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Computed Tomography in the Planning and Evaluation of Therapeutic Stereotaxic Surgical Procedures of the Brain

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Preoperative and serial postoperative computed tomographic (CT) scans were obtained in 38 patients having either stereotaxic thalamotomy, mesencephalotomy, or a cingulumotomy to determine the relevance of the preoperative examination and to evaluate the site and evolution of the surgical lesion. Of the preoperative examinations, 55% were abnormal. The consequences of the stereotaxic procedure were identified in the first 2 postoperative weeks in 26 of the 28 patients having thalamotomy, six of the nine patients having mesencephalotomy, and all three patients having cingulumotomy as a diffuse low-density area (mean attenuation value, 18 Hounsfield units). A central core of increased attenuation due to an intralesional hemorrhage was present on the early postoperative scan in 17% of patients. The late residual lesion studied in seven patients was considerably smaller than the early abnormality. The appearances and temporal sequence of the postsurgical CT changes suggest the early abnormality is due to edema and the late lesion to coagulative necrosis. The findings suggest CT has a role in the preoperative assessment of patients having stereotaxic surgery in defining the target and predicting the stereotaxic coordinates, while postoperatively it documents the site of the lesion and can determine the basis of a complication.

There are several generally recognized indications for therapeutic stereotaxic operations in the brain, including extrapyramidal disorders, spasticity, convulsive disorders, and intractable pain. There is also a limited indication for stereotaxic surgery in the management of patients with emotional and behavioral disorders [1–6].

The lesion produced by stereotaxic cryosurgical or temperature-controlled high-frequency coagulation has been studied pathologically. The early lesion has a necrotic focus, some hemorrhage, and associated phagocytic reactive astrocytosis. The late lesion is associated with an area of localized encephalomalacia resembling an infarct that diminishes as its scar is formed. The anatomic location of the lesion plays a role in its size and extent. The different electrical resistance between white and gray matter is probably responsible for the larger lesion observed in the white matter with the same lesion parameters [7–10] (fig. 1).

Until recently, the consequences of the stereotaxic lesion could be assessed only by their clinical manifestations. However, computed tomography (CT) can demonstrate the consequences of stereotaxy directly. It was used in this study of 38 patients to determine the value of the preoperative scar and to evaluate the site, evolution, and complications of the stereotaxic lesion.

Subjects and Methods

Preoperative and postoperative CT scans were obtained in 38 patients having therapeutic stereotaxic procedures. Twenty-six patients had a total of 28 thalamotomies, nine patients had mesencephalotomy, and three patients had bilateral cingulumotomy. The stereotaxic lesions were generated with thermister radiofrequency probes with exposed tips (5.6 × 1.5 mm for thalamotomy, 4.0 × 1.08 mm for mesencephalotomy, and 5.0 × 2.4 mm for cingulumotomy). Standard lesion parameters were used: thalamotomy (30 sec at 70°C–60 sec at 75°C), mesencephalotomy (30 sec at 75°C), cingulumotomy (30–60 sec at 70°–80° × 4). Conray or metrizamide ventriculography aided stereotaxic localization.

Four patients were scanned on the operative day but most postoperative CT scans were obtained in the first 2 weeks. Five patients were restudied at 1 year, one patient at 3 years, and one patient was studied 11 years after surgery. The preoperative and postoperative CT scans were analyzed. Any abnormality was noted on the preoperative study and the location, size, and attenuation value of the stereotaxic lesion site was determined on the postoperative study. The CT findings and clinical results were then correlated.

Results

The preoperative scan was abnormal in 14 of 26 patients having thalamotomy. Ten of these had diffuse atrophy and four had focal low densities consistent with infarction or posttraumatic encephalomalacia. Four of the nine patients having mesencephalotomy had CT findings consistent with an old middle cerebral artery infarction. The preoperative examination of the cingulumotomy patients was normal.

