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 thrombati of a laminated clot, thus preventing rupture. 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. 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 homogeneous 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 aneurysms with strong enhancement.
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.

THOMAS N WARD
JAMES L BERNAT\(^*\)
ABE S GOLDSSTEIN

Section of Neurology
Dartmouth-Hitchcock Medical Center,
Hanover, NH

Section of Neurology
VA Hospital,
White River Junction, Vermont, \(^*\) USA

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

References


Accepted 24 March 1984

**Absence of herpes simplex virus antigen in brain in encephalitis lethargica**

Sir: Encephalitis lethargica was an acute encephalitis 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 trace an infectious agent were undertaken and some claims were made that afilterable agent had been isolated.\(^1\) One such claim was made by Levaditi et al 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).\(^2\) 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 encephalitis\(^3\) 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