Movement disorders following nonfunctional neurosurgery

JOACHIM K. KRAUSS, M.D., JAN J. BORREMANS, M.D., THOMAS POHLE, M.D., AND NELSON GODOY, M.D.

Department of Neurosurgery, Inselspital, University of Berne, Berne, Switzerland; and Department of Neurosurgery, Albert-Ludwigs-University, Freiburg, Germany

Object. Knowledge is scarce about movement disorders that follow neurosurgical operations other than functional stereotactic surgery. The cases of 14 patients who suffered from movement disorders secondary to craniocebral or spinal surgery are analyzed. None of these patients was initially treated by any of the authors.

Methods. Twelve patients underwent surgery for cerebrovascular diseases. Nine of these patients harbored tumors and three patients had neurovascular disorders. Two patients underwent spinal surgery for cervicothoracic epidermoidoma or for multiple cervical disc herniations. Twelve of the 14 patients had immediate postoperative side effects such as hemiparesis, ataxia, and somnolence. In all but two patients, movement disorders became manifest only after a delay.

Dystonic movement disorders developed in eight patients, unilateral tremors in three patients, unilateral facial myokymia in one patient, and hemichorea–hemiballism in two patients. The mean delay of onset for tremor was 5 weeks and that for dystonic movement disorders was 5.5 months. Movement disorders were transient in three patients; however, they were persistent in 11 patients at a mean follow-up period of 5 years. These movement disorders caused marked persistent disability in four patients. Lesions of the contralateral striatum were identified in patients with dystonic syndromes and lesions of the dentatothalamic outflow in patients with tremors. In three patients who had postoperative basal ganglia lesions after partial removal of astrocytomas, tumor regrowth was later documented. Medical treatment in patients with persistent movement disorders rendered only limited benefit. Two patients improved with botulin injections. In one patient postoperative hemidystonia was alleviated by contralateral thalamotomy.

Conclusions. Dystonic syndromes and tremors are the most common movement disorders that occur after craniocebral and spinal surgery. Postoperative movement disorders can lead to various degrees of functional disability. The pathoanatomical correlations are similar to those described in other patients with secondary movement disorders.

KEY WORDS • brain neoplasm • postoperative complication • dystonia • surgery • tremor

The occurrence of postoperative morbidity after neurosurgical procedures has gained widespread attention within the past few years. In particular, in developing strategies for avoidance of surgical side effects, it is important to recognize and understand underlying mechanisms. Such knowledge has helped to reduce surgical morbidity significantly over the years. Although motor deficits, sensory dysfunctions, and cognitive and behavioral sequela are well recognized complications of neurosurgical procedures, the occurrence of movement disorders, in general, has received very little attention. Apart from akinetic mutism, which has been recognized as a possible side effect of transcallosal approaches, information on movement disorders occurring after neurosurgical operations other than functional stereotactic procedures is only available from scattered case reports and incidental notations. There has been only one small previous series in three patients that focused on this subject.41

The purpose of this report is to document clinical and radiological features in a larger series of patients who suffered postoperative movement disorders. These movement disorders often became manifest only after a delay of several weeks or even months. We think that this postoperative side effect often may not be appreciated within the appropriate context and that it has been underreported so far.

Clinical Material and Methods

We present the cases of 14 patients who developed movement disorders such as tremor, dystonia, hemichorea–hemiballism, and myokymia following neurosurgical operations for various cerebral and spinal pathological disorders. Involuntary movements occurring after functional stereotactic surgery were not considered for this report nor were movement disorders that were most likely related to complications of anesthesia. The patients described here were seen over a period of 12 years, from 1986 to 1998, at different institutions. Four patients were referred to one of the authors (J.K.K.) for consultation with regard to further treatment of the movement disorder or for other reasons. None of the patients had been surgically treated by any of the authors of this article.
Only patients with a conclusive cause-and-effect relationship were accepted in this series. Data were collected from the patients’ charts and clinical notes. Whenever possible, the movement disorder was documented by videotaping. Follow-up interviews were obtained in 13 instances. Follow-up periods ranged from 3 months to 11 years. Functional disability at the last follow-up examination was rated according to the Karnofsky Performance Scale.22 One patient was reported previously in a case report.4

