THE LATE PROGNOSIS OF SUBARACHNOID HAEMORRHAGE*

BY

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It is generally recognized that many patients with spontaneous subarachnoid haemorrhage die in a first attack of bleeding. Walton (1952a) reports a series of 312 cases, 45% of which succumbed in the first haemorrhage or in a recurrence of bleeding during the first eight weeks of the illness. Until comparatively recently little information has been available on the fate of patients who survive; despite the lack of adequate data, some authors (Sands, 1941; Baumoel, 1941) have suggested that recurrent bleeding after an interval is the rule. This somewhat gloomy conception of the outlook has remained general; commonly, patients have been advised of the possibility of recurrence, and so have sought less arduous work, often with financial hardship. The position has been greatly clarified by the valuable follow-up studies, giving information on the remote prognosis of the condition, which have been reported by Magee (1943), Wolf, Goodell, and Wolff (1945), Hamby (1948), Hyland (1950), and Ask-Upmark and Ingvar (1950). In this paper the results of a similar study are recorded; before recounting these in detail it may be of value to consider the findings of other authors.

Magee reported that 66 of his 150 patients (44%) survived the illness, but of this group 21 (14%) were severely crippled, only 45 (30%) being able to pursue relatively normal activity. Twenty-two patients were re-examined from six months to four years later, and seven were unable to work satisfactorily owing to residual symptoms.

Of the 46 patients reported by Wolf *et al.*, 30 survived, six with severe neurological sequelae, and 21 were still alive after an average follow-up period of three to four years; only one had died of recurrent haemorrhage since discharge.

Of Hamby's 130 patients, 63 (48.5%) survived the initial illness; 14 (22%) of these died of recurrent subarachnoid bleeding after periods varying from a few days to eight years. In five of these patients, however, the fatal recurrence occurred within the first two weeks after leaving hospital, the patients having discharged themselves against advice. Thus only nine patients (16%) died of a recurrence after the initial breach in the wall of the aetiological vascular anomaly had been given adequate time to heal. Three other patients died of unrelated conditions. Eleven of the survivors were crippled, another 13 were working though handicapped by neurological deficiencies, and only 21 (16.2%) of the original series of patients had survived intact.

Hyland reported a follow-up investigation on 91 patients (out of a series of 191 cases) who survived an attack of subarachnoid haemorrhage between the years 1928 and 1942. Sixty-seven of the survivors were traced; 14 (21%) had died of a recurrence of bleeding after an average interval of 6.1 years, and seven of other causes; 43 were able to work, though 14 had mild or moderate disabilities; and three remained completely crippled.

Finally, 100 of the 138 cases reported by Ask-Upmark and Ingvar survived the initial illness; 23 (23%) died of recurrent bleeding, usually within five years of the initial attack, but on occasion the second episode was delayed for as long as 20 years; and 13 died from other diseases. Of the remaining 64 cases, approximately half were able to carry on their occupation as usual, whereas the others were more or less severely crippled.

Material

During the years 1940 to 1949 inclusive 312 cases (156 males, 156 females) of "spontaneous" subarachnoid haemorrhage were admitted to the medical wards of the Royal Victoria Infirmary, Newcastle-upon-Tyne. The criteria used in diagnosis and the clinical features of the illness are described elsewhere (Walton, 1952a). Of these patients 140 (45%) died either as a direct result of the first haemorrhage or from a recurrence of bleeding within the first eight weeks. Of the survivors, 90 were males and 82 females. All but two of these 172 patients have been traced, and 120 of them were still alive at the time of follow-up (late 1951). The methods used in the investigation are described in the Appendix; the length of time which had elapsed since the original illness is given in Table I.

TABLE I.—*Duration of Period of Follow-up*

	Males	Females	Totals
1–2 years ..	2	0	2
2–3 ,, ..	11	2	13
3–4 ,, ..	15	5	20
4–5 ,, ..	4	5	9
5–6 ,, ..	8	1	9
6–7 ,, ..	6	8	14
7–8 ,, ..	4	9	13
8–9 ,, ..	12	5	17
9–10 ,, ..	0	3	3
10–12 ,, ..	8	12	20
Totals ..	70	50	120

Results

It will be noted from Table I that 20 of the male patients and 32 of the females have died since discharge from hospital. Ask-Upmark and Ingvar pointed out that in their series recurrent bleeding was three times as common in female as in male patients, a point not previously recorded. Clearly it is important first to consider in detail the patients who have died, in order to assess the frequency of recurrent bleeding and to see whether this occurs more often in females.

Deaths Since Discharge

(*a*) *Recurrent subarachnoid haemorrhage* with a fatal outcome has occurred in 35 patients (20.6%) since discharge (13 males, 22 females). It will be seen that this incidence is comparable to the figures of 22%, 21%, and 23% reported by Hamby, Hyland, and Ask-Upmark and Ingvar respectively. Six male patients and four female patients recovered from a recurrence of bleeding, but of this group five subsequently died in another attack and are included above. Clearly, therefore, recurrent bleeding occurred more often in female patients (27 as against 19 males), and almost twice as many females as males died of recurrent bleeding. Though not so pronounced as in the series of Ask-Upmark and Ingvar, the difference between the sexes is significant. The reason for the discrepancy is obscure.

