

# Focal Seizures: An Unusual Presentation of Giant Intracranial Aneurysms

## A Report of Four Cases with Comments on the Natural History and Treatment

Ian R. Whittle, M.B., B.S., John L. Allsop, F.R.A.C.P., and G. Michael Halmagyi, F.R.A.C.P.

Department of Neurology and Neurosurgery, Royal Prince Alfred Hospital, Camperdown, New South Wales, Australia

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Four patients with partial epileptic seizures for several years, but without permanent neurological deficits, were found to have giant aneurysms of the middle cerebral artery. None had a history of subarachnoid hemorrhage. Operative findings included compression of the medial temporal and subfrontal cortex, infarction of the superior temporal gyrus, and evidence of previous hemorrhage from the aneurysms. In two patients, clipping of the aneurysm with decompression of the adjacent temporal lobe cured the seizure disorder. In the other two patients, the aneurysms could only be wrapped, and the seizures continued. It is postulated that focal compression of temporal lobe structures and local hemodynamic phenomena caused by the giant aneurysm predispose to epilepsy in these patients.

**KEY WORDS:** Giant intracranial aneurysm; Complex partial seizure; Electroencephalogram; Subarachnoid hemorrhage; Computed tomography scanning; Cerebral angiogram

Approximately 5% of all intracranial aneurysms are more than 2.5 cm in diameter [8,13]. These giant intracranial aneurysms behave differently from smaller aneurysms and pose different problems in management [2,4,8,13]. In contrast to smaller cranial aneurysms, only 40% of giant aneurysms present with subarachnoid hemorrhage [13]; the other 60% may cause diagnostic difficulties when they present in one of the following ways [2,4,8,13]:

1. As a slowly enlarging intracranial mass causing progressive cranial nerve, brainstem, or cerebral hemispheric dysfunction.
2. As transient ischemic attacks or cerebral infarction.

*Address reprint requests to:* I. R. Whittle, Lecturer in Surgical Neurology, University of Edinburgh, Western General Hospital, Edinburgh, Scotland EH4 2XU.

3. As progressive dementia with or without features of diencephalic amnesia.
4. As recurrent partial epileptic seizures without permanent neurological deficit.

This article presents four patients with unruptured giant aneurysms of the middle cerebral artery who presented with focal seizures. The relationship between the seizure disorder and the giant aneurysms and implications for treatment are discussed.

