A SURVEY OF SUPRATENTORIAL GLIOMAS AND MENINGIOMAS

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During the 10-year period 1952 - 1961, 434 patients with histologically proven intracranial tumours were seen in the Departments of Neurology and Neurosurgery at Groote Schuur Hospital, Cape Town. 41 of these patients had metastatic deposits from extraneural sites. Of the remaining 393 patients, 93 with infratentorial and 61 with miscellaneous tumours (pituitary, emendymal, choroid plexus and vascular tumours) were excluded from consideration. This left 156 who had supratentorial gliomas and 83 with meningiomas above the tentorium cerebelli. These 2 categories constituted the subject matter of our study.

INCIDENCE OF GLIOMAS AND MENINGIOMAS

Both types of tumour result in a progressive clinical picture. As a consequence most patients would seek medical advice and would be referred by their doctors to specialized diagnostic neurological and neurosurgical services. In the 10-year period under discussion, the vast majority of such patients in the Cape Province would have been referred to Groote Schuur Hospital, as this hospital provided the main neurosurgical service in the Province.

Because of the uncertainty inherent in the above supposition, we have not attempted to present the true incidence of these tumours in our population, but rather to indicate the relative ratio of their frequency in the White, Coloured and Bantu groups referred to Groote Schuur Hospital.

There were 118 gliomas and 36 meningiomas in the White group, 25 gliomas and 39 meningiomas in the Cape Coloured group, and 13 gliomas and 8 meningiomas in the Bantu group.

For the purpose of comparison, these figures were expressed as the number per million of each population group. There were 997,377 Whites, 1,334,635 Cape Coloured and 2,967,827 Bantu in the Cape Province according to the 1960 census.

When expressed thus, it is evident that gliomas are much more frequently seen in the White population (118/million) than in the Cape Coloured population (19·2/million) or the Bantu group (4·3/million). By contrast meningiomas occur with approximately similar frequency in the White (36/million) and the Cape Coloured group (30/million), while the figure remains low for the Bantu (2·6/million).

The equal incidence of meningiomas in the White and Coloured groups is in striking contrast to the difference in incidence of gliomas in these groups. The claim may be made that the low incidence of gliomas in Coloured and Bantu groups represents a failure of Coloured and Bantu patients to reach the large centres from the more remote rural areas. We have, therefore, analysed in similar fashion the incidence per million of the population of cerebral gliomas and meningiomas in the Cape Town area where medical facilities and hospitalization are certainly available to all racial groups.

As may be seen from Table I, the incidence per million Whites in Cape Town was 107 gliomas and 32 meningiomas. The comparable incidence per million Coloured was 18 gliomas and 44 meningiomas. There were 33 gliomas and 17 meningiomas per million in the Bantu. It will be seen that the number of Bantu cases is too small for analysis. In the White and Coloured groups there is a reasonable correlation between the incidence of both sorts of tumour in the Cape Town population and in that of the Cape Province. These figures show that gliomas occur 3 times less frequently in the Coloured than in the White group.

TABLE I. THE INCIDENCE/MILLION OF GLIOMAS AND MENINGIOMAS IN THE 3 POPULATION GROUPS IN THE CAPE PROVINCE AND CAPE TOWN*

	Gliomas		
	White	*Coloured*	*Bantu*
Cape Province ..	118 (118)	19·2 (25)	4·3 (13)
Cape Town ..	107 (30)	18 (7)	33 (2)
	Meningiomas		
	White	*Coloured*	*Bantu*
Cape Province ..	36 (36)	30 (34)	2·6 (8)
Cape Town ..	32 (9)	44 (17)	17 (1)

*The number of cases are shown between brackets.

Table II shows that when gliomas and meningiomas are expressed as a percentage of all intracranial tumours a similar proportion to that of most large European and American series is seen only in our White group. The incidence of gliomas in the Coloured group is disproportionately low.

