MEDICAL PRACTICE

Occasional Survey

Intracranial Aneurysms

W. R. HENDERSON, D. S. MEHTA

British Medical Journal, 1973, 2, 30-35

Summary

A reappraisal of case histories of patients with ruptured intracranial aneurysms emphasized the importance of clinically recognizing severe spasm that contraindicates early angiography and large cerebral haematomas that require immediate evacuation. Observation from the day of haemorrhage is important; most recurrent episodes with cerebral signs in the first 10 days were due to spasm; haemorrhage was more common during the following two weeks. In many attacks the signs were not sufficiently distinctive for diagnosis of spasm or haematoma.

Introduction

This paper reviews the clinical manifestations of arterial spasm and intracerebral haemorrhage in cases of ruptured aneurysm. It is important clinically to recognize these complications, especially in severe cases, in order to avoid excessive reliance on angiography, which may be fatal in the presence of severe spasm.

Necropsy records studied by Richardson and Hyland¹ showed that the commonest cause of death was haemorrhage into the brain. Infarction was recognized later,² and with the increasing use of

angiography it was established in the 1950s that spasm can cause reversible ischaemia with temporary cerebral signs or infarction resulting in permanent disability or death. It was also concluded that angiography may aggravate spasm and should sometimes be delayed until after clinical improvement in spite of the risk of recurrent haemorrhage.³⁻⁸ Recurrent clinical attacks were at one time thought to be due invariably to haemorrhage, and the importance of prevention sometime led to early angiography and operation, even within the first day or two. This also ensured that large, potentially fatal haematomas would not be missed, but mortality was high. Cerebral signs in many early recurrent attacks and occasionally after first haemorrhages are, however, due to spasm. In statistical analyses spasm was rarely separated from haemorrhage.

The present study is based on 323 patients who were admitted to hospital or seen by one of us during 1948-68; nine other patients (seven of whom died) whose records were missing or inadequate are excluded (tables I and II). Clinical attacks were classified as mild—that is, with or without loss of consciousness for up to two hours—and severe—that is, with loss of consciousness lasting for several hours or longer and usually associated with cerebral signs. Spasm sometimes caused focal signs without headache or impairment of consciousness. Lumbar puncture was used after most recurrent attacks. Absence of fresh blood usually indicates spasm but it also occurs with a purely intracerebral (or subdural) haematoma; on the other hand, cerebral signs accompanying subarachnoid haemorrhage may be due to severe spasm.

The cerebral manifestations of aneurysm at each of the three common sites are well known. It is often possible to recognize severe spasm from focal signs due to involvement of areas supplied by the middle cerebral artery (face and arm) or the anterior cerebral artery (leg) and large haematomas by deepening coma (and local, usually temporal, signs). In early management the cause of cerebral signs—haematoma or spasm ischaemia—is more important than the site and visual proof of presumed aneurysm; this knowledge allows consideration of other cerebral lesions.

Leeds General Infirmary, Leeds LS1 3EX

W. R. HENDERSON, F.R.C.S., Honorary Consultant Neurosurgeon

D. S. MEHTA, F.R.C.S., Senior Neurosurgical Registrar (Present appointment: Associate Professor of Neurosurgery, Maulana Azad Medical College and Irwin and G.B. Pant Hospitals, New Delhi, India).

Spasm

Spasm is presumably a protective reflex to control bleeding, but the reduced blood flow may result in distal ischaemia or infarction. Spasm is sometimes triggered later in the absence of fresh bleeding possibly by further weakening of the aneurysmal wall.

