Journal of Neurology, Neurosurgery, and Psychiatry, 1979, **42**, 485–492

Difficulties in diagnosis of supratentorial gliomas by CAT scan

B. E. KENDALL, J. JAKUBOWSKI, P. PULLICINO, AND L. SYMON

From the Lysholm Radiological Department and Gough Cooper Department of Neurological Surgery, The National Hospital for Nervous Diseases, London

SUMMARY The false positive and false negative computed tomography diagnoses of glioma made using an EMI 1010 machine on a consecutive series of patients seen over a period of two years are recorded. About 1.5% of gliomas were not detected on initial CAT scan, 6.5% were misdiagnosed as benign lesions, and in 6.5% of the cases identified as glioma a non-malignant condition was subsequently diagnosed.

Computed tomography (CAT) generally reveals some abnormality in patients with intracranial tumours at the time of clinical presentation and at an earlier stage of the disease than any other radiological procedure (Wende *et al.*, 1977). This has modified the spectrum of neurosurgical investigation and has virtually replaced isotope encephalography and invasive procedures as a screening test. In previous studies (Claveria *et al.*, 1977, 1978) the advantages and some of the limitations of CAT in the diagnosis of malignant tumours were evaluated. The experience gained from these studies has been applied to routine examination of scans carried out over the past two years. Diminished use of other diagnostic methods for confirmation of the nature of lesions shown by CAT has occurred even though reliance on CAT may contribute to misdiagnosis in a few cases.

We considered that an analysis of the accuracy of CAT in the diagnosis of the supratentorial gliomas examined during this period would be of interest, together with a critical appraisal of diagnostic errors.

Method

The clinical and pathological records of all patients in the National Hospital for Nervous Diseases with a diagnosis of supratentorial glioma during the period of 1976 and 1977 were reviewed. All CAT scans in which a diagnosis of supratentorial glioma had been suggested during this period, were also reviewed and the data were correlated, giving four groups:

1. CAT diagnosis correct.
2. Glioma present but no abnormality detected by CAT.
3. Glioma present but incorrectly diagnosed as a different pathology by CAT.
4. Another pathology incorrectly diagnosed as glioma by CAT.

Results

During the period of study there was a total of 314 patients with proven gliomas. All of them had CAT examination, and 274 (87.3%) were correctly diagnosed on the initial scan. The remaining 40 cases (12.7%) were either considered to be normal (five cases), misdiagnosed as another pathology (26 cases), or diagnosed only as a mass lesion (nine cases). The wrong diagnoses included meningioma (eight cases), metastasis (six cases), inflammation (four cases), cerebral infarction (three cases), intracranial haemorrhage (three cases), and vascular malformation (two cases).

Detailed histological examination revealed that high grade astrocytomas (grade 3–4) were present in 67.5% (27 patients) of the 40 misdiagnosed cases. The remaining 32.5% (13 patients) suffered from low grade astrocytomas (six), oligodendroglioma (five), or microglioma (two) (Table 1).

Address for reprint requests: Dr B. Kendall, Lysholm Radiological Department, The National Hospital, Queen Square, London WC1N 3BG.

Accepted 27 December 1978

Table 1 *Histology of gliomas not detected or misdiagnosed on CAT*

Misdiagnosed	*(Total)*	*Low grade 1–2*	*High grade 3–4*	*Oligodendroglioma*	*Microglioma*
Unspecified mass	(9)	2	5	2	—
Meningiomas	(8)	—	6	2	—
Metastases	(6)	—	6	—	—
Normal scans	(5)	1	4	—	—
Inflammations	(4)	—	3	1	—
Infarct	(3)	1	1	—	1
Intracerebral haemorrhage	(3)	—	2	—	1
Vascular malformation	(2)	2	—	—	—
Total	(40)	6	27	5	2

Angiography was performed in 29 cases, and in the majority (25) it showed an obvious abnormality. However, the correct diagnosis was established by showing glioma vessels in only nine cases, and in five cases the angiographic diagnosis was misleading (Tables 2, 3).

A follow-up scan revealed the glioma in four out of five cases with normal scans (Fig. 1) and EEG (two cases), and pneumoencephalography (one case) suggested the diagnosis occasionally. In 60% (24) of the cases the correct diagnosis was not established until histological examination.