In view of the bearing which the recurrence rate may have upon the advice given to a patient after recovery from this condition, it was thought important to discover the length of time elapsing before fatal recurrent bleeding. This information is given in Table II. Furthermore, in view of the widespread belief that exertion may provoke recurrent

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TABLE II.—*Interval Elapsing Before Recurrent Bleeding*

Period	Recurrence with Recovery	Fatal Recurrence
Less than 3 months	1	9
3–6 months	0	8
6 months–1 year	0	4
1–3 years	4	7
3–4 ,,	3	3
4–5 ,,	1	2
Over 5 years	2 (8 years in one case)	2 (10 years in one case)

bleeding, information obtained from patients' relatives was analysed to see whether this was the case in the present series.

From Table II it will be seen that half the fatal recurrences occurred within six months of discharge from hospital, whereas only one patient recovered from a recurrence during this period. Thus the incidence of fatal bleeding in later years in the 153 patients who were still alive six months after the original illness was 11.8%. Admittedly, fatal recurrent haemorrhage did occur after long intervals (10 years in one case), and Rosen and Kaufmann (1943) have reported such an event after an interval of 27 years, while Dandy (1945) reported intervals of 15 and 22 years between the first and fatal attacks.

Adequate information concerning the onset of the fatal recurrence of bleeding was obtained in 22 of the 35 cases, and in only four was the illness clearly precipitated by exertion; in three of these patients this occurred within six months of the illness in hospital. It is suggested elsewhere (Walton, 1952a) that exertion or a temporary rise of blood pressure, from whatever cause, may be the final exciting factor in producing haemorrhage from an aneurysm or angioma which would ultimately have ruptured spontaneously. There is no reason to suggest that this tenet does not apply equally to patients with recurrent bleeding after an interval.

(*b*) *Death from Other Causes.*—Three female patients who were aged 63, 65, and 70 years at the time of the original illness remained severely crippled and were bedridden for periods of five months, four years, and five years respectively after the illness in hospital; they died as a result of infection of the respiratory or urinary tract. In these three cases death was clearly a sequel of the original illness. One patient (Case 204), a woman aged 40, has died recently, 12 years after her subarachnoid haemorrhage, following cranial exploration carried out in an attempt to remove a suprasellar aneurysm of the internal carotid artery. Six other female patients and five males have died after discharge from unrelated conditions, including cardiac infarction, cardiac or renal failure, accidents, respiratory infections, and malignant disease. Each of these patients had apparently recovered completely from the subarachnoid haemorrhage.

Condition of Surviving Patients

Of the 120 surviving patients, 106 have been questioned and examined by me either in hospital or at home; the remaining 14, who are now inaccessible, have supplied information by letter.

Of the 103 at work (including housework), 79 are in the employment they followed before the illness, and 24 in some lighter occupation. Another 12 have retired but remain able to pursue relatively normal activity for age; only five are completely disabled and unable to work. On the other hand, only 36 patients are completely free from symptoms since the illness; in 40 the symptoms which have followed it have been relatively trivial, but in the remaining 39 the sequelae have been disturbing at times, although all these patients have been able to return eventually to some useful occupation. This information is collected together for ease of reference in Table III.

TABLE III.—*Present State of the Survivors of 312 Cases of Subarachnoid Haemorrhage*

State of Patient	No. of Cases	Percentage of Survivors	Percentage of Whole Series
Full employment	79	65·8	25·3
Lighter occupation	24	20·0	7·8
Retired but active	12	10·0	3·8
Unable to work	5	4·2	1·6
Total	120	100·0	38·5
Completely well	36	30·0	11·5
Slight sequelae	40	33·3	12·8
Moderate or severe sequelae	39	32·5	12·6
Completely disabled	5	4·2	1·6
Total	120	100·0	38·5

It will be convenient to consider in detail the various sequelae which have been noted.

Residual Paralysis

Of the surviving 120 patients, 26 showed some signs of neurological defect on discharge from hospital; in 14 cases these signs have resolved completely, whereas in the remaining 12 some residua remain.

Complete Disablement

Case 60 (a man of 38) showed a profound left-sided hemiplegia on discharge from hospital, and four years later the arm is still completely paralysed, though the leg has improved a little and he can walk a few yards with a stick. A complete right hemiplegia with severe dysphasia was present in Cases 80 (a man of 36) and 116 (a man of 53) on discharge from hospital; in Case 80 slight improvement has taken place, so that he can go out in a wheel-chair eight years after his illness. Case 116 remained a completely helpless, bedridden invalid five years later. Both Cases 60 and 80 show severe emotional lability.

Case 23 (a man of 54) had three attacks of bleeding between December 12, 1948, and February 10, 1949. On discharge he had a left sixth-nerve palsy and a spastic paresis of the right leg. Diplopia cleared up within three months, but the right leg remained weak, and although able to walk he was also troubled by persistent pain in sciatic distribution in both legs. He has been completely unable to work, and his progress was further retarded by an attack of coronary thrombosis in September, 1950.

Case 251, a girl aged 18 with multiple hereditary haemorrhagic telangiectasia, recovered from subarachnoid haemorrhage but later developed a paraplegia, presumably as a result of a telangiectatic lesion. It is doubtful whether the paraplegia resulted from the same lesion as that which caused the subarachnoid haemorrhage, so perhaps the severe residual paraplegia should not be regarded as a sequel of the original illness.

