can often be found during life by arteriography. The value of block excision of the malformation in patients with a proved tendency to recurrent haemorrhage, uncontrollable seizures, and increased intracranial pressure, provided the lesion is of size and in a location which makes it approachable, is now generally acknowledged. The value of this method as compared with conservative measures, including x-ray therapy, has not been determined.

The hypertensive intracerebral haemorrhage continues to be the most serious problem amongst the haemorrhagic brain diseases. The responsible arterial abnormality still eludes us. Recent clinical studies indicate that this type of haemorrhage is somewhat more frequent than pathological data would suggest and is fatal in about 75% of cases. The diagnosis of small haemorrhages gives us much trouble, for they may not cause the deepening coma, dense hemiplegia, and the sanguineous cerebrospinal fluid so characteristic of massive brain haemorrhage. No satisfactory method of treatment has been found. Early aspiration of the cerebral clot, hypothermia, and prolonged hypotension are not of proved value, but these methods have had relatively limited trial and may not have been used correctly.

Occlusive Cerebrovascular Diseases

Pathological studies show thrombosis (40% of stroke cases) and embolism (25%) to be the most frequent vascular lesions. The vascular occlusion is the primary cause of this group of diseases. Anastomotic vessels, of which there are three groups—the pre-Willisian and Willisian, the meningeal-arterial, and the capillary anastomoses—are of importance. The number, size, and location of these anastomoses

determine the dimensions of a given infarct, and, at times, even whether it will occur at all. These and other variables, such as systemic blood pressure, oxygenation, or vascular spasm, all of which may be called the "ischaemia-modifying factors," must also be important, but their role is more difficult to assess. Once occlusion occurs there is no evidence that stellate ganglionectomy or drugs which cause diffuse cerebral vasodilatation improve the blood flow to the ischaemic zone, and it is possible that some of them may actually decrease it. Studies of total cerebral blood flow by the Schmidt-Kety and other methods which have been so illuminating in the field of metabolic diseases of the brain have failed thus far to provide useful information concerning any of the focal vascular diseases of the human brain. The local tissue needs under different conditions of activity, the state of the vessels and of vascular reflexes in any given region of the brain, are beyond the scope of these methods.

Clinical and pathological studies indicate the surprisingly high frequency of embolic vascular disease of the brain. In some statistics embolism is almost as frequent as thrombosis. Embolic occlusion presents special problems in neuropathology. Even in proved cases one often cannot find the embolic material proximal to a cerebral infarct, especially if it be haemorrhagic. Embolism thus appears to account for a significant number of puzzling cases of extensive fresh infarction without vascular occlusion. Haemorrhagic infarction, exclusive of venous occlusion, which is relatively rare, usually proves to be related to embolism and seldom to thrombosis. It appears to be due to resumption of the circulation through the necrotic tissues. The explanation of the haemorrhagic character of the infarct is not altogether clear; but shattering and migration or dissolution of the embolic material after ischaemic necrosis of brain tissue has been induced, and release of extensive segmental spasm induced by the sudden impact of embolic material in a healthy artery, are possible factors. Failure to distinguish between haemorrhagic infarcts and massive brain haemorrhage has been a source of confusion in all statistical analyses of pathological material.

Relatively little has been learned in recent years about the treatment of an embolic infarct, and it is impossible to recommend any method at the present time. Many embolic strokes are transitory, lasting only minutes or hours. In the few available studies of therapy no comparison or control groups have been included. With respect to the prevention of further embolic strokes more can be said. The use of prolonged anticoagulants in groups of patients with rheumatic heart disease who have had their first stroke or in groups of patients with either arteriosclerotic or rheumatic heart disease (Wright and McDevitt, 1954; Foley *et al.*, 1955) has led to such a striking reduction of vascular attacks that there seems little doubt about the value of the method. Moreover, it has been shown that the protracted use of dicoumarol is a practical and relatively safe procedure.