Clinical manifestations of spasm are characterized by the relatively silent onset of focal signs, without headache and often without loss or impairment of consciousness. Sometimes, however, the patient is anxious and restless, and there may be temporary drowsiness or confusion. Focal cortical signs may be transient—that is, lasting for only a few minutes or hours—and occur either at the moment of haemorrhage or later. Such brief signs, which are easily missed, indicate the presence of severe spasm and are a very important warning of possible danger from angiography. They emphasize the importance of observation, which is, in effect, cardinal in diagnosis (after lumbar puncture in a neurosurgical centre from the day of haemorrhage. In more severe attacks the signs persist for several days or weeks, sometime from the time of haemorrhage; usually, however, they appear during the first or second week, often without fresh haemorrhage. The signs may be maximal from the start but sometimes they increase in stages during one or more days; later they subside over a few days or weeks but the decline may be interrupted by exacerbations or their disappearance may be followed by a short recurrence. Remissions, with return to full alertness, differ from fluctuations due to a large haematoma in the semicomatose patient, who at best is very drowsy. Signs that will disappear after temporary ischaemia cannot at the time be distinguished from those of infarction. Infarction is not as rapidly fatal as haemorrhage, except sometimes after angiography. Angiographic evidence of spasm was graded as slight, moderate, or severe; only severe spasm is likely to produce clinical effects. Severe spasm with very narrow, threadlike, or invisible arteries is rarely seen later than two weeks after the beginning of the last attack of haemorrhage or spasm; it may remain symptomless but it is potentially dangerous, and the first anteroposterior films show whether angiography should be abandoned. The prevention of exacerbation of spasm is as important as forestalling further haemorrhage.

MIDDLE CEREBRAL ANEURYSMS

The syndrome of sensory impairment and weakness of the face and arm, with dysphasia and hemianopia, occurs with middle cerebral aneurysms (and with some posterior communicating) aneurysms. In this series dysphasia was the most severe and persistent effect; next came sensory loss and weakness in the hand and face and, less often, partial or complete hemianopia, though this may have been only an inattention defect. Arm monoplegia without signs in the leg in a conscious patient is virtually diagnostic of spasm. When severe spasm spreads to the anterior cerebral artery there may be additional signs in the leg which generally recover quickly. Hemiplegia with aphasia and hemianopia in a conscious patient cannot be distinguished clinically from that more often due to spasm from anterior or posterior communicating aneurysms, which are more dangerous during angiography. The following case illustrates a typical history of spasm.

Case 1.—A man aged 41 had a mild subarachnoid haemorrhage without signs. After three days there was severe dysphasia for several hours, and two days later another transient attack of dysphasia occurred with weakness of the right hand. Eight days after haemorrhage there was a pronounced change: he remained conscious and alert but there was total aphasia and hemianopia, facial weakness, reduced pain sensation and paralysis of the right arm, and moderate weakness of the leg. Lumbar puncture was negative for fresh blood. Bilateral angiography after two days

showed left middle cerebral aneurysm and extreme spasm of the middle cerebral artery and of the proximal anterior cerebral artery, which in a later angiogram was found to bifurcate. The leg recovered in four days but improvement in speech and in the arm was slower and continued after operation eight days later.

Several patients had symptoms lasting only a few hours during the first week (case 1). Five patients had more persistent symptoms of spasm which appeared 7–11 days after haemorrhage, apparently without recurrent bleeding, as indicated by negative lumbar puncture (four recovered). In a further five patients similar focal signs occurred immediately after bilateral angiography 8–12 days after haemorrhage; three of them had middle cerebral infarction (two died). Eight of the nine angiograms showed severe spasm.

Angiography in the second week in four patients with vessel displacements due to temporal haematoma, which had produced drowsiness and hemiparesis from the time of the initial haemorrhage, also showed severe spasm; the spasm had apparently remained clinically silent except possibly in one patient, in whom after 10 days the hemiparesis progressed to hemiplegia for a few days without impaired consciousness and lumbar puncture was negative. Signs that persist after haemorrhage are more likely to be due to haematoma than to spasm (table II), and this must be assumed when the signs are not distinctive enough—for example arm monoplegia—to diagnose spasm or when earlier clinical examinations were inadequate for assessment. The late appearance in a fully conscious patient of dysphasia, sensory loss or weakness of the hand, or hemiplegia is unlikely to be due to haemorrhage.

ANTERIOR COMMUNICATING ANEURYSMS

These aneurysms, at the midline and close to both ventricles, are the most dangerous (table I). Severe spasm usually involved both anterior cerebral arteries but occasionally it was unilateral (case 2); surprisingly the middle cerebral artery was rarely involved in patients with hemiplegia. Clinical signs may be confined to one leg or may present as hemiparesis maximal in the leg or as paraparesis; maximal involvement of the leg in aneurysm cases is strongly suggestive of spasm (it also occurs with posterior fossa aneurysms⁹). Ten case histories recorded transient focal cortical signs during mild initial haemorrhages. Other transient attacks—non-focal but because of their brief duration characteristic of spasm—sometimes occurred after a few days; the predominant effects were sudden restlessness, disorientation, drowsiness, loss of consciousness, or paresis lasting only hours.