### Case Reports

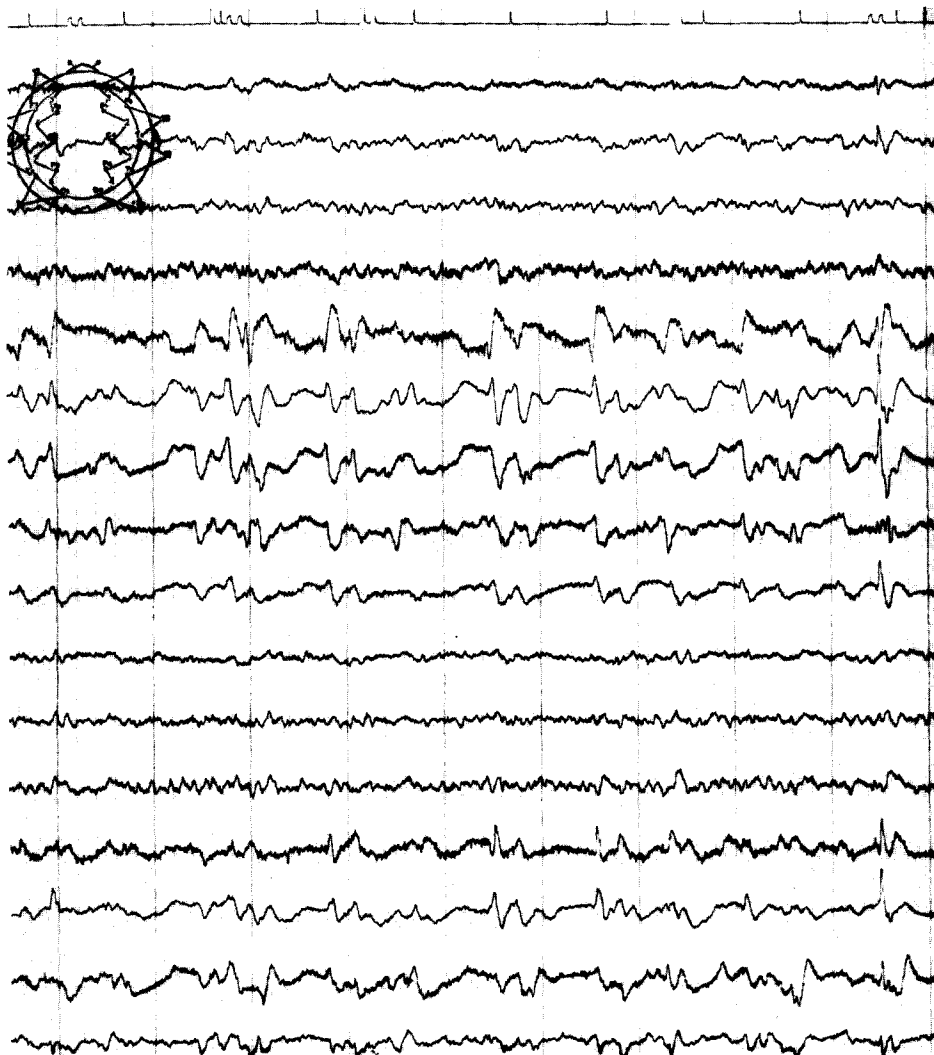
#### *Case 1*

A woman aged 50 years presented in July 1980, with a history of confusion and unusual behavior for 3 weeks. Her family said that over the previous 10-15 years, she had experienced many similar episodes but none had lasted for more than 1 day. She had steadfastly refused to seek medical advice on each occasion. There was no history of alcohol abuse. The episode leading to admission had caused concern because she had become particularly withdrawn and uncommunicative and had suffered from jerking movements of the right side of the body.

On examination, she was alert and behaving normally, but was unable to understand any written or spoken language and was unable to say anything apart from "yes" and "no." There were no involuntary movements and except for a suggestion of a right lower facial weakness, neurological examination was normal. A computed tomography scan showed only a small indefinite region of contrast enhancement in the medial part of the left temporal lobe, which was interpreted as a small area of cerebral infarction. An electroencephalogram showed periodic lateralized epileptiform discharges over the left inferior frontal and temporal regions (Figure 1).

It was decided that her aphasia was due to status epilepticus of partial type [6,7]. She was treated with phenytoin and carbamazepine.

