# Spontaneous Intracranial Haemorrhage

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## Introduction:

Spontaneous intracranial haemorrhage (SICH) can cause one of the most dramatic presentations of human illness; potentially it can also be a highly destructive event. The diagnosis of such a mishap tends to be arrived at clincally with relative ease: a combination of such symptoms and signs as sudden onset of headache, sudden deterioration in the level of consciousness, meningism and neurological deficit makes for rapid interpretation. However, not all cases of SICH present with florid symptoms and signs and a high degree of clinical suspicion must be present to detect the "warning haemorrhages" that often precede the catastrophic one.

## Broad Diagnosis:

Various aetiological factors may give rise to SICH (Table 1) and, in turn, SICH may cause various pathophysiological processes (Table 2). Therefore, the term SICH merely describes a broad diagnosis as does, for example, the term 'anaemia'. The diagnostic process must not end at this point: a specific diagnosis must be sought. In the patient presenting with dyspnoea on mild exertion (pathophysiological process), clinical methods lead to the diagnosis of anaemia (a broad diagnosis); further clinical acumen and investigations reveal the anaemia to be of iron-deficiency type due to chronic blood loss from a carcinoma of the caecum (specific diagnosis). An identical sequence has to be embarked upon in the case labelled as SICH.

This exercise entails:

1. the mundane art of history taking;
2. the 'hunt-down' attitude in the clinical examination;
3. the judicious use of investigative procedures;
4. adequate neurosurgical intervention.

I do not intend to review the aetiology, symptomatology, investigations and management, text-book fashion, of SICH; there are several such reviews available. My goal is to highlight certain features of SICH under the above four headings and illustrate them with relevant cases.

## The Mundane Art of History Taking:

The importance of history taking is pointed out to the student as soon as he joins the medical fraternity and a medical practitioner forgets this to his peril and to that of his patient. From Table 1. it is obvious that a past history of hypertension, anticoagulant therapy, epileptic seizures (that would suggest the presence of an arteriovenous malformation) etc. are important features but more important still is the course of events immediate to the attack.

## Case 1:

S.H., a 31 year old right-handed Asian male was admitted at the G.P.'s request because of headache persisting over a few days; the headache was of sudden onset and severe. Examination was non-contributory; a L.P. was performed and examination of the c.s.f. showed a mixed white cell reaction without evidence of fresh blood or xanthochromia; no organisms were seen in the c.s.f. but cultures were instituted. A tentative diagnosis of tuberculous meningitis was entertained and appropriate treatment commenced. Over the next few days, the patient had two further episodes of sudden exacerbation of his headache. One morning he suddenly lost consciousness and developed a right dilated pupil. He was referred to the neurosurgical unit where examination revealed a deeply unconscious patient with a right extensor response to pain, a fixed dilated right pupil and a dilating left pupil. An urgent CT scan showed a massive right temporo-parietal haematoma. He was immediately taken to theatre and more than 80 mls of clotted haematoma were evacuated (LVZ: 8/10/80). During the next two days the patient regained consciousness with signs of a left hemiparesis that improved steadily. Angiography proved negative. At present, the patient has no

TABLE 1:

## CAUSES OF SPONTANEOUS INTRACRANIAL HAEMORRHAGE

1. Rupture of vascular structures:
   (aneurysms,, arterio-venous malformations, capillary telangiectasia, cavernous angiomas);

2. Hypertension;

3. Blood Dyscrasias:
   (Sickle cell disease, polycythaemia rubra vera, leukaemia, thrombocytopoenic purpura; Waldenstrom's macroglobulinaemia;
   Hypofibrinogenaemia, hypoprothrombinaemia especially due to anticoagulant therapy; haemophilia);

4. Various causes:
   (encephalitis eg. Herpes simplex;
   arteritis e.g. S.L.E.;
   haemorrhage into tumors;
   haemorrhagic infarcts);

5. No cause found



(The most comprehensive analysis of intracranial haemorrhage is that of Pakarinen (Acta Neurol. Scan. Suppl. 29: 1 - 128, 1967). The prevalence rate of ruptured aneurysms in people less than 60 years was found to be 9.6/100,000 population; the incidence of intracranial haemorrhage due to all causes was 16.8/100,000. It is also estimated that the prevalence rate of angiomas is 10% of that of aneurysms. Therefore, in Malta, one would expect 32 cases of ruptured intracranial aneurysms in people less than 60 years, 3 cases of arterio-venous malformations, including telangiectasis and 25 cases of spontaneous intracranial haemorrhage due to other causes per year.)

