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THE PROGNOSIS AND MANAGEMENT OF SUBARACHNOID HÆMORRHAGE*

JOHN N. WALTON, M.D.(Durh.), M.R.C.P.(Lond.),†
Newcastle upon Tyne, Eng.

AN INCREASING AMOUNT of attention has been paid, in recent years, to the problem of management posed by cases of subarachnoid hæmorrhage. A flurry of recent publications has advised methods varying from the ultra-radical approach (on the part of some neurosurgeons) to an extreme conservatism (advocated by some neurologists). From the welter of conflicting opinions, some based upon an uncritical acceptance of ill-digested information, it is difficult for the physician who is confronted with a patient suffering from this disorder to know which path to follow in the interests of his patient. I cannot hope this evening to present to you any final decision upon this highly controversial subject, but I would like to indicate several facts derived from a study of the prognosis of the illness in a series of patients treated conservatively, facts which seem to me to give us some guidance in our approach to the problem.

The opinions which I shall express are based upon the results which I collected when reviewing a series of 312 consecutive cases of spontaneous subarachnoid hæmorrhage admitted to the Royal Victoria Infirmary, Newcastle upon Tyne, over the 10-year period 1940-1949 inclusive.[1] Of these 312 cases only 30 were observed personally; information concerning the remainder was gleaned from case records. However, in 1951 and 1952 I was able to trace 170 of the 172 patients who survived the original illness and I interviewed and examined all the 120 patients who were still alive.[2] On 82 of the 140 patients who died of the original illness, an autopsy was performed and I was able to examine the pathological material. In addition, a scrutiny of the records of the Pathology Department from 1920 to 1939 yielded another 91 examples of this condition, so that I was able to analyze the pathological findings in a total of 173 fatal cases.

Before relating to you some of the results of my investigation and the conclusions which I have drawn from them, it might be of interest to look for a moment at one or two historical facts. Subarachnoid hæmorrhage is probably a disease of great antiquity. Indeed, there is a reference in the second book of Kings, Chapter 4, to a boy "who said unto his father 'my head, my head'—and when he had taken him and brought him to his mother he sat on her knees till noon and then died." I think that this story is very suggestive of subarachnoid hæmorrhage and there are many references to similar incidents in classical literature and in ancient medical writings. Morgagni's description[3] of the servant who collapsed when running after his master's chariot, and in whom autopsy revealed a tear in the intracranial carotid artery, is a classic example. In general, however, this condition was grouped with the other forms of apoplexy until late in the 19th century. At about this time numerous French authors, including Froin[4] and Hayem,[5] began to report instances of meningeal hæmorrhage, while many reports appeared of cases of intracranial aneurysm, some with subarachnoid and intracerebral hæmorrhage. Although Gull[6] in 1859, Bartholow[7] in 1872 and Bramwell[8] in 1886 had commented upon the frequency of the association between aneurysm and meningeal hæmorrhage it was not until the early 1920's, owing largely to the writings of Fearnsides,[9] Collier[10] and Symonds,[11] that it became generally recognized that the syndrome of spontaneous subarachnoid hæmorrhage was usually due to rupture of an intracranial aneurysm.

DIAGNOSIS AND ETIOLOGY

Here I think it is essential to pause for a moment to define my terms. It is well known that blood can appear in the subarachnoid space as a result of numerous factors, among which trauma and parenchymatous cerebral hæmorrhage of atherosclerotic origin, in addition to intracranial aneurysm, are outstanding. However, in my concept of the clinical syndrome of spontaneous subarachnoid hæmorrhage I exclude traumatic and neonatal cases in addition to those in which atherosclerotic intracerebral hæmorrhage is the primary disorder, with subsequent rupture into the ventricles and subarachnoid

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†Research Assistant, Department of Medicine, Royal Victoria Infirmary, Newcastle upon Tyne; late Nuffield Foundation Travelling Fellow in Neurology, Massachusetts General Hospital and Harvard Medical School, Boston, Mass., present address—Neurological Research Unit, National Hospital, Queen Square, London.

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space. In the latter type of case the patients are usually elderly, while deep coma and dense neurological signs such as hemiplegia are usually present from the outset. In spontaneous subarachnoid hæmorrhage on the other hand, the patients are usually younger, with a peak incidence in the 40 to 60 age group,[1] severe blinding headache and neck stiffness are most often the presenting symptoms, and neurological signs resulting from bleeding into brain substance tend to appear some hours after the ictus, not immediately. I must say frankly, however, that while this differential diagnosis is straightforward in the average case, it may occasionally be very difficult to decide whether a case is one of spontaneous subarachnoid hæmorrhage with or without intracerebral extension, or one of primary intracerebral hæmorrhage of atherosclerotic origin. Despite the occasional difficulty in making this distinction I have attempted it in selecting my series of cases in order to achieve as clearly as possible definition of the clinical features of the syndrome of spontaneous subarachnoid hæmorrhage.

