Tuberculomas of the brain formed about 20 per cent of the intracranial tumors dealt with in Madras. They form a good percentage of intracranial lesions dealt with in countries of Asia, Eastern Europe, Italy, Spain and South America. Since the advent of streptomycin, surgery has been of great benefit to these patients. Hence clinical and radiological diagnosis of tuberculomas of the brain are of practical importance to neurosurgeons.

It is not necessary to perform operation in every case of tuberculoma. Surgery is indicated only when there are signs of space-occupying lesions. If there is no evidence of pressure on the brain medical treatment is fully effective.

A preoperative diagnosis of tuberculoma of the brain is a presumptive diagnosis. When a patient presents focal neurological signs and evidence of increased intracranial pressure, investigation to exclude a tuberculoma is a routine procedure. This is essential in countries in which tuberculomas represent a good percentage of intracranial tumors. The patient is investigated for other evidence of tuberculous infection, such as adenitis (cervical or mediastinal), pleural effusion, active pulmonary lesion, involvement of bones or joints, renal tuberculosis, endometritis, etc. In more than 34 per cent of cases (59 out of 175) there was concomitant evidence of tuberculous infection. In another 36 per cent (64 out of 175) there was a previous history of tuberculosis in the patient or a history of contact with the infection.

The clinical features of a tuberculoma resemble those of other intracranial tumors. As tuberculomas are slow-growing the clinical picture is that of a slowly progressive lesion.

Clinical Examination. It has been our experience that patients who have a tuberculoma of the brain are of a better build, and look better nourished than those who have tubercular meningitis. The fact that a patient obviously is healthy does not exclude the presence of a tuberculoma of the brain. It may be that the increased resistance of the patient prevented formation of widespread tubercular meningitis.

In our experience, patients with tuberculomas of the brain fall broadly into two groups. In the larger first group, the patients have signs of increased intracranial pressure with or without localizing neurological signs. This group is investigated as in any other case of intracranial space-occupying lesion.

In the smaller second group, the patients exhibit progressive neurological disability without increased intracranial tension. There is definite evidence of tuberculous infection elsewhere in the body or a definite history of contact with tuberculosis, and the Mantoux test is positive. When treated with antituberculous drugs, the neurological signs recede. The patients do not show signs of increased intracranial tension for a long time after the onset of neurological signs. They seek treatment early because of the focal
neurological deficit, and with medical treatment they show good improvement. Patient B.B.J. (NS 3720) gave a history of focal fits followed by progressive weakness of the left upper limb. There was no evidence of increased intracranial pressure but she had enlarged cervical glands which on biopsy proved to be tubercular. The patient was put on antituberculous treatment and phenobarbitone. She showed remarkable improvement and the enlargement of the cervical glands disappeared. The fits were controlled and the power in the limb improved though some residual weakness was present. Another patient (NS 6120) was admitted with progressive unilateral cerebellar symptoms without increased intracranial tension. He had an active tubercular lesion in the lung. With antibiotic therapy, the pulmonary lesion and the cerebellar symptoms cleared.

From the marked improvement resulting in the focal neurological condition, it is presumed that these patients had an infiltrating tuberculous process in the brain, which receded with antibiotics. Two of the patients in whom the intracranial pressure increased were operated upon and were found to have an infiltrating type of tuberculoma of the cortex and subcortex. Such lesions are different from those of the first group. The radiological features are discussed below. In such cases surgery is not indicated; it will lead only to a permanent neurological deficit, with no benefit to the patient.

The radiological investigation of tuberculomas of the brain has proved very interesting. Roentgenograms of the chest are taken routinely. In 14 per cent of the cases there was evidence of old or recent involvement of the lung, and in 11 per cent mediastinal adenitis was revealed. Roentgenograms of the skull also are taken routinely. Contradicting the popular idea that tuberculomas of the brain often show calcification, in only 11 out of 175 cases, i.e. 6 per cent, was there evidence of intracranial calcification in tuberculomas. Pineal shift frequently cannot be assessed by us as the pineal body does not calcify as often as reported in the literature of the West.5 Plain roentgenograms often show signs of increased intracranial tension as evidenced by marked digital impressions, separation of sutures, erosion of the posterior clinoid process or a deepening of the pituitary fossa (40 out of 200 cases, or 20 per cent).

When the lesion is diagnosed as supratentorial in location, carotid angiography is done. Tuberculomas of the brain usually are revealed as avascular lesions in the angiogram (Figs. 1 and 2). Depending on its size and location, the tuberculoma causes changes in the vascular patterns. When such an avascular lesion is seen, it is presumed to be either a tuberculoma or, rarely, a chronic abscess. Initially it was presumed that all tuberculomas were avascular. But experience has shown that some of them can show a mildly increased vascularity resembling a low-grade vascular meningioma (Figs. 3, 4 and 5).

The tuberculomas that show vascularity...