QOL between tremor patients and nontremor patients or with respect to tremor severity or aggregate score.

**DISCUSSION**

In this population-based study, the prevalence of tremor in the Olmsted County population was 26%, although severe tremor was only found in 3%. These population-based estimates are lower than those found in an MS clinic. Estimates of tremor based on clinic studies are highly variable. Alusi and colleagues detected tremor in 58% of MS patients randomly selected from an MS unit in London. Tremor severity ranged from minimal in 27%, mild in 16%, and moderate to severe in 15% of cases. Clinic-based studies are accepted to be biased toward more disabled patients.

In our study, we found that tremor in MS is associated with greater impairment, disability, and handicap and occurs more frequently in patients with SPMS. In addition, no patients with severe tremor were employed and none were driving a car. Patients with tremor of any severity were more likely to be retired early on disability (41% vs. 15%) and less likely to be employed (40% vs. 68%), despite their younger age.

In a population-based study of quality of life in MS in Olmsted County, we recently found that most patients with MS were satisfied or better with their qualities of life and the correlation of QOL with disability was less then expected. Similarly in this study, patients with tremor scored themselves the same as to those without tremor on a simple QOL scale of between 0 and 10.

In conclusion, severe tremor is a relatively infrequent complication of MS when examined in a community-based cohort. When analyzed on a population basis, tremor correlated with EDSS, ISS, and ESS, which are global measures of impairment, disability, and handicap, respectively.

**REFERENCES**


**Neuronal Activity in the Globus Pallidus of Multiple System Atrophy Patients**

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Abstract: The pathophysiological changes in neural activity that characterize multiple system atrophy (MSA) are largely unknown. We recorded the activity of pallidal neurons in 3 patients with clinical and radiological features of MSA who underwent unilateral microelectrode-guided pallidotomy for disabling parkinsonism. Findings in these patients were compared with 4 control patients with a clinical diagnosis of Parkinson’s disease (PD). The position, firing rates, and firing patterns of single neurons in the pallidal complex were analyzed in both MSA and PD patients. The mean spontaneous firing rate of neurons in the internal segment of the globus pallidus internus (GPi) was significantly lower in MSA than in PD patients. There were no significant differences between MSA and PD patients, however, in firing rates of neurons in the external globus pallidus (GPe) or in the external segment of GPi (GPIe). In

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addition, no significant differences in firing pattern were found between MSA and PD patients. In conclusion, this study has shown that firing rates of neurons in GPe but not in GPi were different in MSA patients compared with that in PD patients, a finding that may reflect the poor clinical results of pallidotomy reported in patients with MSA.

Key words: MSA; PD; BG; globus pallidus; recordings

Approximately 20% of patients with a clinical diagnosis of Parkinson’s disease (PD) may have neuropathological evidence of alternative causes of parkinsonism. Multiple system atrophy (MSA), once considered a rare disease, is now known to account for up to 10% of patients with parkinsonism. MSA includes adult-onset sporadic olivopontocerebellar atrophy (OPCA), Shy-Drager syndrome (SDS), and striatoniigral degeneration (SNd). Incidence rates for MSA are not well known and may range around 3 new cases per 100,000 persons per year for ages 50 to 99 years. MSA is generally characterized by progressive autonomic failure/urinary dysfunction with cerebellar, pyramidal, and extrapyramidal signs, but the various clinical features of MSA make the diagnosis of the disease difficult, especially in its early stages when signs of the dysfunction of the different systems involved have not yet appeared. A large proportion of patients with MSA present with and have a clinical course dominated by parkinsonism that is often poorly responsive to antiparkinsonian drugs.

Due to uncertainties in clinical diagnosis of degenerative atypical parkinsonism, it is believed that a significant proportion of patients undergoing functional neurosurgical procedures, including unilateral posteroventral pallidotomies (PVP), may have MSA or other neurodegenerative disorders. Posteroventral pallidotomy can result in striking beneficial effects to all aspects of PD but preliminary reports suggest that it usually has a limited benefit in patients with atypical parkinsonism who do not respond to levodopa (L-dopa) therapy. We studied the physiological attributes of pallidal complex neurons in MSA and PD patients to determine if the apparent differences in outcome after pallidotomy in these two conditions correlate with differences in neuronal activity. We carried out a retrospective study of the single-unit microelectrode recordings of 3 patients with clinical and radiological diagnosis of MSA and 4 patients with PD who underwent PVP at the Toronto Western Hospital. The data were compared with the literature and with current models of movement disorders.