Other Paralytic Sequelae

A unilateral third-nerve palsy has remained complete for three years and eleven years respectively in Cases 21 (a man aged 53) and 198 (a woman aged 56), but both patients have been able to continue at work.

Case 279 (a man aged 31) has a monoparesis of the left arm, Case 34 (a man aged 55) a minimal right hemiparesis with persisting dysphasia, and Case 36 (a man aged 28) a persisting slight weakness of the right arm and leg with exaggerated tendon reflexes. All three patients are fully employed, eleven, two, and three years respectively after the original illness. Case 152 (a woman of 23) had had a residual right hemiparesis since the age of 15. Eight years later, in the attack of subarachnoid haemorrhage for which she was admitted to this hospital, a left hemiplegia developed. She was in bed for a year at home, but has since been able to walk, and her activities have increased further since tenotomy of the left tendo Achillis. For the last four years she has done regular housework, despite clear signs of "bilateral residual hemiparesis," more pronounced on the left.

A right temporal lobe angioma is probably present in Case 205, who has had a spastic left hemiparesis ever since her attack of subarachnoid haemorrhage 11 years ago; although much inconvenienced, she is able to walk long distances and to do all her own housework.

Signs Produced by Enlargement of the Aneurysm

As Hyland (1950) has pointed out, the large aneurysms which give rise to symptoms and signs of a progressive intracranial lesion do not usually rupture ; occasionally, however, as occurred in his Case 3, an aneurysm which previously produced subarachnoid haemorrhage may gradually increase in size and may encroach upon important structures.

Case 204 of the present series had a subarachnoid haemorrhage in 1940 at the age of 28 and another attack in 1945, when she suddenly became blind in the left eye. After the latter illness she suffered severe neuralgic pains in the face, and eventually developed progressive optic atrophy in the right eye. In 1946 left common carotid ligation was proposed, but compression of the artery produced a transient hemiparesis and the operation was not proceeded with. On intracranial exploration in December, 1951 (Mr. G. F. Rowbotham), a large suprasellar aneurysm was discovered which was inoperable, and the patient died soon afterwards from a respiratory infection.

Case 246 (a woman of 47) made a good recovery from an attack of subarachnoid haemorrhage in 1944 and was well until 1951, when a progressive paralysis of the right third nerve developed. This patient has been treated successfully by common carotid ligation (Mr. G. F. Rowbotham), as have Cases 138 (a man aged 41) and 277 (a man aged 33), who have had recurrent attacks of subarachnoid bleeding since the attack for which they were admitted to this hospital, but in whom no "tumour" signs developed. In Case 134 (a boy of 11) an angioma of the left temporal lobe was demonstrated and headaches of somewhat increased severity with epileptic manifestations suggested that the lesion might be enlarging ; this patient, too, has been treated surgically, large superficial arteries of supply being ligated.

Epilepsy

Epileptic manifestations occurring as a sequel of subarachnoid haemorrhage have been mentioned by Taylor and Whitfield (1936), but in their paper no indication was given of the frequency of such sequelae. The fact that authors such as Magee (1943), Hamby (1948), and Ask-Upmark and Ingvar (1950) do not mention such manifestations in their follow-up material suggests that they are uncommon.

In the present series, however, epileptic attacks have been noted in 15 patients since the original illness ; if the two patients known to have angiomas are omitted (these patients would possibly have developed epilepsy in any case) it will be seen that this sequel has developed in 13 patients (10.8%) with presumed aneurysmal rupture. In two of these patients the fits have been of focal onset, whereas in the other 11 they have taken the form of generalized convulsions. One patient was aged 29 years, three were between 30 and 39, four between 40 and 49, two between 50 and 59, and three between 60 and 69 ; six were male, and seven female. Thus age and sex have little bearing on the development of this sequel.

Electroencephalograms have been taken from eight of these patients (the two with focal epilepsy and six others) ; focal abnormalities were discovered in the two patients with fits of focal onset and in three others, while in three patients the record was normal. Details of the electroencephalographic findings are given elsewhere (Walton, 1952c).

The fits developed between four months and one year after the illness in 12 cases, but not until six years later in one case (Case 157, a woman aged 36). But for the fact that a focal abnormality was demonstrated in the latter patient, it might have been suggested that the fits were not related to the original illness. She has had two attacks only, but the remaining patients have had multiple seizures. In all cases the attacks have been well controlled by phenobarbitone and/or hydantoinates, and the patients are following their usual occupations.

Headache

Of the survivors, 44 (36.6%) have complained of headache since the attack of subarachnoid haemorrhage. Seven of them were free from headaches six months after the illness, and in another nine who had similar complaints the symptom cleared up after periods varying from one to four years. In these 16 patients the headaches were vague or indefinite in six, but in the remainder they were paroxysmal and usually severe ; the headache was limited to the occipital region in half the patients and was frontal or generalized in the others.

