Short report

Clinico-anatomical correlations in uncomplicated stroke

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SUMMARY A method for making clinico-anatomical correlations by computer superimposition of brain maps derived from CT scan images, described in a companion paper, was tested in 19 patients with an uncomplicated left hemiparesis due to acute stroke. Eight patients with an isolated left hemiparesis had small lesions in the most rostral part of the right internal capsule. The composite lesion maps of 11 patients whose left hemiparesis was complicated only by spinothalamic sensory loss showed a larger area of damage more caudally in the posterior part of the posterior limb of the capsule, abutting the thalamus. The close correlation between the results obtained by this method and those obtained in previous clinico-pathological studies of patients with uncomplicated strokes, confirms the validity of this mapping technique.

The vascular lesions causing pure motor and sensorimotor deficit have been demonstrated in previous CT scan and necropsy studies.^{1–4} Patients with such uncomplicated stroke deficits usually have small deep lesions in the internal capsule with involvement of the adjacent thalamus if sensory loss is persistent. We therefore chose a group of patients with these deficits to validate a new method of computer superimposition of brain lesions (see companion paper).

Patients and methods

Definitions:

Pure motor hemiparesis: Power loss affecting the limbs on one side occurring in a patient who displayed no other neurological deficit.

Simple mixed hemideficit: Power loss affecting the limbs on one side accompanied by impairment of pinprick or light touch sensation ipsilaterally but with no higher cerebral dysfunction, visual loss or other type of sensory deficit.

Patients and CT scan timing: In an unselected group with acute stroke under the age of 76 years,⁵ there were nine patients with left sided pure motor hemiparesis and 12 with left sided simple mixed hemideficit. CT scans were per-

formed at a time ranging from between 1 to 21 days, mean 8 days. One patient from each group had no focal defect on the scan.

Computer superimposition of lesions: This was performed using the method described in a companion article. For each syndrome a composite map for each slice showing the degree of lesion overlap in terms of a percentage of the total number of patients with that syndrome was made.

Results

Of the eight patients with pure motor hemiparesis who had abnormal scans six showed lesions in the rostral internal capsule (fig 1) on slice 5 of the standardised brain map (Companion paper, fig 1). The lesion seen in one patient extended from slice 5 into

Fig 1 Left pure motor hemideficit. Computer drawn outlines of lesions found in slices 4–6 of the standard brain map.

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Fig 2 Left pure motor hemiparesis. Computer composite showing the overlap of lesions seen in fig 1.

slice 4 and in another patient a second lesion was seen in the head of the caudate nucleus in slice 6.

In the two remaining patients in this group the only lesions visible were respectively in slice 3 and slice 7. In the computer superimposition composite (fig 2), the lesions were seen to be maximally overlapping in the posterior half of the most rostral part of the internal capsule, near its origin from the corona radiata.

Of the 11 patients with simple mixed hemiparesis

Fig 3 Left simple mixed hemiparesis. Computer drawn outlines of lesions found in slices 4-6 standard brain map (see text).

Fig 4 Left simple mixed hemiparesis. Computer composites showing the overlap of lesions seen in fig 3. (a) Slice 5, (b) Slice 6.

who had abnormal scans all but one showed lesions restricted to slices 5 and 6 of the standardised brain map. The exception was a patient whose lesion was seen in slices 4 and 5. Six patients had lesions on slice 6 only, three on slices 5 and 6 and one on slice 5 only (fig 3). The lesions were on average larger than those of the patients with a left sided pure motor hemiparesis, and the superimposition composite (fig 4) showed a larger area of overlap occupying two slices. In slice 5 (fig 4a) this overlap is in a similar but more medial position than that of 3 pure motor hemiparesis patients. In slice 6 (fig 4b) the overlap was in the position of the posterior part of the posterior limb of the internal capsule abutting the posterolateral aspect of the thalamus.