SUBJECTS AND METHODS

Data were obtained from 3 MSA patients undergoing unilateral microelectrode-guided PVP at Toronto Western Hospital, University of Toronto between August 1992 and June 1993. These pallidotomies, among the earliest carried out by our group, were done to determine whether this procedure was effective for MSA. The poor clinical outcome has been reported previously. The 3 MSA patients had prominent L-dopa-resistant parkinsonism (the so-called striatoniigral form, MSA-p). One also had disabling dysautonomia (urinary incontinence and symptomatic orthostatic hypotension), encouraging the designation of SDS. All patients had magnetic resonance imaging (MRI) before surgery. An abnormal decrease in signal intensity of the putamen, particularly along the lateral and posterior portions, and some degree of striatal atrophy was evident in all MSA patients. For the comparative study, we analyzed the data obtained from 4 patients with clinical features of PD undergoing unilateral PVP between February and April 1994. PD patients from whom data had been used in previous publications were not included. Table 1 provides details on the patients studied.

Surgical Procedure and Single-Unit Microelectrode Recording

Each patient underwent a standard microelectrode-guided PVP, as outlined previously. Briefly, a Leksell stereotactic frame was applied to the patient’s head and the coordinates of the anterior and posterior commissures (AC and PC, respectively) determined by MRI. Standard stereotactically sagittal maps (20 mm lateral to the midline) based on the Schaltenbrand and Wahren atlas were generated, and scaled according to the length of the patient’s AC-PC distance. The target selected for all 7 patients was the most posteroventral part of the internal globus pallidus (GPi). An electrode guide tube was stereotactically inserted into the brain and a microelectrode was advanced to the target by means of a hydraulic microdrive. All patients were operated on with local anesthesia after overnight withdrawal of their antiparkinsonian medication.

Microelectrode recording usually began within the Globus pallidus externus (GPe), 15 or 20 mm above the base of the internal segment of the globus pallidus. Recordings were carried out in trajectories directed from anterodorsal to posteroventral, and microstimulation (300 Hz, 0.2-msec pulse width, 100 μA) was carried out at regular intervals (1–2 mm) from the target. Platinum-plated tungsten microelectrodes (0.1–1.0 MΩ impedance) were used. Neuronal signals were amplified, filtered, displayed on oscilloscopes, and stored on tape for offline analysis. Units were recorded with the patients at rest. Multiple tracks (one to five) were made along similar trajectory angles, allowing for medial-lateral and AP
adjustments. The AP distance between tracks was 2 or 3
mm.

Data Analysis

The data were analyzed offline using the CED 1401-
plus data acquisition system and the Spike2 analysis
package (Cambridge Electronics Devices, Cambridge,
UK).

For the quantitative calculations, only neurons that
could be discriminated clearly were included. The loca-
tions of the cells were determined from reconstructions
of electrode tracks superimposed on the stereotactic atlas
diagram of the globus pallidus 20 mm lateral to the
midline. These diagrams were scaled such that the pa-
tient’s AC and PC coordinates were superimposable on
those in the standard atlas. In addition, the electrode
track was adjusted if necessary so that the major physi-
ologically determined landmarks corresponded with the
appropriate nuclear and fiber track regions. The main
landmarks utilized were: (1) the location of the top of the
optic tract (OT) as determined from microstimulation-
induced visual percepts or recordings of background
optic tract neuronal responses to flashing light in the
patient’s eye; (2) the location of the internal capsule (IC)
at the posterior border of the pallidum as determined
from lack of neuronal activity (the microelectrodes we
use rarely record axonal action potentials) in conjunc-
tion with microstimulation-induced motor or sensory (pa-
resisphagia) responses; and (3) the presence of “border cells”
and locations of regions devoid of cellular activity as
described previously, which provided additional infor-
mation on the locations of the laminae and borders of the
pallidum and its subnuclei. Only trajectories that could
be localized adequately were included in the study. The
neurons in the GP were subdivided into: (1) those pre-
sumed to be located in the ventral or inner portion GPi
(GPii) based on being approximately 0 to 8 mm above
the dorsal border of the OT and inferior to a silent zone
or border cells, (2) those presumed to be located in the
external portion of the GPi (GPie) lying superior to GPii
and inferior to the border with GPe; and (3) those pre-
sumed to be in GPe, located more than approximately 12
mm above the OT and superior to a lamina characterized
by a zone with no cellular activity or some border cells.

