Movement disorders in astrocytomas of the basal ganglia and the thalamus

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Abstract
In a series of 225 patients with astrocytomas (grades I–IV) of the basal ganglia and the thalamus, 20 had a movement disorder. In all patients the histological diagnosis was verified by stereotactic biopsy. Tremor was observed in twelve patients, dystonia in eight, chorea in three, and chorea/ballismus and myoclonus in one. The tumour involved the thalamus in 16 patients. Corticospinal tract dysfunction was evident in 70% of the patients with movement disorders and in 73% of those without. Demographic, clinical, histological and neuroradiological data of the patients with a movement disorder were compared with the data of patients without. CT data yielded no differences with respect to the involvement of anatomical structures. Movement disorders were significantly associated with low-grade astrocytomas.

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Tumours of the thalamus and the basal ganglia are relatively rare. They are estimated to make up 1–2.5% of intracranial tumours. Most of these neoplasms are benign or malignant astrocytomas. Since craniotomy and partial resection was associated with high mortality and morbidity, stereotactic diagnostic and therapeutic techniques have been favoured in recent years.

As movement disorders (MDs) are often related to dysfunction or structural lesions of the basal ganglia, a high incidence in intrinsical tumours might be expected, but they seem to be infrequent. While in some series they are not even mentioned, they range from 1–7% in recent and from 4–33% in earlier series.

Since 1965, no large series has focused on the subject. To characterise better this relatively uncommon cause of MDs, we reviewed the literature and retrospectively analysed a series of 225 patients with histologically verified astrocytomas of the basal ganglia and the thalamus. Among those we identified 20 with MDs (classified according to criteria published elsewhere) at the time of biopsy.

Results
Twenty (9%) patients with astrocytomas of the basal ganglia and the thalamus displayed an MD at the time of biopsy. The MD was the initial symptom of the tumour in three patients. The mean latency between onset of any symptoms and biopsy was 3 years. Various other clinical symptoms were found (table 1). Signs of raised intracranial pressure at biopsy were evident in patients 2, 5, 6, 8, and 15. Obstructive hydrocephalus at biopsy was found in patients 5, 6, and 8. Five patients had received ventriculoatrial or ventriculoperitoneal shunts before stereotactic biopsy. Two had no MD before the shunt operation, in two others shunting did not alter the MD, and in patient 14 hemiparesis improved while tremor did not.

CT or additional MRI findings were obtained in 18 of the 20 patients (two had died before 1975). The tumour involved the thalamus in 16. Downward extension to the subthalamic or the upper midbrain varied. This could not always be determined exactly due to the compressive effect of the tumour on adjacent structures or due to peritumoural oedema, or because some patients had had only axial CT scans. In 15 patients the tumour...
Table 1  Clinical features, histological grading and neuroradiological findings of 20 patients with movement disorders in basal ganglia and thalamic astrocytomas