Over the next 2 weeks, her language function grad-

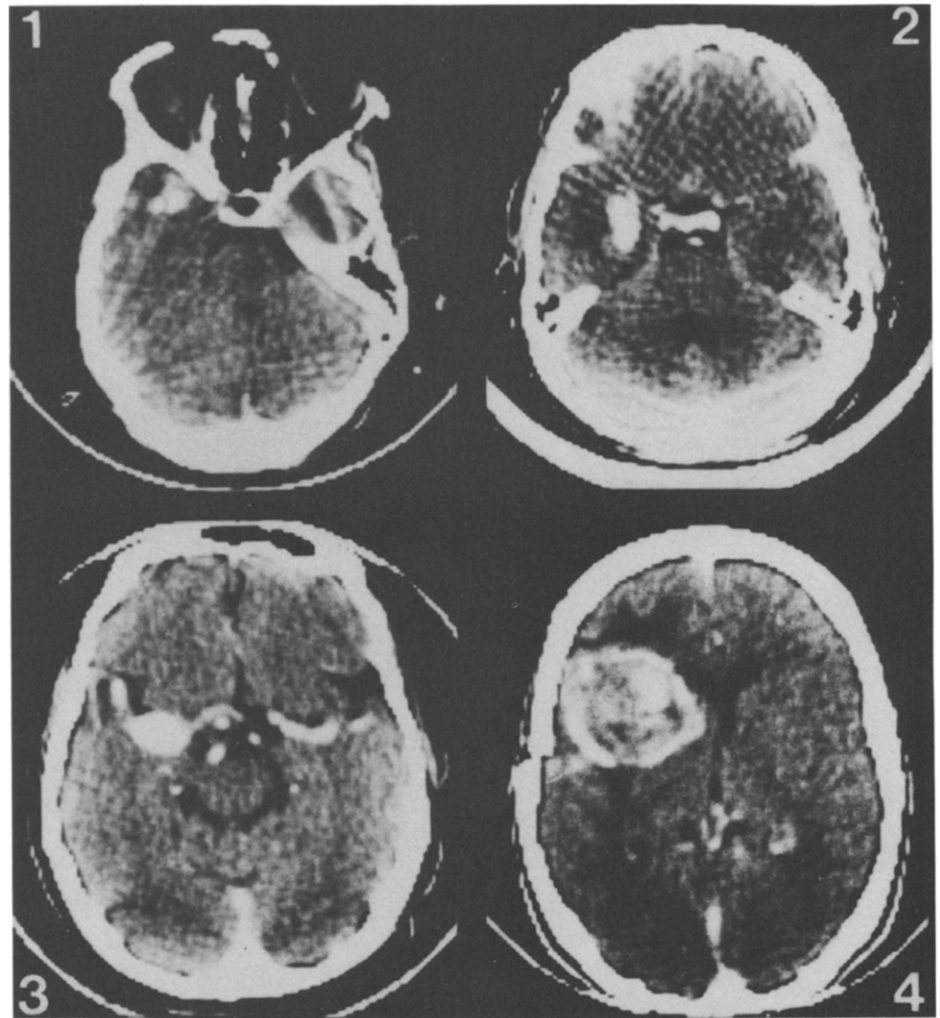


**Figure 1.** Electroencephalogram of case 1 during aphasic episode showing left temporal periodic lateralized epileptiform discharges. Standard electrode placements with the first four channels showing the right parasagittal run, the second four channels showing the left parasagittal run, the third four channels showing the right temporal run, and the last four channels showing the left temporal run.

ually returned to normal. A second electroencephalogram was normal except for some left temporal theta activity. Two months later, the patient again suddenly lost her power of speech. An electroencephalogram again showed left inferior frontal and temporal periodic lateralized epileptiform discharges. A computed tomography scan now revealed a much larger contrast-enhancing mass in the medial part of the left temporal lobe (Figure 2.1), and carotid angiography showed there were two aneurysms in the left middle cerebral artery in this region, one on the trunk of the middle cerebral artery and another larger aneurysm at the bifurcation of the middle cerebral artery near the insula (Figure 3.1).

At craniotomy in October 1980, a chronic subdural membrane was encountered over the left inferior frontal and temporal regions. There were multiple dural adhe-

sions and the brain was discolored. In particular, the anterior part of the superior temporal gyrus appeared to be infarcted. The appearance of the extracerebral blood and its particular location, around the aneurysmal sac, suggested that at least one previous subarachnoid hemorrhage had occurred. After dissection of the Sylvian fissure, the bifurcation aneurysm was clipped. The more proximal aneurysm, however, was significantly larger than suggested by the angiogram. The fundus, which measured at least 2.5 cm, extended medially to the anterior clinoid process, and only with difficulty could a large Drake clip be satisfactorily placed around the aneurysmal neck. A temporal pole artery arising from a region of the neck had to be sacrificed inasmuch as it could not be dissected free from the aneurysmal fundus. Aneurysmectomy and thrombectomy were performed after



**Figure 2.** Contrast-enhanced axial computed tomography scans of the four cases showing the giant aneurysms of the middle cerebral artery. (Note that the computed tomography scan of case 4 has been right-left reversed for purposes of illustration.)

clipping with considerable reduction in mass effect from the lesion. The immediate postoperative course was uneventful, and when last seen 2 years later, the patient was well and had no further episodes of aphasia.

*Comment.* This patient had two episodes of complex partial status epilepticus characterized by ictal aphasia with a left temporal epileptogenic focus. The periodic lateralized epileptiform discharge usually indicates a large structural lesion [3]. In this case, the seizures probably arose from the infarcted superior temporal gyrus adjacent to the giant aneurysm in the middle cerebral artery. Reduction of the aneurysmal mass after clipping was followed by cessation of the seizures.