Twenty-eight patients (23.3%) have continued to suffer from headaches for periods varying from two to eleven years. Three patients who had no such symptoms before the attack have had bouts of classical migraine with visual phenomena, unilateral headache, and nausea ; in two the headache has occurred on either side of the head, but in the other patient it has invariably been right-sided. There are no physical signs in the latter patient to indicate the nature or the position of the causal lesion. These three cases are of interest, since none of the patients who recovered and who had suffered from migraine before the illness experienced similar headaches afterwards. Clearly, as suggested elsewhere (Walton, 1952a), the teleological relationship between migraine and cerebral aneurysm or angioma is by no means well defined.

The headaches experienced by 12 of the remaining 25 patients have been described as "slight," "not severe," or "infrequent," and have caused little disability. Thirteen patients, however (10.8%), have suffered severe, paroxysmal, and frequent headaches since the haemorrhage, having been free from such symptoms previously. Again, in roughly half the cases the headache has been maximal in the occipital region, in the remainder frontal or generalized. Some of these patients have had nausea and vomiting, and often the headache has lasted for a day or more and has been prostrating ; in only two has it ever been unilateral, and no patient in this group has experienced an aura. Six of the surviving 10 patients who suffered from migraine before the illness now experience headaches of this nature ; the other four are free from symptoms. Probably this type of headache is related to migraine, but the exact relationship cannot be defined.

From these figures it will be seen that severe headaches, sometimes disabling, have occurred in 21.6% of the surviving patients ; in 8.5% they cleared up after an interval, but in the remaining 13.1% they have persisted.

Mental Symptoms

Although Taylor and Whitfield (1936) and Richardson and Hyland (1941) referred to the fact that mental symptoms may persist after physical recovery from subarachnoid haemorrhage, and Hyland (1950) has mentioned briefly the occurrence of mental dulling or nervousness in surviving patients, psychiatric sequelae have been paid scanty attention in most reports. It is my view that this is a serious omission, for persisting mental symptoms have proved an important cause of disability in the surviving patients of the present series. Sequelae of this nature can be classified into two broad groups : (1) Mental symptoms presumed to be of organic origin ; and (2) anxiety symptoms.

(1) Mental Symptoms Presumed to be of Organic Origin

Korsakoff's syndrome occurring during the stage of recovery from subarachnoid haemorrhage is discussed elsewhere (Walton, 1952b) and will not be considered here ; it is shown that manifestations of this nature invariably resolve. As already mentioned, three patients (Cases 219, 233, and 177) were demented on discharge from hospital and remained so until death five months, four years, and five years later respectively. Cases 60 and 80, who survive with severe paralytic signs and are completely disabled, show gross evidence of mental deterioration. Case 80, with left hemisphere damage, is severely dysphasic, and both patients are emotionally labile, tending to laugh or cry on the least provocation. They are unable to concentrate or to carry out any but the simplest forms of mental activity. Focal lesions in one or other fronto-temporal region have been demonstrated by the electroencephalogram in these cases.

Similar but less severe symptoms have been noted in another three cases. Thus Case 305 (a man aged 46) shows clear evidence of organic mental deterioration confirmed by intelligence testing with the Wechsler–Bellevue scale. Since his illness he has been unable to concentrate or take responsibility; he has worried over trifles, has been disinclined to leave the house, and when he does go out is afraid to cross the street. Furthermore, he has had periods of severe depression during which everything seems unreal. Case 11 (a man aged 22) has also had feelings of unreality and depressive episodes in which he has contemplated suicide; in addition periods of "pathological drive" have been experienced in which he has been compelled to concentrate on his work for fear that his mind would run away with him. Attacks of acute panic have also occurred when he has been alone in his car or in the bathroom. Psychometric testing in this patient showed no evidence of organic deterioration, but an electroencephalogram taken three months after his illness indicated damage to the left frontal lobe, and there seems to be little doubt that his mental symptoms are organically determined. A similar pathology in Case 305 is probable. Lack of ability to concentrate is also mentioned by Case 34 (a man aged 55), who is unable to think or to calculate as quickly as he did; he is also much more emotional than before his illness and is worried by trifles; he shows some evidence of dysphasia of nominal type. This patient has suffered attacks of grand mal since his subarachnoid haemorrhage and an electroencephalogram taken recently showed a focus of delta and sharp wave activity in the left fronto-temporal region. All three of these patients are working, though with some difficulty, particularly Case 305. Only Case 34, who has a minimal right hemiparesis, shows neurological signs.

In the five patients mentioned above the manifestations are very similar, differing only in degree. Despite Bleuler's (1951) suggestion that it is not possible, as a rule, to attribute any particular abnormal mental state to a localized cerebral lesion, it seems to me that these five patients all show features of the "frontal psycho-syndrome"; in four of them frontal lobe damage has been confirmed by the E.E.G.

Another three patients have mentioned that they have been mentally slower since the illness, and that their memory is worse; similar deterioration of memory has been mentioned by three others. It is possible that in these six patients minimal frontal lobe damage has occurred. Four patients who show no evidence of residual paralysis are permanently dysphasic; in three cases the defect consists simply in occasional difficulty in finding the right word, but the other patient has a moderately severe dyslexia. No evidence of the site or source of the haemorrhage exists in these cases.