To assess the firing patterns of the neurons we used
three different methods: kurtosis, skewness, and bursting
index. Kurtosis and skewness are standard descriptive
statistics that can be used to describe the shape of the
interspike interval histogram. Positive kurtosis indicates
a relatively peaked distribution. The “burst index” used
in this study was the ratio of the mean to modal inter-

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**TABLE 1. Clinical characteristics for each patient**

<table>
<thead>
<tr>
<th>Patient no./</th>
<th>Age (yr)</th>
<th>Symptom duration (yr)</th>
<th>Major symptoms/signs*</th>
<th>Drugs**</th>
<th>l-Dopa response***</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 MSA</td>
<td>40</td>
<td>6</td>
<td>R, hand, leg stiffness; sustained neck flexion; cogwheel rigidity, 4 limbs; swallowing/ speech difficulties</td>
<td>l-Dopa (500 mg) Bromocriptine (37.5 mg) Selegiline (10 mg) Domperidone (40 mg)</td>
<td>Limited</td>
</tr>
<tr>
<td>2 MSA</td>
<td>60</td>
<td>6</td>
<td>R arm bradykinesia; bilateral leg “weakness;” bradykinesia/akinesia, 4 limbs; resting tremor, limb ataxia; dysarthria</td>
<td>l-Dopa (750 mg) Bromocriptine (8 mg)</td>
<td>Limited</td>
</tr>
<tr>
<td>3 MSA</td>
<td>47</td>
<td>4</td>
<td>“Weakness,” bladder difficulty; fainting, autonomic dysfunction; bradykinesia, 4 limbs; rigidity, postural tremor dysarthria, dysphagia</td>
<td>Oxazepam (20 mg) Diazepam (2.5 mg)</td>
<td>Not effective</td>
</tr>
<tr>
<td>4 PD</td>
<td>64</td>
<td>14</td>
<td>Dyskinesias (severe); bradykinesia; resting tremor</td>
<td>l-Dopa (1.150 mg) Pergolide (7 mg)</td>
<td>Effective</td>
</tr>
<tr>
<td>5 PD</td>
<td>42</td>
<td>21</td>
<td>L hand resting tremor; L side dyskinesias (severe); gait instability; depression</td>
<td>l-Dopa (600 mg) Pergolide (1.5 mg) Selegiline (15 mg)</td>
<td>Effective</td>
</tr>
<tr>
<td>6 PD</td>
<td>51</td>
<td>15</td>
<td>Resting tremor; rigidity, bradykinesia; severe dyskinesias; postural instability</td>
<td>l-Dopa (1,200 mg) Bromocriptine (20 mg) Amantadine (200 mg) Selegiline (10 mg) l-Dopa (1,200 mg) Pergolide (2.6 mg)</td>
<td>Effective</td>
</tr>
<tr>
<td>7 PD</td>
<td>68</td>
<td>18</td>
<td>L hand resting tremor; rigidity, bradykinesia; L sided dyskinesias</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Listed by symptom chronology.
**Medication taken at baseline before undergoing surgery; l-Dopa given with a decarboxylase inhibitor (either carbidopa or benserazide).
***l-Dopa responses: Limited, <30% reduction in signs of parkinsonism; Effective, >30% reduction (typically >50%) in signs of parkinsonism.
L-Dopa, levodopa; MSA, multiple system atrophy; PD, Parkinson’s disease; R, right; L, left.

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spike interval and has been used previously to compare firing patterns of pallidal neurons. Analysis of variance (ANOVA) was used to determine the level of significance ($P < 0.05$) when comparing findings across and within patient groups.

**RESULTS**

**Identification of the Globus Pallidus and Cell Distribution**

In total, 61 cells distributed along 7 tracks (8.7 cells/track) in MSA patients and 129 cells distributed along 14 tracks (9.2 cells/track) in PD patients were studied. All cells included in this study fulfilled the inclusion criteria outlined above. The mean ($\pm$ standard deviation [SD]) duration of the recordings used for firing rate determination for each cell was $20 \pm 18$ seconds in MSA and $44 \pm 50$ seconds in PD.