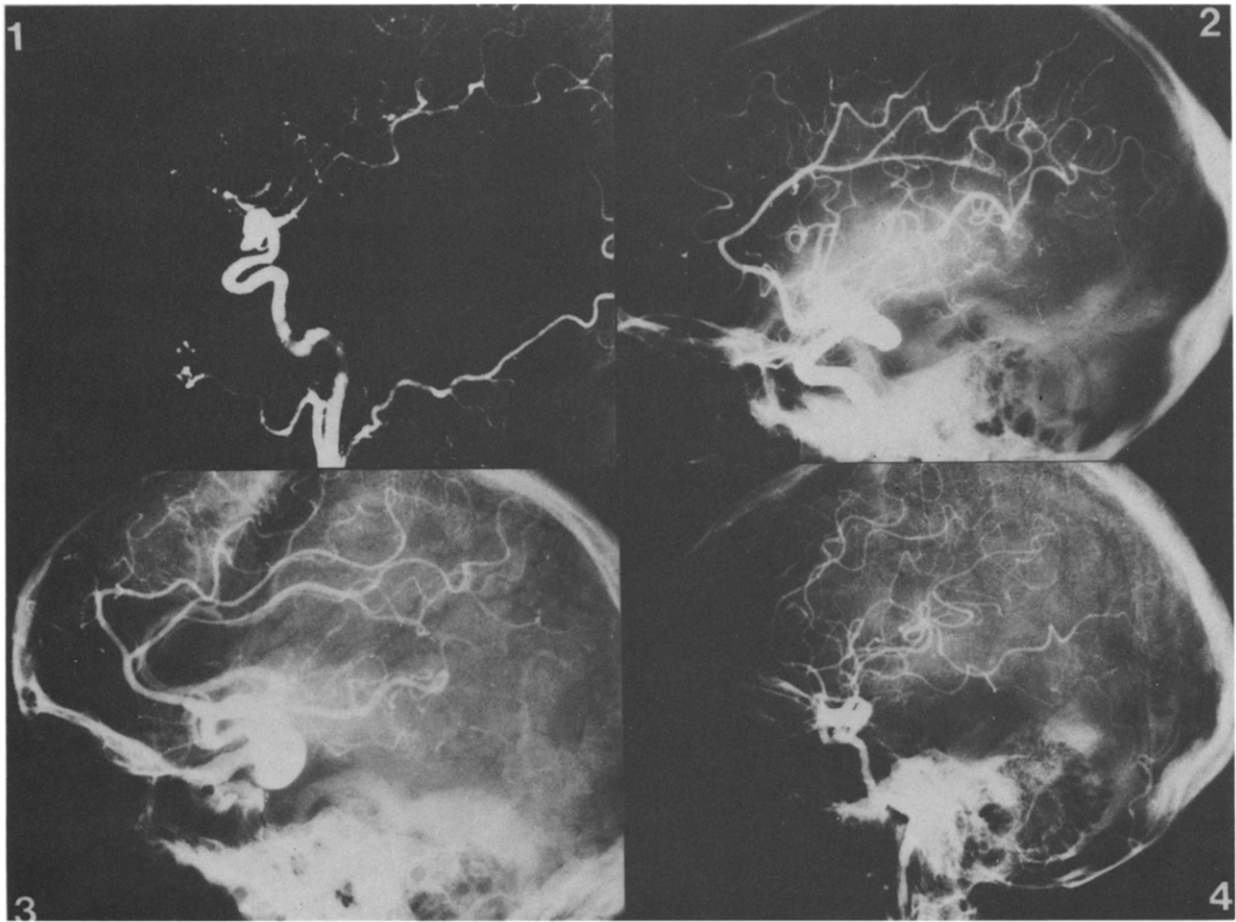
#### Case 2

A 52-year-old man presented in May 1976 after a generalized tonic/clonic seizure. Six years before, he had begun to experience episodes of a sudden overwhelming

pleasant smell, which he likened to roses, lasting about 1 minute. Six months later, the smells had become associated with a sensation of unusual fear. The patient would run to the nearest person so as not to be alone during the episode. The olfactory sensation stopped after 2 years, but the episodes of fear continued.