One other patient (Case 180, a woman aged 28 at the time of the original illness) has noticed a complete alteration in temperament. Whereas beforehand she was quiet and somewhat moody, she is now cheerful, talkative, and "the life and soul of the party." An intracerebral haematoma was evacuated from the right temporal lobe in this patient (Mr. G. F. Rowbotham); during the seven years which have elapsed she has had no other symptoms, and the change in temperament described cannot be regarded as a disability.

From these figures it will be seen that an appreciable number of patients (9% in the present series) who survive an attack of subarachnoid haemorrhage may show permanent mental deterioration of greater or less degree, and this may often be attributable to frontal lobe damage. Two of the patients in this group showed some dysphasia, but the latter sign was also evident in 3% of patients with no other mental symptoms.

(2) Anxiety Symptoms

Symptoms of an anxiety state followed the original illness in 32 cases (26.6%), of whom 15 were males and 17 females. In 30 of these it has been possible, on inquiry, to estimate the nature of the patient's pre-morbid personality; the other two were contacted by letter and no definite conclusions could be reached on this matter. Fourteen patients had shown neurotic symptoms of variable severity before the subarachnoid haemorrhage, but in every case the symptoms were very much worse afterwards; indeed, two patients, although they have worked since the illness, have virtually become chronic psychiatric invalids. In one of these (Case 21, a man aged 53) the clinical picture is flavoured by the addition of a "compensation neurosis," since his attack developed while engaged in heavy manual labour; the other (Case 264) is a housewife aged 49. The pre-morbid personality of the remaining 16 patients appeared to be good.

The anxiety state has been severe and persistent in 15 patients, moderately severe in 12, and only slight in five. All 27 patients in the first two groups have continuing anxiety symptoms after intervals of from two to ten years, although in five of the "moderate" group these symptoms have improved considerably. Four of the five patients in whom the sequelae have been slight throughout recovered completely two to three years after the illness, and in the remaining patient the residual symptoms are relatively trivial.

The exact symptoms professed by these patients vary considerably in type and degree, but one symptom common to all is *fear of recurrence*. In several cases this fear has amounted almost to terror, and several patients mentioned that for months they were afraid to leave the house in case the illness returned; others retired to bed immediately they got a headache and waited for it to increase in severity. For example, Case 114 (a woman aged 54) said that she could not "get the illness out of her mind"; people told her that if she had one attack she would have three, and she sat in the house for almost six months waiting for this to happen. These symptoms were greatly increased when her elder sister died of a second attack of subarachnoid haemorrhage. Seven years after her illness her symptoms are beginning to improve. Similar comments have been made by other patients who have lived in a state of severe anxiety for many months and years.

In many cases anxiety has been heightened by medical advice; thus no fewer than 12 patients mentioned that they had been afraid to exert themselves, as the doctor had told them that exertion might cause the complaint to return. Chance remarks overheard or misconstrued during teaching were also a further source of anxiety; thus Case 289 (aged 22) heard the doctors say that she would probably have another attack, and Case 290 (aged 29) says that she heard one doctor remark that the attacks usually occurred every seven years. Clearly the remark which gave rise to this impression was misunderstood, but the patient was so impressed by the memory of the incident that seven years later she confined herself to the house for two months and was surprised and relieved when the illness did not return. Similarly, Case 242 (a woman aged 48) was constantly anxious and tended to shun company, as she gathered the impression, from a teaching round, that some patients "went suddenly mental" after an attack of subarachnoid haemorrhage.

In many other cases useful descriptive terms were used by the patients. Some said that they were constantly "jittery," that their nerves were "brittle," "shot to pieces," "on edge," or "in an awful state." These symptoms tended to improve as the years went by, but rarely abated before one or two years had elapsed, and in several instances they persisted unchanged for longer periods.

Thus about one-quarter of the patients in this series who survived an attack of subarachnoid haemorrhage developed symptoms of an anxiety state afterwards, and these symptoms lasted for a long time and were disabling in the majority. Admittedly half these patients had suffered neurotic symptoms previously, but nevertheless I feel that complaints of this nature are an important sequel, and have been given insufficient attention in the past.

Other Symptoms

Dizziness since the illness, particularly on stooping or on movement of the head, has been mentioned by 18 patients; no patient has suffered true vertigo. No other notable symptoms of central nervous origin have been recognized. Three patients, in addition to Case 23, who has been mentioned already, have suffered persistent *pain in the legs* in the distribution of one or both sciatic nerves, and Kernig's sign remains positive in this group. It is possible that some degree of plastic arachnoiditis around the cauda equina, resulting from irritation by blood during the original illness, is the cause of these symptoms.

Unrelated conditions developing in individual patients since recovery from subarachnoid haemorrhage included angina of effort (two cases), paroxysmal nocturnal dyspnoea in a hypertensive male, intermittent claudication, tuberculous pleural effusion, diabetes mellitus, osteoarthritis of knees, and a duodenal ulcer.

Comparison with the Late Effects of Head Injury

It will readily be recognized that the sequelae of subarachnoid haemorrhage described above are in many respects very similar to the late effects of severe head injury. It is reasonable to expect that this would be so, since a rapid effusion of blood into the subarachnoid space may have effects similar to those of concussion, while the damage to the cerebral parenchyma which often occurs when an aneurysm or angioma ruptures can be compared to contusion or penetrating wounds.