**Results**

There were five male and nine female patients in the study population. The patients’ ages at operation ranged from 13 to 71 years; the mean age at surgery was 44 years. Twelve patients underwent surgery for cerebral diseases and two patients for spinal diseases (Table 1). The patients with cerebral disorders suffered from a variety of diseases: nine patients had tumors and three patients had neurovascular disorders. The supratentorial compartment was involved in eight patients and the infratentorial compartment in four patients. The basal ganglia were infiltrated by tumor in three cases. Of the two patients with spinal processes, one patient harbored a cervicothoracic ependymoma and the other patient suffered from multiple cervical disc herniations.

Only five patients underwent suboccipital approaches. In six of the nine patients with cerebral tumors, complete tumor removal was achieved. In none of the patients who had tumors invading the basal ganglia was complete tumor resection intended. Remarkably, in 12 of the 14 patients side effects appeared immediately after the operation including hemiparesis (six patients), appendicular ataxia (three patients), somnolence (two patients), and facial palsy (one patient). Movement disorders that were observed included hemidystonia or hemichoreoathetosis (eight patients), unilateral tremors (three patients), unilateral facial myokymia (one patient), and hemichorea–hemiballism (two patients). The movement disorders became manifest only after a delay in all but two patients (Table 1). The delay between surgery and manifestation of the movement disorder in the other patients ranged between 1 week and 1 year. The mean delay in patients with tremors was 5 weeks and the delay in patients with dystonia was 5.5 months. In three patients the causal relationship between the postoperative movement disorder and the previous operation had not been recognized initially and the patients had been supposed to suffer from psychogenic disorders.

One of the two patients with spinal disease developed paroxysmal kinesigenic dystonia of the right arm after a complicated course with reflex sympathetic dystrophy (RSD) following repeated cervical spine surgery for multiple disc herniations. In the other patient a dystonic post-
Scores ranged between 40 and 90. Medical treatment with examination, the patients’ Karnofsky Performance Scale noted over years. Patients were disabled to various degrees by the postoperative movement disorder. The functional impact was mild in three patients, moderate in four notables over several months. Patients improved after functional stereotactic surgery in the ventrolateral thalamus. During the follow-up period there was no recurrence of tumor in any patients who had undergone complete tumor removal, except in one instance (Case 10). In all three patients with astrocytomas invading the basal ganglia, however, tumor regrowth occurred after partial tumor removal. Notably, one of these patients suffered from marked long-term disability as a result of the postoperative movement disorder.

Discrete cerebral lesions responsible for the movement disorders were identified in all patients with cerebral disease for whom postoperative imaging studies were available (Table 2). Lesions in the contralateral caudate and/or putamen were found in the patients with dystonic movement disorders. Lesions of the ipsilateral superior cerebellar peduncle, cerebellar hemisphere, or dentate nucleus were seen in the patients with tremors. In the patient with transient right-sided hemichorea after partial removal of a contralateral parietal astrocytoma invading the basal ganglia was unaware of the movement disorder (Case 1). In the patient in Case 3, a 66-year-old woman who developed right-sided hemichorea–hemiballism, an ipsilateral intraventricular cyst was observed after removal of a meningioma of the lateral ventricle. Magnetic resonance (MR) imaging revealed distortion of the upper brainstem and mesencephalon, which was compatible with involvement of the contralateral subthalamic nucleus. The movement disorder disappeared after operative revision and drainage of the cyst.