During the period under review glioma was diagnosed on 311 CAT scans. Out of this number 68.4% (213) were confirmed by histology and 19.6% (61) cases by other methods, mainly by angiography. A further 2.3% (seven) cases still remained unconfirmed. Thirty cases (9.7%) were proved to have a different pathology. The majority (26) of these (86%) were benign lesions: only four cases had metastases to the brain (Table 4).

Angiography was performed on 21 (70%) patients; 17 studies (80%) showed an obvious abnormality, but in only five out of seven meningiomas, and two out of four arteriovenous malformations did angiography correct the diagnosis. Five angiograms in cases of radionecrosis (two), metastasis (one), tuberculoma (one), or meningioma (one) were mistakenly considered to show glioma circulation (Table 4).

The initial CAT misdiagnosis was corrected by other studies in 16 (53%) cases; in the remaining 14 patients final pathology was established by histological examination (Table 5).

Table 2 *Angiographic studies in gliomas not detected or misdiagnosed on CAT*

CAT diagnosis	*Number of cases*	*Number of angiograms performed*	*Diagnosis corrected by angiography*
Unspecified mass	9	6	2
Meningiomas	8	6	1
Metastases	6	4	2
Normal scans	5	2	0
Inflammations	4	3	1
Infarcts	3	3	1
Intracerebral haemorrhages	3	3	1
Vascular malformations	2	2	1
Total	40	29	9

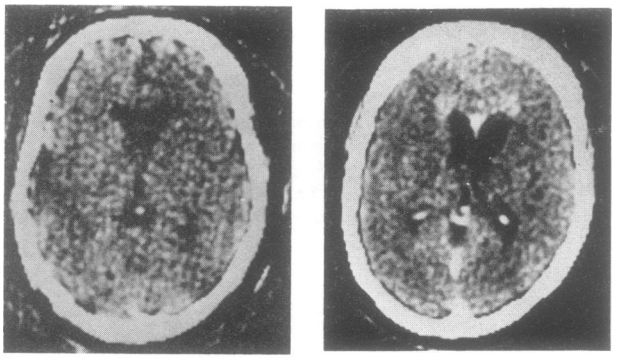
Discussion

Most glial tumours are shown on CAT as masses with attenuation values mixed within and below the normal range. There is usually surrounding white matter oedema. Enhancement is usually irregular and tends to be less than that occurring in meningiomas and metastases. Unenhancing regions of low attenuation suggesting cystic or necrotic changes are present in over half of malignant gliomas.

Homogeneous enhancement occurs in 15–20% of gliomas. When the margins are well defined if may cause appearances on CAT indistinguishable from some solitary metastases or, if situated per-

Table 3 *Angiography in gliomas misdiagnosed on CAT*

Angiographic findings	*(Total)*	*Histology*			
		High grade astrocytoma	*Low grade astrocytoma*	*Oligodendroglioma*	*Microglioma*
Normal	(4)	2	1	0	1
Avascular mass	(11)	7	1	3	0
Glioma vessels	(9)	8	1	0	0
Other pathology suggested	(5)	3	2	0	0
Total	(29)	20	5	3	1



(*a*) (*b*)

Fig. 1 *Glioma corpus callosum. (a) Normal scan. (b) Six months later. Minor dilatation of lateral ventricles. Thickening of septum pellucidum and abnormal enhancement in genu of corpus callosum and adjacent white matter.*

Table 4 *Angiography in cases misdiagnosed as glioma on CAT*

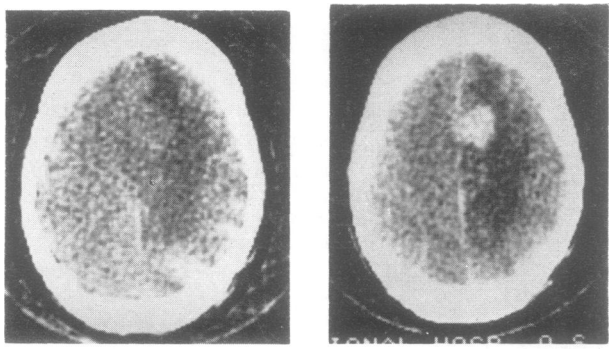
Final diagnosis	*(Total)*	*Angiograms performed*	*Results*			
			Normal	*Correct diagnosis*	*Avascular mass*	*"Glioma" circulation*
Meningioma	(7)	7		5	1	1
Metastasis	(4)	2			1	1
Inflammation	(3)	1				1
AVM	(4)	4	2	2		
Multiple sclerosis	(1)	1	1			
Radionecrosis	(2)	2				2
Arachnoid cyst	(1)	1			1	
Contusion	(1)	1			1	
Infarction	(7)	2	1		1	
Total	(30)	21	4	7	5	5