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex/Age at Onset</th>
<th>Preop. KPS</th>
<th>Movement Disorder</th>
<th>Other Clinical Symptoms at Biopsy</th>
<th>Hemiparesis</th>
<th>Grade of Astrocytoma</th>
<th>Brain Imaging Study Results</th>
<th>Tumour Volume (cm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/25/13</td>
<td>60</td>
<td>Tremor, 3-4 Hz</td>
<td>R hypacusis, nystagmus</td>
<td>—</td>
<td>I</td>
<td>R thalamus, cyst extending to midbrain (circumscribed)</td>
<td>~75</td>
</tr>
<tr>
<td>2</td>
<td>M/3/3</td>
<td>80</td>
<td>Tremor, 3-5 Hz</td>
<td>Papilloedema</td>
<td>—</td>
<td>I</td>
<td>R thalamus, internal capsule, pallidum (circumscribed)</td>
<td>~22</td>
</tr>
<tr>
<td>3</td>
<td>M/50/44</td>
<td>60</td>
<td>Tremor, 3-4 Hz</td>
<td>R optic atrophy, 3rd nerve palsy, dysarthria, gait ataxia</td>
<td>L</td>
<td>II</td>
<td>R postero-basal thalamus, nucleus ruber (circumscribed)</td>
<td>~1.5</td>
</tr>
<tr>
<td>4</td>
<td>F/49/43</td>
<td>60</td>
<td>Chorea face, bilateral I &gt; r</td>
<td>Impaired memory, seizures</td>
<td>L</td>
<td>II-III (3m)</td>
<td>R frontomedial, basal, septum pellucidum, corpus callosum, caudate (diffuse)</td>
<td>&gt;50</td>
</tr>
<tr>
<td>5</td>
<td>F/70/70</td>
<td>40</td>
<td>Tremor, 3-4 Hz</td>
<td>Impaired memory, hypothesia, internal capsule, thalamus, lenticular nucleus, myoclonus, gait ataxia</td>
<td>L</td>
<td>IV</td>
<td>R + L postero medial thalamus, 3rd ventricul, corpus pineale, splenium corporis callois (circumscribed)</td>
<td>~20</td>
</tr>
<tr>
<td>6</td>
<td>M/37/31</td>
<td>60</td>
<td>Tremor, 4-5 Hz</td>
<td>Impaired memory, seizures, aphasia</td>
<td>—</td>
<td>—</td>
<td>R thalamus, internal capsule, pallidum, putamen, corpus callosum, septum pellucidum, r frontal + temporal, I frontal (diffuse)</td>
<td>&gt;100</td>
</tr>
<tr>
<td>7</td>
<td>F/11/10</td>
<td>60</td>
<td>Dystonia I arm: rest, on action</td>
<td>Lethargy, Neurofibromatosis type I</td>
<td>L</td>
<td>I</td>
<td>R pallidum, subthalamic, internal capsule (circumscribed) + quadrigeminal plate + bilateral pons + cerebellum (diffuse)</td>
<td>~12</td>
</tr>
<tr>
<td>8</td>
<td>M/12/9</td>
<td>70</td>
<td>Dystonia I hand: on action</td>
<td>Impaired memory, obsessive hypothesia, internal capsule, thalamus, l hemiparesis, 1th nerve palsy, Parinaud’s syndrome</td>
<td>L</td>
<td>I</td>
<td>R thalamus (circumscribed)</td>
<td>~15</td>
</tr>
<tr>
<td>9</td>
<td>M/42/41</td>
<td>70</td>
<td>Tremor, 4-5 Hz</td>
<td>Nystagmus, headache</td>
<td>—</td>
<td>II</td>
<td>L basal ganglia + hemisphere, corpus callosum, diffus, multifocal</td>
<td>~100</td>
</tr>
<tr>
<td>10</td>
<td>M/25/25</td>
<td>60</td>
<td>Dystonia I arm: rest, on action</td>
<td>Impaired memory, aphasia, behavioural disorder, seizures, r hemiparesis</td>
<td>R</td>
<td>I</td>
<td>L thalamus, subthalamus, caudate, putamen, pallidum, internal capsule, corona radiata (circumscribed)</td>
<td>~40</td>
</tr>
<tr>
<td>11</td>
<td>M/15/13</td>
<td>70</td>
<td>Tremor, 4-5 Hz</td>
<td>Impaired memory</td>
<td>R</td>
<td>I</td>
<td>R pallidum, putamen, corona radiata (circumscribed) — compression of thalamus</td>
<td>~18</td>
</tr>
<tr>
<td>12</td>
<td>F/1/9</td>
<td>70</td>
<td>Chorea I arm: leg marked on action</td>
<td>Behavioural disorder, r hemiparesis</td>
<td>R</td>
<td>II</td>
<td>L subthalamus, internal capsule, medial caudate, postero basal pallidum (circumscribed)</td>
<td>~18</td>
</tr>
<tr>
<td>13</td>
<td>F/10/9</td>
<td>60</td>
<td>Dystonia I arm: leg</td>
<td>Preoccous puberty</td>
<td>L</td>
<td>III (4y)</td>
<td>R subthalamus, thalamus, internal capsule, corona radiata, cr cerebri (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>14</td>
<td>F/12/3</td>
<td>60</td>
<td>Tremor, 3-5 Hz</td>
<td>Seizures, gait ataxia</td>
<td>L</td>
<td>II</td>
<td>R thalamus, quadrigeminal plate (circumscribed)</td>
<td>~25</td>
</tr>
<tr>
<td>15</td>
<td>M/16/15</td>
<td>40</td>
<td>Dystonia + Chorea</td>
<td>Obtundation, 3rd nerve palsy, cachexia</td>
<td>L</td>
<td>IV</td>
<td>R thalamus, upper hemisphere (≥ CT)</td>
<td>~30</td>
</tr>
<tr>
<td>16</td>
<td>F/62/59</td>
<td>60</td>
<td>Tremor, high-frequency bilateral, r + fingers + eyelids</td>
<td>Psychosis, lethargy, dysarthria</td>
<td>R</td>
<td>II</td>
<td>L anterior-ventrolateral thalamus subthalamus, internal capsule, cr cerebri (circumscribed)</td>
<td>~7</td>
</tr>
<tr>
<td>17</td>
<td>F/13/11</td>
<td>70</td>
<td>Dystonia, r arm: rest, on action</td>
<td>Dysarthria</td>
<td>R</td>
<td>I</td>
<td>L hypothalamus, thalamus extending over midline and to midbrain (≥ CT)</td>
<td>~25</td>
</tr>
<tr>
<td>18</td>
<td>F/3/2</td>
<td>70</td>
<td>Myoclonus, r shoulder</td>
<td>Behavioural disorder, anorexia, diminished vision 1 eye</td>
<td>—</td>
<td>I</td>
<td>L hypothalamus, thalamus (circumscribed)</td>
<td>~20</td>
</tr>
<tr>
<td>19</td>
<td>F/5/5</td>
<td>60</td>
<td>Dystonia, r arm: rest, on action</td>
<td>Behavioural disorder, diminished vision 1 eye</td>
<td>R</td>
<td>—</td>
<td>L subthalamus, thalamus, caudate, internal capsule, pallidum, putamen, insula, uncus (circumscribed)</td>
<td>~20</td>
</tr>
<tr>
<td>20</td>
<td>F/12/10</td>
<td>60</td>
<td>Tremor, 3-4 Hz</td>
<td>Impaired memory, behavioural disorder, diminished vision 1 eye</td>
<td>R</td>
<td>I</td>
<td>L thalamus, subthalamus, cr cerebri (circumscribed)</td>
<td>~16</td>
</tr>
</tbody>
</table>

