Figure (a) Mass showing several cystic areas, "soap bubbles", (b) numerous capsulated organisms, PAS.

lesions and the antigen titre has not risen.

Although infection has been described in a range of animals, pigeons are the main source of human infection by cryptococcus. The organism is inhaled in infected dust usually in attics where pigeons roost. Following entry into the lungs, infection may spread to any site, but especially to the meninges. Even if the primary lung lesion resolves, spread to other organs can still occur.¹

Granulomas of the CNS make up 2–3% of all lesions first diagnosed as tumours and may be due to tuberculosis, syphilis, sarcoidosis, fungal or other unusual infections. Cryptococcal granulomas causing space-occupying masses are rare, but may occur in both previously healthy patients, or in those with already diagnosed pulmonary or meningeal cryptococcosis.

Immunocompromised patients, especially those with Hodgkin's disease, other lymphoid malignancies, or AIDS are at risk. Fujita *et al*³ noted a lower incidence of such underlying conditions in patients with intracerebral cryptococcomas (5%), compared with those having cryptococcal meningitis (50%).

We believe that development of a mass lesion may reflect a relatively "competent" host reaction to the organism, with localisation of infection, rather than dissemination in the meninges. Our patient had no evidence of immunosuppression, and had a well marked

inflammatory reaction around the mass. Cryptococcal masses have been described in the cerebral hemispheres, ventricles, brainstem, cerebellum and spinal cord.^{2–5} In Fujita's comprehensive review of the literature,³ 55 intracerebral cryptococcal mass lesions were found, which were multiple in 19 patients. These cases included small (1 cm) lesions found at necropsy, not causing space-occupying symptoms. Only 18 of the 55 patients had intracerebral masses without meningitis. Seventeen of the 55 had lung involvement. Six had no abnormality on neurological examination, and CSF was negative in some cases.

There is no established treatment for this unusual condition.^{2,4,5} In our patient, the response to steroid therapy was dramatic, almost certainly due to suppression of inflammation and oedema around the lesion. Although the treatment of CNS cryptococcosis does not usually include corticosteroids, in our patient the effect was beneficial. The response to steroids, lack of fever, chest signs or meningitis, meant that the diagnosis was not considered before surgery. In our patient, removal of the mass was probably curative. Six other patients have had successful surgical resection without adjuvant chemotherapy.¹ None had meningitis.

Detailed follow up on these patients is not given. In other patients, surgery and therapy

with Amphotericin B and 5 fluoro-cytosine were combined, mostly in cases with meningitis or predisposing underlying diseases. We plan to review our patient regularly, and if there is clinical or antigenic evidence of disease activity chemotherapy will be given.

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A simple method for localisation and removal of small subcortical brain tumours

In recent years the widespread utilisation of computerised tomography (CT) has made it possible to reach an early diagnosis of cerebral lesions which are often too small to be found at conventional open surgery without damaging brain tissue.

We present a method to localise and remove small subcortical brain tumours, even those seated in critical areas.

This is a very simple method that needs only a CT scanner and a circular piece of hollow rubber (2 cm in diameter, 7 mm thick). The advantage of rubber is that it is dense and radiopaque without creating artifacts on the CT scan.

Before surgery the patient has a CT scan with the rubber piece attached to the shaven scalp overlying the probable site of the tumour. The rubber piece is moved and scanning is repeated until the centre of the scalp marker exactly overlies the middle of the tumour. After this the rubber is removed and a small mark with a dermatographic pencil is left in its place, providing the landmark of the tumour on the scalp.

At operation a smaller than usual skin and bone flap is made so that the scalp mark is in the centre of the flap. The flap is drawn on the scalp after accurately measuring the distance from the tumour marker.

It is very important to preserve the relation between the scalp mark and the tumour. Neither the scalp nor the brain must be displaced during the initial stage of operation. For this reason it is necessary to take the following precautions: a) Raney scalp clips should be used for haemostasis of the wound, because the weight of the haemostatic forceps might cause the scalp to slide on the skull; b) the periosteum of the flap edges should not be taken off the skull for the same reason; c) the patient's head should be positioned so that an imaginary line passing through the scalp

Figure CT scan of a 59 year old man with metastatic adenocarcinoma in the right parietal lobe. Left: preoperative CT scan after contrast infusion showed an intracerebral lesion with oedema and mass effect. Note that the hollow midportion of the rubber piece is placed on the scalp to correspond with the middle of the tumour. Right: 45 days after surgery a contrast enhanced CT scan did not reveal any residual tumour.

mark and the brain tumour is perpendicular to the floor of the operating room; d) osmotic drugs that produce excessive cerebral dehydration should not be administered during the operation; c and d are carried out to avoid the brain moving with gravity.

After the dura has been opened, the removal of the tumour is carried out with the help of an operating microscope. If the tumour is not visible at the cortical surface, it is necessary to confirm its position by accurately measuring the distance between the wound edges and the site of the corticotomy; this must be equal to the distance between the wound edges and the scalp mark. Furthermore, a careful evaluation of the relationship between the tumour and the near cortical sulci, shown both on high resolution CT scan and on the operating field, allows confirmation of the site for corticotomy. In some cases this evaluation permits removal of the tumour through a microsurgical transsulcal approach (fig).