TABLE 2:

## LIST OF MAIN PATHOPHYSIOLOGICAL PROCESSES CAUSED BY SICH

1. Meningeal irritation: headache, neck stiffness, backache;

2. Deterioration in level of consciousness, alteration in behaviour, alteration in level of mentation;

3. Focal neurological deficit that may help in localizing the lesion or that may be false localizing due to the general increase in intracranial pressure or due to diffuse arterial spasm secondary to the haemorrhage;

4. Mass effect of an intracerebral haematoma resulting in cerebral herniation syndromes;

5. Epilepsy that may be of the grand mal or Jacksonian type;

6. Development of hydrocephalus due to impairment of c.s.f. flow.

7. Cerebral infarction and localized cerebral atrophy.

detectable neurological deficit and is managing his own TV repair shop. (Figures 1 and 2 show a CT cut before operation and at a later date).

In retrospect, this patient must have had at least three "warning bleeds" that remained intracerebral without the blood dissecting into the subarachnoid space, the c.s.f. thereby only giving a white cell reaction. The final haemorrhage was massive with resulting tentorial herniation. The negative angiogrpahy puts this case as one of SICH in which no cause is found. This is a small group estimated to comprise some 12-18% of cases of spontaneous intracerebral haemorrhage.

I have also come across cases which have been diagnosed initially as cervical spondylosis and given a cervical collar because of the neck stiffness resulting from a minor subarachnoid haemorrhage, others diagnosed as 'Head-Injury' cases because their ictus happened whilst they were driving and others who were diagnosed as 'stroke'.

**The 'Hunt-Down' attitude in the Clinical Examination:**

The clinical examination is consciously directed to finding clues to the aetiology of the SICH e.g. auscultation for a cranial bruit.

**Case 2:**

While sitting at table, a 9 year old left-handed girl (T.G.) complained of sudden onset of headache followed by numbness of the right leg and then the child went into a convulsion. She was immediately referred to the neurosurgical unit. On examination on the unit (LVZ 13/4/80), the child was fully conscious and complained of a severe headache. There was a right hemiparesis with diminished sensation also on the right side; speech was fluent, visual fields were full, there was no neck stiffness, no fundal changes and no cranial bruits were audible. However, there were multiple circumoral and cheek lesions; suggestive of Hereditary Haemorrhagic Telangiectasia. The clinical diagnosis was of a high left hemisphere intracerebral haemorrhage due to a telangiectatic lesion. The CT scan appearance (figure 3) of a left para-ventricular haemorrhage was consistent with this diagnosis. The child's general condition and the scan appearances were encouraging and neurosurgical intervention was not warranted; indeed neurosurgery would be meddlesome. The immediate problem proved to be allaying the mother's anxiety, not only about the prognosis of the child but also about the welfare of the mother herself. The child's maternal grandfather had also suffered from a bleeding diathesis and the mother, who suffered from the occasional nosebleed, started to suspect that inherited factors were present and that she herself might eventually develop a 'stroke' and, of course, the mother was right: hereditary haemorrhagic telangiectasia is an autosomal

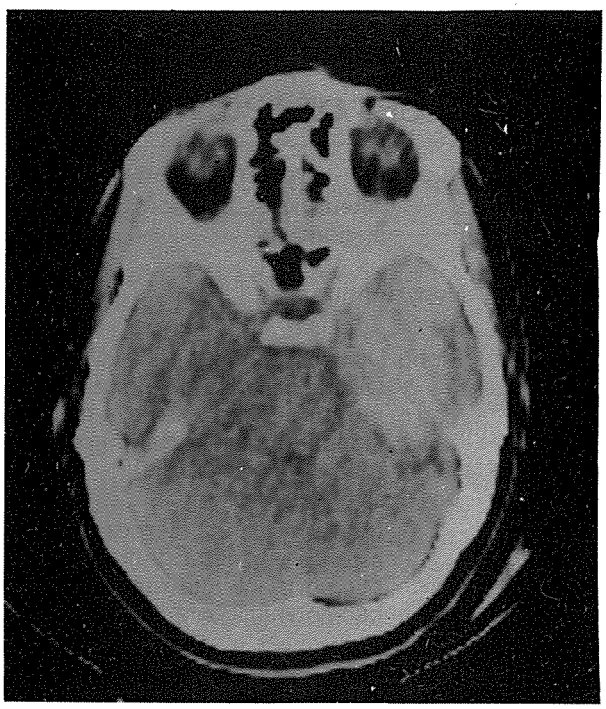

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*Figure 1: Right temporal intracerebral haematoma: Case 1;*