I must make it clear that I did not confine myself to consideration of cases of aneurysmal rupture. This syndrome may result from a variety of pathological lesions and another of my aims was to obtain accurate information concerning the incidence of the various pathological entities responsible for the production of the clinical picture. A patient may present with the characteristic picture of spontaneous subarachnoid bleeding but it is then the duty of the physician to apply his clinical acumen and whatever diagnostic tests he may consider necessary and profitable in order to determine the cause. I reported last year[12] a series of cases of unusual etiology—subarachnoid hæmorrhage resulting from glioblastoma multiforme, from meningioma, from intracerebral metastases, from acute ulcerative endocarditis, from blood diseases and from intracranial sinus thrombosis, as well as some instances of primary spinal subarachnoid hæmorrhage resulting from vascular anomalies and tumours of the cord. In some of these patients the primary disease was evident before the bleeding episode, in others the subarachnoid hæmorrhage was the initial manifestation. In most of them the clinical picture was indistinguishable from that due to rupture of an aneurysm.

Within recent years, angiographic studies have revealed the importance of previously asymptomatic intracranial angiomas as a cause of this syndrome. In some series,[13, 14] as many as 20 to 30% of cases have shown such lesions. The incidence of angioma is much less in autopsy material (3% of my fatal cases) but this may be related to the fact that angiomas tend, in general, to bleed little and often and the prognosis in most such cases is less grave than that of aneurysmal rupture. My conclusion from my own series and from a review of the literature is that the syndrome of spontaneous subarachnoid hæmorrhage is a result of aneurysmal rupture in about 80% of cases; it is caused by rupture of an intracranial angioma in perhaps 10% and by a variety of pathological conditions in the remaining 10%. I think it is worth pointing out here that the aneurysms which are large and give rise to signs of a space-occupying lesion, the so-called "paralytic" group of Meadows,[15] do not often rupture to give this clinical picture. Large aneurysms within the cavernous sinus do bleed, but when this occurs, hæmorrhage is confined by the walls of the sinus, giving the typical picture of pulsating exophthalmos, but there is no extension as a rule to the subarachnoid space. Most aneurysms giving rise to subarachnoid hæmorrhage are previously asymptomatic and the same applies to a considerable proportion of angiomas. I make these points since the information I have given you about etiology will clearly have a bearing upon management.

I do not propose to dwell upon the clinical and pathological features of this illness as I am sure most of them will be familiar to you all. However, one or two points are worthy of brief mention in view of the influence they have upon the course of treatment to be adopted. In particular it is important to consider what clinical features may be of help in deciding upon the nature and situation of the causal lesion. Firstly, when confronted with a patient suffering from this clinical syndrome the possibility that the condition may be due to one of the unusual causes already mentioned should be borne in mind. Like so many other conditions, this is one in which we should remember that as physicians and surgeons we are concerned with the whole patient, and not just with that portion which lies within the skull and spinal canal. In such a case careful history-taking and a complete

physical examination may reveal signs of anæmia or of bleeding into skin or mucous membranes to suggest a blood disease. On another occasion a history of vague headaches, of personality change or clumsiness of a limb may bring to mind the possibility of an intracerebral neoplasm as a cause of the bleeding. Or fever and cardiac murmurs may raise the possibility of bacterial endocarditis. Many other variations occur from time to time, but I do not wish to be too particulate; it is sufficient to say that one should be on the alert for such signs of general medical disease which may be the cause of the hæmorrhage and may require definitive treatment. In the absence of features of this type it is justifiable to assume that the bleeding is due to a ruptured aneurysm or angioma. Can one then differentiate on clinical grounds between these two? It is often quite impossible to make this distinction; several authors have pointed out that many bleeding angiomas are very small and otherwise asymptomatic. Of course it is true to say that a history of multiple previous bleeding episodes, of focal seizures and progressive neurological deficit (e.g. hemiplegia) favours angioma, while a cranial bruit, if heard, is virtually diagnostic. Similarly, a long history of typical attacks of migrainous headache, invariably occurring on one side of the head, is professed by some patients with intracranial angiomas, but occasionally the same is true of patients with intracranial aneurysms, particularly those on the internal carotid artery. Furthermore, many angiomas, being situated within cerebral substance, tend to produce more direct brain damage and hence neurological signs when they bleed than do aneurysms; although this comparison is statistically valid in a large series of cases it is of little value in the assessment of an individual case. I think we can conclude that whereas occasionally there are clinical features to suggest an angioma rather than an aneurysm as the cause of bleeding, in the great majority it is impossible to be sure which is present. Of course, as my figures have shown, the odds are 8 to 1 in favour of aneurysm.

CLINICAL LOCALIZATION OF THE BLEEDING POINT

Now let us look at the clinical features to see whether we can localize the bleeding point with any degree of accuracy. On careful analysis of my cases the only symptoms of any localizing value proved to be headache or facial pain. When these were unilateral the causal aneurysm was invariably discovered on the same side of the head. What of the physical signs? Forty-five per cent of the cases which I reviewed had some neurological signs, varying from an isolated cranial nerve palsy, say of the 3rd or the 6th nerve, to a dense hemiplegia or quadriplegia. A hemiplegia was present in 20%. These findings must be correlated with the fact that in my pathological material 69% of patients with ruptured aneurysms had some destruction, through bleeding, of brain tissue; of course, the incidence of intracerebral bleeding is much less in those who recover.