M = male, F = female, KPS = Karnofsky Performance Scale, R = right, L = left. Tremor is qualified as: rest—tremor at rest, post—postural tremor and int—intention tremor.

appeared to be more or less circumscribed. In patients 6 and 9 (fig 1) the neoplasm involved the basal ganglia and large parts of the hemisphere diffusely. The tumour volume ranged from 1.5 to more than 100 cm³. It was located in the contralateral basal ganglia in patients with unilateral MDs.

Twelve patients had tremor. Only patient 5 had Parkinsonism with bilateral rigidity, resting and postural tremor, akinesia, masked face, and loss of postural reflexes, though tremor was also present on action. In this case the tumour was located in the midline and involved both thalami, but exerted marked compression on the midbrain. Patient 16 had a high-frequency tremor of fingers and eyelids, presumably essential tremor, which was less severe on the right, slightly hemiparetic side contralateral to the tumour. The tumour preemminently involved the anterior and ventrolateral thalamus (fig 2). Ten patients had unilateral tremor, who in two was only present on action, in two others on action and on maintaining antigravity posture, and in six a combined resting-postural-intention tremor was found. These tremors had a frequency of 3 to 5 Hz. The slight increase in tone in some patients with an ipsilateral hemiparesis had features of spasticity rather than of rigidity. Tremor in these patients was not associated with akinesia or impaired postural reflexes. In nine of these patients the tremor involved the
thalamus. In the tenth (case 11) the tumour
was predominantly located in the pallidum
and putamen, sparing the thalamus, but nev-
theless exerted considerable compression on
the thalamus. In patient 3 the tumour was
located mainly in the red nucleus and to a
lesser degree in the postero-basal thalamus
(fig 3). In patient 14 the astrocytoma extended
from the thalamus to the quadrigeminal plate
(fig 4).

Hemidystonia or hemiathetosis was seen in
eight patients, in five of whom it was par-
ticularly marked on action. The arm was affected
in all these patients, with additional involve-
ment of the leg in two. Patients 2 and 8 with
involvement only of hand and fingers had an
astrocytoma confined to the thalamus. In
patients 10 and 19 the tumour mainly involved
the caudate and putamen, to a lesser degree
the thalamus. In cases 13, 15, and 17 the main
tumour mass was found in the thalamus,
whereas in case 7 the thalamus only was
compressed.

Choreatic MDs were seen in four patients.
Two had hemichorea, one left hemichorea
involving the face markedly on the left side,
and one hemichorea/hemiballismus. Patients
13 and 15 presented with hemichorea and
dystonia appearing as "choreoathetosis". Hemichorea was associated in two
patients with involvement of the head of the caudate. In
the patient with hemichorea/hemiballismus,
the thalamic tumour involved the subthalamic
nucleus. Choreatic hyperkinesias disappeared
in all patients in the long term.