**Firing Frequencies**

The mean firing rate of GPi neurons in MSA patients was $60.7 \pm 37.7$ Hz and was significantly lower than that in PD patients ($81.6 \pm 44.9$ Hz) ($P < 0.005$) (Mann–Whitney rank sum test). Figure 1 plots the mean firing rate of neurons recorded in 1-mm intervals as a function of the location above the physiologically verified superior border of the optic tract for each group of patients. This plot demonstrates that the major differences in firing rate between the two patient groups occurred in the ventral part of GPi. This was confirmed by comparing the firing rates of neurons in GPii, GPie, and GPe, shown in Figure 2. The mean firing rate of neurons in the GPii in MSA patients was significantly lower than that in PD patients ($48.9 \pm 32.1$ MSA vs. $94.5 \pm 48.7$ PD; $P < 0.05$, one-way ANOVA and Tukey’s test). There were no significant differences, however, between firing rates of neurons in GPie and in GPe in MSA compared with that in PD patients (GPie, $74.0 \pm 39.7$ in MSA vs. $63.0 \pm 31.1$ in PD patients; GPe, $55.4 \pm 61.4$ in MSA vs. $54.2 \pm 38.8$ in PD patients; one-way ANOVA and Tukey’s test) (Fig. 2). The neurons in the GPii of PD patients fired at a significantly higher rate than did neurons in the GPe ($P < 0.05$), but this did not hold true for neurons in MSA patients (one-way ANOVA and Tukey’s test) (Fig. 2). The differences between MSA and PD patients in GPii are also apparent when comparing the distribution of cells according to their mean firing rate, which showed that there are many more neurons with low firing rates in MSA patients.

There were no significant differences between the firing patterns of neurons in MSA and PD patients based on the three indices: kurtosis ($37.0 \pm 42.5$ MSA vs. $43.3 \pm 75.8$ PD), skewness ($3.4 \pm 2.4$ MSA vs. $4.1 \pm 2.9$ PD), and burst index ($3.9 \pm 7.0$ MSA vs. $3.7 \pm 15.0$ PD). However, in PD patients the mean kurtosis was found to be higher for neurons in GPie than in GPii ($P < 0.05$).

**DISCUSSION**

Based on the current model of the basal ganglia, one might have expected to find higher neuronal activity in the GPi of MSA patients due to their loss of inhibitory direct striatopallidal fibers. Furthermore, the severity of the aki-
netic-rigid state in MSA patients, at least in theory, could be secondary to increased inhibitory outflow from GPi. We found that neurons in GPi of MSA patients had lower firing rates, especially those in GPii, whereas neuronal activity in the GPe and GPie was comparable to that found in PD patients. These findings are consistent in general with those of two recent studies that compared firing rates of neurons in GPi of PD patients with that in dystonia and Huntington’s disease patients, and failed to find any significant differences, suggesting that the model may not be useful in predicting pallidal firing rate alterations based on various pathologies.

The conclusions of this study are based on findings from only 3 MSA patients and thus a limited number of electrode penetrations and a relatively small number of cells, especially in GPe. It is thus possible that our sample is not entirely representative of pallidal firing in MSA patients and that we may have missed a region or regions with different characteristics. We feel that this is unlikely, however, because our extensive experience in more recent recordings from pallidal neurons in other patient groups strongly suggests that properties of pallidal neurons are very similar in different parts of each subregion and between patients with a similar diagnosis. Furthermore, there were no obvious differences in firing rates of neurons recorded in different electrode tracks and between patients in the current study. In view of the unique nature of these data and the fact that we are unlikely to have additional opportunities to record from MSA patients, we feel that it is valid and worthwhile to present these limited data.