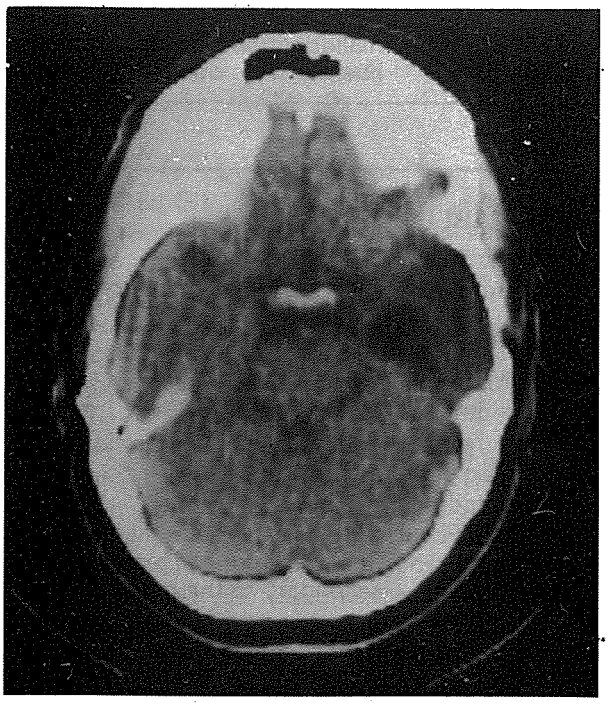

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*Figure 2: Follow-up scan of figure 1 showing an area of low attenuation in the right temporal region after evacuation of haematoma: Case 1;*

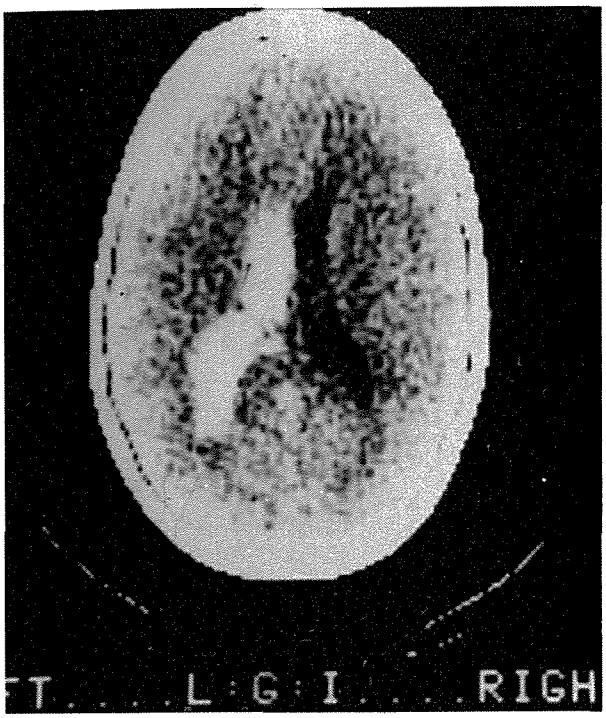


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*Figure 3: Left Paraventricular Haemorrhage in a case of Hereditary Haemorrhagic Telangiectasia: Case 2*

