compression of the adjacent brain by the enlarged aneurysm or ischaemic steal from the main middle cerebral artery owing to the large amount of blood pooling into the giant aneurysm.

It is proposed that such a large aneurysm developed from a small saccular aneurysm by continuous dilation due to blood pressure or turbulent blood flow. The large size of aneurysms of congenital origin seems to favour the formation of a laminated clot thus preventing rupture.⁸ Other investigators consider giant serpentine aneurysms originate from fusiform aneurysms which are most likely caused by a degenerative process of the arterial wall from infection or arteriosclerosis.⁹ This case suggests that it is probably of congenital origin and the aneurysm may have enlarged slowly.

The possibility of a giant aneurysm must be considered when a nonenhanced CT study shows a well circumscribed high density mass without oedema at the adjacent area of brain and contrast infusion reveals homogenous strong enhancement in the lumen. Sometimes it is difficult to confirm the diagnosis before angiographic study. Giant aneurysms can be divided into three types: partially thrombosed, completely thrombosed, and nonthrombosed.^{3,10} Our case belongs to the nonthrombosed group and showed a well circumscribed homogeneous mass without perifocal oedema and diffuse enhancement in the lumen with contrast infusion. For the total excision of a giant aneurysm in early infancy, it should be remembered that the patient may have an acute cardiopulmonary insufficiency. In a situation of poor general condition, it is reasonable to proceed with the surgery using the extracorporeal heart-lung machine and hypothermia. It is important to make efforts to minimise the damage of the adjacent functional area accompanying surgical procedures; this principle is especially important in the surgery for a giant aneurysm deep in dominant hemisphere. During resection of a giant aneurysm in the infant it is imperative to avoid damage of the adjacent areas surrounding it because the compressed immature brain has strong potential for restoration of function. The postoperative course of giant aneurysm in the infant is quite different from that in the adult or in the elderly group, because the underlying brain, which is compressed by a large aneurysmal sac, is in a relatively immature state. The marked improvement of the neurologic deficit following an excision of the giant aneurysm in this patient may also

be attributed to restoration of adequate blood flow and prevention of steal phenomena and the resultant return of intracranial contents to their normal position.

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Occlusion of the anterior choroidal artery

Sir: Occlusion of the anterior choroidal artery is seldom reported. Its clinical syndrome is controversial and some authors have even questioned its existence.^{1,2} First described in 1891 by Kolisko (cited by Masson *et al*) and later by Foix, the anatomy of the anterior choroidal artery

and the consequences of its ligation have been generating considerable dispute.⁴ Four cases of the syndrome with CT scan documentation were recently reported.⁵ We report a fifth case of anterior choroidal artery occlusion, with confirmatory CT scan findings.

A 64-year-old right-handed male was admitted because of left hemiparesis. He was known to have hypertension and diabetes mellitus, and to smoke. The evening prior to admission he developed paresthesiae of the left hand. Over the next 12-24 hours he developed "pins and needles" over the remainder of his left arm, left leg, and the left side of his face. His left hand was weak and "clumsy." He fell due to weakness of his left leg. He denied a history of transient ischaemic attacks. He had no nausea, vomiting, headache, or visual loss. On examination, his mental status was normal. Visual fields were intact. There was a left central facial paresis and dysarthria. He was moderately weak on the left side in a pyramidal distribution. Tone was normal. There was a left pronator drift. Sensation was diminished to all modalities over the entire left side of his body. Romberg sign was not present. Rapid, rhythmic alternating movements were slower on the left. Reflexes were symmetric and the plantar response was equivocal.

The next day he was much weaker on the left side and manifested anosognosia. There was a new left homonymous hemianopia. A CT scan was obtained, and intravenous heparin therapy was initiated. The patient's condition stabilised and heparin therapy was discontinued after several days. Three months after discharge he still had a "pins and needles" sensation over the entire left side of his body as well as diminished vibratory sensation. He had a slight left central facial paresis and a left homonymous hemianopia. He had left-sided weakness in a pyramidal distribution with a pronator drift and slight unsteadiness of gait requiring a cane.