TABLE I—Cause of Death

Site of Aneurysm	No. of Patients	No. of Hospital Deaths	Cause of Death (Necropsy Cases)		
			Haematoma	Infarction	Operation
Middle cerebral	74	22	12 (5 Vent.)	4	4 (2 Infarct)
Anterior communicating	106*	41	22 (18 Vent.†)	13	4 (4 Infarct)
Posterior communicating	119	26	11 (9 Vent.)	7	6 (Carotid ligation)
Carotid bifurcation ..	11	3	1 Vent.	1	—
Carotid-ophthalmic	5	1	—	—	1
Basilarvertebral	8	3	3 Vent.	—	—
Total	323	96	49 (36 Vent.)	25	15

* Includes 8 with anterior cerebral aneurysm on the fornix.

† Three had previous severe infarction.

Vent. = Intraventricular haemorrhage.

Leg monoparesis.—The signs in four patients were confined to one foot or leg, perhaps with some mental confusion and incontinence but not loss of consciousness. One patient died six hours after too-early (18-hour) angiography.

Hemiparesis.—The early confident recognition of spasm

hemiparesis is important in order to prevent exacerbation by angiography. Twelve patients had hemiparesis or hemiplegia which developed suddenly or over a day or two beginning in the leg and without loss of consciousness. Severe weakness was maximal in the foot, which recovered last, sometimes with permanent slight weakness; there was corresponding impairment of pain and joint sensations. Hemiplegia (sometimes called late hemiplegia) was total in seven patients, three of whom had virtually complete recovery. Partial or complete hemianopia was recorded as present in four patients and absent in four. It may be several days before improvement begins and several weeks before good recovery (four cases). At some stage there may also be slight weakness of the other leg or pronounced mental apathy and incontinence due to involvement of the other frontal lobe—the latter symptoms may be due to a small frontal haematoma, but the leg signs are due to spasm. The following case illustrates spasm hemiparesis.

Case 2.—Man aged 23. During eight weeks there were three almost certain but unproved mild subarachnoid haemorrhages. Five days after the last attack the left leg suddenly "gave way," and this was followed later by numbness and weakness of the left arm. During the next two days the weakness increased to hemiplegia with sensory loss but there was no hemianopia. The patient was never unconscious and was fully alert. After four days bilateral angiography showed on the right side severe spasm and on the left an anterior communicating aneurysm but no spasm. The aneurysm was clipped and obliterated (Mr. A. E. Wall) with good recovery. The absence of spasm on one side probably reduced the risk of early angiography.

Angiography was followed by coma in four conscious patients with hemiplegia (see case 3); clinically two patients had an orbital bruit, two had had transient signs of spasm, and angiography was performed too early (12 hours in two, three days in two). It seems that during angiography the effects of severe spasm may extend to the other frontal lobe, producing infarction with coma and paraparesis (two cases), whereas ischaemia in middle cerebral territory is more likely to remain reversible. Hemiplegia in the following conscious patient might have disappeared without angiography.

Case 3.—Man aged 46. Six days after a mild proved subarachnoid haemorrhage without signs there was the onset in stages over a few hours of left hemiplegia beginning in the leg and hemianopia. The patient remained conscious and co-operative though lethargic and lumbar puncture was negative. There was an orbital bruit. Bilateral angiography 12 hours after the onset showed an anterior cerebral aneurysm on the fornix and severe bilateral spasm. Angiography was followed immediately by coma. Later he was awake but mute and both legs were weak; he died after six weeks. Necropsy showed bilateral anterior cerebral infarction and slight infarction in right middle cerebral territory.

In seven patients necropsy showed anterior cerebral infarction on one side; middle cerebral infarction was found in four (two slight), and there was infarction in the opposite anterior cerebral area in four.

Intracranial (orbital) bruit is an occasional, very important sign of severe spasm in cases of aneurysm but it is more common with large cerebral angiomas, when it is sometimes heard also over the scalp.¹⁰ Bruit is a strong contraindication to early angiography.

Paraparesis.—Seven patients had signs in both legs. In attacks of moderate severity there is the possibility of recovery when angiography is deferred, as in the following case.