Such signs as high fever, hypertension, albuminuria and glycosuria are of no localizing value, but probably indicate that bleeding is severe and has irritated centres in the floor of the third ventricle. Subhyaloid hæmorrhage, too, was of no localizing value; it was often unilateral but on one occasion the causal aneurysm was on the opposite side of the circle of Willis. It is certainly not always due to a carotid aneurysm as Dandy[16] suggested. The same is true of an isolated 3rd nerve palsy—this is a common sign of an expanding supraclinoid aneurysm of the carotid artery, as is well known. However, in my series of cases, although it was sometimes produced in this way, equally often it was due to intracerebral bleeding above the tentorium with herniation of the temporal lobe, and in some cases it appeared to be due simply to direct damage to the nerve through dilatation of or bleeding from aneurysms arising from several different arteries in the suprasellar region. When this sign appears in a conscious patient with no symptoms and signs of subarachnoid hæmorrhage, it practically always indicates a supraclinoid carotid aneurysm. However, when developing after an acute episode of bleeding its significance is much more difficult to evaluate and it has little localizing value.

Focal epileptic seizures, which occurred in 3% of cases, visual field defects, indicating bleeding into the temporal lobe, and neurological signs in the extremities do, of course, lateralize the bleeding point and generally indicate accurately the site of intracerebral bleeding. On the other hand, they cannot be said to localize the aneurysm with any degree of certainty, as hæmorrhage into the frontal lobe can occur medially from the

anterior communicating or anterior cerebral arteries, infero-laterally from the middle cerebral, or into its undersurface from the region of the carotid bifurcation. Hæmorrhage into the temporal lobe can also occur from at least three directions. It must also be remembered that a dense hemiplegia can occur in patients whose bleeding is confined to the subarachnoid space. In some such cases simple pressure of the effused blood in the subarachnoid space may be responsible, but in others (1.7% of all cases in my series) neurological signs are due not to bleeding but to coincident infarction which may be in the distribution of the artery on which the aneurysm lies or in a situation entirely remote.[17] The possibility of subdural hæmatoma, which develops in about 2% of cases of aneurysmal rupture,[18] should also be borne in mind. It is, of course, true that intracerebral bleeding and consequent neurological signs most frequently occur when the aneurysm is closely related anatomically to cerebral substance (e.g. middle cerebral and anterior communicating) and are less common when the aneurysm lies free in the subarachnoid space (e.g. internal carotid) (Table I). Again this is an observation which is valid when considering a large series of cases but is of no help in the management of an individual case.

All of these observations, based upon a detailed analysis of my cases, have led me to the conclusion that in the great majority of cases of subarachnoid hæmorrhage clinical observations are of practically no value when one is attempting to localize the bleeding point. Very rarely is it possible to do more than to indicate upon which side of the head the bleeding is occurring, and even this is often impossible. Hence, if one's scheme of management should require accurate diagnosis and localization of the causal lesion, ancillary investigations are needed. Prominent among these is cerebral angiography, which will, in a high proportion of cases, give all the information that is required. I will discuss the place of this procedure when we come to management. Very occasionally straight radiography of skull may be of some value, revealing calcification in the wall of an aneurysm or within an angioma.[19] Electroencephalography deserves brief mention. Often one finds diffuse generalized delta activity, which is of no real help. However, a delta wave focus usually indicates intracerebral bleeding in the given situation. Unfortunately this is a relatively uncommon finding. Minor degrees of lateral asymmetry of the alpha rhythm which many authors have stressed[20] seem to me to be a very tenuous basis for localization; indeed in many of the published reports the asymmetry described has appeared to be well within the limits of normal variation. I have found[21] that the electroencephalogram often reveals spikes or other focal abnormalities in patients suffering from seizures as a sequel to subarachnoid and intracerebral bleeding. I do not feel, however, that it is a dependable tool in the localization of a bleeding aneurysm, except in very occasional cases.

TABLE I.

THE INCIDENCE OF INTRACEREBRAL AND SUBARACHNOID HÆMORRHAGE RESULTING FROM RUPTURE OF ANEURYSMS IN DIFFERENT SITES

Site of aneurysm	*Intracerebral hæmorrhage*	*Hæmorrhage confined to the subarachnoid space*
Internal carotid	2	6
Carotid bifurcation	5	9
Anterior cerebral, proximal to anterior communicating	8	3
Anterior cerebral distal to anterior communicating	7	0
Anterior communicating	18	8
Anomalous anterior cerebral	2	0
Middle cerebral	33	7
Posterior communicating	5	1
Basilar	1	1
Vertebral	1	2
Posterior cerebral	1	0
Cerebellar arteries	2	1
Site not stated	0	1
Totals	85	39

PROGNOSIS

(a) *Immediate prognosis.*—In Table II, I compare the mortality rate in my series with that reported in several other published studies. The figure I give is of deaths occurring within eight

TABLE II.