Guttmann (1946) divided the manifestations of the chronic stage of head injury into five groups. First, largely objective defects as a result of irreparable damage to cerebral tissue were noted, including sensory-motor defects such as hemiparesis, ideomotor defects such as aphasia, and disturbances of intellectual efficiency, including impairment of reasoning and change of character. Epilepsy was also an important sequel, as were the heterogeneous group of subjective manifestations (headaches, dizziness, fatigue, insomnia, irritability, and disturbed concentration) which could be collectively described as the "post-traumatic" syndrome. Finally, purely psychogenic reactions were described, and less commonly a psychosis was precipitated. The difficulty of differentiating between psychogenic reactions and the so-called "post-traumatic state" was stressed.

Clearly the objective defects noted in the patients in the present series are very similar to those described above. Epilepsy has been described in 2.5% (Rowbotham, 1942) and 3.5% (Russell, 1942) of patients after recovery from closed head injury, and after penetrating wounds the reported incidence has varied between 7.9% (Penfield and Shaver, 1945) and 49.5% (Credner, 1930), the average being about 40%. The incidence of 12.5% reported in the present series of patients who have recovered from subarachnoid haemorrhage lies between the figures for open and closed injuries; this would be expected, since in an unknown proportion of the survivors the cerebral parenchyma would have been damaged when the aneurysm or angioma bled. It is shown elsewhere that the electroencephalographic findings in these patients resemble those of post-traumatic epilepsy (Walton, 1952c).

Further examination of the state of the survivors indicates other grounds for comparison, since many of the manifestations of the post-traumatic state and symptoms similar to the psychogenic sequelae of injury have been noted. To take headache alone, it will be remembered that 21.6% of patients have had severe and frequent headaches since the illness, and of Denny-Brown's (1945) series of 200 patients with closed head injury, 63 (31.5%) had headaches of some type lasting for more than two months after the injury. Both figures are much greater than the incidence of "severe and frequent" headaches in the normal population, which has been assessed at 9% (Weider, Mittelmann, Wechsler, and Wolff, 1944).

There is now general agreement that patients with an unsatisfactory previous personality are more likely to develop the post-traumatic syndrome. Symonds and Russell (1943) found that a predisposition to mental disorder was of decisive importance. In a series of cases studied in the R.A.F. it was found that the invaliding rate in patients with a history of mental instability in the family was twice as high as other cases, and it was low in air crews. However, there is no doubt that environmental factors also play a part: Denny-Brown remarked that in his series "symptoms associated with prolonged disability, whether severe or mild, were predominantly anxiety symptoms. Environmental factors were more important in accounting for disability than were factors indicative of the severity of the injury."

Guttmann (1946) compared the chronic stage of head injury with the common neuroses and found that the conditions differed only in minor details: a history of previous mental upset was much less common in the patients with head injury, and it was thus possible to say that, whereas the post-traumatic syndrome was particularly likely to occur in patients with a predisposition to mental disorder, it was also seen in a large number of individuals whose previous personality was good. He also stressed the importance of environmental factors, pointing out that the head plays an important part in the body image, and that all people are "head-conscious"; fear of mental impairment may be a potent source of anxiety. Unskilful management may stimulate the formation of neurosis; thus "if a patient feels subjectively well he is liable to conclude, when kept in bed for several weeks, that the doctor must fear serious consequences, and this fear is easily transferred to the patient himself. Such apprehensions, or rather misapprehensions, are often expressed to the patient, who cannot fail to be impressed and to start watching for after-effects." After quoting illustrative cases to support this view, Guttmann emphasized the importance of reassurance and careful management.

Clearly it is possible to say that all the manifestations of the post-traumatic syndrome referred to above have been recognized in patients in this series, occurring as a sequel of subarachnoid haemorrhage. As in the case of head injury, anxiety symptoms have been pronounced in patients with an unsatisfactory pre-morbid personality, but have also occurred in individuals of good biological stock with no previous history of mental upset. It is therefore reasonable to conclude, in view of Guttmann's observations, that adequate reassurance and careful management are likely to pay a dividend.

Recommendations on Management

It has been shown that fatal recurrences of bleeding have occurred in 20.6% of patients in this series since recovery from the original illness and discharge from hospital, but that almost half of these recurrences occurred within the first six months. Furthermore, it has been stressed that, whereas in isolated cases exertion may appear to have provoked fatal recurrent bleeding, in most cases no such precipitant could be incriminated. Of the 120 surviving patients, 115 are able to pursue some useful activity, although 49 are moderately disabled. The disability in about half the latter group is the result of paralysis, epilepsy, severe headaches, mental symptoms of organic origin, or some combination of these features, whereas in the remainder it is the result of anxiety and of fear of recurrent bleeding in particular.

It will be remembered that in many instances anxiety symptoms verging on misery and even terror could be attributed, at any rate in part, to medical advice and to the suggestion that exertion should be avoided in order to prevent recurrence. It is important to consider what purpose such advice may serve. In the present series there is no evidence to suggest that careful regulation of activity and studious avoidance of exertion was ever successful in avoiding recurrent bleeding, which occurred in several patients who had followed such instructions to the letter. Indeed, at least six patients who had been advised to find

light employment ignored such advice either through boredom or for financial reasons, and returned to heavy work after a reasonable interval; two served in the Army as infantrymen, and one fought in the Anzio landing. In none of these were there any untoward sequelae. As one patient (Case 125, a man aged 49) remarked: "I was told not to exert myself, not to smoke, and not to drink. I sat in the house for months feeling miserable and moping because I couldn't work, and life didn't seem worth living. Eventually I couldn't stick it any longer, so I went back down the mine and haven't lost a day's work for five years. I feel fine."