TABLE II. PERCENTAGES OF MENINGIOMAS AND GLIOMAS IN PREVIOUS AND PRESENT SERIES OF CEREBRAL TUMOURS

	Cushing[3] *1932*	*Cairns*[4] *1941*	*Olive-crona*[5] *1932*	*Walshe*[6] *1931*	*Present Series (White)*	*Present Series (Coloured)*
Gliomas ..	42·6	47	47·9	41	40·8	21·7
Meningiomas	13·4	14·5	19·2	13	11·7	34

Proctor[1] compared Bantu with European patients. He found, from necropsy material, that primary cerebral tumours appeared in the Bantu with one-fifth the European frequency. Higginson and Oettlé[2] confirmed that the incidence of brain tumours was significantly lower in the Bantu than expected and that 'this restriction in incidence affects gliomas particularly'.

It is difficult to account for the low incidence of gliomas in the Coloured population studied. An explanation which we considered was the lower life expectancy in the Coloured as compared with the White group. This fact failed to explain the disproportionate incidence in our series. We hope that more exact information will be provided by a registry of all cases of cerebral tumours which is soon to be established in Cape Town.

Site of the Tumours

We have analysed the site of occurrence of these tumours according to operative descriptions where these were sufficiently detailed and explicit. As tumours are not always conveniently localized to anatomical subdivisions of the brain we have divided their situations into *prefrontal* (anterior to precentral sulcus), *central* (pre- and post-

central regions), *temporal, parietal, occipital* and *parieto-temporal* (involving both these areas) localities.

In the meningiomas such subdivision is particularly difficult and the operative notes were extensively used to determine which lobe of the brain was being compressed. Thus olfactory-groove meningiomas were grouped with the frontal tumours while those of the sphenoidal ridge were largely found to involve the temporal lobe. The meningiomas of the sellar region and of the optic foramina were excluded in this aspect of the study. 108 gliomas and 64 meningiomas were so subdivided as shown in Table III.

TABLE III. PERCENTAGE OF GLIOMAS AND MENINGIOMAS IN EACH SITE

	Pre-frontal	*Central*	*Temporal*	*Parietal*	*Temporo-parietal*	*Occipital*
Gliomas ..	23·1%	14·8%	17%	26%	8·7%	12%
			64·8%			
Meningiomas	23·4%	29·7%	8%	32·8%	0%	6·25%
			70·5%			

The prefrontal region contained 23% of the meningiomas and a similar proportion of gliomas. In the occipital region there was a very low incidence of both tumours as has been reported. The temporal, parietal and central regions contained 64·8% of gliomas and 70·5% of the meningiomas analysed.

Symptomatology

There are many excellent studies dealing with the symptomatology of gliomas and meningiomas.[3, 7-10] In nearly all of these reports the occurrence of symptoms at any stage of the illness was considered. In this paper we have analysed only the initial symptom or symptoms where 2 or more symptoms co-existed from the onset of the illness. Hence our results are not strictly comparable with those of most others. This fact should be borne in mind in all subsections of this paper.

TUMOURS OF THE PREFRONTAL REGION

There were 25 gliomas and 15 meningiomas localized in the prefrontal region.

Symptoms

Epileptic seizures were present at the onset of the illness in only 20% of patients with prefrontal gliomas and in 20% of patients with prefrontal meningiomas (Table IV). Penfield[11] found seizures in 53% and White[12] in 33% of patients with *frontal* tumours. Baker[13] says that seizures occur only 'occasionally' with prefrontal meningiomas and does not mention them when discussing symptoms of olfactory-groove meningiomas. Although these percentages differ considerably, all authors are in agreement that the incidence of seizures rises as the tumour site approaches the central fissure.

Headache, on the other hand, was the original symptom in 64% of patients with gliomas and 60% with meningiomas. In a few patients in each group the headaches were lateralized, but in the majority they were bilateral and compatible with the commonly accepted description of high intracranial-pressure headaches. This is in general agreement with the studies of others.

Personality change or *memory disturbance* is another important symptom of prefrontal gliomas and occurred in 60% of the cases. Meningiomas in this situation resulted in mental changes much less frequently (33%). The high incidence of mental changes in frontal tumours is general experience, and its greater frequency in the intrinsic gliomas rather than in the compressive meningiomas has been pointed out by Northfield and Russel.[14]

Localizing symptoms such as hemiparesis, anaesthesia or dysphasia were uncommon and occurred in only 16% of cases with gliomas and 13% of those with meningiomas. Similarly, symptoms due to disturbances of the visual pathways were, as would be expected, rare. They did not occur with the gliomas and were present in 2 cases with olfactory-groove and optic foramen meningiomas.