The four patients studied on the operative day had focal areas of low density in the operative site; these had a maximum diameter of 10–20 mm and a mean attenuation value of 18 Hounsfield units (H). Twenty-six of the 28 thalamotomy patients had oval areas of low density in the thalamus when scanned within 2 weeks of surgery (mean, 18 H; 10–25 mm maximum diameter) (fig. 2). In 10 patients the low density extended into the internal capsule; several of these patients developed a transient neural deficit and a transient worsening of a preexisting dysarthria (fig. 3). In nine patients a small central area of lower density (mean, 10 H) could be differentiated from the diffuse area of low density after 10 days. Four patients had a central core of increased density (mean, 35 H) associated with the diffuse area of low density (fig. 4). In one of these patients a transient left central facial weakness and hemiparesis developed.

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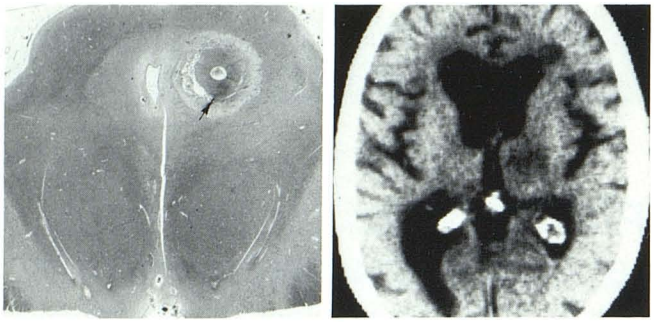

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Fig. 1.—Transverse section of mesencephalon 6 months after mesencephalotomy. Discrete area of necrosis (*arrow*) at lateral edge of central gray matter.

Fig. 2.—5 days after thalamotomy. Low-density region in ventrolateral thalamus (mean attenuation value, 18 H) encroaches on internal capsule. Associated cerebral atrophy.

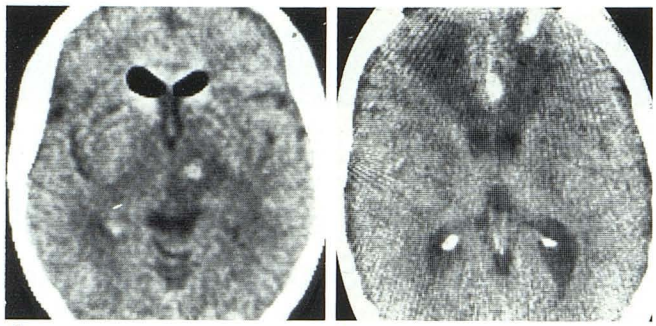

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Fig. 4.—Small intralesional hemorrhage associated with postthalamotomy low density 2 days after surgery.

Fig. 5.—Cingulumotomy. Mixed high-/low-density lesion 2 days after cingulumotomy. Extension of low density into white matter of frontal pole.

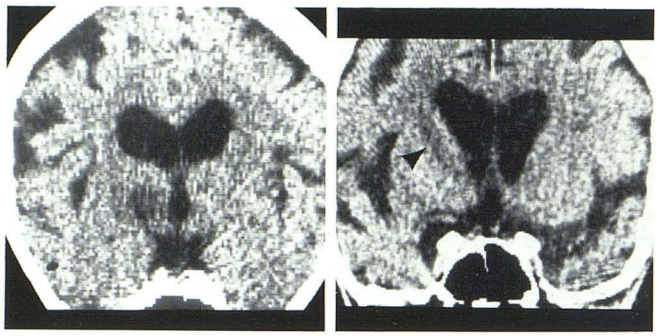

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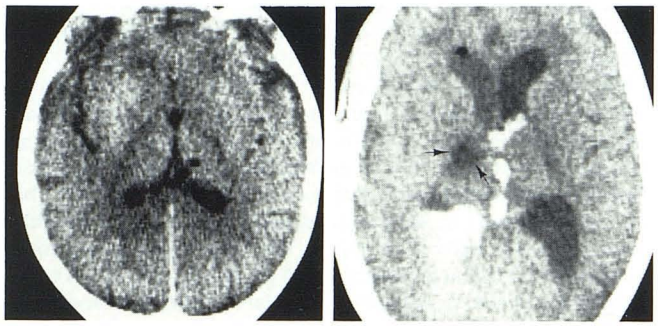
Fig. 3.—6 days after thalamotomy. Low density (mean attenuation value, 18 H) in thalamus, extending into internal capsule (*arrowhead*).