Clearly each patient must be considered individually, but as a rule undue caution is unnecessary when a patient has made a good recovery from a subarachnoid haemorrhage. It is probable that much misery and anxiety may be avoided if words of advice are chosen with care; the danger of unguarded discussion at the patient's bedside must also be stressed. Certainly the causal lesion must be given time to heal, and for this reason adequate rest and graduated activity during the first few months after the illness should be advised. Some patients may be fit to return to work after three months, others not for a longer period, but, as Rowbotham and Ogilvie (1945) have said: "It is wrong to allow the sword of Damocles to hang too closely above one's head." It is my view that the possibility of recurrent bleeding should never be mentioned, except perhaps to certain foolhardy individuals who may be willing to take unnecessary risks soon after the original illness.

Once a patient has survived for a period of six months he should be encouraged to live a perfectly normal life, and to do heavy work and play games if he so desires. If he inquires about the possibility of recurrence, it is probably justifiable to reassure him that it will not occur, since where doubt exists severe and disabling anxiety may breed. It has been a revelation to see the look of relief produced by such reassurance in certain patients even many years after a single attack of subarachnoid haemorrhage. It is true that further bleeding will probably take place in an appreciable number of cases, and that in about 12% it will be fatal, but since warnings and restrictions are unlikely to prevent such an event it is surely preferable that these individuals should be allowed to live free from the haunting fear of recurrence.

The place of surgery in the treatment of a patient with subarachnoid haemorrhage is considered elsewhere (Walton, 1952a); it is doubtful whether such treatment has any part to play in a patient who has survived the original illness by more than six to eight weeks, since after that period the risks of craniotomy are almost as great as those of recurrence. Ligation of the carotid artery in the neck may sometimes be considered when an aneurysm of the carotid artery has been demonstrated, but the procedure is not free from risk, and it is by no means certain that without subsequent intracranial attack it will prevent further bleeding. Certainly if symptoms or signs suggesting enlargement of the causal lesion develop, surgery may be indicated, and this may apply in other special circumstances, but treatment of this nature should not be advised as a routine measure in patients who have recovered from the initial illness. The major aim of surgery must be to reduce the mortality from the first attack of bleeding or from recurrent bleeding within the first few weeks.

Summary

The results are reported of a follow-up study of the survivors of 312 cases of subarachnoid haemorrhage occurring between 1940 and 1949. Of the 172 patients who survived the initial illness, 170 have been traced, 120 being still alive two to eleven years later.

Recurrent subarachnoid haemorrhage has occurred in 40 patients (23.5%) with a fatal outcome in 35 (20.6%), of whom 13 were males and 22 females; half of the fatal recurrences occurred within six months of the initial episode. Exertion did not play a significant part in the provocation of recurrent bleeding. Four patients made an incomplete recovery from the illness and died after intervals varying from five months to twelve years. Eleven patients died of unrelated conditions.

Of the surviving patients, five (4%) are completely disabled, but the remaining 115 (96%) are able to pursue some useful activity; 79 (66%) have returned to their previous employment, 24 (20%) to lighter work, and 12 (10%) have retired. Only 36 patients (30%) are completely symptom-free; in half of the remainder (33%) symptoms are relatively trivial, but in the others (33%) they are disabling.

Important residua include paralytic manifestations (10%), epilepsy (12.5%), headaches (36.6%, severe and persistent in 13.1%), mental symptoms presumed to be of organic origin (9%, severe in 4%), and anxiety symptoms (26.6%). Five patients (4%) have required surgical treatment either owing to enlargement of an aneurysm or angioma or because of repeated attacks of bleeding.

It is shown that the physical and mental sequelae of this illness are similar to those of severe head injury. Graduated activity is advised during the first few months after the haemorrhage, but, since there is no evidence to show that restriction of activity is of value in preventing recurrence, patients should be encouraged to live a normal life. Undue caution is valueless and may be positively harmful. Confident reassurance is an essential part of rehabilitation, and may even require the assertion that recurrent bleeding will not occur. Management along these lines is likely to reduce the incidence of psychoneurotic sequelae.

APPENDIX

Follow-up Methods

In the first instance a circular letter was sent to the address given by each patient on admission to hospital asking for information on the whereabouts of the individual concerned. It was realized that many patients would have removed from their former address, and for this reason the letter was written impersonally, and was addressed to, say, "John Smith or Occupier." In addition to asking for information concerning the patient's present whereabouts, it inquired whether, if still living at the same address, he or she would be willing to attend hospital for an interview. The purpose of the investigation was mentioned briefly, and a stamped addressed envelope was enclosed for reply. Another letter was sent to the general practitioners of the patients concerned, asking for information about their progress and present health.