Disturbances of vision owing to papilloedema and secondary optic atrophy occurred in 12% of patients with gliomas and 47% of those with meningiomas. This high incidence in the meningiomas was noted by Baker who described the symptoms of meningiomas of the anterior third of the sagittal sinus as 'Headaches, mental apathy and decreased visual acuity', which 'results from papilloedema, followed at times by secondary optic atrophy'.

The relative rarity of symptoms of post-papilloedematous visual failure with gliomas is probably the result of their more rapid growth and rapid extension to other areas resulting in the diagnosis being established or death occurring before post-papilloedematous atrophy can occur. This is borne out by the fact that 65% of prefrontal gliomas were diagnosed within 6 months of their initial symptom compared with only 23% of meningiomas.

In summary, epilepsy occurred with equal incidence in both sorts of tumour and was much less common than headache in either type or personality change in the

TABLE IV. COMPARISON BETWEEN INITIAL SYMPTOMS AND THEIR SITUATION*

	Pre-frontal %	*Central* %	*Temporal* %	*Parietal* %	*Temporo-parietal* %	*Occipital* %	*Total* %
Epilepsy							
Gliomas	20	37·5	55	33	9	15·4	35
Meningiomas	20	52·6	80	62	—	25	48·4
Headache							
Gliomas	64	50	39	59	66	61·5	58
Meningiomas	60	68	20	33	—	75	51·5
Personality change							
Gliomas	60	31	28	15	33	38·5	38
Meningiomas	33	10·5	0	4·8	—	0	7
Localizing symptoms							
Gliomas	16	56	5·5	48	0	7·7	30·5
Meningiomas	13·2	26	40	57	—	25	34

*Percentage of patients with tumours in each situation showing a particular initial symptom.

gliomas. While the meningiomas had a lower incidence of mental changes, they showed a higher incidence of symptoms of post-papilloedematous visual failure. Localizing features such as paralysis, dysphasia or symptoms referrable to visual field defects were uncommon presenting symptoms in both tumour types. These features are relatively easily understood when the distance to the sensory motor cortex, the speed of growth and the difference between compressive and invasive tumours are taken into account.

TUMOURS OF THE CENTRAL SULCUS

There were 16 gliomas and 19 meningiomas localized in the vicinity of the central sulcus.

Symptoms

Epilepsy occurred as the initial symptom with greater frequency in this situation than with the prefrontal tumours. It occurred more frequently with meningiomas (52·6%) than with gliomas (37·5%).

Headache was common with both types of tumour, being slightly more frequently found with meningiomas (68%) than with gliomas (50%).

Localizing symptoms occurred more commonly with the gliomas (56%) than with meningiomas (26%) and personality changes, although uncommon in both, occurred more often with the gliomas (30%) than with meningiomas (10%). Symptoms referable to disturbances of the optic pathways were very rare, while defective vision owing to post-papilloedematous optic atrophy was again much less common with gliomas than meningiomas.

The higher incidence of seizures in this group of tumours is compatible with the findings of Penfield *et al.*,[15] who noted that the nearer a tumour is to the rolandic zone, the higher the incidence of seizures.

TUMOURS OF THE TEMPORAL REGION

There were 18 gliomas and 5 meningiomas in the temporal region.

Symptoms

The incidence of *epileptic seizures* was highest with tumours in this region. 55% of patients with temporal gliomas and 4 of the 5 with temporal meningiomas had seizures as the first symptom.

Headaches, personality change, localizing symptoms, as well as visual disturbances of all sorts were relatively much less common initial features of both varieties of tumour (Table IV).

Although the incidence of epileptic seizures with tumours of the temporal lobe was less than that of the rolandic region in all other reports (White[12] 39% ; Penfield[15,17] 48% ; Kolodny[8] 55%), Penfield noted that, when the number of seizures was correlated with the volume of each region of the brain, their incidence was highest in tumours of the temporal lobe. In Penfield's map[15] of the location of tumours which give rise to seizures, the area showing the highest incidence covers the rolandic region and overlaps onto the temporal lobe. Moreover, he commented that 'the nearer the lesion is to the rolandic zone, *especially its lower end,* the higher the incidence of fits'. Kolodny[8] also drew attention to the importance of fits as a symptom of temporal lobe tumours.