Table 5 *Cases misdiagnosed as glioma on CAT*

Final diagnosis	*(Total)*	*Diagnostic study*					
		Angiography	*Control CAT*	*Isotope scan*	*EEG*	*Chest radiograph*	*Histology*
Meningioma	(7)	5		1			1
Metastasis	(4)					1	3
Inflammation	(3)				1	1	1
Infarction	(7)		2	1			4
AVM	(4)	2					2
Multiple sclerosis	(1)		1				
Radionecrosis	(2)						2
Arachnoid cyst	(1)						1
Contusion	(1)		1				
Total	(30)	7	4	2	1	2	14

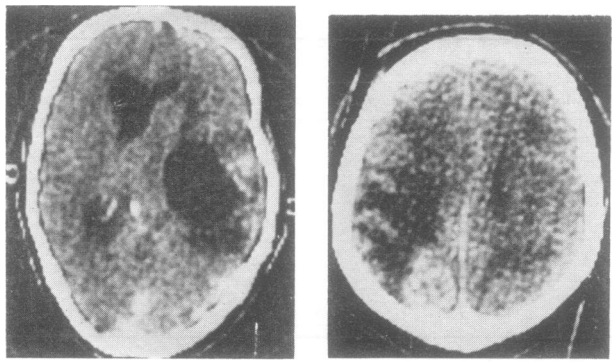
ipherally, from meningiomas (Fig. 2). Both these tumour types most frequently cause well-circumscribed masses of increased attenuation. Metastases commonly cause more extensive oedema than gliomas relative to their size, and meningiomas less oedema. However, there is considerable overlap of all these features with each type of tumour (Fig. 3), and about 10% of meningiomas cause extensive intracerebral oedema (Fig. 4). Realisation of this fact led to nine gliomas being diagnosed merely as intracranial tumour on CAT with the implication that further study was necessary to determine the type. The meningiomas diagnosed as glioma were either intraventricular (one), irregular cavitating masses (Fig. 5), or relatively small and associated with extensive oedema so that the tumour itself was either not identified (two) or else its extracerebral attachment was not appreciated (two) (Fig. 4).

About 20% of recent infarcts have obvious mass effect (Constant *et al.*, 1977). They are usually diffuse low attenuation lesions involving grey and white matter and without the defined perifocal white matter oedema typically found with tu-



(a) (b)

Fig. 2 *Astrocytoma grade 3–4. (a) Plain scan. (b) After intravenous contrast medium. An isodense mass adjacent to the falx enhances homogeneously. There is considerable oedema in the white matter. Diagnosed as falx meningioma.*



(a) (b)

Fig. 3 *(a) Metastasis from carcinoma of bronchus. Enhanced scan. There is a low attenuation mass in the right posterior temporal and low parietal regions which contains a well demarcated cyst medially. There is increased enhancement in its lateral part. There is considerable mass effect but only a small amount of white matter oedema. Diagnosed as glioma. (b) Glioma grade 3–4. After intravenous contrast medium. There is a well-defined enhanced mass in the left occipital region with quite extensive oedema in the white matter anterior to it. Diagnosed as metastasis.*

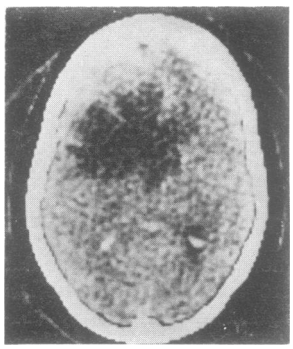
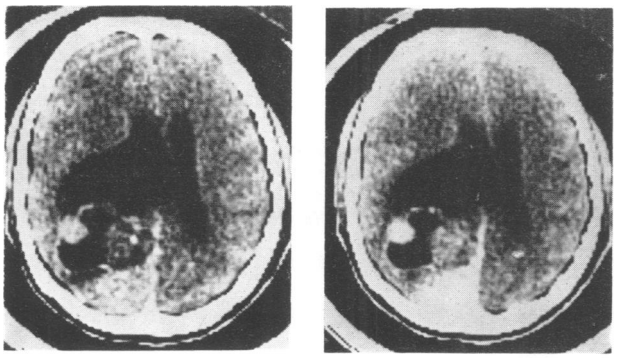