Discussion

A number of problems arise when making clinico-anatomical correlations from CT scans performed in the acute phase of patients' strokes. The volume of abnormal tissue radiodensity seen on a scan at this time is likely to be larger than the lesion seen at post-mortem after pathological stabilisation of the lesion. An unknown proportion of the low attenuation lesions seen on the scan is due to tissue oedema and may have the potential to recover.⁶ These factors probably account for the larger size of the lesions found in our group of patients with uncomplicated strokes compared with those found at necropsy long after the clinical ictus by Fisher and others.¹⁻⁴ Also not discernible on the CT scan image and having an unknown functional significance are the blood flow and metabolic changes that have been observed to occur at some distance from the area of the brain directly involved in the vascular insult.⁷

A further difficulty in comparing different patients scans is the variability of head proportions and the actual plane of the CT section. It was for this reason that we transcribed the outline of the lesions onto standard maps rather than attempt direct consumption of CT scan images. Clearly in these circumstances it was important that the patients' clinical deficit was not known to the radiologist.

Patients with simple neurological deficits were chosen to demonstrate our method since the anatomical basis for such deficits has been extensively researched and they tend to have small deep lesions. The motor fibres of the internal capsule are traditionally shown spread throughout the posterior limb.⁸ However modern studies⁹⁻¹² have shown that they are densely localised in the posterior end of the posterior limb of the internal capsule. Mohr *et al* and others have described the pathological lesions in patients with sensorimotor deficits corresponding to

our "Simple Mixed Hemideficit".^{3,4} These lesions were in very similar positions to the area of overlap found in our patients with this deficit (fig 3).

Thus in these simple deficits our method of mapping the functional anatomy of the brain has produced results which concur with previous anatomical, neurophysiological and pathological studies. It seems legitimate therefore to advocate the use of this method to explore clinico-anatomical correlations in other deficits whose anatomical basis is less well established, especially neuropsychological syndromes.

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Note on the preparation of the Figures 2 & 4

For the purposes of reproduction the dotted areas of the original computer output have been overlaid with appropriate densities of shading. Prints of the original computer output are available from the authors.

References

- 1 Fisher CM, Curry HB. Pure motor hemiplegia of vascular origin. *Arch Neurol* 1965;36:65-73.
- 2 Groothuis DR, Duncan GW, Fisher CM. The human thalamocortical sensory path in the internal capsule: evidence from a small capsular hemorrhage causing a pure sensory stroke. *Ann Neurol* 1977;2:328-31.
- 3 Mohr JP, Krase CS, Meckler RJ, Fisher CM. Sensorimotor stroke due to thalamo-capsular ischaemia. *Arch Neurol* 1977;34:739-41.
- 4 Donnan GA, Tress BM, Bladin PF. A prospective study of lacunar infarction using computerized tomography. *Neurology (Minneapolis)* 1982;32:49-56.
- 5 Allen CMC. Clinical diagnosis of the acute stroke syndrome. *Q J Med* 1983;52:515-23.
- 6 Alcalá H, Gado M, Torack RM. The effect of size, histologic elements and water content on the visualization of cerebral infarcts: a computer tomographic study. *Arch Neurol* 1978;35:1-7.
- 7 Kuhl DE, Phelps ME, Kowell AP, Metter EJ, Selin C, Winter J. Effects of stroke on local cerebral metabolism and perfusion: mapping by emission computed tomography of ¹⁸FDG and ¹⁵NH₃. *Ann Neurol* 1980;8:47-60.
- 8 Williams PL, Warwick R. *Gray's Anatomy* 36th ed. Edinburgh: Churchill Livingstone, 1980:1039.
- 9 Marie P, Bouttier H, Bertrand I. Etude anatomique d'un cas de sclérose latérale amyotrophique à predomi-

- nance hemiplegique. *Bull Soc Med Hop Paris* 1923;**47**:481-5.
- ¹⁰ Hirayama K, Tsubaki T, Toyokura Y, Okinaka S. The representation of the pyramidal tract in the internal capsule and basis pedunculi a study based on 3 cases of amyotrophic lateral sclerosis. *Neurology (Minneap)* 1962;**12**:337-42.
- ¹¹ Ross E. Localisation of the pyramidal tract in the internal capsule by whole brain dissection. *Neurology (Minneap)* 1980;**30**:59-64.
- ¹² Hanaway J, Young RR. Localisation of the pyramidal tract in the internal capsule in man. *J Neurol Sci* 1977;**34**:63-70.