TUMOURS OF THE PARIETAL REGION

There were 27 gliomas and 21 meningiomas localized in the parietal region.

Symptoms

Epilepsy occurred as a presenting symptom in 33% of patients with parietal gliomas and 66% of those with meningiomas in this region.

Headache showed the opposite distribution being more frequent in the case of gliomas (59% compared with 33% in meningiomas).

Personality changes were unusual in both groups while aphasia, paresis or sensory disturbances occurred frequently in patients with both tumour types (48% and 57% respectively for gliomas and meningiomas). Symptoms referable to disturbances of the optic pathways were not encountered and those resulting from post-papilloedematous visual failure were uncommon.

In summary, localizing symptoms were common to both sorts of tumour, while headache was more common in gliomas and epilepsy in meningiomas.

In the series of Gibbs[16] and White[12] epilepsy occurred with greatest frequency in tumours of the parietal lobe. In Penfield's cases[15, 17] 68% of tumours in the parietal region had epileptic seizures.

TUMOURS OF THE OCCIPITAL AND PARIETO-OCCIPITAL REGIONS

13 gliomas and 4 meningiomas occurred in the occipital and parieto-occipital regions.

Symptoms

Epilepsy was an uncommon initial symptom with occipital lobe tumours in our series, as in those of Penfield[15, 17] and Allen.[7]

Headache was the initial symptom in 61·5% of the patients with gliomas and 75% of those with meningiomas.

Personality changes occurred in 38% of patients with occipito-parietal gliomas but were not encountered in patients with meningiomas in this region.

TABLE V. DURATION OF PRESENTING SYMPTOMS BEFORE DIAGNOSIS*

	0–3 months	*3–6 months*	*6–12 months*	*12–24 months*	*More than 24 months*
Epilepsy					
Gliomas	16·0	13·5	15·5	13·5	40·6
Meningiomas	9·7	6·4	19·3	13·0	51·6
Headache					
Gliomas	68·5	11·0	11·0	1·8	7·4
Meningiomas	20·0	17·0	5·7	31·4	25·8
Personality change					
Gliomas	73·0	11·5	3·8	3·8	7·6
Meningiomas	50·0	20·0	10·0	20·0	0
Localizing symptoms					
Gliomas	75·0	20·8	4·0	0	0
Meningiomas	31·5	10·5	15·0	26·0	15·0

*Percentage of patients presenting within each time period.

Localizing symptoms and disturbances of vision of all sorts were uncommon initial symptoms of either type of parieto-occipital tumour.

It may seem strange that visual symptoms should be so uncommon as initial symptoms of occipital lobe tumours, but this is the general experience. Allen[7] noted that only 15% of patients noted visual loss as an *initial* symptom while headache accounted for 35% of cases. In the rest of the clinical course headache occurred in 95% while symptoms suggesting a field defect occurred in only 16% of patients. By contrast, the physical examination showed that 94% of his patients had visual field defects and 70% papilloedema. His conclusion that symptoms of increased intracranial pressure dominate the clinical picture from the beginning, was confirmed in the present study.

A COMPARISON BETWEEN THE INITIAL SYMPTOMS AND THE TIME BETWEEN THEIR APPEARANCE AND THE ESTABLISHMENT OF A DIAGNOSIS OF CEREBRAL TUMOUR (Table V)

The delay before diagnosis was analysed for each initial symptom. The abscissa in Graphs 1a and b represents the time of diagnosis and the ordinate the percentage of cases diagnosed in each of these time periods (Fig. 1). Graph 1a shows that 70% of patients with gliomas who commence their illness with headaches, personality change or localizing symptoms are diagnosed within 3 months of the onset of symptoms. By contrast, with meningiomas (Graph 1b), there is a constant proportion of patients with these initial symptoms reaching diagnosis at each interval. This is represented in the graph as an almost straight line parallel to the abscissa (Fig. 1).

Cases having epilepsy as an initial symptom (Graph 1c) showed a similar distribution for both meningiomas and gliomas where 51% and 40% of cases respectively were diagnosed *more* than 2 years after the original seizure. In the gliomas the distribution is especially noteworthy.