Movement disorders were persistent in 11 patients at a mean follow-up period of 5 years (Table 2). They were noted, in general, to be progressive over several months. In individual patients increases in severity were even noted over years. Patients were disabled to various degrees by the postoperative movement disorder. The functional impact was mild in three patients, moderate in four patients, and marked in four patients. At the last follow-up examination, the patients’ Karnofsky Performance Scale scores ranged between 40 and 90. Medical treatment with a variety of drugs rendered only limited benefit. Two patients experienced transient improvement after botulinum toxin injections. The hemidystonia in the patient in Case 9 improved after functional stereotactic surgery in the ventrolateral thalamus. During the follow-up period there was no recurrence of tumor in any patients who had undergone complete tumor removal, except in one instance (Case 10). In all three patients with astrocytomas invading the basal ganglia, however, tumor regrowth occurred after partial tumor removal. Notably, one of these patients suffered from marked long-term disability as a result of the postoperative movement disorder.

Discrete cerebral lesions responsible for the movement disorders were identified in all patients with cerebral disease for whom postoperative imaging studies were available (Table 2). Lesions in the contralateral caudate and/or putamen were found in the patients with dystonic movement disorders. Lesions of the ipsilateral superior cerebellar peduncle, cerebellar hemisphere, or dentate nucleus were seen in the patients with tremors. In the patient with unilateral facial myokymia, an ipsilateral pontine lesion was identified.

**Table 2**

**Follow-up data obtained in 14 patients with movement disorders after neurological surgery**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex†</th>
<th>Movement Disorder</th>
<th>Imaging Findings</th>
<th>Duration of Movement Disorder</th>
<th>Disability</th>
<th>Outcome &amp; Follow Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>38, F</td>
<td>rt hemichorea (arm &gt; leg)</td>
<td>cavity extending into lt posterior putamen/pallidum</td>
<td>transient (3 days), neglect for movement disorder</td>
<td>mild</td>
<td>alive, KPS 90 (2 yrs), growth of residual tumor</td>
</tr>
<tr>
<td>2</td>
<td>40, M</td>
<td>lt hemichorea–athetosis</td>
<td>cavity extending into rt posterior putamen</td>
<td>transient (2 wks)</td>
<td>mild</td>
<td>alive, KPS 80 (3 yrs), growth of residual tumor</td>
</tr>
<tr>
<td>3</td>
<td>66, F</td>
<td>rt hemichorea–hemiballism</td>
<td>cyst in rt lat ventricle w/ edema &amp; brainstem compression</td>
<td>transient (3 wks), disappearance after cyst drainage</td>
<td>marked</td>
<td>alive, KPS 90 (3 yrs)</td>
</tr>
<tr>
<td>4</td>
<td>21, M</td>
<td>lt myoclonic hemidystonia</td>
<td>cavity at site of AVM including rt anterior striatum</td>
<td>persistent (increase over 6 yrs)</td>
<td>marked</td>
<td>alive, KPS 50 (6.5 yrs), AVM cured after radiosurgery</td>
</tr>
<tr>
<td>5</td>
<td>68, F</td>
<td>rt intention tremor</td>
<td>rt cerebellar defect extending into dentate nucleus &amp; peduncles</td>
<td>persistent (5 yrs)</td>
<td>mild</td>
<td>alive, KPS 70 (3 yrs)</td>
</tr>
<tr>
<td>6</td>
<td>37, M</td>
<td>lt myoclonic hemidystonia</td>
<td>cavity extending into rt posterior putamen</td>
<td>persistent (2.5 yrs)</td>
<td>marked</td>
<td>alive, KPS 60 (3 yrs)</td>
</tr>
<tr>
<td>7</td>
<td>52, F</td>
<td>lt kinetic tremor</td>
<td>defect in lt floor of 4th ventricle &amp; cerebellar peduncles</td>
<td>persistent (3 yrs), spontaneous improvement</td>
<td>mod</td>
<td>alive, KPS 60 (3 yrs)</td>
</tr>
<tr>
<td>8</td>
<td>39, F</td>
<td>lt kinetic tremor</td>
<td>lesion of lt cerebellar hemisphere &amp; superior cerebellar peduncle</td>
<td>persistent (5 yrs)</td>
<td>mod</td>
<td>alive, KPS 70 (5 yrs)</td>
</tr>
<tr>
<td>9</td>
<td>59, M</td>
<td>rt hemidystonia</td>
<td>hypodensity at lt putamen (CT scan)</td>
<td>persistent (10 yrs), improvement after thalamotomy</td>
<td>marked</td>
<td>alive, KPS 60 (11 yrs)</td>
</tr>
<tr>
<td>10</td>
<td>31, F</td>
<td>lt facial myokymia</td>
<td>defect of lt floor of 4th ventricle</td>
<td>persistent (6 yrs)</td>
<td>mild</td>
<td>alive, KPS 90 (7 yrs), tumor recurrence at 4 yrs</td>
</tr>
<tr>
<td>11</td>
<td>71, M</td>
<td>lt hemichorea–athetosis</td>
<td>hypodensity at rt caudate (CT scan)</td>
<td>persistent (3 mos)</td>
<td>mod</td>
<td>alive, KPS 70 (3 mos)</td>
</tr>
<tr>
<td>12</td>
<td>13, F</td>
<td>lt hemidystonia</td>
<td>NA</td>
<td>persistent (1 yr)</td>
<td>mod</td>
<td>lost to follow up</td>
</tr>
<tr>
<td>13</td>
<td>53, F</td>
<td>rt hand dystonic posture</td>
<td>atrophy of cervicothoracic medulla</td>
<td>persistent (6 yrs)</td>
<td>mild</td>
<td>alive, KPS 90 (6 yrs)</td>
</tr>
<tr>
<td>14</td>
<td>33, F</td>
<td>rt paroxysmal kinesigenic dystonia (arm)</td>
<td>no abnormalities of basal ganglia on MR imaging</td>
<td>persistent (4 yrs)</td>
<td>marked</td>
<td>alive, KPS 50 (4 yrs)</td>
</tr>
</tbody>
</table>