Probably the most important advance in our understanding of atherosclerotic vascular disease has been the recognition of a relatively new clinical entity—the *recurrent cerebral ischaemic attack*. These are brief episodes, formerly termed "vasospastic crises" but not well described. Usually they occur as a series of almost identical attacks of focal disorder of brain function. Hemiparesis or hemiplegia, quadriplegia, paraesthesias of face, tongue, arm, or leg, dim vision, diplopia, vertigo, dysarthria, dysphagia, impaired hearing, ataxia, or veering to one side are seen with atherosclerotic disease of the vertebral-basilar system; monocular blindness (amaurosis fugax), contralateral hemiplegia, hemianaesthesia, aphasia, confusion, visual distortion, and other selective impairments of intellectual function, distinguish the carotid cases. The patient may have a few or several hundred attacks over a period of weeks or months, each one lasting a few minutes and leaving no residual signs. The mechanism of the attacks is unknown. Their clinical importance is that they often herald an oncoming stroke. All of the patients seem to have severe atherosclerotic narrowing of the brain arteries, and some have already suffered one or more thrombotic occlusions.

Temporary reduction of systemic blood pressure and presumably of cerebral blood flow on a tilt table, although often altering the electroencephalogram, usually does not reproduce the attacks, and this casts doubt on episodic hypotension as a factor. Anticoagulants have resulted in a dramatic termination of the attacks within a few days in over 90% of cases (Millikan and Siekert, 1955; Millikan *et al.*, 1955); and, although adequate control groups are not available, the striking results in cases treated by Millikan and his colleagues and by Fisher (1957), as well as the low mortality rate at the end of two years of continuous anticoagulant medication (50% dead in the untreated group, 10% in the treated, with more than 200 cases in the total series), are impressive. These observations inform us of the importance of these attacks in the diagnosis of atherosclerotic brain disease (60–65% of cases diagnosed as cerebral thrombosis) and the unexplored factors of hypercoagulable and hyperthrombotic states and hyperviscosity (attacks may occur in polycythaemia and be relieved by bleeding) of blood in occlusive vascular disease.

Another interesting contribution to cerebral atherosclerosis has been the studies of Fisher (1957) on the site of atheroma formation with relation to the state of distal vessels within an infarct. Frequently atherosclerotic occlusion of the carotid, anterior, and middle cerebral arteries will result in an endarterial obliteration of long segments of meningeal arteries. Inasmuch as this has been taken to be one of the pathological criteria of so-called cerebral Buerger's disease, a condition which has been encountered in our post-mortem material only in atherosclerotic occlusion of the carotid or cerebral arteries (rare cases of granulomatous or giant-cell arteritis excepted), the validity of this form of vascular disease of the brain must be seriously questioned.

The treatment of thrombotic infarction is as unsatisfactory as that of embolism. The use of anticoagulants (heparin and dicoumarol) at the beginning of a thrombotic stroke is not of proved value. In some patients there has been progression to a dense hemiplegia and death despite anticoagulants. However, one has the impression that this is the exception rather than the rule, and that the anticoagulated group have done better. Early embolectomy or early or late excision of thrombosed segments of brain arteries will surely have limited application, for the brain is usually irreparably damaged before the procedure can be carried out, and the operations are difficult and hazardous. The prevention of recurrent thromboses by the use of anticoagulants is now being intensively studied in several clinics with proper controls. No data are yet available except those of Wright and his colleagues (where embolic and thrombotic cases were not separated), of Fisher (1957), and of Millikan *et al.* (1955), where the mortality rate seems to have been lowered.

Conclusion

It may be said that our whole orientation towards the stroke case is changing. No longer are we thinking simply in terms of plugging or of vasospasm, but many other mechanisms are being explored. A premium is now placed on early and accurate diagnosis, and the neurological physician must retain competence in internal medicine if he is to have a part in the development of this important field of medicine. But in doing so he merely heeds the admonition of Hughlings Jackson (1882), who in addressing a group of pathologists on the subject of brain disease remarked:

"There are very few nervous diseases in the sense that the essential elements of nervous organs, cells and fibres, go wrong primarily; in most cases they suffer indirectly. For precision in pathology, and, consequently, in therapeutics, we do not think of nervous organs as being made up solely of nervous elements. The ingredients of a nervous organ are not only nerve-cells and fibres but also the subordinate elements, blood-vessels and connective tissues... the nervous tissues are innocent but suffer. Most of them are

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.