There were no neurological or systemic abnormalities on examination. Skull and chest x-ray studies were normal. A nuclear brain scan revealed slightly reduced flow in the left middle cerebral artery in the dynamic phase, but the static study was normal. An electroencephalogram showed symmetrical 8-9 Hz alpha waves, with some sharp and slow wave complexes in both temporal regions, more marked on the left than the right. A diagnosis of complex partial seizures was made and he was treated with phenytoin and methylphenobarbitone. The attacks of fear continued despite adequate serum levels of anticonvulsants.

A repeat electroencephalogram in August 1976 showed random sharp and theta waves over both temporal lobes.



A



B

**Figure 3.** (A) Anteroposterior and (B) lateral angiograms of the four cases showing the giant aneurysms of the middle cerebral artery. (Note that the angiogram of case 4 has been right-left reversed for the purposes of illustration.)

A computed tomography scan in October 1976 with contrast enhancement was normal. However, a further scan in May 1978 revealed a  $1.5 \times 2.5$ -cm area of contrast enhancement in the region of the left uncus (Figure 2.2). There was no contiguous brain edema or mass effect, and the lesion appeared to be related to the middle cerebral artery. Angiography of the carotid arteries revealed a giant aneurysm in the left middle cerebral artery (Figure 3.2). At craniotomy in June 1978, the anterior segment of the superior temporal gyrus was noted to be atrophic and discolored. After dissection of the Sylvian fissure, the giant aneurysm was noted to be compressing the medial temporal lobe. There was no evidence of previous subarachnoid hemorrhage. Three aneurysm clips were required to clip the neck of the giant aneurysm. Satisfactory occlusion of the fundus of the aneurysm from the middle cerebral artery was confirmed by collapse of the lesion after needle aspiration.

Postoperatively, there was a transient global dysphasia associated with micrographia and clumsiness of handwriting, but angiography showed normal filling of the middle cerebral artery and almost total obliteration of the aneurysm. Within 4 months, however, the motor disability had resolved and the patient had returned to work with only a mild residual dysphasia.

When last reviewed 4 years after operation, his speech was essentially normal, but his writing was still abnormal. He has continued to take phenytoin and methylphenobarbitone and has experienced no further seizures.

*Comment.* This patient had complex partial seizures with electroencephalogram abnormalities for 8 years before the discovery of the giant aneurysm of his middle cerebral artery. The aneurysm enlarged over the 2 years before the operation, without rupture; however, it did compress the medial temporal lobe and cause focal infarction of the superior temporal gyrus. Decompression of the medial temporal lobe by clipping of the aneurysm was followed by cessation of the fits.

### Case 3

A 59-year-old man was admitted to hospital in March 1981, after two attacks of loss of memory and confusion. He had been treated for at least 12 years for arterial hypertension and had a history of peptic ulcer. The first attack occurred 2 months before admission when he awoke one night and was, according to his wife, confused

and did not know where he was or who his wife was. The episode lasted several minutes and the following morning he could not remember anything about it. A week later while visiting his family, he suddenly looked perplexed, asked where he was and what he was doing, and who the people around him were. Again the confusion lasted several minutes. On specific questioning, he remembered that he had had many episodes over several years of "déjà vu" and over the previous 18 months had experienced at least six episodes of a sudden bad taste in his mouth lasting several minutes, but not associated with any other symptoms. His wife added that during the 6 months before admission, there had also been a definite general deterioration in his memory.

Neurological examination showed bilateral palmo-mental reflexes and a poor short-term memory. His arterial pressure was 170/100. A computed tomography scan showed an ovoid  $3 \times 5$  cm enhancing lesion related to the left middle cerebral artery (Figure 2.3), and this was confirmed on cerebral angiography to be a giant fusiform aneurysm in the middle cerebral artery (Figure 3.3). There was also diffuse cerebrovascular ectasia and a 5-mm aneurysm in the anterior communicating artery. An electroencephalogram showed occasional phase-reversed sharp waves and delta waves in the left temporal area. At craniotomy in March 1981, it was found that the aneurysm could not be obliterated but only wrapped. The aneurysm had split the Sylvian fissure and was compressing the adjacent insula and temporal operculum. His immediate postoperative course was uneventful, but 1 month later he was readmitted after multiple pulmonary emboli and was treated (uneventfully) with anticoagulants.