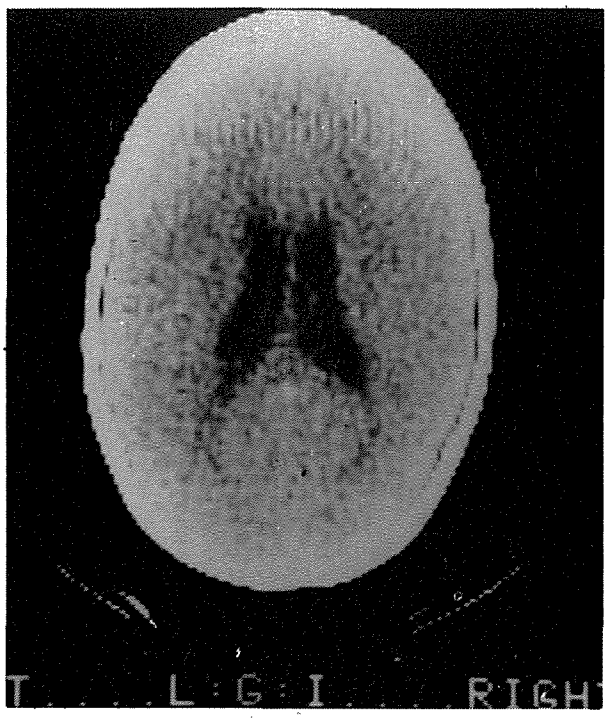


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*Figure 4: Follow-up scan of figure 3 showing resolution of the intracerebral Haematoma: Case 2.*

dominant transmitted disease and the mother did exhibit a few circumoral lesions.

The child remained well till the second day when she went into very resistant status epilepticus that could only be controlled by clonazepam infusions. After this stormy period, the child's condition steadily improved and her hemiparesis recovered. Angiography was eventually carried out but proved negative; the central nervous system lesions in Hereditary Haemorrhagic Telangiectasia are usually either so small as to escape detection by angiographic means or the lesion disrupts itself by the very act of bleeding. The child is now back at school and leading a normal life with no demonstrable neurological deficit. Her epileptiform seizures are extremely well controlled by anticonvulsant therapy. Figure 4 is a CT cut from a routine check scan.

**The Judicious Use of Investigative Procedures:**

Sound clinical judgement is essential to decide as to when and in which order investigative procedures need be performed in the management of SICH. The main procedures include:

1. Plain Radiology of the Skull;
2. C.S.F. Analysis;
3. C.T. Scanning;
4. Cerebral Angiography.

1. **Skull X-Ray** is non-contributory in most cases of SICH. However it may occasionally show:

(a) lateral shift of the calcified pineal due to mass effect of an intracranial haematoma;
(b) areas of calcification that arouse the suspicion of an arteriovenous malformation or tumour:
(c) curvilinear calcification that suggests the presence of a giant aneurysm.

2. **C.S.F. Analysis:** A lumbar puncture (L.P.) often helps to establish the 'broad diagnosis' of SICH and is indicated in a conscious patient who exhibits meningism.
However:

(a) L.P. *need not* be done if funduscopy shows subhyaloid haemorrhages to be present in a patient who exhibits meningism. The presence of such a clinical picture is pathognomic of subarachnoid haemorrhage and a L.P. is therefore superfluous. The same applies to the case where a CT scan shows clear signs of blood in the subarachnoid spaces.

(b) a L.P. should *preferably not* be done before at least six hours have elapsed since the attack. This interval would allow blood to disperse throughout the subarachnoid space and for xanthochromic changes to develop. Should the L.P. prove difficult and

traumatic, the presence of xanthochromia would then allow for no doubt to be entertained about the reality of an intracranial haemorrhage. This is not a hard and fast rule and a L.P. may be performed earlier if it is done by an expert and infective meningitis cannot be ruled out on the basis of the history and clinical findings alone.

(c) a L.P. *should not* be done if, either meningism is not present or the patient is unconscious and exhibits a focal deficit. In the former case, extravasated blood has not dispersed adequately throughout the subarachnoid space. If the patient is unconscious and exhibits a neurological deficit, the physician cannot be certain that a mass effect due to an intracranial haematoma is not operative and, in this situation, a L.P. is, of course, contra-indicated.

It must be remembered that meningism may be absent in fulminant cases of SICH despite the presence of blood in the subarachnoid space. This is due to the general decrease in tone that sometimes accompanies the catastrophe.