Fig. 4 *Left frontal convexity meningioma. Plain scan. There is extensive low attenuation in the left frontal white matter extending across the corpus callosum into the right frontal lobe. The meningioma itself was not identified before or after intravenous contrast medium. Diagnosed as glioma.*

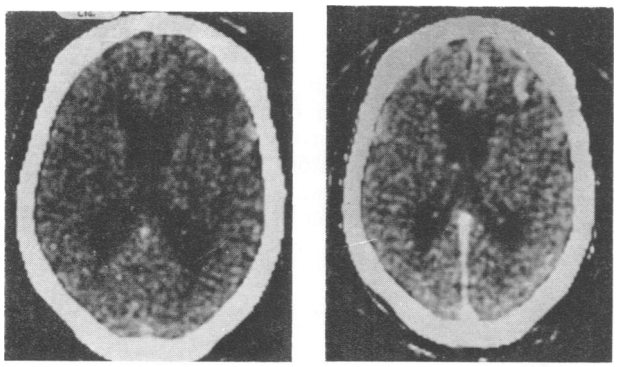
mours. Enhancement may occur in any part of the lesion which has not been completely deprived of its blood supply and when this is irregular the appearances may simulate a glioma (Fig. 6). In contrast, if mass effect and oedema accompanying a glioma are slight, infarction may be simulated.

Recent intracerebral haematoma is evident on CAT as a well-demarcated, high attenuation mass. There is usually only a narrow well-defined surrounding rim of low attenuation. Enhancement, which may occur about one–16 weeks after the bleed, is confined to the margin of the lesion. Earlier and more extensive enhancement should suggest an underlying tumour or angiomatous malformation, and could possibly have led to the correct diagnosis in one of our cases. The other two were indistinguishable from spontaneous haemorrhages. Absorbing haematomas which are isodense or of lower attenuation than brain usually have little or no mass effect, but ring enhancement may suggest an intracerebral tumour. The history is usually of a sudden episode with improvement and should lead to consideration of the diagnosis.



(*a*) (*b*)

Fig. 5 *Meningioma. (a) Plain scan. (b) After contrast medium. Mixed high and low attenuation mass with partial enhancement. Diagnosed as cystic glioma.*



(*a*) (*b*)

Fig. 6 *Infarct. (a) Plain scan. (b) After contrast medium. Low attenuation mass right frontal lobe with patchy enhancement. Diagnosed as glioma.*

The enlarged vessels of an angiomatous malformation (AVM) show typically as round, oval, or serpiginous aggregations of increased attenuation, depending on their orientation to the CAT section, often with intervening low attenuation from gliosis and microcyst formation. There is frequently local atrophy, and calcification may be present in the walls of blood vessels or damaged brain. Enhancement is usual within the malformation or its connecting vessels though it may be absent in some cases in which the lesion is thrombosed (Fig. 7). Mass effect is present in less than 20% of angiomas without a history of recent haemorrhage (Kendall and Claveria, 1976), and it is not usually marked, though it was moderate in two of our cases misdiagnosed as glioma. The AVM did not outline at angiography in two cases, including one in which enhancement was considerable and extensive on CAT; both were totally thrombosed, the enhancement being caused by alteration of the blood-brain barrier (Kramer and Wing, 1977). Both of the gliomas diagnosed as AVMs on CAT had a history of recent intracranial haemorrhage. They were markedly enhancing (Fig. 8) and partially calcified with relatively less mass effect than expected from the size of the lesions. In one of them angiographic evidence of large tumour vessels with arteriovenous shunting corrected the diagnosis.