* KPS = Karnofsky performance scale score; mod = moderate; NA = not available.
† Age, in years, at operation.

**Illustrative Cases**

**Transient Movement Disorder After Brain Surgery**

**Case 1.** This 38-year-old woman had mild problems with short-term memory that increased over 6 months. She then developed a mild right-sided weakness. Computerized tomography (CT) scanning revealed a tumor in the left frontoparietal white matter extending to the posterior putamen/pallidum and the internal capsule (Fig. 1 left and center). The tumor was slightly hyperdense and had a...
diameter of approximately 4 cm. There was mild peri- 

tumoral edema and a slight midline shift to the right side. 

The neurological examination showed mild right-sided 
hemihypesthesia and hemiparesis. The tumor was re- 
moved via a parietal postcentral approach with the aid of 
intraoperative ultrasound imaging. Computerized tomog- 

raphy scans obtained on the 1st postoperative day con- 
firmed subtotal removal of the tumor including the portion 
located in the posterior putamen/pallidum (Fig. 1 right). 

Histopathological studies revealed a World Health Organ- 

ization Grade II protoplasmatic astrocytoma.

Postoperatively, the patient’s hemiparesis was un- 
changed. On the 2nd postoperative day, choreic move- 

ments of the right extremities appeared. The patient was 
completely unaware of the dyskinesias. Further examina- 

tion showed she had neglect for the movement disorder. 

The involuntary movements subsided spontaneously 3 

days later. They did not occur during follow-up exami- 
nations performed during the next 2 years or on regrowth of 

the residual tumor.

Persistent Movement Disorder After Brain Surgery

Case 4. This 21-year-old man was admitted for treat- 

ment of a large frontotemporal arteriovenous malfor- 
mation (AVM). He had a 5-year history of intermittent 

headaches. His neurological examination yielded normal 
results. Computerized tomography scans revealed an 

AVM in the right frontotemporal region extending medi- 

ally just to the caudate and the anterior putamen (Fig. 2 

upper left and upper center). Magnetic resonance imaging 
demonstrated involvement of the anterior insula. The 

maximum diameter of the AVM was approximately 5 cm. 