In the past few years the possibility of integrating the precision of stereotactic methods with new sophisticated instruments, has resulted in the successful treatment of small cerebral lesions.

Unfortunately, these facilities are not available to most neurosurgeons, and small subcortical brain tumours can be easily removed by free-hand microsurgery even when located within or near critical cerebral areas. To do this it is necessary to know the exact location of the tumour. This is not a difficult problem when the lesion is in a cerebral area easily recognisable both on the CT slices and at operation (that is, the poles of cerebral lobes). When the tumour is located in other sites, especially in or near critical areas, the identification and removal of the tumour without risk of damaging healthy brain tissue can be very difficult. In these cases it is essential to localise the lesion as accurately as possible both on the scalp and on the cortical surface.

Many methods have been proposed to relate the data provided by the CT scan to the patient's scalp using bony landmarks^{1,2} or reference markers attached to the scalp.^{3,4} Other techniques have been described to locate the tumour, as seen on the CT slices, to skull radiographs.^{4,5} Nevertheless, all these methods are prone to error.

Our method has the following advantages:

a) it is simple, safe, inexpensive and reliable in localising small subcortical brain tumours; b) the exact localisation of the tumour permits the smallest possible craniotomy and corticotomy to be performed. Apart from the necessity of exact tumour localisation on the scalp there is the need for careful surgical technique.

Whilst our method is useful for removal of subcortical tumours, stereotactic procedure is the treatment of choice for deep-seated tumours.⁶

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Mononeuropathy associated with hyperthyroidism

Thyrotoxic polyneuropathy has been reported by some authors,^{1,2} but mononeuropathy associated with hyperthyroidism has received little attention. We report two patients with thyrotoxicosis who presented with mononeuropathies.

Case 1 was a 37 year old male cook who had a three month history of hyperthyroidism and noticed distal weakness of his legs with gait disturbance and hypoaesthesia and dysaesthesia of his feet for two months. Before administration of thiamazole, hypoaesthesia and dysaesthesia developed on the lateral aspect of his thighs. He had minimal diffuse weakness of the proximal muscles of the arms and legs including the shoulder and hip girdle. However, weakness of the tibialis muscles were out of proportion to the muscle weakness elsewhere, causing complete foot drop of the right and impairment of dorsiflexion of the left. The deep tendon reflexes were brisk. Hypoaesthesia and dysaesthesia of his feet and thighs were localised to the sensory distributions of the peroneal and lateral cutaneous nerves of the thigh. Taps on the inguinal ligament just medial to the anterior superior iliac spine (ASIS) and on the fibular head resulted in an electric tingling sensation in each of the mentioned nerve distributions. The combination of peroneal nerve palsy and meralgia paraesthetica suggested mononeuritis multiplex. Tinel's sign of median nerve was positive bilaterally without sensory disturbance on his fingers. Diagnosis of typical Graves' disease was made on the basis of laboratory findings (TSH, T₃, T₄, and anti-TSH receptor-antibody) and thyroid scintigram.

Electromyographic studies of sampled muscles showed a proximal myopathy and denervation potentials in the anterior tibial muscles. Nerve conduction studies revealed asymmetry of peroneal nerve motor conduction velocities and low amplitude evoked responses (right: 40.5 m/s, 1.0 mV; left: 53.0 m/s, 0.9 mV). The other nerves were normal. Muscle biopsy of the rectus femoris showed mild myopathy. The clinical course was marked by a resolution of neurological symptoms and electrophysiological findings that paralleled the remission of Graves' disease after oral treatment with 30 mg thiamazole daily. The asymmetry of right to left peroneal nerve conduction velocity diminished and the amplitude of evoked responses normalised.

Case 2 was a 36 year old male industrial worker who was admitted because of a 12 month history of hyperthyroidism, weight loss and fine finger tremor. He had difficulty in carrying objects because of weakness in his arms. Two weeks before admission, he noticed dysaesthesia on the lateral aspect of the left thigh with decreased superficial sensation. He showed mild weakness of proximal muscles. The deep tendon reflexes were brisk. All modalities of sensation were preserved except for the lateral aspect of the left thigh. A tap on the left inguinal ligament just medial to the ASIS resulted in an electric tingling sensation in the lateral aspect of the thigh. Tinel's sign of median nerve was present bilaterally. Laboratory findings revealed hyperthyroidism with low TSH level and positive anti-TSH receptor-antibody. Thyroid scintigram by ^{99m}Tc showed increased uptake and enlargement of the thyroid gland. Electromyographic studies of sampled muscles showed mild myopathy of proximal muscles. Muscle biopsy of the rectus femoris showed mild myopathy with changes that suggested neurogenic atrophy. Sensory nerve conduction studies revealed asymmetry of the lateral cutaneous nerve of the thigh, right: 63.1 m/s 7.5 μ V, left: 45.6 m/s 0.8 μ V. The other nerves had normal conduction velocities. Treatment with