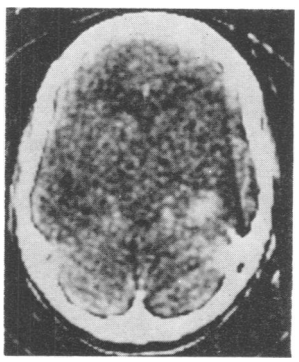
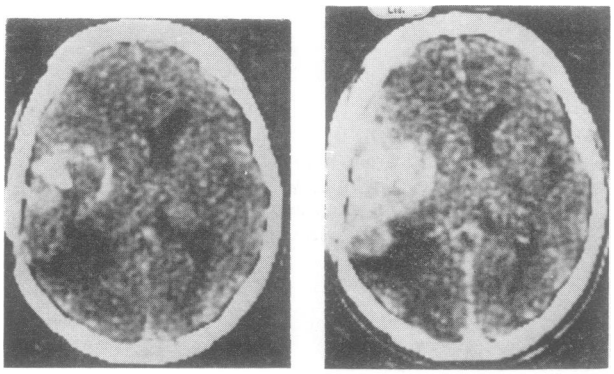


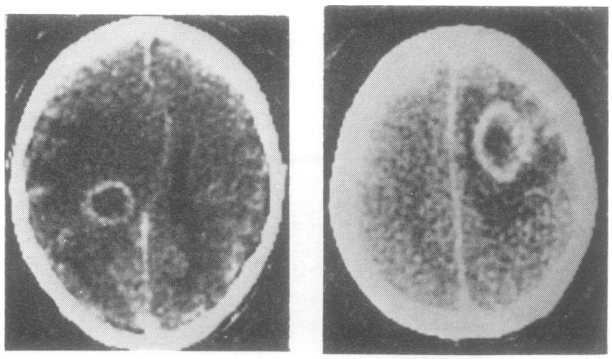
Fig. 7 *Thrombosed angiomatous malformation. Plain scan. High attenuation lesion posteriorly in right temporal lobe. It did not enhance. Diagnosed as low grade glioma.*



(a) (b)

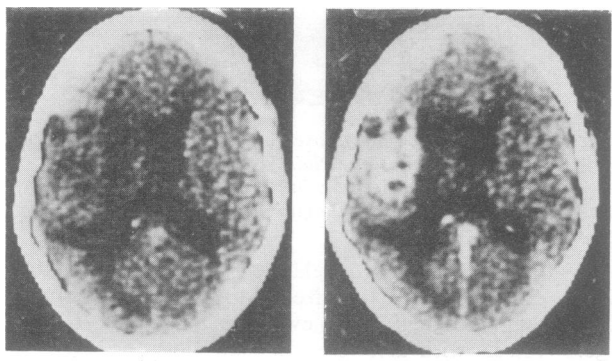
Fig. 8 *Astrocytoma grade 2. (a) Plain scan. (b) After contrast medium. Mixed attenuation mass, left temporal lobe, with marked enhancement. Diagnosed as angiomatous malformation.*

Most cerebral abscesses are diagnosed clinically. CAT is confirmatory by showing an enhancing capsule of almost even thickness enclosing low attenuation pus and surrounded by intracerebral oedema. Cavities in necrotic gliomas usually but not always have walls of irregular thickness; sometimes solid tumour is also evident elsewhere. However, either lesion may occasionally have features more commonly found in the other (Fig. 9). Two solitary tuberculomas with oedema causing mass effect were mistaken for gliomas (Fig. 10), and two locally calcified gliomas were thought to be tuberculous (Fig. 11) partly because the patients were immigrants with pulmonary tuberculosis. It is interesting that in only one of these gliomas was the diagnosis corrected by angiography and, in the one inflammatory lesion (tuberculoma) in which angiography was performed, there was a mistaken confirmation of tumour on the basis of doubtfully abnormal vessels.