The following data were analysed compar-
atively for patients with and without MDs. The
female/male ratio was 1:2:1 (11 females/9
males) for MD patients and 1:1 for those
without (102/103). The 20 astrocytomas of the
MD patients and the 205 astrocytomas of
those without were distributed according to
histological grading as follows: WHO I—12
(60%)/41 (20%); WHO II—6 (30%)/71
(35%); WHO III—0/56 (27%); and WHO
IV—2 (10%)/37 (18%). In patient 16 the
tumour was graded histologically as an astro-
cytoma WHO II, however, focal anaplastic
transformation could not be excluded. The
frequency of MDs in patients with low-grade
(I + II) astrocytomas was 15%, in patients
with high-grade (III + IV) astrocytomas
21%. This difference was statistically sig-
nificant (p = 0.0017). The mean age of
patients with MDs at the time of biopsy was
lower (24 years) than that of the patients
without (36 years), however, the proportion of
low-grade astrocytomas was higher in MD
patients and the mean age correlated with
histological grading as follows: WHO I (mean
age in years, MD patients/patients without)—
13/20; WHO II—36/35; WHO III—42/42; and
WHO IV—43/47. There was also a trend for
the mean age in patients with astrocytomas

Figure 1 Axial enhanced CT scans of patient 9 with
right resting, postural and
intention 4 Hz tremor
show a diffusely spreading
WHO grade II
astrocytoma of the left
basal ganglia and
hemisphere extending
contralaterally via the
corpus callosum.

Figure 2 Enhanced CT scans of patient 16 with essential
tremor showing a WHO grade
II astrocytoma (focal anaplastic transformation could not be excluded) located
predominantly in the left anterior/ventrolateral thalamus and the genu of the internal
capsule.

Figure 3 Axial enhanced CT scans (lower row) with
sagittal (upper row) and coronal (middle row)
reconstructions of patient 3 with left resting, postural and
intention 3–4 Hz tremor showing a relatively small
circumscribed WHO grade I astrocytoma, predominantly
located in the right red nucleus and posterobasal thalamus.
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WHO I to be lower in MD patients (15 years) compared with those without (20 years).

Analysis of the CT data of patients with and without MD yielded no difference concerning the involvement of different anatomical structures. Comparing tumour volumes there was a different distribution, which reached statistical significance: MD patients more often had tumour volumes ranging from 16 to 25 ccm (55%) than those without (17%). However, large tumours were found more often in astrocytomas WHO III and IV.

Seventy per cent of patients with MDs and 73% of patients without had a hemiparesis of variable degree contralateral to the tumour. In the astrocytoma WHO I group there was a tendency for MD patients to present more often with hemiparesis (75%) than those without (61%). The median Karnofsky Performance Score$^{20}$ (KPS) in MD patients was 60. Considering the interrater variability the distribution of the KPS of patients with and without MD was similar.

**Discussion**

The frequency reported for MDs in patients with tumours of the basal ganglia and the thalamus varies considerably. It ranged from 1 to 33%$^{1-7,9,10,21-24}$ (table 2) in previous studies and our study was 9%. However, in some studies a histological diagnosis was not established in all patients and different histological diagnoses were grouped together without specifying separately those with astrocytomas, so that an exact comparison cannot be made. Only three series on tumours of the basal ganglia and the thalamus which mentioned patients with movement disorders dealt with histologically verified astrocytomas for the total study population or a defined subgroup.$^{7,10,21}$ Since the involvement of the basal ganglia and thalamus in astrocytomas is different from that in other neoplasms, it may be better to consider the former tumours separately. The incidence of MDs seems to be generally lower in recent than in earlier studies. This might be explained by the availability of CT and MRI which also reveal smaller and oligosymptomatic tumours, and by the use of stereotactic techniques to establish an earlier histological diagnosis. We chose biopsy as the timepoint for calculating the incidence of MDs, since later MDs may be altered by, or even result from, therapeutic measures.

Previous series on basal ganglia and thalamic tumours differ considerably with respect to the proportion of low- or high-grade astrocytomas in MD patients. While some series report that the majority of patients had “malignant gliomas”$^{23}$ or glioblastomas,$^{10}$ others stress that most of the tumours producing “extrapyramidal” symptoms grow “very or rather” slowly.$^{22}$ In our study MDs were found significantly more often in low-grade astrocytomas.

Although well documented cases of basal ganglia and thalamic tumours presenting with MDs and ipsilateral hemiparesis had been published earlier,$^{21,26}$ sparing of the corticospinal tract was regarded as essential for MDs to occur.$^{23,25}$ In contrast, our data show that tremors as well as dystonia and chorea may be associated with a mild or moderate degree of hemiparesis. Comparing the patients with and without MDs we did not find a significant difference in the frequency of corticospinal tract dysfunction.