There was very little mass effect. Cerebral angiography 

revealed that the AVM was fed mainly by branches of the 

right opercular and anterior cerebral arteries and by two 
enlarged lenticulostriate arteries. Venous drainage ran into 

the superior sagittal sinus and the basal and internal cere- 

bral veins. Several feeding arteries were embolized using 

histoacryl via a transfemoral approach in five subsequent 

sessions. It was estimated that approximately 50% of the 

blood flow through the AVM had been reduced. Because 

further endovascular interventions were considered too 

risky the patient was scheduled for operative removal of 

the vascular tumor. Surgery was planned to remove the lat- 
lateral portion, but to leave the portion extending to the ante- 
rior basal ganglia for radiosurgical treatment if possible. 
The AVM was approached via a right pterional approach. 
The lateral portion of the AVM was resected after isola- 
tion of the feeding arteries. Because there were consider-
able difficulties obtaining hemostasis, it was also neces- 
sary to remove medial portions of the mass that were 
supplied by a lenticulostriate artery. Postoperatively, the 
patient experienced left-sided hemiparesis. Computerized 
tomography scans showed bleeding in the region of the 
head of the caudate and in the right lateral ventricle (Fig. 2 

upper right and lower left). The paresis resolved almost 
completely over the next 10 days. Cerebral angiography 

performed 1 month after the operation revealed small 

remnants of the AVM in the right hippocampus, the insu- 
lar subcortex, and the gyrus rectus. To control the residual 

AVM the patient was scheduled for stereotactic radio- 
surgery 8 months postoperatively. Stereotactic angiogra- 

phy performed at that time showed a small nidus in the 

right gyrus rectus, which was treated by radiosurgery.

At the time of the radiosurgery a mild dystonic posture 
and jerking movements of the left hand were described. 

Approximately 1 month later, the patient reported invol- 
untary rotation of his head to the left side. Over the next 

few months, the jerking and the dystonic posture in- 
creased markedly. Eighteen months postoperatively, the 
patient presented with cervical dystonia and a left-sided 
myoclonic hemidystonia. Initially, the movement disorder 
was thought to be psychogenic and the patient underwent 

a variety of psychotherapeutic therapies. Medical treat- 

ment with administration of trihexyphenidyl, benzodiaz- 
epines, and baclofen did not result in amelioration of the 
dystonia. The cervical dystonia was treated successfully 

by repeated injections of botulinum toxin. The hemidys- 
tonia progressed slowly. There were no AVM remnants 

on cerebral angiography. Five years postoperatively, CT 
scans demonstrated a cavity at the site of the previous 

AVM that communicated with the anterior horn of the 

right lateral ventricle (Fig. 2 lower right). The defect

![Fig. 1. Case 1. Computerized tomography studies obtained in a woman with transient right-sided hemichorea after she underwent partial removal of a left protoplasmatic astrocytoma when she was 38 years of age. Left and Center: Preoperative scans revealing the tumor in the left frontoparietal white matter extending into the posterior putamen/pallidum and the internal capsule. Right: Postoperative scan obtained after subtotal tumor removal demonstrating a defect extending into the posterior putamen/pallidum.](image-url)
included the head of the caudate and the anterior putamen. A functional stereotactic operation was suggested.

Case 6. This 37-year-old man had an unremarkable medical history until he suffered a series of tonic-clonic seizures. Both CT and MR imaging studies showed a right-sided insular tumor extending into the frontal and temporal white matter and medially into the posterior limb of the internal capsule and the putamen. The patient’s neurological examination yielded normal results. The tumor was partially removed via a frontotemporal approach. The neuropathological report provided the description of a World Health Organization Grade II fibrillary astrocytoma. Postoperatively, the patient exhibited a marked left-sided hemiparesis. The weakness improved over the next few months to the extent that he was able to ambulate without help and to hold things with his left hand.