6 7

Fig. 6.—Late lesion. Low-density lesion (mean attenuation value, 8 H) 11 years after thalamotomy.

Fig. 7.—24 hr after stereotaxic thalamotomy. Residual metrizamide from localizing ventriculogram has penetrated cerebral-extracellular space but has been excluded from site of surgical lesions (*arrows*). This is probably due to rapid bulk flow of plasma filtrate from edematous site about surgical focus inhibiting normal migration of metrizamide.

In five of the patients having mesencephalotomy a low-density area (mean, 18 H) of 4–15 mm diameter was visualized on the scan in the first 2 postoperative weeks. All of the patients having mesencephalotomy had a satisfactory clinical result despite the fact that four patients had lesions not detected by CT.

After cingulumotomy there were large areas of reduced density extending from the white matter of the cingulum into the forceps minor and the adjacent white matter of the frontal pole; associated areas of high density were seen in the cingulum bilaterally (fig. 5).

In seven patients the late consequence of thalamotomy was evaluated by CT after one or more years; it appeared smaller (2.5 mm maximum diameter), was of cerebrospinal fluid density, and was more sharply demarcated from adjacent tissue than in the first 2 weeks (fig. 6). The lesion was reevaluated 9 months after cingulumotomy in one patient; it was recognized by a slight reduction in the attenuation of the white matter of the cingula.

Discussion

The preoperative CT scan was abnormal in 14 of 26 patients having thalamotomy and in four of nine undergoing mesencephalotomy; it is clearly important for the accurate preoperative planning of a stereotaxic procedure.

The early lesion demonstrated by CT appears to be largely due to edema. The observed low density in the early postoperative period often extended into adjacent white matter, but this extension had resolved by 2 weeks. These are both characteristic features of edema as visualized by CT [11, 12]. Further support for the view that the early lesion is due to edema comes from four patients studied on the operative or postoperative day. These patients had had intraventricular metrizamide instilled during the stereotaxic localization procedure. Their CT scans showed the water-soluble contrast agent distributed through the cerebral-extracellular space but excluded from the site of the lesion. This probably reflected a rapid bulk flow of plasma filtrate from the surgical site and inhibition of the normal migration of metrizamide. It is consistent with the view that the early lesion is largely due to edema [13] (fig. 7). It is of clinical interest that the extension of edema into adjacent white matter, which was noted in 10 patients after thalamotomy, was associated in only one patient with an immediately obvious clinical consequence.

After 10 days a smaller central area of low density (mean attenuation value, 12 H) was observed in the thalamotomy patients and could be differentiated from the more diffuse area of reduced density. This smaller focus appears to represent the focus of coagulative necrosis and is the basis of the residual lesion visible at 1, 3, and 11 years. Nonvisualization or disappearance of a lesion

was noted in seven patients, however, in six of these the clinical result of the procedure had been satisfactory. Nonvisualization of a lesion may be due to its being too small to be visualized or having an attenuation value similar to that of adjacent brain. Passerini et al. [14] were unable to visualize three of the lesions in 12 patients studied and like us found that nonvisualization was not associated with the quality of the result.

Complications, when they occurred, were usually but not necessarily associated with the presence of an area of increased attenuation within the low-density area. This presumably reflected an area of hemorrhage and edema at the lesion site.

In this study we have shown that CT can demonstrate the consequences of a stereotaxic lesion. The findings in the first 2 weeks are largely due to edema around the lesion site and involve a greater volume of tissue than the later resultant of the lesion. Nonvisualization of a lesion is not necessarily associated with an unsatisfactory clinical result. Focal areas of hemorrhage can sometimes be identified—they may explain a clinically manifest complication—but they were also noted in our series in patients without clinical complications.

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