He was admitted in July 1981 with severe headaches. Neurological examination was unchanged and there was no sign of meningism. The cerebrospinal fluid was clear. Repeat cerebral angiography showed that the giant fusiform aneurysm had extensively thrombosed to form a giant serpentine aneurysm [12]. Four months later he was finally admitted after a sudden onset of global aphasia and dense right hemiplegia. His wife said that, during the previous 3 months, he had had several attacks of expressive dysphasia lasting up to 30 minutes. Furthermore, despite phenytoin and methylphenobarbitone, he continued to have intermittent complex partial seizures. Repeat cerebral angiography again showed a giant serpentine aneurysm; in addition, there was now poor perfusion of the entire territory of the left middle cerebral artery.

It was considered that he had suffered multiple episodes of embolization from the thrombosed giant aneurysm of the middle cerebral artery, resulting eventually in major infarction. The patient continued to deteriorate neurologically and died from pulmonary em-

bolism 1 month later. Postmortem examination confirmed the large recent left temporoparietal infarction and also demonstrated compression and focal infarction of the medial temporal lobe by the giant aneurysm (Figure 4).

*Comment.* This patient's aneurysm caused not only complex partial seizures but eventually transient ischemic attacks and a large infarction in the territory of the affected middle cerebral artery. The aneurysm, however, did not bleed even when the patient was heparinized to prevent pulmonary emboli. The aneurysm could not be obliterated and the seizures did not cease.

#### Case 4

A 48-year-old woman presented to the hospital in 1976 complaining of three attacks of jerking involving the left

**Figure 4.** Basal view of the brain at postmortem examination of case 3. The anterior pole of the temporal lobe has been amputated to expose the giant fusiform aneurysm of the middle cerebral artery (arrows). Gauze, which was placed on the aneurysm during operative exploration, can be seen partially covering the aneurysm. Compression of the mesial temporal lobe can clearly be seen.



arm in the previous 2 months. There had been no other neurological illnesses and there were no neurological abnormalities on examination. She had been noted to have arterial hypertension for 6 months. Investigations at that time included a normal electroencephalogram and a nuclear brain scan which showed a large area of increased isotope uptake in the right frontal area. An angiogram of the carotid arteries showed a 2-cm multilobed aneurysm of the right middle cerebral artery contiguous with a large avascular temporal lobe mass (Figure 3.4). At operation a 4 × 3 cm giant aneurysm of the middle cerebral artery arising from the bifurcation was present. There was also a smaller aneurysm arising from an insular branch of the middle cerebral artery. The large aneurysm could only be wrapped but the smaller one was clipped.

About 1 year after the operation, she began to complain of episodes of a putrid smell together with a bad taste. She compared the smell to peeled chokos. She also had occasional visions of a lady vampire. A computed tomography scan at the time showed a large area of increased density in the frontotemporal area, the rim of which enhanced with contrast (Figure 2.4). An electroencephalogram now showed theta, delta, and phase-reversed sharp waves in the right frontotemporal area. A repeat angiogram showed the large aneurysm to be unchanged and to be surrounded by a large avascular mass. She continues to have occasional olfactory hallucinations, despite adequate blood levels of several anticonvulsants at different times including phenytoin, methylphenobarbitone, carbamazepine, and sodium valproate.

*Comment.* This patient had focal motor seizures probably due to the larger aneurysm that could not be obliterated. Postoperatively she developed complex partial seizures which did not really respond to anticonvulsants. Six years after the onset of the seizures, the aneurysm, which has probably been there for much longer, remains unchanged and unruptured and the patient remains otherwise well.

#### Discussion

These observations consolidate previous reports of unruptured giant intracranial aneurysms causing focal seizures [2,4,8,10]. More specifically they link complex partial seizures to giant aneurysms of the middle cerebral artery. They also emphasize that patients presenting with recurrent complex partial seizures, without fixed focal neurological deficits and without a history of subarachnoid hemorrhage, may still have a giant aneurysm of the middle cerebral artery as a cause of the seizures. Because the appearances on computed tomographic scanning of