THE MORTALITY RATE OF SUBARACHNOID HÆMORRHAGE

Author	*No. of cases*	*Deaths*	*Percentage*
Taylor & Whitfield (1936)	81	51	63.0
Sands (1941)	120	41	34.0
Fetter (1943)	68	27	39.0
Magee (1943)	150	84	56.0
Sahs and Keil (1943)	64	18	28.0
Wolf *et al.* (1945)	46	15	33.0
Hamby (1948)	130	67	51.5
Hyland (1950)	191	100	53.0
Ask-Upmark & Ingvar (1950)	138	38	28.0
Present series	312	140	45.0
Totals	1,300	581	44.7

weeks of the ictus. It will be seen that the proportion of fatal cases in my series, 45%, is almost identical with that obtained by collecting together 1,300 reported cases. Further analysis of the fatal cases reveals several points of interest. Ninety-one patients (66%) died in or as a result of the first attack of bleeding, whereas 47 (34%) succumbed in a recurrence of bleeding within the first eight weeks and two died of unrelated conditions. Table III gives the total

TABLE III.

DURATION OF THE ILLNESS IN THE FATAL CASES

Death in the first hæmorrhage	*Number of cases*
Within 12 hours	33
12 - 24 hours	13
1 - 2 days	3
2 - 4 days	11
4 - 7 days	12
7 - 14 days	11
over 14 days	8
Death in a recurrence	*Number of cases*
Within 1 week	7
1 - 2 weeks	22
2 - 3 weeks	8
3 - 4 weeks	6
4 - 8 weeks	4

duration of the illness in the fatal cases; those dying in a first attack are considered separately from deaths due to a recurrence. It will be seen that half the patients dying due to the first hæmorrhage did so within 24 hours, the remainder at varying times within the third to the fourteenth days. Correlation with pathological findings (Table IV) revealed that rapid death

TABLE IV.

DURATION OF FATAL ILLNESS IN PATIENTS WITH INTRACEREBRAL HÆMORRHAGE COMPARED WITH THOSE IN WHOM BLEEDING WAS CONFINED TO THE SUBARACHNOID SPACE

Duration of illness	*Intracerebral hæmorrhage*	*Confined to subarachnoid space*
0 - 24 hours	18 (46%)	8 (40%)
1 - 7 days	0	6 (30%)
7 days	21 (54%)	6 (30%)
Totals	39	20

was equally as common in patients with intracerebral hæmorrhage as in those whose bleeding was confined to the subarachnoid space. However, none of the patients with intracerebral hæmorrhage died between the second and seventh days. Hence death may be delayed by intracerebral bleeding. Another important point, which has a very considerable bearing upon management, is evident from the second half of Table III. The peak incidence of fatal recurrent bleeding was in the second week after the first hæmorrhage.

In any disease such as this, in which a fatal outcome is relatively common, attempts will be made to develop criteria which may be used for predicting the outcome in an individual case. Subarachnoid hæmorrhage is no exception to the rule, but in fact, as Richardson and Hyland[22] have pointed out, it is impossible to make such a forecast with complete accuracy. However, certain factors do exist which may be of value. Hyland[23] showed that the prognosis was worse with increasing age, Hamby[24] noted the adverse influence of recurrent bleeding, and Robertson[18] showed clearly that the outlook was worse in patients with severe neurological signs. In my own series I endeavoured to assess the significance in this connection of 80 individual symptoms, physical signs and methods of treatment. I did so by assessing the incidence of each factor in the fatal cases and in the recoveries, making corrections for possible faults in selection, such as inaccuracy or incompleteness of the history in comatose patients. The two patients who died from unrelated conditions were excluded. The incidence in the two groups was then compared by the chi-squared test and the results are given in the next three tables. Table V shows that the prognosis is undoubtedly worse with increasing age; there is a suggestion in each age group that it is worse in females, but the sex difference is certainly not significant. Table VI reveals that epileptic manifestations, loss of consciousness, an abnormal mental state, neurological signs, rapid pulse and high fever, hypertension, prolonged coma and recurrent bleeding are all unfavourable signs. On the other hand Table VII shows that the nature of the onset, a history of previous attacks occurring more than six months before the present one, papillœdema, subhyaloid hæmorrhage, glycosuria, and treatment by repeated lumbar puncture, appeared to have no influence upon the outcome. Although these results may be of some value in predicting the outcome of most cases, they are by no means infallible. All the signs may indicate a favourable outcome in a certain patient, but sudden death from catastrophic recurrent bleeding may yet occur without warning. Unfortunately we have no means of knowing in which cases this will happen.

TABLE V.

PROGNOSIS ACCORDING TO AGE AND SEX

Age in years	Deaths Male	Deaths Female		Recoveries Male	Recoveries Female	Totals Male	Totals Female
0 - 9	1	1		0	0	1	1
	} 33.0%	} 50.0%					
10 - 19	3	2		8	3	11	5
20 - 29	7	5		12	10	19	15
	} 36.4%	} 38.2%					
30 - 39	9	8		16	11	25	19
40 - 49	17	10		20	23	37	33
	} 43.8%	} 45.1%					
50 - 59	15	22		21	16	36	38
60 - 69	10	13		11	15	21	28
	} 50.0%	} 57.2%					
70 - 79	3	11		2	3	5	14
80 - 89	0	1		0	1	0	2
Totals	65	73		90	82	155	155

TABLE VI.