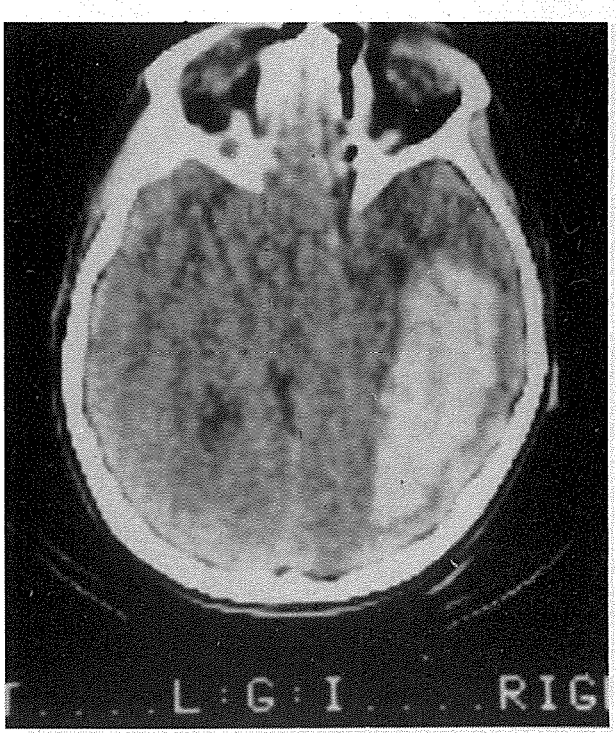


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*Figure 5: Massive right occipito-parietal intracerebral haematoma: Case 3.*

3. **C-T Scanning:** In the mid 70's, computed tomography was described as "coming of age"; since then, this investigative procedure has proved of such a great benefit to the neuro-sciences in particular and to medical science in general that, by the late 70's, it became clear that this is the "age of the scan". The August 1981 issue of the journal *Neurosurgery* published an article entitled *Neurosurgeons, Computed Tomographic Scanners and the Health Bureaucracy.* The author writes:

> "It is critical that... neurosurgeons and others emphasize the increasing wide applications of CT scanning to all parts of the body... We must acknowledge that our colleagues in some other specialities are not aware of the critical nature and diversified applications of these diagnostic procedures... But the question the planner must ask is whether the corresponding benefits justify the expenditures. Mounting evidence is that they do... There are unmeasurable savings in accurate diagnosis that saves lives... and there are measurable savings in shortened hospital stays... We must have them in acute general hospitals were there are seriously ill patients deserving an appropriate quality of medical care."

In SICH, CT scanning may show blood in the subarachnoid spaces if performed during the first few days after the attack: also, by locating the site of even a small haematoma, it could indicate the probable aetiology e.g. anterior communicatng, internal carotid, middle cerebral artery aneurysm, arteriovenous malformation or bleeding into a tumour

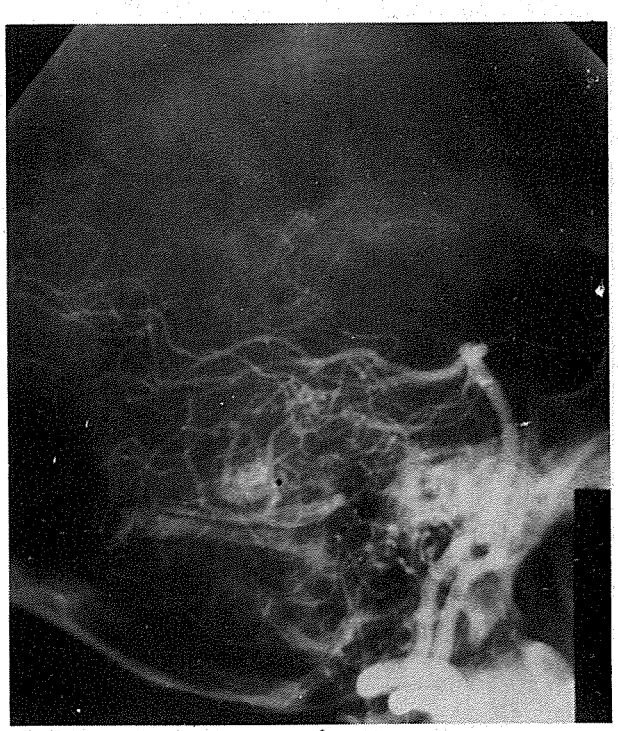

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*Figure 6: Vertebral Angiogram: Lateral View: showing tangle of abnormal blood vessels constituting an arterio-venous malformation: Case 3.*