Case 4.—A 47-year-old woman had a mild, almost certain subarachnoid haemorrhage at home, and four days later a more severe, proved haemorrhage occurred with brief unconsciousness. On admission the following day she was conscious but apathetic, confused, unco-operative, and incontinent, both legs were weak, especially the right, and the right arm sagged; there was no hemianopia. Angiography was deferred for nine days, when it showed extreme spasm of both proximal anterior cerebral arteries

and an anterior communicating aneurysm but no filling of distal vessels. Three days later she became mentally alert, but after a few days confusion, incontinence, and weakness in the right leg, sometimes spreading to the arm and other leg, returned and continued to fluctuate with complete remissions for several weeks. Operation was not performed. She remained well six years later.

In more severe attacks, with paraplegia and weakness of one or both arms, the patients were mute and unresponsive to pain, though still awake; the signs progressed to near-quadruplegia but the pupils remained normal until terminal coma supervened shortly before death 5-16 days after haemorrhage. Infarction cannot be recognized clinically in the later stages when there is semicoma without adequate history of onset or definite involvement of both legs. Angiography after three to six days had no obvious immediate aggravating effect, comparable to the change from conscious hemiplegia to coma paraparesis (case 3), possibly because both frontal territories were already involved, as in the following case.

Case 5.—A man aged 41 had a severe subarachnoid haemorrhage with loss of consciousness for several hours followed by drowsiness, paralysis of both legs and the left arm, and left hemianopia; after a day or two he spoke a few words. Five days later, although awake, co-operation was poor. There was quadriplegia, apart from slight movement in the right fingers, and bilateral loss of pain sensation but the pupils were normal. Next day bilateral angiography showed an anterior communicating aneurysm but only slight spasm. He died 24 hours later. Necropsy showed bilateral anterior cerebral infarction. It is assumed that initially very severe spasm produced anterior cerebral infarction and middle cerebral ischaemia and then subsided. In retrospect the near-quadruplegia contraindicated angiography (in 1952).

In six cases necropsy showed severe bilateral anterior cerebral infarction affecting the corpus callosum but no naked-eye middle cerebral infarction.

In summary, weakness of an arm and a leg may be regarded simply as hemiparesis if the other foot is not carefully examined, especially when co-operation is poor. The diagnosis of spasm requires greater confidence in conscious patients with hemiplegia than in patients with paraparesis and recovery seems to be more likely, but deterioration aggravated by angiography is more obvious. Infarction involves one or both frontal lobes, but middle cerebral ischaemia seems often to be at the preinfarction stage whether there is hemiparesis or paraparesis. Sixteen necropsies (three after terminal intraventricular haemorrhage) showed bifrontal infarction in 10 patients (also slight middle cerebral in two); unilateral anterior and middle cerebral infarction in only two; infarction confined to one anterior cerebral territory in one; and cerebral oedema (possibly infarction too early to be seen with the naked eye) in three patients, all previously conscious, who died six or seven hours after angiography.

POSTERIOR COMMUNICATING ANEURYSMS

Severe spasm involves mainly the proximal arteries and affects (like carotid ligation) middle cerebral territory more often than anterior; the risk from angiography is greater than when similar signs from a middle cerebral aneurysm are present. Both distal anterior cerebral arteries usually fill well from the other carotid when the anterior communicating connexion is adequate, but extension of severe spasm across the midline is apt to produce signs similar to those of spasm from anterior communicating aneurysms.

Eight patients had severe unilateral signs which in one case were confined to the leg and in the others were primarily of middle cerebral distribution, varying from dysphasia or signs in the arm to hemiplegia; four patients recovered in a few days or weeks but four died after angiography. Angiography was followed in 10 patients by the appearance of or exacerbation of symptoms (seven died); it should be abandoned when

severe spasm is found on the first films (carotid ligation must in any event be postponed).

Necropsy in seven patients (no carotid ligation) showed middle cerebral infarction in four and bilateral anterior cerebral infarction in two; one necropsy following death 12 hours after angiography showed only oedema.

ANGIOGRAPHY

Consideration of the clinical effects of spasm requires mention of possible effects of angiography. It may be difficult to recognize deterioration when the patient is already in coma or when angiography is followed immediately by operation (these important factors are not always mentioned in statistical analyses), but there can be no doubt when there is sudden noticeable change with new signs, and especially coma in a previously alert patient.