The response to these letters was very gratifying, since replies were received from 142 patients or their relatives and from 134 doctors, and from these letters it was possible to assess in some degree the progress or present health of 153 of the 172 patients who were discharged from hospital. Most of the surviving patients who replied agreed to come to hospital, and were interviewed; a few who were prevented from attending by age or infirmity were visited where possible. Eight patients had removed to other parts of the country, but replied, and later, in response to a second letter, gave details of their progress since the illness; in each case the letter had been forwarded by the present occupier of the house in which they had lived previously. One patient eventually received the letter and replied after it had gone to five separate addresses. Two other patients had emigrated —one to Israel, the other to Australia. The former patient replied to the original letter, which was forwarded to her by relatives, and the latter was eventually traced through the good offices of the Commonwealth Immigration Officer at Australia House. One patient was a Canadian Service-man when admitted to this hospital in 1944; his home address in Canada was recorded in his case notes, and he too replied. In the case of patients who had died since discharge, rela-

tives or neighbours were, as a rule, willing to give information in response to a tactful inquiry concerning the fatal illness; in several other cases the general practitioner supplied such details. Sometimes the present occupier of the house spontaneously made inquiries and was able to trace the whereabouts of the patient. In no case was information refused. Only three patients who were physically able to attend the hospital refused to do so, but they were later visited at their homes.

In certain instances it was possible to trace individual cases on very scanty information. Thus, to cite an example, the occupier of the house where Case 255 formerly lived said that he did not know where the patient was, but he did know that his brother was a jockey with Mr. X, at Newmarket. A telephone call to a firm of local turf commission agents was successful in obtaining the trainer's address. A letter was written to the trainer, who passed it on to the jockey concerned. The jockey replied that he had not seen his brother for years, but gave his mother's address. A letter to the mother was forwarded by her to the patient, who visited the hospital the following week. Similar manœuvres were necessary in tracing other cases, but eventually, with persistence, 159 patients were traced. Three of the remaining 13 cases were found with the help of the Tyneside Council of Social Service; representatives visited the neighbourhood and were able to obtain the required information. Another eight were traced with the aid of a final-year student of the social studies department of King's College, who visited the patient's previous address and by tactful inquiry in the neighbouring houses and streets was able to discover what had happened to the patient in every case. Thus, in all, 170 patients of the 172 who were discharged from hospital were traced; the only two who could not be found were serving in the Army when admitted to this hospital, and, although letters were forwarded to their last known addresses via the War Office, no replies were received.

Of the surviving 120 patients, 104 were questioned and examined by me, and the remainder, who were inaccessible, forwarded whatever information was asked of them.

I wish to thank the physicians of the Royal Victoria Infirmary for allowing me to consult their records and to examine their cases, and in particular I am grateful to Professor F. J. Nattrass for his advice and encouragement. I was able to examine the records of the Department of Neurosurgery, Newcastle General Hospital, through the courtesy of Mr. G. F. Rowbotham. I am also grateful to the many general practitioners in the North-eastern region who supplied information about their patients, and to Miss G. Nicholson, lady almoner of the Royal Victoria Infirmary, Miss Lois Armstrong, of the Department of Social Studies, King's College, and to the numerous others who gave invaluable aid in tracing surviving patients. I must also thank Mr. C. F. Naylor and the staff of the Records Department, the Royal Victoria Infirmary, for their help, and Miss Audrey Cairns, who carried the main burden of the large amount of clerical work involved.

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INTRA-ARTERIAL INJECTIONS IN THE TREATMENT OF PERIPHERAL VASCULAR DISEASE

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The arterial route for the introduction of therapeutic substances has been used since the turn of the century in a wide field of disease. In 1899 Parlaveochio treated a severe infection of the limbs by injection of antiseptic solutions into the brachial and femoral arteries. Leriche and others used intra-arterial injections from 1913 onwards for the administration of specific antisera. The pioneer work of Sicard and Forestier (1923) in the introduction of relatively innocuous radio-opaque materials into the arterial tree encouraged the use of this route for therapeutic agents. Intra-arterial vasodilator substances were apparently first used by Singer in 1943, and since this time numerous workers have used this method to improve the peripheral circulation with varying success. Their cases were varied, but all showed features of ischaemia. When success was obtained it was believed to be due to reduction of vasoconstriction as well as to the opening up of collateral channels.

Present Investigation

The present series consists of patients with organic occlusive arterial disease, subdivided into two broad groups: those presenting symptoms primarily of intermittent claudication and those in whom ischaemic necrosis was paramount. All patients with manifest coronary artery disease were excluded. A number of vasodilator substances have been used, with the aim of improving exercise tolerance in intermittent claudication or limiting the development of ischaemic necrosis. Table I shows the total number of cases which received treatment by this means, divided into the two above-mentioned groups for age and sex.

TABLE I

	Over 50		Under 50		Total
	Male	Female	Male	Female	
Intermittent claudication ..	49	2	6	0	57
Ischaemic necrosis ..	18	8	1	0	27

The success of this method can be assessed in a number of ways, and we employed the following: (1) clinical; (2) variation in oscillometry; (3) exact estimation of exercise tolerance (walking) by means of the claudicometer; and (4) immediate thermometric response.

Clinical.—Assessment by the patient of betterment was received critically. Good clinical criteria of improvement were: increased warmth, lessened pain both at rest and after exercise, nearer approach to normal colour, and evidence of healing.