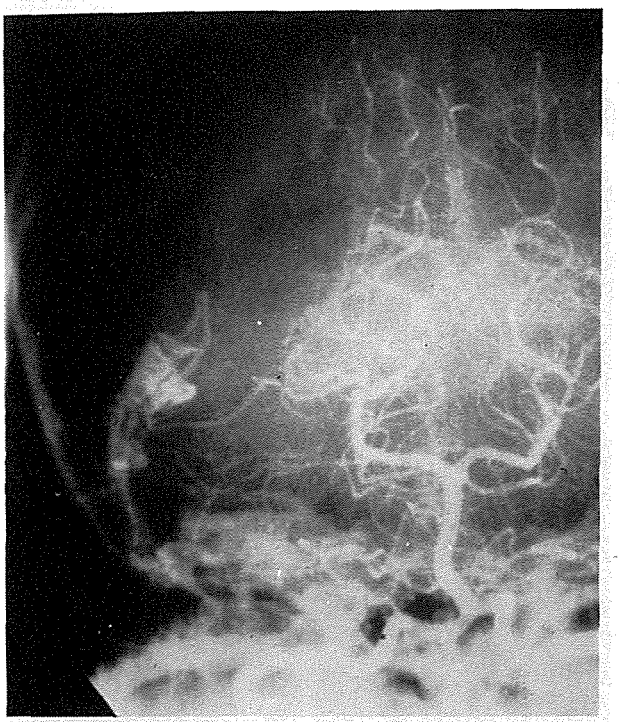

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*Figure 7: Vertebral Angiogram: Towne's View: showing the arterio-venous malformation: Case 3.*

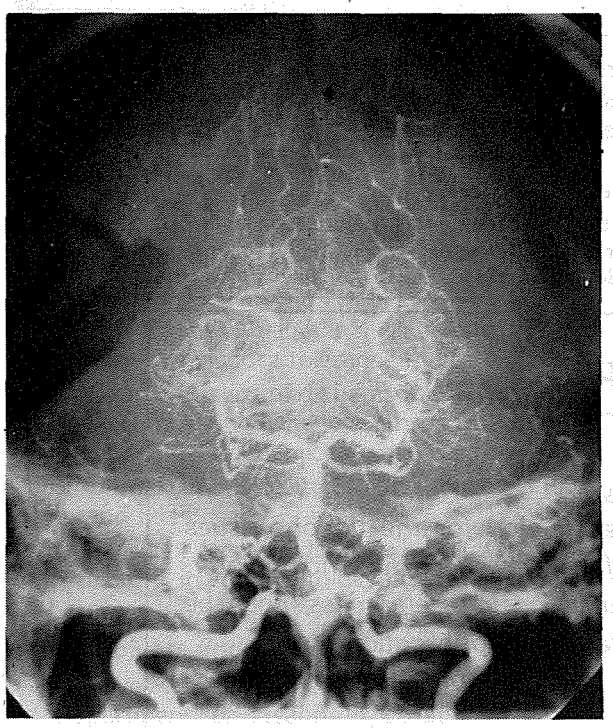

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*Figure 8: Post-operative check Vertebral Angiogram: Towne's View: showing the craniotomy and the absence of abnormal vascular patterns: Case 3.*

mass. However, the main use of CT scanning in SICH is in indicating the pathophysiological processes secondary to the SICH e.g. the site and mass effect of an intracranial haematoma, the presence of cerebral oedema or infarction, the development of hydrocephalus.

**Case 3:**

A 41 year old female (R.M.A.) was admitted to the neurosurgical unit via Accident and Emergency. The patient had been found collapsed at her home and on examination, she was deeply unconscious with an extensor response to painful stimuli on the right more than on the left; the right pupil was fixed and dilated, the left one was small but also fixed. There was no meningism. A presumptive diagnosis of a right hemisphere intracerebral haematoma was confirmed by an emergency CT scan (figure 5). This showed a massive right parietoccipital haematoma, the site of which suggested an arterio-venous malformation as the aetiological factor. Also, the density of the haematoma on the CT scan in Hounsfield units suggested that the haematoma was only partially clotted. The patient was immediately taken to theatre and 90mls of blood were aspirated through a right occipital burr hole (LVZ 24/5/81). This decompression relieved the cerebral herniation and the patient recovered consciousness over the next 48 hours with a residual left homonymous hemianopia.

4. **Cerebral Angiography:** Just as CT scanning demonstrates the pathophysiological processes ensuing after SICH, cerebral angiography elucidates the aetiology, e.g. arterio-venous malformation, aneurysm.

**Case 3 continued:** When the patient's condition had stabilized, cerebral angiography was performed and this showed an arterio-venous malformation in the right occipital region (figures 6 and 7): vertebral angiogram showing the A.V.M.). A right occipital craniotomy was performed and the AVM was excised completely (LVZ 12/6/81) as proved by post-operative check angiography (figure 8). The patient was discharged fully well apart from a left homonymous hemianopia and is currently pursuing post-graduate studies in arts.