Most of the patients (289) had angiography (24 angiograms were missing or poor). Out of 177 angiograms taken in the first two weeks after the beginning of the last attack of haemorrhage or spasm 46 showed severe spasm. Only seven patients had angiography within 24 hours of the onset of symptoms; five died from infarction (four were previously conscious). Only one flap craniotomy (out of 116) and 14 carotid ligations (in 89 patients) were performed on the same day as the angiography, partly because of a general policy of allowing recovery from angiography before operation. Only one of the 28 patients found to have severe infarction at necropsy (table I) had had a craniotomy; four had not had angiography. It seems from surgical statistics that some "post-operative" infarctions had already occurred before operation and were not due to surgical technique. Six other necropsies after craniotomy on patients with milder signs and not in coma showed cortical infarction and also, unlike most unoperated cases, naked-eye infarction in basal ganglia due to operative damage to arteries.

Angiography was followed immediately by clinical deterioration (case 3) in 22 patients (14 previously had no signs), of whom 17 became comatose or mute (two were already in semi-coma); 15 died (seven in 5-12 hours). The prevention of complications from angiography rests on (1) clinical selection—avoiding especially too-early timing; orbital bruit; transient or continuing signs of spasm; and general detrimental factors (age, low haemoglobin, general vascular disease)—and (2) radiological technique—avoiding excessive injections when the first films show severe spasm or when there is difficulty in visualizing an aneurysm.

Intracerebral Haemorrhage

Intracerebral (or subdural) haemorrhage produces clinical signs from the onset. Medium-size haematomas with local

cerebral signs are not necessarily urgent, but when larger, with continuing drowsiness of fluctuating semicoma, chest complications may supervene if operation is unduly delayed, whereas large unruptured (into the ventricle) haematomas with high intracranial pressure and deepening coma require early evacuation within the first two days or even hours. Intraventricular rupture with profound coma is usually rapidly fatal within hours or even minutes. Haematomas are more common, and are more easily diagnosed, in the temporal lobe than in the frontal lobe (table II).

TEMPORAL HAEMATOMAS

Most temporal lobe haematomas are from middle cerebral aneurysms, are superficial, and when large can be aspirated. Blood from posterior communicating aneurysms is more likely to enter the temporal horn, but sometimes it misses the ventricle and continues laterally to form a large haematoma.

Medium haematomas produced loss of consciousness for hours or days followed by apathy, drowsiness, or confusion similar to that with frontal lobe haematomas but distinguished by the presence of temporal signs. All left-sided haematomas produced dysphasia. There was usually hemianopia, which may be confined to the upper or lower quadrant. These temporal signs are more severe than the frequent additional hemiparesis and sensory impairment that usually involve the arm more than the leg.

Large haematomas produced coma and severe signs of sudden or rapid onset during several hours that continued until, in five cases, the high intracranial pressure was relieved within the first three days by burrhole aspiration of 20 to 30 ml of blood after or without angiography. When consciousness returned re-examination confirmed the previously obvious severe hemiparesis and reduced pain sensation and also showed dysphasia and hemianopia; there was noticeable improvement before craniotomy a week or two later. By far the most important sign, and the indication for immediate aspiration, was continuing coma and not the severe long tract or temporal lobe signs which alone could be due to spasm and are not signs of urgency. A haematoma should be excluded when consciousness does not return within two days but operation may be required much earlier for deepening coma, a dilating pupil, or bradycardia, as in the following case.

Case 6.—A 37-year-old woman had a sudden severe headache with vomiting at midnight; at 6 a.m. she was found unconscious. When seen six hours later pain stimulation showed paralysis of the right arm and partial weakness of the leg. The pupils were normal. Three hours later respiration was irregular and the left pupil was dilated and fixed. Temporal haematoma was suspected, and without angiography an immediate left temporal burrhole aspiration (15 hours after the onset) produced 20 ml of blood from inside the temporal lobe (Miss C. M. Bannister). Next day she was conscious and the pupil was normal; there was aphasia and hemianopia. The