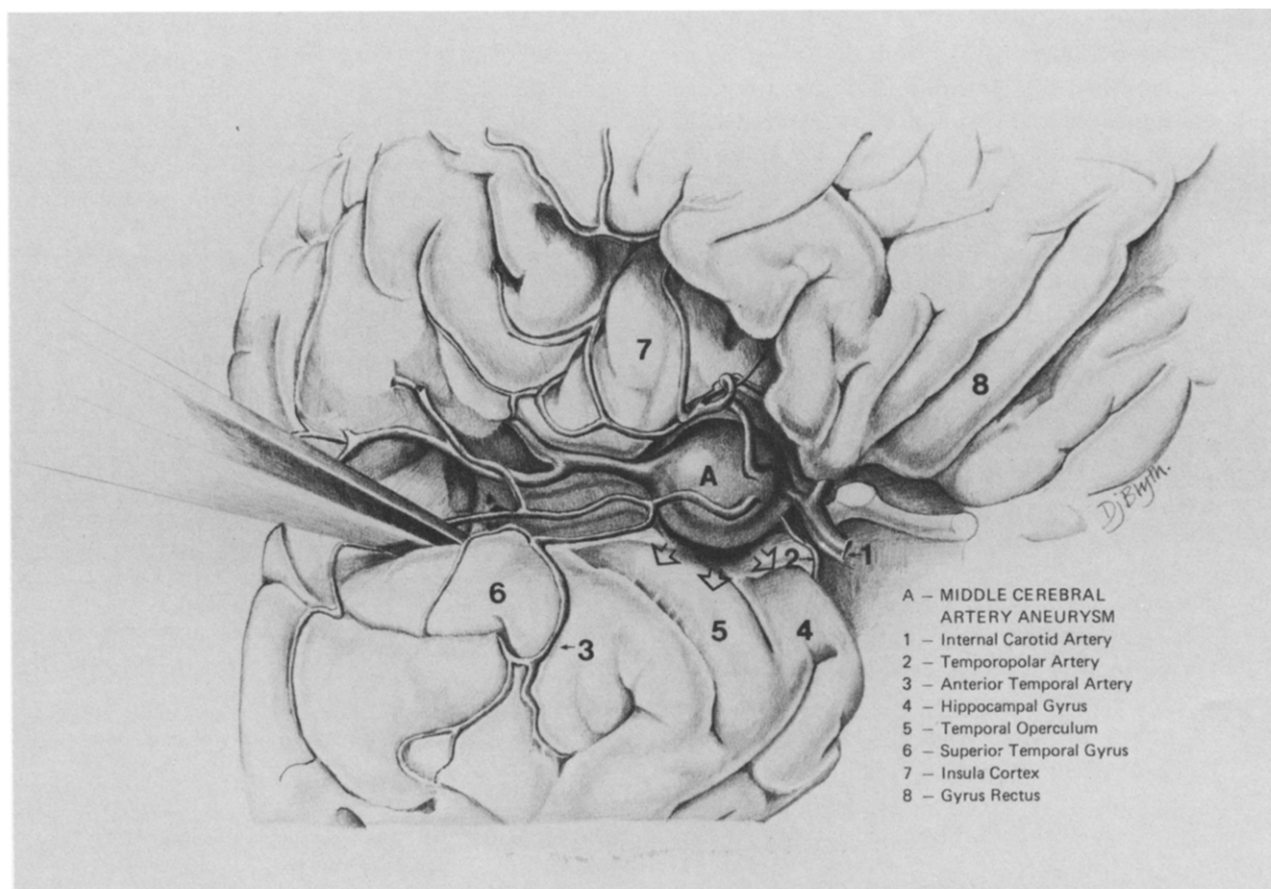
giant aneurysms may mimic an intracranial neoplasm owing to intra-aneurysmal thrombosis, angiography of the carotid arteries should be included in the preoperative investigation of these patients [12].