Parallel to the improvement of the hemiparesis was the development of a left-sided hemidystonia that was accompanied by jerking movements. The patient was severely impaired by the movement disorder, which did not improve in response to a variety of medications. Magnetic resonance imaging studies performed 16 months postoperatively demonstrated a large cavity in the temporal lobe extending into the posterior ventral putamen (Fig. 3). Adjacent to the defect, a residual tumor was found in the frontal white matter invading the basal ganglia. A routine repeated MR study performed 1 year later revealed growth of the residual tumor, which now extended to the thalamus. The patient then underwent percutaneous radiotherapy. Six months later there was an increase of his hemiparesis. The patient refused further diagnostic evaluations and treatment.

Case 7. This 52-year-old woman suffered from recurrent paresthesias of the right hemibody for several years. Magnetic resonance imaging revealed a cavernoma located in the left floor of the fourth ventricle and in the adjacent left middle cerebellar peduncle. The results of the patient’s neurological examination revealed mild ataxia and dysmetria of the left arm and a tendency to fall to the left when standing with her eyes closed. The cavernoma was removed via a suboccipital transventricular approach. Postoperatively, the patient experienced right-sided hemihypesthesia, a left-sided incomplete facial palsy, and a gait disturbance.

Over the next few days the patient’s truncal and appendicular ataxia increased and a kinetic low-frequency tremor of the left arm developed. The patient was referred to a neurorehabilitation program. At a follow-up visit 18 months later, her condition was improved. There was a persistent left-sided appendicular tremor and ataxia. The
the hand and fingers. The spasms occurred up to 20 times
the shoulder, extension of the elbow, and dorsal flexion of
right arm adopted a dystonic posture with abduction in
arm and by cold temperature. During these attacks the
voluntary spasms of her right arm. The dystonic spasms
were evoked by active forceful movements of her right
arm and C-6 was performed. As a result, the patient experi-
enced transient relief of her pain. Three months later, MR
imaging demonstrated persistent anterior compression of
the C-6 nerve root in the right foramen at C5–6. This time
the cervical disc at the level of C5–6 was removed via an
anterior approach and the segment was fused with autolo-
gous iliac crest. Postoperatively, the patient was
free from pain, which adopted a burning character
affecting her entire right arm as well as the upper lateral
wall of her right thorax. The results of the patient’s neuro-
logical examination revealed a ‘shoulder–arm syndrome’
with mild weakness of all muscles of the right arm dis-
tal to C-5. Sensory deficits, hyperhidrosis, and allodynia
were evident in the region in which the patient experi-
enced spontaneous pain. Repeated temperature measure-
ments showed that the right arm was considerably colder
than the left arm, with a mean difference of 8°C. A diag-
nosis of RSD was made. Anesthetic infiltration of the
right stellate ganglion resulted in temporary relief. The
pain was relieved to a certain extent after implantation of
a morphine pump. One year later, however, there was an
increase in the spontaneous pain and allodynia.

At the same time the patient developed paroxysmal in-
volutary spasms of her right arm. The dystonic spasms
were evoked by active forceful movements of her right
arm and by cold temperature. During these attacks the
right arm adopted a dystonic posture with abduction in
the shoulder, extension of the elbow, and dorsal flexion of
the hand and fingers. The spasms occurred up to 20 times
daily and had a variable duration, lasting from 2 minutes
to approximately half an hour. There was no change in
the paroxysmal kinesiogenic dystonia over the next years.
Medication with a variety of drugs was ineffective. Mag-
netic resonance imaging studies of the brain were unre-
markable.

**Discussion**

Movement disorders such as dystonia, chorea, tremors,
and hypokinetic syndromes may be secondary to a variety
of causative factors including neurodegenerative disor-
ders, vascular events, metabolic diseases, infections, tra-
uma, and mass lesions.8,10,24,25,27–34,37,38 It is well known that
movement disorders may also occur after functional ste-
reotactic procedures. Hemichorea or hemiballism sec-
ondary to thalamotomy was a common problem in the
1960s, during which it was reported to occur in 0.3 to 9%
of patients in the early postoperative period.39,40 In rare
cases such movement disorders were persistent over years
and were a source of additional disability for the patient.
Movement disorders resulting from neurosurgical opera-
tions other than functional stereotactic procedures, how-
ever, have received much less attention. In this regard it
is important to rule out the existence of these movement
disorders preoperatively. We have found an incidence of
tumor-induced parkinsonism in 0.5% of patients with sup-
ratentorial tumors.28 In patients with thalamic and basal
ganglia astrocytomas tumor-induced movement disorders
were present in 9%.29