TABLE II—Severity of Initial Haemorrhages and Causes of Severe Attacks

Site of Aneurysm	No. of Patients	Initial Attacks			Severe Recurrent Attacks			Severe Initial and Recurrent Attacks of Unknown Cause
		Mild	Severe (Known Cause)		Haematoma	Spasm or Infarction		
			Haematoma	Spasm or Infarction		Before Angiography	After Angiography	
Middle cerebral	74	45 (6)	21	—	16 (5 Vent.)	5	5	12
Anterior communicating ..	106	87 (20)	5	4	27 (18 Vent.)	19	7	20
Posterior communicating ..	119	108 (32)	4 (1 Vent.)	1	13 (8 Vent.)	8	10	11
Carotid bifurcation	11	11 (1)	—	—	1 Vent.	1	—	2
Carotidophthalmic	5	5 (1)	—	—	—	1	—	1
Basilarvertebral	8	6	1 Vent.	—	2 Vent.	—	—	1
Total	323	262 (60)	31 (2 Vent.)	5	59 (34 Vent.)	34	22	47

Mild attacks include unproved recent haemorrhages at home (in parentheses) and oculomotor palsies without lumbar puncture. The cause of severe attacks is known from necropsy or operation findings or is assumed from angiograms showing severe spasm without vessel displacement; small (1-2 cm) haematomas discovered at operation or necropsy and transient attacks of spasm are not included. In the unknown group cerebral signs were not investigated or early case records were inadequate; the signs later were usually slight or had disappeared and no patients died (except 7 without necropsy—table I). Numbers refer to attacks not to patients.

Vent. = Intraventricular haemorrhage.

signs decreased before angiography 12 days later showed a large middle cerebral aneurysm which was wrapped. There was good recovery apart from slight dysphasia.

A unilateral dilating pupil does not always occur but when present it is very important. When dysphasia and hemianopia cannot be shown, continuing coma and hemiparesis could be due to a haematoma in the frontal lobe or elsewhere. Angiography is not always necessary because unruptured large temporal (or frontal) haematomas are usually situated laterally and can be found through a burrhole (which excludes also most subdural haematomas). If temporal aspiration is negative a frontal burrhole should then be made. Aspiration without angiography can be tried when the patient is very ill.

FRONTAL HAEMATOMAS

Haematomas from anterior communicating aneurysms are commonly in the inferomedial quadrant of the frontal lobe, where they easily enter the ventricle, but sometimes the blood goes more laterally and forms a large unruptured haematoma. Haematomas from aneurysms on the fornix are higher in the frontal lobe, and from middle cerebral aneurysms are situated laterally. Medium haematomas of about 5 to 10 ml produced "frontal lobe" symptoms recorded as mental confusion, incontinence, apathy or drowsiness, and sometimes slight hemiparesis but without loss of consciousness after the first few hours. Large haematomas produced continuing coma or fluctuating semicoma and slight or severe hemiparesis.

INTRAVENTRICULAR HAEMORRHAGE

Haemorrhage from anterior or posterior communicating aneurysms is more likely to enter the ventricle than is haemorrhage from middle cerebral aneurysms, which more often produces an unruptured haematoma (table II). Intraventricular haemorrhage with profound coma caused 36 deaths—20 within 4 hours and 9 within 6-16 hours; in 7 cases death occurred 36-72 hours after the haemorrhage, the reason for which is uncertain without detailed clinical records to show whether there might have been an early recurrent haemorrhage with sudden change just before death.

Equally rapid deaths were occasionally due to unruptured temporal haematoma (four cases) or subdural haematoma (four cases); five patients died within three hours and three after 8-13 hours, but even the latter patients might have been moribund from the start from brain-stem haemorrhage, which is apt to occur with these types of compression. All were in deep coma, and apart from possible dilating pupil it seems that such rapidly fatal haematomas cannot be distinguished from intraventricular haemorrhage. A decision about very early aspiration for deep coma is difficult; it is rarely possible when death occurs in a few hours but early burrhole aspiration, which should find most operable haematomas, may be justified after, say, 12 hours when most deaths from intraventricular haemorrhage have already occurred.