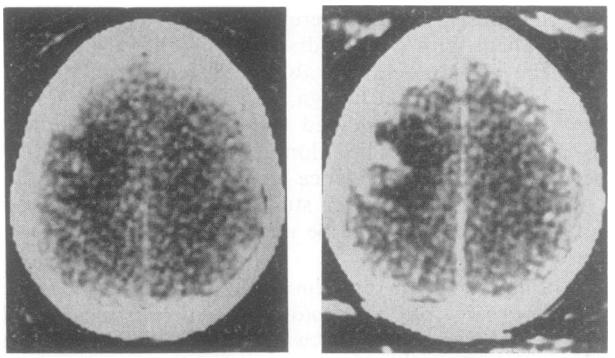
(a) (b)

Fig. 9 *(a) Glioma grade 3–4. After contrast medium. Low attenuation mass left parietal lobe with even ring enhancement. Diagnosed as abscess. (b) Pyogenic abscess. After contrast medium. Low attenuation mass with isodense enhancing slightly irregular ring within it. Diagnosed as glioma.*



(a) (b)

Fig. 10 *Tuberculoma. (a) Plain scan. (b) After contrast medium. Mass of mixed attenuation in the left frontoparietal operculum with considerable mass effect which shows marked but irregular enhancement after intravenous contrast medium. Diagnosed as glioma.*

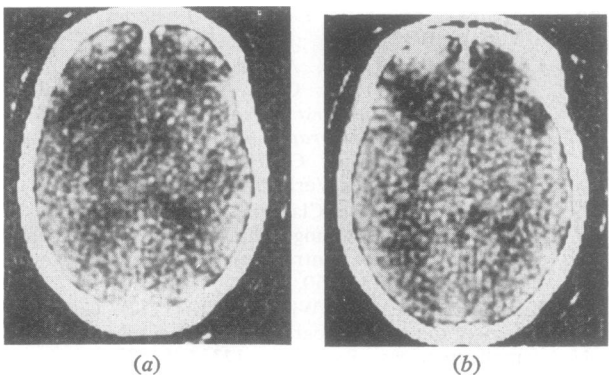


(a) (b)

Fig. 11 *Glioma grade 3–4. (a) Plain scan. (b) After contrast medium. Mixed attenuation mass left parietal lobe which contains a little calcification and shows irregular enhancement. There is some surrounding oedema. The patient was an immigrant from India with pulmonary tuberculosis; diagnosed as tuberculoma.*

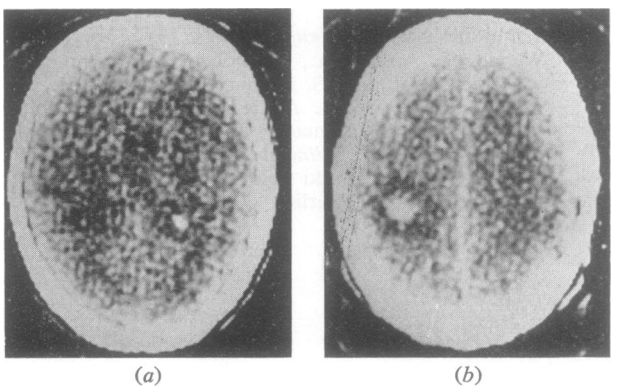
Radionecrosis may reasonably be assumed when a central non-enhancing low attenuation region appears or increases within a treated mass. When a new low attenuation mass showing homogeneous or mixed enhancement appears in a therapy field, it may be due to tumour or a granuloma caused by deep X-ray treatment (Fig. 12). Angiography showed small irregular vessels around such lesions which led to inaccurate confirmation of tumour in our two cases. In appropriate conditions such masses are best treated by excision, and histology appears to be the only reliable method of diagnosis.

Acute plaques of multiple sclerosis may cause enhancing low attenuation lesions with little mass effect (Fig. 13). The large plaques are usually in the central white matter often adjacent to the lateral ventricles. This distribution, and the tendency to be associated with asymptomatic lesions elsewhere and with cerebral atrophy, usually suggest the correct diagnosis (Radue and Kendall,



(a) (b)

Fig. 12 *Radiation granuloma. (a) Plain scan. (b) After contrast medium. There is low attenuation in both frontal lobes more extensive on the left with increased attenuation at the left frontal pole. An enhancing mass is shown at the periphery of the left anterior frontal region after intravenous contrast medium. The patient had had a previous resection of a right frontal glioma followed by radiotherapy. Diagnosed as recurrent glioma.*



(a) (b)

Fig. 13 *Multiple sclerosis. (a) Plain scan. (b) After contrast medium. Focal low attenuation in left parietal lobe with dense central enhancement after intravenous contrast medium. Diagnosed as glioma. Scan returned to normal within one year after which time disseminated lesions were evident clinically, and the patient was confirmed to have multiple sclerosis.*

1978). In one unconfirmed case diagnosed as glioma there was an extensive low attenuation enhancing lesion, with only slight mass effect centrally in both hemispheres and in the corpus callosum. There was a previous history of optic neuritis but the presenting lesion has not progressed or tended to resolve. The diagnosis is sub judice but may well be a demyelinating disease. A further course towards resolution is most helpful in elucidation of the nature of multiple sclerosis, infarction, haemorrhage, and contusion, when more active therapy is not mandatory.

