Association of Ipsilateral Motor Automatisms and Contralateral Dystonic Posturing

A Clinical Feature Differentiating Medial From Neocortical Temporal Lobe Epilepsy

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Background: Clinical features that may help to differentiate medial temporal lobe epilepsy (MTLE) from neocortical temporal lobe epilepsy (NTLE) are lacking.

Objective: To investigate the localizing and lateralizing value of the association of ipsilateral motor automatisms and contralateral dystonic posturing in patients with medically refractory temporal lobe epilepsy.

Patients and Methods: Videotapes of 60 patients with well-defined MTLE, NTLE, or both were reviewed to assess the presence and the localizing value of unilateral dystonic posturing associated with motor automatisms.

Results: Twenty-eight of the 60 patients exhibited unilateral dystonic posturing. This sign was observed in patients with MTLE and NTLE. It was mostly contralateral to the seizure focus in patients with MTLE and exclusively ipsilateral in patients with NTLE. Unilateral motor automatisms occurred in 26 of the 60 patients with MTLE or NTLE. It was predominantly ipsilateral to the seizure focus in patients with MTLE and exclusively contralateral in patients with NTLE. The association of ipsilateral motor automatisms and contralateral dystonic posturing was found in 14 patients with MTLE but in none of the patients with NTLE. Two patients who had medial and neocortical seizure onset also exhibited this clinical feature. This association was not significantly correlated with the postoperative outcome in patients with MTLE.

Conclusions: The association of ipsilateral motor automatisms and contralateral dystonic posturing may help to differentiate MTLE from NTLE with a reliable lateralizing value. This clinical association may reflect a specific pattern in the spread of the ictal discharge.

Arch Neurol. 1999;56:927-932

CTAL BEHAVIOR may be useful in determining the side of seizure onset, especially in temporal lobe epilepsy (TLE). In clinical practice, dystonic posturing of limbs contralateral to the epileptogenic focus and unilateral motor automatisms ipsilateral to the focus are usually considered as valuable lateralizing signs. Nevertheless, the reliability of these signs has been questioned in the literature, as they may provide false lateralization and are subject to interobserver variability. Furthermore, the localizing value of these signs is seriously diminished as they can also occur in complex partial seizures of temporal, parietal, or frontal lobe origin.

In the present study, the association of contralateral dystonic posturing and ipsilateral motor automatisms, defined as the simultaneous or successive occurrence of a sustained posturing and a stereotyped, nonpurposeful, involuntary movement of the upper extremities, was evaluated in patients with TLE. The occurrence of these motor signs in patients with medial TLE (MTLE) was compared with their occurrence in patients with neocortical TLE (NTLE) to assess the lateralizing and localizing value of these signs within the temporal lobe. The aim of this study was to determine whether these motor signs could reflect a specific pattern in the spread of the ictal discharge and, thus, provide reliable information on the site of seizure onset.

RESULTS

CLINICAL FINDINGS

Dystonic Posturing

Dystonic posturing was present in 24 (53%) of 45 patients with MTLE. The

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PATIENTS AND METHODS

PATIENTS

The study population comprised 60 patients with TLE who underwent preoperative evaluation for intractable seizures between 1991 and 1996 in 3 epilepsy centers: Salpêtrière Hospital Epilepsy Unit, Paris, France; Gent University Hospital Epilepsy Unit, Gent, Belgium; and Notre-Dame Hospital Epilepsy Unit, Montreal, Quebec. All patients underwent complete presurgical evaluation, including medical, neurologic, and neuropsychological examination. Monitoring with closed-circuit television and scalp electroencephalography (EEG) was continued until typical seizures had been recorded for each patient. Eighteen patients were monitored with closed-circuit television and intracranial EEG recording. All patients underwent magnetic resonance imaging, and 50 underwent interictal positron emission tomography with fluorodeoxyglucose examinations. Patients were separated into 3 groups: (1) MTLE, (2) NTLE, and (3) association of MTLE and NTLE.