Clinical and Pathological Features of MSA

The basal ganglia pathology of MSA is characterized by cell loss, loss of myelinated fibers, and gliosis in both the striatum and substantia nigra, as well as widespread presence of glial cytoplasmic inclusions. The putamen is affected most, and may be the primary site of lesion in SND. The substantia nigra pars compacta (SNc) exhibits hypopigmentation and variable loss of dopaminergic neurons, accompanied by a marked depletion of immunoreactivity of striatonigral projection fibers. The GP, especially the external part, shows similar but considerably less involvement of both neurons and nerve fibers, whereas some autopsied cases showed no significant cell loss. An extensive review of pathologically proven MSA cases showed correlation between the presence of akinesia and the severity of cell loss in the putamen, substantia nigra, and GP, but pathological abnormalities (cell loss and gliosis) only ranged between 7.5 and 36.6% in GP, and were not related to the severity of putaminal damage. These findings are consistent with the present results in that we did not observe any marked reduction in the number of cells per track in MSA compared with that in PD, although our techniques preclude quantitative assessments. Immunohistochemical studies, used to demonstrate projection fibers from the caudate nucleus and the putamen, have shown marked loss of immunoreactivity in the GP corresponding exclusively to areas of striatal degeneration, which is mostly the posteroverentral parts of the GP (ventral part of the internal and ventral part of the external GP). The subthalamic nucleus seems to be spared in MSA.

PD is characterized by the progressive death of heterogeneous populations of neurons, including the neuromelanin-laden dopaminergic neurons of SNc, selected aminergic, cholinergic, and catecholaminergic nuclei and small cortical neurons. The pattern of cell loss in the SNc, greatest in the ventrolateral tier, followed by the medial-ventral tier and dorsal tier, is relatively specific to PD. Consequently, the dorsal and intermediate subdivisions of the putamen show greater dopamine loss, although depletion of dopamine is found in all subdivisions. There have been few structural studies of GP pathology in PD. The GPe in PD patients may not differ significantly from that of control subjects, whereas a significant reduction in the proportion of neurons containing the calcium-binding protein parvalbumin has been described in the GPi in PD patients compared with that in normal controls, but this was not accompanied by significant evidence of neurodegeneration.
In summary, pathological findings suggest that in MSA there is a predominantly subregional deafferentation of the GP and substantia nigra pars reticulata (SNr) from degenerated putaminal inputs. In PD, however, there is essentially loss of dopaminergic innervation of somewhat different subdivisions of the striatum and consequently dysfunctional striatal output to the GP and SNr. This may explain the differences between the two conditions in responsiveness to dopaminergic therapy.33,34

The Basal Ganglia Model

The GPi discharge rate of MSA patients in this study was lower than that in PD patients and similar to that reported for normal monkeys.35 This finding suggests an overactive inhibitory input to the internal segment of the GPi. There are two major sources of afferent inhibition to GPi: the direct striatopallidal fibres (from striatal neurons containing predominantly D1 receptors) and projections from GPe. In simple terms, one could speculate that because there was no hyperactivity in GPe, a relative increase in the inhibitory input of the direct pathway would be the most likely explanation, although due to the small number of GPe neurons recorded in the MSA patients, this finding may not be representative. Previous studies have shown the loss of striatal D2 receptors in MSA.36,37 In some studies, this loss was greater than was the reduction of D1 receptors,38 although other studies have emphasized striatal D1 receptor decline in SND.39

On the other hand, striatal D2 binding potential, which is normal or raised in untreated patients with PD, has also been reduced in patients with PD and fluctuating L-dopa response.40 Immunohistochemical studies in MSA have shown an almost complete disappearance of both substance P and Met-enkephalin immunoreactivity (markers of striatal projection fibers to GPe and GPi, respectively) in the lateroventral GP,25 in accordance with the apparent predominance of the severe neuronal degeneration taking place in the matrix of dorsolateral striatum.24 These facts would predict that, if it exists, increased direct pathway striatal inhibitory input to GPi might be seen only in the anterior-medial (limbic and associative) segments of the GP rather than in the sensorimotor posteroverentral portion that we studied.

One other possible explanation for our findings is a deficient STN excitatory input to GPi, because subthalamopallidal projections greatly influence pallidal neuronal activity.41 Nonetheless, because the STN is not involved significantly by the pathology of MSA, this would also involve more complexity than the double circuit model we have now,41 with afferents to STN dominated largely by excitatory cortical and inhibitory GPe inputs. Other projections to STN, such as thalamic (parafascicular nucleus) and ascending projections from the brainstem (substantia nigra, pedunculopontine tegmental nucleus, and dorsal raphe nucleus) may exist and be abnormal in MSA. More data are needed regarding these issues.