Nearly all deaths from haemorrhage occurred during recurrent attacks after ample time for investigation of previous haemorrhage. Out of 44 intraventricular and rapid haematoma deaths only two occurred during initial attacks (many such patients die before they can reach hospital) and only five during the first 10 (2, 2, 5, 7, and 7) days, so that it should have been possible greatly to reduce this large number of haemorrhage deaths. We believe, as has been emphasized by many surgeons, that it should be general policy to investigate all mild initial haemorrhages, assuming that there is no general contraindication to operation. The timing can be fairly routine—observation for, say, three or four days when angiography should be safe provided that there is restraint when it shows severe spasm, followed by operation next day.

NATURAL HISTORY OF RECURRENT HAEMORRHAGE OR SPASM

Four out of five initial attacks were mild; most fatal haemorrhages and dangerous spasm occurred in later attacks (table II). Spasm caused nearly a third of all (129) severe attacks of known cause and a quarter of the hospital deaths. Although spasm was overall much less frequent than haematoma, during the first 10 days it was three times as common as haemorrhage (table III), thus emphasizing the need for caution before early angiography. Most of the severe recurrent haemorrhages occurred after 10 days, many in the following two weeks.

TABLE III—Interval before Recurrent Severe Attack of Known Cause

Cause	2-6 Days	7-10 Days	After 10 Days	Total
Haemorrhage ..	4	5	50	59
Spasm ..	11	16	7	34
Total ..	15	21	57	93

Intervening mild (subarachnoid) haemorrhages and transient manifestations of spasm are not included.

The timing of investigation, with least risk of another haemorrhage or exacerbating spasm, depends mainly on assessing the cause of existing signs and partly on the likely interval before another attack, and also on whether signs of spasm indicate involvement of middle or anterior cerebral territory. With maximal involvement of middle cerebral territory the first films, after, say, five to seven days, are likely to show either a middle cerebral aneurysm with distal spasm that is diagnostic and warrants exploration (full angiography can be completed after operation) or a potentially more dangerous aneurysm on the carotid, which necessitates a delay of 7-10 days before completing angiography. With anterior cerebral signs a delay of five to seven days is probably sufficient when milder "frontal lobe" symptoms are suggestive but not diagnostic of spasm, but with limb signs maximal in the leg or mutism a delay of 10 days is advisable, or longer when recurring transient signs or bruit indicate continuing severe spasm. With cerebral signs of uncertain cause angiography probably should be delayed at least five days to avoid converting reversible ischaemia into local infarction.

Conclusion

The problems, often conflicting, in management are prevention of another haemorrhage, recognizing and allowing severe spasm to subside, and early evacuation of a large haematoma. Angiography cannot be routine because of the danger of spasm; its timing depends on clinical assessment of the history and existing signs. In this series deaths from spasm aggravated by angiography emphasized the importance of clinically recognizing signs due to severe spasm with sufficient confidence to defer angiography.

Coma has always been regarded as the main criterion of severity and of prognosis. It occurs with all severe haemorrhages but not often with spasm, which produces predominantly cortical signs that may be severe without impairment of consciousness; it is easy to under-assess the potential danger of spasm in conscious patients with focal signs. Coma due to spasm occurs mainly in the later stages after several days or after angiography. Early investigation is inadvisable and is, we believe, unnecessary, because necropsy shows that large unruptured haematomas, the only indication for very early operation, are situated laterally (usually temporal, occasionally frontal) and can usually be aspirated (or excluded) through a burrhole without angiography; other haemorrhages with coma are into the ventricle or have damaged deep structures. Thus patients with severe focal signs, whether conscious or in coma, rarely require early angiography.

It has long been known that most recurrent clinical episodes occur in the first three to four weeks, and it was believed, before the effects of spasm were appreciated and often without evidence of repeat lumbar puncture, that they were always due to fresh haemorrhage, sometimes perhaps small but regarded as a sign of increasing urgency; it seems, however, that rapidly recurring small haemorrhages, or leaks, are uncommon and would not produce cerebral signs. Very early operation to forestall recurrent haemorrhage has a higher mortality than later operations due partly to the greater risk of operation haemorrhage and partly to angiography. Conservative treatment, on the other hand, does not prevent deaths from recurrent haemorrhage. We believe that better overall results can be obtained by relying less on angiography and more on clinical observation and selection before investigation, in spite of possible fatal haemorrhage while waiting for spasm to subside. This entails recognizing the small minority of very severe cases, either with large haematoma requiring immediate aspiration or with signs of severe spasm necessitating delaying angiography up to even several weeks. Most patients not in coma who are improving and have moderate signs due either to medium-size haematoma or to less severe spasm that often

cannot be differentiated clinically can have careful angiography after about a week before recurrent haemorrhage becomes more frequent. Perhaps the overriding consideration in the management of subarachnoid haemorrhage is referral immediately after the initial, usually mild haemorrhage.