**Case 4:**

A 52 year old female (D.V.) was admitted with a history of sudden onset of severe headache followed by loss of consciousness for a few minutes. By the time of admission, she was fully conscious; examination revealed severe meningism but no focal deficit. A presumptive diagnosis of subarachnoid haemorrhage was confirmed by lumbar puncture. Cerebral angiography was eventually performed: carotid angiography showed a right posterior communicating artery aneurysm and also a pericallosal artery aneurysm (figure 9); vertebral angiography was negative. The aneurysms were clipped using the operating microscope via a right

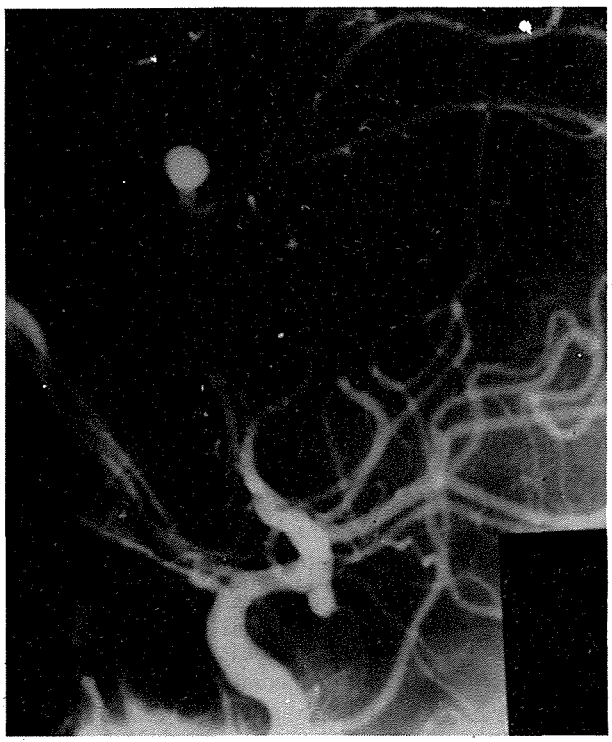

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*Figure 9: Carotid Angiogram: Lateral View: showing a posterior communicating artery aneurysm and also a pericallosal artery aneurysm.*

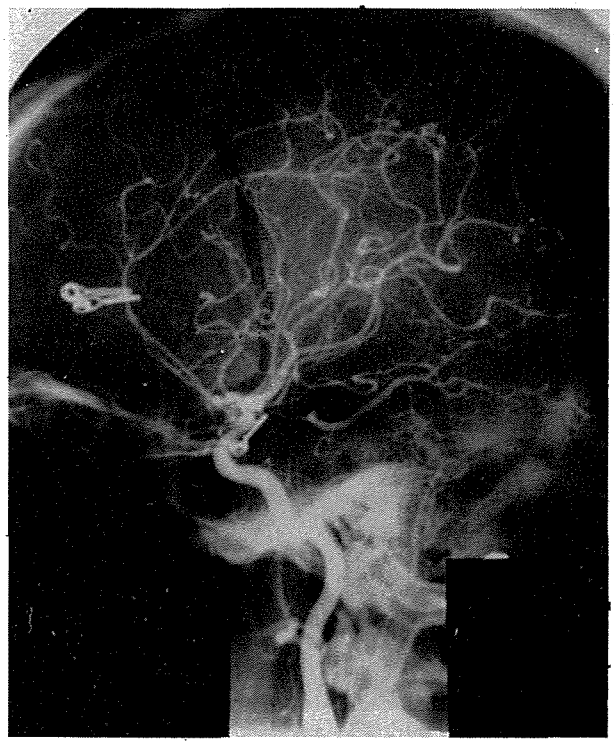

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*Figure 10: Post-operative check angiogram: Lateral view showing frontotemporal craniotomy and Scoville aneurysm clips in a good position (two clips are on the pericallosal aneurysm, a single clip is on the posterior communicating artery aneurysm).*

fronto-temporal approach (LVZ 15/5/80). Post-operative check angiography showed the clips to be in a very satisfactory position (figure 10).

**Conclusion:**

There are cases of spontaneous intracranial haemorrhage where the clinical events are so catastrophic that resuscitative measures with subsequent tolerable neurological deficit is beyond present medical skill. However, with adequate management, the modbidity and mortality of this devastating human ailment can be greatly diminished.

**Acknowledgment:**

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*EDITOR*