FEATURES SIGNIFICANTLY RELATED MORE OFTEN TO DEATH OR TO RECOVERY

Symptom or sign	No. of cases with the feature: Deaths	Recoveries	Significance	More frequent in
	Total 138	Total 172		
Onset with loss of senses	40	25	Probable	Deaths
Loss of senses before admission	96	69	Probable	Deaths
Epileptic manifestations	28	17	Probable	Deaths
Coma or semicoma on admission	82	16	Very probable	Deaths
Normal mental state	3	41	Very probable	Recoveries
Pulse >100/min	17	2	Probable	Deaths
Moderate or severe hypertension	68	33	Very probable	Deaths
Abnormal neurological signs	110	21	Very probable	Deaths
Hemiplegia	37	16	Very probable	Deaths
Temp. >102°F	23	4	Very probable	Deaths
Considerable albuminuria	17	8	Possible	Deaths
Rapid improvement	11	80	Very probable	Recoveries
Recurrent bleeding	47	13	Very probable	Deaths
Coma less than 1 hour	2	31	Very probable	Recoveries
Coma 12 - 48 hours	32	13	Very probable	Deaths
Coma over 48 hours	54	14	Very probable	Deaths

(b) *Late prognosis*

Before going on to consider management, it is also important to consider what happens to those patients who recover from the illness. The results of a follow-up study of my cases were published in detail in 1952.[2] Subarachnoid hæmorrhage recurred between eight weeks and 12 years after the initial episode in 24% of my cases and in 85% of these (i.e. 20% of all survivors) the recurrence was fatal. Half of the fatal recurrences occurred in the first six months. Four patients made an incomplete recovery from the illness and died after varying intervals while another 11 died of unrelated conditions. Of the 120 surviving patients 4% were completely disabled but the remainder were able to pursue some useful activity. However, of these a third had residual symptoms which were more or less disabling, a third had relatively trivial sequelæ and only a third were symptom-free. Important sequelæ included paralytic manifestations in 10%, epilepsy in 13%, headaches in 37% (but severe only in 13%), organic mental deterioration in 9% and anxiety symptoms in 27%. These physical and mental sequelæ are similar to those of severe head injury. The study also revealed that restriction of activity after the first three months was of no value whatever in preventing recurrent bleeding. In view of these results I concluded that activity should certainly be graduated during the first few months after the illness, but after this time patients should be encouraged to live a normal life. Undue caution is valueless and may be positively harmful, resulting in severe psychoneurotic sequelæ. Confident reassurance is an essential part of rehabilitation and may even require the assertion that bleeding will not

TABLE VII.

FEATURES WHICH DID NOT OCCUR SIGNIFICANTLY MORE OFTEN IN EITHER GROUP

Symptom or sign	*Number of cases with the feature*	
	Deaths Total = 138	*Recoveries Total = 172*
Sudden onset	115	146
Gradual onset	11	13
Vomiting	75	108
Previous history of subarachnoid hæmorrhage	16	17
Papillœdema	19	21
Subhyaloid hæmorrhage	12	11
Glycosuria	9	8
More than one lumbar puncture	21	22
Repeated lumbar puncture	7	11

recur; I say this knowing full well that it does recur in over 10% of patients after the first six months, but it is far better that they should be unaware of this fact than that a lurking fear should provoke a severe anxiety neurosis. This had certainly occurred in some of the patients whom I first saw several years after the illness.

MANAGEMENT

Until recently it was commonly accepted that the essential measure in treatment of a case of subarachnoid hæmorrhage was a prolonged period of rest in bed with careful nursing and avoidance of undue physical strain. In the past the only fertile source of dispute has been the place of lumbar puncture in treatment. The advent of active surgical therapy in such cases following upon the work of Dandy,[16] Falconer,[25] Norlén,[26] Poppen,[27] Mount,[28] Hamby[29] and many others is now compelling a reorientation of opinion. The optimum role to be played by surgery is by no means clearly defined; it is a very controversial point and the most important aspect of the problem which concerns us this evening. However, this method is not yet firmly established as a method of routine management, and the comment of Wechsler and Gross[30] that "conservative treatment is a euphonious justification for doing nothing" is by no means justified. At the present time, surgical treatment is possible in only a few special centres and in the great majority of cases there is little alternative to conservative treatment. Care taken in management may pay valuable dividends and for this reason I believe we should spend a moment considering briefly the principles of conservative management.