It is a great pleasure to thank Dr. D. G. F. Harriman, neuropathologist, for his reports and helpful comments.

References

- ¹ Richardson, J. C., and Hyland, H. H., *Medicine*, 1941, 20, 1.
- ² Robertson, E. G., *Brain*, 1949, 72, 150.
- ³ Falconer, M. A., *Proceedings of the Royal Society of Medicine*, 1954, 47, 693.
- ⁴ Logue, V., *British Medical Journal*, 1956, 1, 473.
- ⁵ Small, J. M., *Proceedings of the Royal Society of Medicine*, 1957, 50, 289.
- ⁶ Gillingham, F. J., *Annals of the Royal College of Surgeons*, 1958, 23, 89.
- ⁷ Connolly, R. C., *Annals of the Royal College of Surgeons*, 1962, 30, 102.
- ⁸ Pool, J. L., *Journal of Neurosurgery*, 1962, 19, 378.
- ⁹ Dimsdale, H., and Logue, V., *Journal of Neurology, Psychiatry and Neurosurgery*, 1959, 22, 202.
- ¹⁰ Henderson, W. R., and Gomez, R. de R. L., *British Medical Journal*, 1967, 4, 571.

Hospital Topics

Thyroxine "Addicts"

RICHARD F. HARVEY

British Medical Journal, 1973, 2, 35-36

Summary

Four patients are described who were surreptitiously taking thyroxine-containing tablets. In two cases this caused considerable diagnostic problems. The diagnosis should be considered in patients with clinical features of hyperthyroidism (but without goitres or proptosis), who appear psychiatrically abnormal and whose thyroid function tests show conflicting results.

Introduction

Difficult diagnostic problems may be posed by patients who take unusual medications without telling their doctor. This report draws attention to one such instance—namely, the development of apparent thyrotoxicosis due to surreptitious self-administration of thyroxine. Four patients attended the Middlesex Hospital in the course of a 14-month period with clinical features suggestive of hyperthyroidism, and a combination of thyroid function tests indicated that all were taking exogenous thyroxine.

Middlesex Hospital, London W.1

RICHARD F. HARVEY, M.D., M.R.C.P., Medical Registrar (Present address: University Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HW)

Case Reports

Case 1.—A 51-year-old woman was referred to the outpatient clinic with a number of complaints, of which malaise and depression were the most prominent. There was a history of psychiatric disturbances for at least 15 years, unrelieved by electrical treatment. She had previously been investigated at at least two other London teaching hospitals and variously believed to have an overactive thyroid gland (unsubstantiated by tests) and hypopituitarism. She denied taking any medication apart from chlor-diazepoxide (Librium). She was hyperkinetic and had a pulse rate of 120/min, but no goitre or eye signs of thyrotoxicosis were present. Thyroid function tests showed a grossly raised serum thyroxine level (269 ng/ml) and a paradoxically low thyroid uptake of radioiodine (3% at 24 hours) (see table). On her second visit she was confronted and eventually admitted to taking "antidepressant" tablets from a private doctor, who was treating her without the knowledge of her own general practitioner.

Case 2.—A 21-year-old student nurse presented with loss of weight and grossly hysterical behaviour. She was hyperkinetic, with a resting pulse rate of 100/min, but no goitre or eye signs or thyrotoxicosis were present. She denied taking any medication. Serum thyroxine was 176 ng/ml and there was virtually no thyroid uptake of radioiodine (4% at 24 hours). When confronted, she admitted to taking tablets, prescribed by the same doctor as in case 1, as a combined medication for slimming and depression. She later had a major psychiatric episode requiring inpatient treatment at a mental hospital.

Case 3.—A 25-year-old woman was referred by her general practitioner for investigation of weight loss. There was a long history of various social problems and abnormal eating habits, and at the age of 14 she had been thought to have anorexia nervosa, with refusal of all fattening foods and loss of 3 st. (19 kg) in weight. She had also been treated as an inpatient at a mental