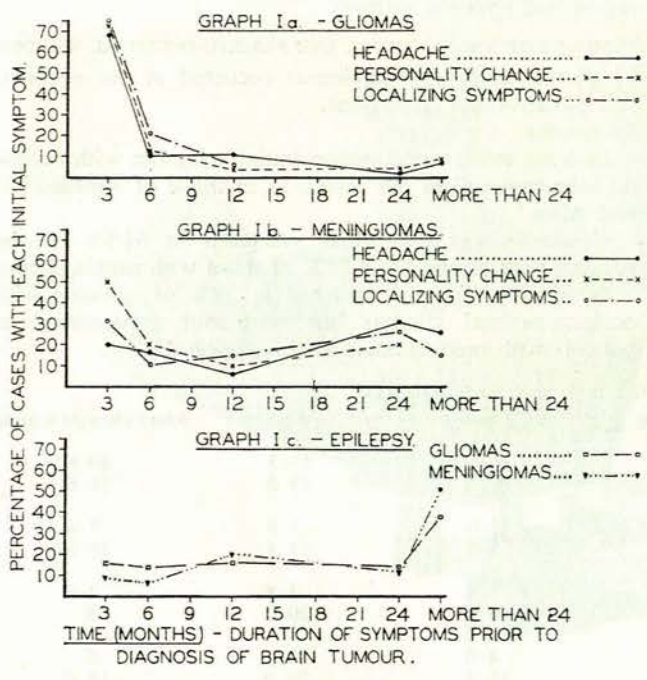

Fig. 1. See text.

The delay in diagnosis of tumours presenting with epilepsy was previously noted by Penfield *et al.*[11, 15, 17] who, after analysis of tumour growth and structure, concluded that epilepsy was associated with the slower growing tumours. The relationship between epilepsy and the slower growing tumours is also apparent in that epilepsy constituted the presenting symptom in 50% of oligodendrogliomas, 48% of meningiomas, and 33% of astrocytomas, but only 17% of glioblastoma multiforme in the present series. This is similar to the distribution reported by White *et al.*[12] Walshe[6] also noted that meningiomas and the *'slower* growing gliomata' tended to present with epilepsy.

Although it is true that epilepsy occurs more frequently with slow-growing tumours, it is as much the nature of the epileptic process as the slowness of growth of the tumour which accounts for the delay in diagnosis. Epilepsy, unlike all the other symptoms discussed previously, results from a disturbance of *function* of cells—an 'irritative' as opposed to a destructive phenomenon. It is also clear from the recent research into temporal lobe epilepsy, that seizures may result from very small lesions. By contrast, the other symptoms of supratentorial tumours only occur when a tumour is of sufficiently large size to cause raised intracranial pressure or fairly extensive destruction of brain tissue. Thus, it is clear that epilepsy may appear as the sole symptom of tumour growth, while the tumour is quite minute, and long before it manifests any 'space-occupying' features. This was certainly noted in many of our patients, whose seizures were fully investigated both clinically and radiologically on one or more occasion, with negative results; only to be followed months or years later by the appearance of a tumour in the region of the previous EEG focus. This is also implicit in Walshe's remarks:[6] 'Generalized epileptiform convulsions, recurring at irregular intervals, may for years be the sole manifestation of tumour, and even when the age of the patient awakens the suspicions of the observer, repeated clinical and radiological investigations may prove negative until the final and rapid development of signs of intracranial tension'. Many other observers (Lund[18]) have noted this feature.

It seems, therefore, that the presence of epileptic seizures as the initial manifestation of a tumour is not only a function of its site and slowness of growth, but also of the fact that it may be excited by a very small lesion.

CONCLUSIONS

1. *Incidence*

The incidence of gliomas in the Cape Coloured group in Cape Town was only a third of the figure for the White group. Meningiomas occurred with almost equal frequency in the White and Coloured groups.

2. *Site of Tumours*

60 - 70% of both varieties of tumours occurred in the temporo-parietal and central regions. 23% occurred frontally while the occipital lobe was involved least often.

3. *Symptomatology*

(a) *Headache* as an initial symptom of gliomas was very common with tumours of all regions, except the

temporal lobe where epilepsy was the commonest presenting symptom. With meningiomas headache was common in all regions except the temporal and parietal regions where epilepsy and localizing features were more frequent initial symptoms.