Our three surviving patients had seizures for 15 (case 1), 8 (case 2) and 7 (case 4) years without clinical subarachnoid hemorrhage. The fourth patient had complex partial seizures for at least 4 years before he died after complications of thrombosis of the aneurysmal middle cerebral artery. He also did not suffer a clinical subarachnoid hemorrhage. These four cases may be added to other cases in the literature in which recurrent seizures occurred for many years without clinical manifestations of subarachnoid hemorrhage from the giant aneurysm. Frankel and Alpers [5] describe one patient (their case 10) with a giant aneurysm of the middle cerebral artery, who had presented with complex partial seizures that had persisted for the 3 years prior to her death after aneurysmal surgery. Bull's series [2] of 22 giant aneurysms included three patients who presented with complex partial seizures (his cases 6, 7, and 11). None had aneurysms directly operated upon, and 4, 5, and 12 years after the seizures had begun, none had experienced subarachnoid hemorrhage. The series of

Morley and Barr [18] of 28 giant aneurysms included three patients who presented with seizures (their cases 15, 24, and 28). Two of these had seizure disorders for many years prior to successful surgery of the aneurysm. Seizure disorders of many years' duration are also described in the series of Drake [4] and Sengupta and colleagues [10]; however, in these series, three patients are described who suffered fatal subarachnoid hemorrhage from their giant aneurysm shortly after presentation with seizures. Therefore, although it would appear that giant aneurysms that present with focal seizure disorders form a discrete clinicopathological subgroup which has a low propensity to rupture, clinical management should not be undertaken on this spurious assumption.

This cautious approach would seem justified by Sahs and colleagues [9], who described seizure disorders in 95 of 2621 patients who later suffered a first subarachnoid hemorrhage. Although this cooperative series refers to aneurysms of all sizes, the operative findings

Figure 5. Diagrammatic representation of local vascular and cerebral anatomy surrounding an expanding aneurysm of the middle cerebral artery.



suggestive of previous aneurysmal hemorrhage in two of our patients, neither of whom had ever had clinical manifestations of subarachnoid hemorrhage, suggested that the same natural history may occur in some giant aneurysms. Obliteration of the aneurysm by clipping and aneurysmectomy or aneurysmotomy and thrombectomy would appear to be a requisite for postoperative control of the seizure disorder as well as removing the risk of a later subarachnoid hemorrhage. Most patients in whom giant aneurysms were successfully managed by direct surgery have been seizure-free, although they remain on anticonvulsants postoperatively [4,8,10]. In contrast, those patients who underwent indirect aneurysmal surgery, carotid artery ligation, wrapping or exploration of the aneurysm continued to have seizures, deteriorated neurologically, or suffered later fatal subarachnoid hemorrhage [2,4].

It is not obvious how giant aneurysms of the middle cerebral artery cause complex partial seizures, but both compression and infarction of temporal lobe structures seem important. At operation all four patients had obvious compression of the temporal lobe with distortion of the insula, uncus, temporal operculum, and subfrontal neocortex (Figure 5). The mesial temporal lobe structures have a low seizure threshold and are particularly epileptogenic if damaged. Cortical compression may potentiate other pathophysiological processes that increase the risk of epilepsy. Minor leaks from the adventitia of the giant aneurysmal fundus could cause focal cortical atrophy and gliosis. Focal cerebral ischemia related to the pressure effect of the aneurysm could lead to further focal neuronal atrophy, particularly in the basolateral nucleus of the amygdala and the Sommer sector (h1) and the endofolium (h3-5) of the hippocampus [1].

In two patients successful obliteration of the aneurysm and decompression of the temporal lobe cured the seizure disorder, suggesting that compression was at least aggravating a seizure tendency. Distortion of the temporal lobe and focal effects from the giant aneurysm would not appear to be the only factors in the genesis of the epilepsy in that up to 36% of aneurysms of all sizes in the middle cerebral artery may present with seizures [9,10]. The finding of focal superior temporal lobe infarction in three patients in this series suggests vascular phenomena related to the hemodynamics of the aneurysm contributed to the epileptogenic tendency. Transient or permanent focal disturbances in blood flow to the temporal lobe could be caused by aneurysm of the middle cerebral artery in several ways. The anterior

temporal or temporal-polar artery ostia could be mechanically occluded or hemodynamically compromised by the aneurysm or flow disturbances emanating from it (Figure 5). Intra-aneurysmal thrombus or vasoconstrictor substances such as thromboxane A<sub>2</sub> and prostaglandin A<sub>2</sub>, both of which may be formed in an aneurysm [11], may embolize distally along either of the vessels supplying the temporal lobe. These vascular phenomena may aggravate the seizure tendency caused by the mass effect on giant aneurysm.

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