The frequency of postoperative movement disorders
remains unclear. It is not possible to estimate their occur-
rence based on data available at this time. The patients
described in this report were surgically treated in different
hospitals and some were referred because of their move-
ment disorder. We think that the incidence of postope-
rative movement disorders may be higher than one could
infer from previous scattered reports. Such movement dis-
orders may not be recognized for several reasons. The
delay between the causative event and the appearance of
such movement disorders can be quite variable. In this
series the delay of onset was up to 1 year in individual
patients. Similar delayed onset have been observed in
patients with movement disorders following head in-
jury.13,17,21 We have found a delay of onset ranging from 2
weeks to 6 months in patients with posttraumatic tremor
and from 2 months to 2 years in patients with posttrau-
matic dystonia.29 Patients with posttraumatic and postop-
erative movement disorders, however, differ markedly: in
general, after head injury patients often suffer from mul-
ple lesions and have more widespread cerebral damage,
whereas patients with postoperative movement disorders
harbor more circumscribed lesions.

Diagnostic accuracy is another problem in patients with
postoperative movement disorders. Frequently, dystonia is
incorrectly labeled as spasticity and intention or kinetic
tremors are mistaken for appendicular ataxia. In other
cases the organic nature of the movement disorder is sim-
ply overlooked by physicians who are inexperienced with
such syndromes. As occurred in three of our cases, the
movement disorder might have been thought to be psy-
chogenic, resulting in unnecessary and costly therapeutic
interventions.

**Persistent Movement Disorder After Spinal Surgery**

Case 14. This 38-year-old woman was referred for fur-
ther evaluation of severe pain and a paroxysmal dystonic
movement disorder of her right arm. At age 33 years, she
had noted radiating pain and mild weakness of her right
arm. Myelography revealed disc herniations at C-3–4 and
C-5–6 on her right side. She underwent right-sided fo-
raminotomies at both levels via a dorsal approach. Post-
operatively, increased weakness of her right arm was evi-
dent and an operative revision with laminectomies of C-4
and C-5 was performed. As a result, the patient experi-
enced transient relief of her pain. Three months later, MR
images revealed a circumscribed defect in the floor of
the left fourth ventricle and the adjacent middle and supe-
rior cerebellar peduncles (Fig. 4).
The most frequently observed movement disorders in our series were dystonic syndromes and tremors. Few instances of dystonic movement disorders and tremors secondary to craniocerebral surgery were reported previously. Postoperative movement disorders other than tremor and dystonia appear to be exceptional and include unilateral facial myokymia (this series), hemichorea–hemiballism (this series and others), parkinsonian syndromes, palatal myoclonus, segmental branchial and spinal myoclonus, and clonic perseveration. In addition, dystonia, tremor, and myoclonus rarely have been described as sequelae of spinal surgery.

The dystonic movement disorders secondary to craniocerebral surgery in our series were associated with lesions of the caudate and/or the putamen. This is in accordance with previous reports on pathoanatomical correlations in secondary dystonia. With regard to the currently accepted model of basal ganglia function, it is thought that dystonia in such cases results from abnormal activity in the pallidothalamicocortical loop as a consequence of striatal lesions. Other mechanisms are involved in the dystonic movement disorders in our two patients after spinal surgery. Paroxysmal kinesigenic dystonia is an unusual movement disorder that can be induced by sudden movements. Sporadic cases of this movement disorder are rare and the underlying mechanisms have not been elucidated so far. In our case it is most likely that the movement disorder was precipitated by the postoperative RSD. Dystonic postures and tremors have been observed occasionally after RSD. It has been postulated that such movement disorders might be related to altered input to the basal ganglia from the periphery, possibly associated with subcortical reorganization in predisposed patients. One patient with familial paroxysmal nonekinesigenic dystonia was previously reported to have developed persistent dystonia after laminectomy. The dystonic hand posture observed after removal of the cervicothoracic intramedullary ependymoma in the patient in Case 13 may present a case of “spinal pseudoathetosis.” The distinction between spinal pseudoathetosis and dystonia has been considered to be chiefly semantic. “Pseudoathetosis” is associated with a marked loss of proprioception, which has been postulated to follow disinhibition at a higher level of motor processing. There is a report of a previous case in which pseudoathetosis was found after removal of a cervical low-grade glioma and cavernoma.