The diagnosis of MTLE was based on clinical and neuroimaging criteria adapted from Engel. The clinical criteria included a family history of epilepsy or febrile convulsions, presence of an aura characterized by autonomic or psychic symptoms, complex partial seizures beginning with arrest and stare with presence of oroalimentary automatism, and unilateral or bilateral independent anterior temporal EEG spikes with maximal amplitude in basal electrodes on surface EEG monitoring. The following neuroimaging criteria were also required: hippocampal atrophy visible on magnetic resonance imaging scans without any other structural abnormalities, characteristic temporal lobe hypometabolism on interictal fluorodeoxyglucose–positron emission tomographic scans, or both. Forty-five consecutive patients met these criteria (17 men and 28 women; mean age, 33 years). When information about this first phase was not sufficiently congruent or clear, a second phase consisting of intracranial recordings with depth electrodes could be undertaken. As previously described,13 the electrode location was determined according to the hypotheses resulting from the previous phase. The intracranial electrographic criterion for diagnosis was the presence of a well-lateralized mesial temporal lobe seizure onset. In 6 patients, the intracranial ictal EEG abnormalities were ipsilateral to the hippocampal sclerosis. In 3 patients, the abnormalities were bilateral, but predominated ipsilaterally to the hippocampal sclerosis. All the patients underwent surgery (28 left- and 17 right-sided resections): anterior temporal lobectomy in 37 patients, amygdalohippocampectomy in 6, and anterior temporal lobectomy sparing the hippocampal formations because of a Wada test failure in 2. Pathological examinations of the resected hippocampal formations were not available for all the patients and, thus, did not constitute a diagnostic criterion. The postoperative follow-up was at least 2 years. Postoperative outcome was evaluated according to the Engel classification.12

The diagnosis of NTLE was based on case history (no history of complicated febrile seizures or history of a presumed etiologic insult), clinical features (presence of an aura characterized by auditory illusions or hallucinations or absence of early oroalimentary automatism), and interictal and ictal EEG data and magnetic resonance imaging scans (absence of hippocampal atrophy or presence of a temporal neocortical structural abnormality). In 10 cases, additional fluorodeoxyglucose–positron emission tomographic examinations revealed a severe localized lateral temporal lobe hypometabolism without hippocampal hypometabolism. The location of seizure onset was assessed either by intracranial recordings with depth electrodes or by the results of surgery (ie, patients seizure free after neocortical temporal lobe resection). Thirteen patients had NTLE (4 men and 9 women; mean age, 36 years). Six patients had intracranial recordings that demonstrated a seizure onset in the neocortical temporal lobe. Structural abnormalities on magnetic resonance imaging scans, pathological examination, or both were found in 9 cases (Table 1). To date, 11 of the 13 patients have undergone surgery: lesionectomy, 8; temporal lobectomy, 1; and temporal neocortectomy, 2. All had good postoperative results.

Two patients had associated MTLE and NTLE. The diagnosis of associated MTLE and NTLE was assessed based on intracranial recordings that demonstrated the existence of 2 independent seizure foci within the temporal lobe. The first patient (aged 26 years) contracted meningitis at the age of 9 months. Seizures began at 3 years. He had 2 types of complex partial seizures: seizures beginning with (1) complex auditory hallucinations and (2) anxiety and swallowing. Medial and lateral temporal lobe seizure onset was diagnosed by ictal intracranial EEG recordings. Magnetic resonance imaging results showed isolated left amygdala hyperintensity on T2-weighted sequences. The second patient (aged 46 years) experienced febrile convulsions at age 4 years. Seizures began at 12 years; initially, she exhibited tonic-clonic seizures. These were replaced by complex partial seizures characterized by early oral and motor automatism. Magnetic resonance imaging results showed left hippocampal sclerosis and a left lateral temporal dysembryoplastic neuroepithelial tumor. Concurrent left medial and lateral temporal lobe seizure onset was also detected on ictal intracranial EEG recordings. The patient underwent a left temporal lobectomy including the lateral lesion and the hippocampus. Following surgery, she experienced a worthwhile reduction in seizures.

SEIZURE DATA

The videotapes of all patients were reviewed. One to 10 (mean, 2.7) seizures per patient were blindly analyzed by 2 of us (S.D. and F.S.). The occurrence of motor signs (motor automatisms and dystonic posturing) was studied. The side of these motor signs and their association, whether simultaneous or successive, were analyzed. Unilateral motor automatisms were defined as stereotyped, nonpurposeful, and involuntary movements of one hand (as repetitive raising, picking, or fumbling). Unilateral dystonic posturing was defined as sustained, unnatural posturing of one upper extremity.