Functional Procedures for Parkinsonian Symptoms in MSA

Since 1992, there has been renewed interest in posteroverentral pallidotomy (PVP).42 PVP sometimes produces striking improvement in patients with advanced PD, mostly by reducing peak-dose dyskinesias or dystonia and akinesia, rigidity and tremor in the off period. The benefits from PVP, however, as well as from deep brain stimulation (DBS) of the GPi or STN in PD patients, seem to be restricted to the L-dopa-responsive symptoms.44–46 We could identify two reports regarding thalamotomy in patients with SND and four reports of STN deep brain stimulation in patients with MSA, but none that referred specifically to pallidotomy.47–50 The effects of STN DBS, as in PD, is correlated in general with the extent of the ongoing response to L-dopa. We carried out PVP in MSA patients, hoping to achieve results similar to those in PD.44 As we have reported previously,10 however, the benefits of the procedure proved to be minimal and of short duration. It is possible that the poor results achieved in the MSA patients is related to the unexpected low firing rates in GPi, because the common belief is that PVP and DBS produce relief of PD symptoms by decreasing a hyperactive GPi. Recent studies, however, suggest that GPi firing rates may not be the major determinant of motor symptoms due to basal ganglia pathology17,18 and that DBS51,52 may not reduce the firing rate of GPi neurons. The poor PVP and DBS results52 in MSA patients thus suggest that the main symptoms of the disease involve other components of the motor system to a greater extent than in PD.

In conclusion, this study has shown a reduction in GPi activity in MSA in contrast to the hyperactivity expected in the parkinsonian state. The mean GPi firing rate in our akinetic-rigid MSA patients was similar to that reported in hyperkinetic disorders.53 A more complex model of basal ganglia function in health and disease states is needed to explain some results observed in this and other studies.

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REFERENCES

Natural History of Posttraumatic Cervical Dystonia

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Abstract: We studied a case series of 9 patients with posttraumatic cervical dystonia, in whom involuntary muscle spasms and abnormal head postures occurred within 7 days after cervical injury. Patients were examined, treated with botulinum toxin as necessary, and were followed up to 5 years. Based on our observations of these cases, we propose that complex regional pain syndrome (CRPS) could represent a variant of posttraumatic cervical dystonia that may develop over time after the initiation of dystonia. © 2004 Movement Disorder Society

Key words: posttraumatic torticollis; complex regional pain syndrome

The most common form of focal dystonia is torticollis or rotation of the neck to one side. Most cases of torticollis are idiopathic, although some may result from a number of identified causes including brain injury, brain tumor, stroke, cervical cord injury or lesion, drugs including levodopa (L-dopa) and neuroleptics, multiple sclerosis and, in some cases, genetic abnormalities.

A small number of patients report a history of trauma before onset of dystonia. Severe head trauma associated with brain injury that produces identifiable lesions, particularly in the putamen, has been known to cause hemidystonia.1 Peripheral trauma as a precipitating cause of dystonia is not accepted universally,2– 4 although one of the earliest reported cases of torticollis, the Birmingham mummy, is thought to have been caused by an arrow in the neck.5

Posttraumatic cervical dystonia is thought to be a unique syndrome, distinct from idiopathic torticollis. Some of its unusual features include a limitation in cervical range of motion, fixed posture, absence of “geste antagonistique,” persistence of symptoms during sleep, lack of improvement after sleep (the “morning honeymoon” effect), dominant laterocollis, and poor response to botulinum toxin injections.6,7

Complex regional pain syndrome I (CRPS-I) has been known to occur in posttraumatic cervical dystonia, and dystonia has been seen in CRPS.8– 10 CRPS-I is defined as a pain syndrome that usually develops after an initiating noxious event, is not limited to the distribution of a single peripheral nerve, and is disproportional to the inciting event.11 It is associated with edema, changes in blood flow, abnormal sudomotor activity in the region of pain, and allodynia or hyperalgesia. Schott12 first proposed a possible relationship between peripheral trauma-induced movement disorders and causalgia or reflex sympathetic dystrophy in 1986. We describe 9 additional patients, 3 of whom exhibit CRPS, with posttraumatic cervical dystonia after apparent peripheral trauma, who share characteristics unique to the syndrome.

PATIENTS AND METHODS

Patients with dystonia of the cervical musculature who presented to the Parkinson’s and Movement Disorder Clinic in Fountain Valley, CA were systematically asked for history of trauma at their first interview. Inclusion criteria2 were as follows: (1) onset of dystonia within 3