Kinetic or intention tremors in our series were associated with lesions of the cerebellar hemisphere, the dentate nucleus, and the superior cerebellar peduncle. Similar tremors are also observed in patients with diffuse axonal injury. Because such lesions concern the predecussational dentatothalamic pathway, the tremor is ipsilateral to the side of the lesion. More rarely such tremors may also be observed with contralateral midbrain lesions when the postdecussational dentatothalamic pathway is involved. Kinetic tremors can be very disabling because they interfere with virtually any daily routine activities. In one patient paroxysmal jerking of the right arm and trunk at a frequency of 6.1 Hz was described after excision of a right cerebellar hemangioblastoma. Remarkably, the postural axial tremor in this patient developed only after a delay of 7 years. An area of hyperintense signal in the left ventral upper medulla was interpreted as compatible with secondary olivary degeneration. Recently, a patient was reported to have presented with palatal myoclonus after removal of a cerebellar low-grade astrocytoma. In this case the olivary pseudohypertrophy demonstrated by MR imaging was also thought to be secondary to denervation of the dentatothiery nuclear loop.

Several mechanisms can be involved in the pathogenesis of postoperative movement disorders. Most frequently direct lesioning of structures such as the striatum or the cerebellar outflow pathways is the responsible cause in patients with intraaxial or extraxial tumors. Lesioning of the substantia nigra by the catheter tip of an Ommaya reservoir resulting in subsequent contralateral hemiparkinsonism has been reported in a 28-year-old woman. Ischemia secondary to intraoperative occlusion of vessels is another mechanism that can be relevant. It can be the primary cause in patients in whom movement disorders develop after clipping of aneurysms. Treatment with diphenylhydantoin may also contribute to the development of movement disorders. Structural cerebral lesions were identified in several patients who presented with movement disorders while receiving diphenylhydantoin. In particular, chronic movement disorders were described more frequently in such patients.

The question arises as to how the postoperative occurrence of movement disorders can be avoided. Although postoperative movement disorders are transient and only mildly disabling in some patients, they may be the source of marked disability and interfere with daily activity in other patients. In this regard it is important to note that basal ganglia lesions that appear to be almost identical may result in severely disabling movement disorders in one patient and an unremarkable neurological status in another. Definitely, postoperative movement disorders were “unexpected” sequelae in the majority of cases in this series. In other instances, however, the use of preoperative planning that takes into account the possibility of such side effects might have prevented their occurrence. In patients with low-grade gliomas invading the basal ganglia, for example, it might be feasible not to attack the latter portions, in particular when complete tumor removal will not be achieved anyway.
Conclusions

Our series demonstrates that movement disorders can be the consequence of craniocerebral and spinal surgeries other than functional neurosurgical procedures in patients with various underlying pathological disorders. Postoperative movement disorders may be a source of considerable disability in individual patients. Increased awareness of this possible complication could result in its more frequent diagnosis. In some patients its occurrence can be avoided by using appropriate planning strategies.

References


Manuscript received July 27, 1998.
Accepted in final form November 5, 1998.
Address reprint requests to: Joachim K. Krauss, M.D., Department Neurochirurgie, Inselspital, University of Berne, 3010 Berne, Switzerland. email: jkrauss@insel.ch.

890
J. Neurosurg. / Volume 90 / May, 1999