(A) CONSERVATIVE MANAGEMENT

It is agreed that a period of strict bed rest with careful nursing and care of the bladder and bowels is essential. Many authors have suggested periods of bed rest as long as eight weeks. In my view a period of about four weeks is normally sufficient in the uncomplicated case, or two to three weeks after headache and signs of meningeal irritation have subsided. The remaining aims of treatment are firstly to relieve headache, secondly to reduce the intracranial pressure, thirdly to control excitement and epileptic seizures, and fourthly to provide adequate fluid and nourishment. It is possible that hypotensive drugs such as hexamethonium compounds of Arfonad may prove to be of some value in suppressing bleeding but these remedies have not yet been given to sufficient patients for their worth to be proven.

Many drugs have been used for headaches, but for effective relief powerful analgesics are required. In my series there was no evidence that morphine had any detrimental effect, but since this drug is known to raise the intracranial pressure, it is probably preferable to use demerol, or as it is called in England, pethidine, which does not have this effect.[31] Reduction in the intracranial pressure by mechanical means is often most effective in relieving this symptom. We have found detensifying therapy of little value in achieving this aim, though rectal magnesium sulphate can do no harm. Undoubtedly, the most efficient agent is lumbar puncture, but this procedure has its dangers, particularly when there is intracerebral hæmorrhage above the tentorium. The great risk is that of tentorial herniation; I doubt whether the removal of cerebrospinal fluid is ever responsible for recurrent bleeding. On the other hand, it is equally evident that, apart from their effect in relieving headache, repeated punctures are of no therapeutic benefit. Hence I have come to believe, as does Meadows,[15] that the procedure should be used sparingly. It is essential for diagnosis, but even this should be done with very great care, only a few drops of fluid being removed, if there is a unilateral fixed pupil or 3rd nerve palsy or other signs suggestive of a supratentorial intracerebral hæmorrhage. A narrow-gauge needle should be used to diminish the risk of persistent leakage from the puncture in the dura and arachnoid. In other cases, it is prob-

ably justifiable to reduce the pressure to normal at the initial tap. Thereafter the procedure should be repeated only when there is continuing severe headache, restlessness, meningeal irritation, profound coma, or signs of recurrent bleeding.

The control of excitement and of epileptic seizures can be dismissed quickly. In some cases intramuscular phenobarbitone may be of value but I believe that intramuscular paraldehyde surpasses all other sedatives in such cases. So far as the provision of fluid and nourishment is concerned, it is probably wise to avoid overhydration in view of the possibility of pulmonary complications and for this reason intravenous fluid is generally contraindicated. In most patients this is rarely an important problem, since coma is of relatively brief duration. In others, fluid may have to be given rectally or subcutaneously with hyaluronidase and tube-feeding may be necessary. One must also be on the look-out for complications such as pulmonary and urinary infections. Indeed in most comatose patients it is wise to give prophylactic penicillin, and in occasional instances other antibiotics may be required.

(B) SURGICAL TREATMENT

As far as surgical treatment is concerned, I feel that it would be presumptuous to consider this in detail since most of you know far more about this question than I. However, I think it is generally accepted that two main methods are in use, namely arterial ligation and intracranial attack. The former method, though by no means free from complications, is less fraught with danger than the latter. On the other hand, it is doubtful whether carotid ligation is completely effective in the prevention of recurrent bleeding, particularly from those aneurysms which are not situated upon the carotid artery itself. So far as aneurysms upon the basilar and vertebral arteries are concerned, here surgery has very little to offer, save in rare instances. Generalizations are dangerous in considering such a subject but an increasing volume of evidence appears to suggest that, when surgical treatment is decided upon, aneurysms on the trunk of the internal carotid artery or at its bifurcation are best treated by carotid ligation, followed, where possible, by intracranial attack. For aneurysms on the anterior cerebral, anterior communicating, middle cerebral, posterior communicating and posterior cerebral arteries, intracranial operation is even more necessary, while those on the vertebral, basilar and cerebellar arteries are difficult to treat surgically with any measure of success. Carotid ligation appears to be ineffective and even dangerous in the case of angiomas;[34] these anomalies require direct attack.

(C) CONSERVATIVE TREATMENT OR SURGERY?

Finally, then, we come to the most vexed question of all—that of the indications for surgical treatment. Many neurosurgeons, notably Falconer[25] and Mount,[28] have expressed the view that all cases of this condition should be managed and treated surgically. There can be no doubt that at first sight the results and survival rates in the surgically treated series of cases reported by Dandy,[16] Jaeger,[32] Poppen,[27] Falconer[25] and Norlén[26] are infinitely better than any recorded in conservatively treated series of cases of subarachnoid hæmorrhage. But here it is essential to add a word of caution, for the comparisons made by several of these authors are by no means valid. This can best be illustrated by analysis of the figures quoted by Mount[28] in 1951. He reviewed a total of 469 cases treated surgically with a mortality of 14% and compared this figure with the mortality rate for conservative treatment which he assessed at 48%; in my series, you will remember, it was 45%. However, in Mount's surgical series intracranial aneurysms and angiomas were listed indiscriminately and many single cases were included which had probably been reported solely because of the success of surgical treatment. Even more important, a very large proportion of the aneurysms which he included were unruptured. There is no doubt that surgery within a few days of a subarachnoid hæmorrhage is much more hazardous than a "cold" operation, whatever method is used, and for this reason Mount's comparisons are valueless.