Unlike previous series and case reports on histologically verified astrocytomas of the basal ganglia and the thalamus the distribution of MDs differs in our study$^{2,7,8,10,12,21-23,25-41}$ (table 3). We observed comparatively more cases with dystonia and chorea. In patients with dystonia the thalamus was involved more frequently than the caudate or putamen. However, there may be secondary putaminal involvement due to the mass effect of the tumour. Clinicopathological correlations reported previously demonstrate primary lesions in caudate or putamen in the majority of cases with secondary dystonia.$^{12,42,43}$ Choreatic hyperkinesias, which were transient in our patients, seem to be rare in basal ganglia tumours. We found only one case in the literature and six of “choreoathetosis”. Hemi-ballismus was described in a cystic midbrain glioma,$^{14}$ but as far as we know, not in thalamic or basal ganglia astrocytomas.

Tremor was the most frequent MD in our

![Figure 4: Sagittal SE 1040/40 and axial SE 1200/80 MR images of patient 14 with leaft resting, postural and intention 3-5 Hz tremor reveal a WHO grade II astrocytoma, predominantly located in the right thalamus and the quadrigeminal plate.](http://jnnp.bmj.com/)

Table 2: Large series of patients with tumours of the thalamus and the basal ganglia

<table>
<thead>
<tr>
<th>Literature Review (by first author)</th>
<th>Number of patients with movement disorders/Total number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ody, 1932</td>
<td>4/25 16%</td>
</tr>
<tr>
<td>Globus, 1942</td>
<td>3/21 * 14%</td>
</tr>
<tr>
<td>Arseni, 1958</td>
<td>5/50 * 10%</td>
</tr>
<tr>
<td>Roth, 1958</td>
<td>7/34 * 21%</td>
</tr>
<tr>
<td>McKissock, 1958</td>
<td>8/24 ** 33%</td>
</tr>
<tr>
<td>Tobi, 1961</td>
<td>2/49 *** 4%</td>
</tr>
<tr>
<td>Tolosa, 1965</td>
<td>15/61 25%</td>
</tr>
<tr>
<td>Cheek, 1966</td>
<td>7/51 *** &quot;rare&quot;</td>
</tr>
<tr>
<td>Paillas, 1970</td>
<td>13/55 23%</td>
</tr>
<tr>
<td>Hirose, 1975</td>
<td>4/18 ** 22%</td>
</tr>
<tr>
<td>Bernstein, 1984</td>
<td>4/60 *** 7%</td>
</tr>
<tr>
<td>Beta, 1987</td>
<td>7/27 *** ?</td>
</tr>
<tr>
<td>Kelly, 1989</td>
<td>1/72 1%</td>
</tr>
<tr>
<td>Krauss, 1992</td>
<td>20/225 9%</td>
</tr>
</tbody>
</table>

* Series with different types of histological diagnoses, but mainly astrocytomas I-IV.
** Histological diagnosis was not obtained in all patients.

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We think that clinicopathological correlations in thalamic and basal ganglia astrocytomas should be made only with certain reservations. Interaction of different mechanisms may or may not lead to clinical symptoms. Functional lesions of the structures involved primarily by the tumour might result from "internal" compression of neurons or neural pathways or from altered metabolism by reduced vascular supply. But tumours or a peritumoural oedema may also induce symptoms by "external" compression of adjacent structures or their vascular supply. The question has been raised as to why tumours of the basal ganglia are not associated with MDs more often. The situation becomes more complex, since these tumours may also involve structures of the basal ganglia which known to abolish or to reduce MDs when lesioned (predominantly the ventrolateral thalamus, the zona incerta, the pulvinar thalam and their fibre connections). Again these structures may be functionally damaged by "external" compression.

Neither necropsy findings nor the present comparative analysis of in vivo CT data showed topographical differences in thalamic and basal ganglia astrocytomas that do or do not cause MDs. However, tumours which are apparently identical on imaging studies may nevertheless differ in functional terms. These aspects might be least partly explained using models such as that of parallel distributed processing provided more detailed information were available. However, functional state of the basal ganglia in brain tumours causing MDs has only rarely been studied. In a case of a left frontal meningioma with right hemi-Parkinsonism PET indicated impaired oxygen metabolism and tissue perfusion in the striatopallidal region. In one case of Parkinsonism secondary to a craniopharyngioma biochemical findings on necropsy revealed considerably reduced striatal catecholamines and severely decreased dopamine receptor binding sites in the caudate. Further functional studies are needed and should provide deeper insights into the pathophysiological processes underlying the generation of movement disorders.

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