Comparison of the patient's CT scan with those recently published by Mason *et al*,⁴ and with the template published by Damasio,⁵ reveals that his lesion corresponded with the area supplied by the anterior choroidal artery. This vessel most often arises from the internal carotid artery as its last branch, although it is sometimes a branch of the posterior communicating or middle cerebral artery. The small vessel crosses the optic tract, then runs laterally to the inferior horn of the lateral ventricle to supply the choroid plexus. Its anastomoses include the posterior choroidal artery. The

Fig CT scan showing hypodense area in distribution of right anterior choroidal artery.

territory supplied by the anterior choroidal artery and its branches usually includes the posterior 2/3 of the posterior limb of the internal capsule, portions of the medial and intermediate segments of the globus pallidus, the lateral 1/2 of the lateral geniculate body and the proximal geniculocalcarine tract, the middle 1/3 of the basis pedunculus, the uncus, the amygdala, the anterior hippocampus, and the dentate fascia. In a given specimen, the area of supply may be slightly greater or slightly less.⁶⁻⁸ Total occlusion of this vessel has been reported to cause contralateral hemiplegia and hemianesthesia involving all sensory modalities but to varying degrees, hemianopia, and ipsilateral pupillary dilatation with sluggish pupillary light reaction.⁷ Occurrence on the non-dominant side may cause a syndrome of the minor hemisphere with anosognosia, visual neglect, constructional apraxia, and motor inpersistence. Occurrence on the dominant side has been reported to cause a mild aphasia with paraphasias and decreased fluency.⁹ The extent and location of lesions produced by occlusions of the anterior choroidal artery are variable owing to its anastomoses with the posterior choroidal artery, interpeduncular plexus, and posterior communicating artery. It is not an end artery.¹⁰ Cooper, in his report of ligation of this vessel for Parkinsonism in eight cases found no instance of contralateral hemiplegia nor hemianesthesia.¹¹ However, his

cases clearly had pre-existing disease in the region supplied by the anterior choroidal artery, and ligation of blood vessels is not physiologically equivalent to thrombosis.⁷ We believe this is a case of occlusion of the anterior choroidal artery because of CT confirmation of the predicted anatomic lesion.

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Absence of herpes simplex virus antigen in brain in encephalitis lethargica

Sir: Encephalitis lethargica was an acute encephalitic disease which occurred in Europe and America in epidemic proportions in the 2nd and 3rd decades of this century. A causal organism was not identified but the neuropathology of the condition as well as the epidemiological features were suggestive of a viral cause. Numerous attempts to isolate an infectious agent were undertaken and some claims were made that a filterable agent had been isolated.¹ One such claim was made by Levaditi *et al*² who obtained an agent which was passed to rabbits and was found to have the properties of herpes simplex virus. As herpes simplex virus antigens can be identified in formalin-fixed, paraffin-embedded material we decided to examine stored material from a case of encephalitis lethargica with an antibody to herpes simplex virus using the immunoperoxidase technique.

The patient was a boy aged 17 years who had died in The London Hospital in 1920 after an illness of one week's duration consisting of headache, drowsiness and fever progressing to coma. At post-mortem examination (LH PM 15/20) there was swelling of the brain with widespread petechial haemorrhages. Stored paraffin-embedded blocks from the cerebrum and brain stem were recut; the sections showed intense inflammation and congestion in the midbrain with destruction of many neurons in the substantia nigra and periaqueductal grey matter. Unstained sections from this block were treated with an antibody to herpes simplex virus raised in rabbits (Dako) as previously described.³ No evidence of herpes simplex virus antigen was found.

We thought it worth placing this negative finding on record. As herpes simplex encephalitis is one of the commonest identifiable causes of sporadic encephalitis⁴ it would not be surprising if some cases of this disorder occurred and were investigated during the encephalitis outbreak. Although the distribution of damage in herpes simplex encephalitis differs from that of encephalitis lethargica the pattern of damage now recognised as typical for herpes simplex encephalitis had not been defined at that time and so it may well have been confused clinically and pathologically with encephalitis lethargica. As herpes simplex virus is only one of several viruses which retain some antigenicity after routine formalin fixation and paraffin