Indeed the only surgical results which can justifiably be compared with those of conservative treatment have been achieved by Falconer.[25] In 1951 he reported 69 conservative cases of subarachnoid hæmorrhage which were investigated and treated surgically. The mortality rate was 18%, and 66% of patients made a complete recovery. Even in this series there was certainly some degree of selection, since all cases were referred from physicians, some from distant hospitals an appreciable time after the ictus. As I have pointed out already (Table III), a consider-

able number of patients with subarachnoid hæmorrhage die within the first 24 hours. Such cases would never reach Falconer's unit, and it is quite possible that other methods of selection were used in collecting his cases. It is my impression that if Falconer had applied his methods of investigation and treatment to my series of cases, the results would have been far less satisfactory than those he achieved in New Zealand. Indeed, by surveying my fatal cases individually and by making an arbitrary assessment of the possible value of operation in each one, I came to the conclusion that if one excluded from consideration the possibility of complications and untoward events during surgery, the very best one could expect would be to reduce the mortality rate of the disease from 45 to about 30%. If such a result were achieved, this would be a considerable improvement upon the results of conservative treatment, though by no means as great as some surgeons would have us believe. I make this point in order to sound a word of caution, and in order to stress the fact that in my opinion the results of indiscriminate surgery in cases of subarachnoid hæmorrhage cannot at present be good enough to make surgery a *sine qua non* of treatment. Having made this point, let us now consider the pros and cons of surgical treatment to see if we can formulate some rational criteria of management.

If we take the debit side first, it is essential to consider the shortcomings of angiography, without which an informed surgical approach is impossible. To be really thorough, if no clinical indications exist as to the site of the bleeding point, one would have to take bilateral carotid and vertebral arteriograms. Carotid arteriography is not without risk, particularly in elderly, atherosclerotic individuals and in comatose subjects; it has been known to produce hemiplegia in up to 7% of patients, some of whom were relatively young and apparently in good condition.[33] The incidence of complications of vertebral angiography is considerably higher. It must also be remembered that in more than 20% of cases[13, 37] the arteriogram might fail to reveal the causal lesion, and in such cases there would be no alternative to conservative treatment. It is true that such cases with a negative arteriogram usually carry a good prognosis. In many of them the bleeding aneurysm has thrombosed and hence fails to show, while in others some pathological process more benign than aneurysm may have caused the bleeding. However, it cannot be assumed that all patients with negative arteriograms recover. Another point worth making is that in my autopsy series of 145 intracranial aneurysms, 15% were situated on the vertebral-basilar system of vessels; in this situation effective surgery is often very difficult. Only 25% were upon the internal carotid artery at its bifurcation, while 30% arose from the middle cerebral artery in the Sylvian fissure and another 28% from the anterior communicating. Surgical treatment is most satisfactory in the case of carotid aneurysms and is much more difficult in the other two situations. It must also be remembered that in the cases of unusual etiology, surgical treatment would be impossible in some and would require a different approach in others, particularly those harbouring intracranial neoplasms. So far as angiomas are concerned, much the same problems apply as in the case of aneurysms; some can be excised,[34] others are too extensive for surgery.[35] Finally, it cannot be denied that surgical methods carry considerable hazards. Even carotid ligation may be followed by hemiplegia in about 8% of cases[27, 36] while any operation upon intracranial structures involves a substantial risk. These risks are far greater in ill or comatose patients who have bled recently.

Now what of the credit side? Firstly, it is clear from the figures I have quoted that a distinct improvement upon the immediate results of conservative treatment is possible with effective surgery. More important still is the probability that surgical measures would prevent most of those fatal recurrences of bleeding which frequently occur in the second week after the ictus, as well as fatal recurrent bleeding after a substantial interval, as occurred in 20% of my cases. Furthermore, increasing experience with carotid arteriography has shown that in experienced hands the procedure has increasingly few complications, except in elderly, hypertensive and atherosclerotic patients. Surgical technique is improving rapidly and the use of hypotensive drugs has greatly diminished the dangers of operation. As I pointed out earlier, there are many clinical signs which suggest a favourable prognosis in subarachnoid hæmorrhage; in my series 90% of patients under the age of 40, with no severe alteration of the sensorium, normal blood pressure and no neurological signs, recovered. However,

in the remaining 10% unpredictable and catastrophic bleeding recurred. It seems to me that the only possible way in which such tragedies can be averted is by the judicious application of surgical methods of treatment. Indeed this is probably the only way in which one can hope to reduce the mortality of the disease.