Although angiography may be diagnostic, in many of these unusual cases it has failed to elucidate the specific nature of the lesion. Tumour vessels were shown in only nine of the 29 gliomas: in five of the seven meningiomas and two of the four AVMs the diagnosis was established. Vessels suggesting malignancy were also shown in one metastasis, but they were recorded in four benign lesions (one meningioma, one tuberculoma, and two granulomas after radiotherapy). Thus angiograms performed in 50 cases were diagnostically helpful in 16 (32%), misleading in four (8%), and non-contributory in 60%.

Although the series presented is relatively small it shows that over the last two years, despite being aware of the difficulties of CAT diagnosis from the analysis of our earlier material, about 1.5% of gliomas were not detected on initial CAT, 6.5% were misdiagnosed as benign lesions, and 6.5% of lesions diagnosed as gliomas were benign.

The misdiagnosis of a glioma for another pathology is not particularly important since in most cases it does not influence significantly the natural course of the disease. Sometimes it is even beneficial when simple observation of the clinical and CAT course delays potentially damaging surgery. Occasionally emergency biopsy is precipitated when brain abscess is suspected. Misdiagnosis is more significant when a benign pathology is mistaken for a glioma. Thus three meningiomas in this series were discharged without surgery to outpatient follow-up though happily the tumours were removed successfully at a later date. In one patient, operation on an abscess was delayed, and one with infarction had an unnecessary resection.

Computed tomography, despite its advantages, by no means invariably provides final and reliable diagnosis. It is, therefore, important that no patient with a CAT diagnosis of glioma in which the appearances are also consistent with, though less typical of a benign, surgically treatable lesion should be disqualified from surgery on clinical and CAT findings alone. In such cases positive confirmatory evidence may be obtained from angiograms or other studies but, if necessary, an open biopsy should be made.

Figure 13 is reproduced by permission of the *Journal of Neurology, Neurosurgery, and Psychiatry* where it was originally published in August 1978.

References

Claveria, L. E., Kendall, B. E., and du Boulay, G. H. (1977). Computerised axial tomography in supratentorial gliomas and metastasis. In *The First European Seminar on Computed Axial Tomography in Clinical Practice*, pp. 83–93. Edited by G. H. du Boulay and I. F. Moseley. Springer Verlag: Berlin.

Claveria, L. E., du Boulay, G. H., and Kendall, B. E. (1978). The diagnostic limitations of computerised axial tomography in hemispheric tumours. In *The Diagnostic Limitation of Computerised Axial Tomography*, pp. 2–16. Edited by J. Bones. Springer Verlag: Berlin.

Constant, P., Renou, A. M., Caille, J. M., and Vernhiet, J. (1977). CAT studies in cerebral ischaemia. In *The First European Seminar on Computed Axial Tomography in Clinical Practice*, pp. 227–236. Edited by G. H. du Boulay and I. F. Moseley. Springer Verlag: Berlin.

Kendall, B. E., and Claveria, L. E. (1976). Use of computed axial tomography for the diagnosis and management of intracranial angiomas. *Neuroradiology*, **12**, 141–160.

Kramer, R. A., and Wing, S. D. (1977). Computed tomography of angiography occult cerebral vascular malformations. *Radiology*, **123**, 649–652.

Radue, E. W., and Kendall, B. E. (1978). Iodide and xenon enhancement of computed tomography in multiple sclerosis. *Neuroradiology*, **15**, 153–158.

Wende, S., Aulich, A., Schindler, E., Grumme, T., Meese, W., Lange, S. Kasner E., Steinhoff, H., and Lanksch, W. (1977). A German multicentre study of intracranial tumours. In *Computerised Axial Tomography in Clinical Practice*, pp. 111–117. Edited by G. H. du Boulay and I. F. Moseley. Springer Verlag: Berlin.