(b) *Mental changes* occurred with tumours of all regions but were most common initial symptoms in tumours of the prefrontal region with or without involvement of the corpus callosum. They were very much less in evidence with extrinsic compressive lesions (meningiomas) than with the infiltrating gliomas.

(c) *Localizing symptoms* (dysphasia, paresis or sensory loss) were commonest with lesions involving the central region and the parietal lobe, especially of the dominant hemisphere.

(d) *Visual symptoms* resulting from visual-field defects were rare with tumours of any part of the cerebral hemispheres while those from post-papilloedematous optic atrophy were not uncommon with meningiomas in the prefrontal and central regions. Even with occipital lobe tumours causing marked visual field defects, symptoms arising from these defects were rare.

4. *Epilepsy as an Initial Symptom of Gliomas and Meningiomas*

The figures presented here support the conclusions reached by Penfield *et al.*[11, 15, 17] and White *et al.*[12] that the incidence of epileptic seizures increased as the tumour approached the central sulcus. However, the highest incidence of epileptic seizures in our material occurred with lesions in the temporal lobe and it was rarest with occipital lobe lesions.

When the nature of each symptom was contrasted with the delay in diagnosis, it was evident that most delay occurred with epilepsy—whether caused by gliomas or meningiomas. It was also noted that epilepsy tended to exist as an isolated symptom much more frequently and for longer periods than any other symptoms. The views of Penfield, White and others were discussed; the conclusion reached by them being that the presence of epilepsy is a function of the slowness of growth of the tumour as well as of its situation. We have suggested another factor to account for the delay, namely that epilepsy, by virtue of its pathogenesis, may be caused by a lesion so small that it will not cause space-taking or significant destructive effects for a considerable period of time after the onset of seizures.

The slowness of growth of tumours evoking epilepsy does not necessarily indicate a good prognosis, because, as Walshe pointed out (see above), they are often only discovered when raised intracranial pressure makes its appearance.

It seems reasonable to conclude that, as long as surgical therapy remains the treatment of choice, more attention should be paid to the careful investigation of focal seizures, including, if needs be, exploratory craniotomy.

SUMMARY

239 supratentorial gliomas and meningiomas have been analysed as to their racial incidence, site, initial symptomatology and delay in diagnosis.

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REFERENCES

1. Proctor, N. S. P. (1955): In *Proceedings of the 2nd International Congress on Neuropathology, London*, p. 99.
2. Higginson, J. and Oettlé, A. G. (1960): J. Nat. Cancer Inst., **24**, 589.
3. Cushing, H (1932): *Intracranial Tumours*. Springfield, Ill.: Thomas.
4. Cairns, H. and Jupe, M. H. (1951): *A Textbook of X-ray Diagnosis*. London: Lewis.
5. Olivecrona, H. (1952): J. Neurosurg., **9**, 317.
6. Walshe, F. J. M. (1931): Quart. J. Med., **24**, 587.
7. Allen, I. M. (1930): Brain, **53**, 194.
8. Kolodny, A. (1928): *Ibid.*, **51**, 385.
9. *Idem* (1929): Arch. Neurol. Psychiat. (Chic.), **21**, 1107.
10. Penman, J. and Smith, M. C. (1954): *Intracranial Gliomata*. Spec. Rep. Ser. Med. Res. Coun. (Lond.), 284.
11. Penfield, W. and Jasper, H. (1954): *Epilepsy and Functional Anatomy of the Human Brain* London: Churchill.
12. White, J. C., Ching Tung Liu and Mixter, W. J. (1948): New Engl. J. Med., **238**, 891
13. Baker, A. B. (1962): *Clinical Neurology*. New York: Hoeber.
14. Northfield, D. W. C. and Russel, D. *in* Feiling, A., ed. (1951): *Modern Trends in Neurology*. London: Butterworths.
15. Penfield, W., Erickson, T. C. and Tarlow, I. M. (1940): Arch. Neurol. Psychiat. (Chic.), **44**, 300.
16. Gibbs, F . A. (1932): *Ibid.*, **28**, 969.
17. Penfield, W. and Erickson, T. C. (1941): *Epilepsy and Cerebral Localization*. Springfield, Ill.: Thomas.
18. Lund, M. (1952): Acta psychiat. scand., suppl. 81.