However, it is important to stress once more that indiscriminate surgery is likely to bring the method into disrepute, and for this reason the exercise of clinical judgment and careful selection of cases is essential. In order to achieve this aim it is essential to have as much information as possible, and for this reason I feel that angiography is indicated as a routine measure in such patients unless there are very real contraindications. Certainly it is doubtful if surgery could ever save patients dying within 24 hours of the ictus or even in the first two or three days, but in individuals who survive this period I feel that bilateral carotid arteriography should be done. I say bilateral because of the appreciable number of patients who have multiple vascular anomalies.[37] Not only will this reveal in many cases the situation and nature of the lesion causing bleeding, but it may also indicate the presence of other complications such as intracerebral or subdural hæmatoma which may require surgical treatment in their own right. If the arteriograms are negative, there will be no satisfactory alternative to conservative treatment. I do not feel that it is profitable to proceed to vertebral angiography in the average case, in view of the high incidence of complications of this procedure and since aneurysms lying upon this system are rarely amenable to surgery. If, however, an aneurysm or angioma is revealed, this is the time when consultation between physician and surgeon is of paramount importance. Each individual case deserves careful consideration and a plan of management should be formulated depending upon the site, size and nature of the bleeding anomaly, and the age and condition of the patient. In some cases where the aneurysm is large and accessible, surgery may be clearly indicated; in others, when all clinical pointers suggest a favourable outcome, or when the aneurysm or angioma will clearly prove difficult to treat effectively, it is probably preferable to temporize. Being possessed of all relevant information, the surgeon is in a position to operate should the patient's condition deteriorate or recurrent bleeding occur. In Table VIII I have collected figures showing clearly that the patient's chance of survival with conservative treatment increases steadily with passage of time after the initial bleeding. The two danger periods are the first 24 hours, when surgery is unlikely to help, and the second week of the illness. It follows that if operation is decided upon, this should preferably be done within the first ten days of the illness, since so many recurrent hæmorrhages occur during the second week. When the second week has passed, the advantages to be gained by surgical treatment, unless the aneurysm is very favourably situated, become progressively less with the passage of time. Should a patient be first seen four weeks or more after the ictus, conservative treatment is generally advisable unless there is clear evidence of inadequate recovery from the bleeding, persisting intracerebral hæmorrhage, subdural hæmatoma or aneurysmal dilatation. In certain cases, of course, operation will be required in the subsequent weeks and months when there are signs to indicate enlargement of the aneurysm or angioma. However, in general, a patient who is seen some six months or more

TABLE VIII.

THE MORTALITY RATE IN PATIENTS WITH SUBARACHNOID HÆMORRHAGE AT DIFFERENT INTERVALS AFTER THE ICTUS

Time after the ictus	*Total surviving patients*	*Died later as a result of the first hæmorrhage*	*Died later of recurrent bleeding*		*Survived period of follow-up*
			Recurrence within 8 weeks	*Late recurrent bleeding*	
24 hours	264	45 (17%)	41 (18%) Total 82 (31%)	35 (13%)	137 (52%)
1 week	231	19 (8%)	40 (17%) Total 75 (32%)	35 (15%)	137 (60%)
2 weeks	198	8 (4%)	18 (9%) Total 53 (27%)	35 (18%)	137 (69%)
4 weeks	176	—	4 (2%) Total 39 (22%)	35 (20%)	137 (78%)
6 months	154	—	—	17 (11%)	137 (89%)

after complete recovery from a subarachnoid hæmorrhage does not require investigation or consideration of surgery, for at this time the combined risk of angiography and operation is just as great as that of fatal recurrent bleeding.

From the evidence I have presented to you, one must conclude that we have as yet no absolutely clear-cut criteria to indicate the place of surgery in the treatment of patients with this disease. I do hope, however, that I have managed to convey to you my feeling that this condition is one in which the utmost co-operation and consultation between neurologist and neurosurgeon will undoubtedly pay dividends. I am confident that such a liaison, together with improvements in surgical and anæsthetic techniques, will eventually lead to a substantial reduction in the mortality of the disease.

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APPENDICITIS—A REPORT OF TWO SEVEN-YEAR SURVEYS

BURNS PLEWES, M.S., F.R.C.S., and L. TESKEY, Jr., M.D., *Toronto*

THE MORTALITY from appendicitis has been reduced to two deaths per 1,000 cases from 26 per 1,000 fifteen years ago. This is shown by a survey of all appendectomies at the Toronto East General Hospital during the years 1946-1952 inclusive as compared with a similar study of the 1933-1939 period. In each series the same classification was used, cases being divided into three main groups: (1) acute, (2) chronic and (3) incidental. Acute appendicitis was divided into unruptured and ruptured. "Chronic appendicitis" included cases which were considered acute by the surgeon but reported otherwise by the pathologist.

During the interval between 1939 and 1946 many changes occurred both in the field of medicine generally and in the Toronto East General Hospital. Antibiotics, a clearer understanding of fluid and electrolyte balance, improved anæsthesia and early ambulation were great advances in treatment. At this hospital, the restriction of surgical and anæsthetic privileges to specialists and improved postgraduate and residency training especially contributed to the better care of surgical patients.

The overall reduction in the mortality of appendicitis is shown in Table I. In the "thirties" patients with acute appendicitis had a 95 out of 100 chance of surviving while in the "forties" such a patient had a 99.7% chance of recovery. The removal of the uninflamed appendix showed a mortality rate of nearly 1% in the first series and one-tenth of 1% in the second. Those subjected to appendectomy during other surgi-