Any relationships among the occurrence of these motor signs, intracranial EEG data, and postoperative outcome were investigated.
occurrence of unilateral dystonic posturing is given in Table 2. Bilateral dystonic posturing was noted in 1 (2%) of 45 patients with MTLE. This sign was easily and consistently identified by the 2 observers. It usually consisted of flexion of the wrist and elbow with extension of the fingers. In patients with MTLE, unilateral dystonic posturing was contralateral to the epileptic focus in 22 and ipsilateral to the seizure focus in 1; this latter patient is discussed below. In the 13 patients with NTLE, dystonic posturing was always unilateral and was ipsilateral to the seizure onset. In both patients with associated MTLE and NTLE, dystonic posturing was always unilateral and was contralateral to the seizure onset.

**Table 1. Characteristics of the Patients With NTLE**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Site of Seizure Onset</th>
<th>Structural Abnormality on MRI and/or Pathological Examination</th>
<th>Localization of the Structure Abnormality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>R T2 to T3†</td>
<td>Dysembryoplastic neuroepithelial tumor§</td>
<td>R posterior T2 to T3</td>
</tr>
<tr>
<td>2</td>
<td>L polar region and T2†</td>
<td>None</td>
<td>...</td>
</tr>
<tr>
<td>3</td>
<td>L T2†</td>
<td>None§</td>
<td>...</td>
</tr>
<tr>
<td>4</td>
<td>R temporal‡</td>
<td>Cavernous angioMa§</td>
<td>R T1 to T2</td>
</tr>
<tr>
<td>5</td>
<td>L Heschl gyrus†</td>
<td>None</td>
<td>...</td>
</tr>
<tr>
<td>6</td>
<td>R posterior temporal‡</td>
<td>Gliosis§</td>
<td>R polar region</td>
</tr>
<tr>
<td>7</td>
<td>R T1†</td>
<td>Tuberculacia granuloma§</td>
<td>R T1</td>
</tr>
<tr>
<td>8</td>
<td>R middle temporal‡</td>
<td>Cystic astrocytoma§</td>
<td>R polar region</td>
</tr>
<tr>
<td>9</td>
<td>R medial and posterior part of the temporal neocortex‡</td>
<td>Oligodendroglialoma§</td>
<td>R polar region</td>
</tr>
<tr>
<td>10</td>
<td>R temporal‡</td>
<td>Cavernous angioMa§</td>
<td>R polar region</td>
</tr>
<tr>
<td>11</td>
<td>R posterior T2†</td>
<td>None§</td>
<td>...</td>
</tr>
<tr>
<td>12</td>
<td>R posterior temporal‡</td>
<td>Oligodendroglialoma§</td>
<td>R polar region</td>
</tr>
<tr>
<td>13</td>
<td>R anterior and middle temporal‡</td>
<td>Cavernous angioMa§</td>
<td>R anterior lateral</td>
</tr>
</tbody>
</table>

†Site of seizure onset ascertained from intracranial electroencephalographic recordings. §Pathological examination.

**Table 2. Occurrence of Dystonic Posturing**

<table>
<thead>
<tr>
<th>Patient Description</th>
<th>Patients With Dystonic Posturing, Total</th>
<th>Type of Dystonic Posturing</th>
</tr>
</thead>
<tbody>
<tr>
<td>With MTLE (n = 45)</td>
<td>24 (53)</td>
<td>1 (4) 22 (92) 1 (4)</td>
</tr>
<tr>
<td>With NTLE (n = 13)</td>
<td>3 (23)</td>
<td>3 (100) 0 0</td>
</tr>
<tr>
<td>With associated MTLE and NTLE (n = 2)</td>
<td>2 (100)</td>
<td>0 2 (100) 0</td>
</tr>
</tbody>
</table>

Data are given as the number (percentage) of patients. MTLE indicates medial temporal lobe epilepsy; NTLE, neocortical temporal lobe epilepsy.

**Motor Automatisms**

Motor automatisms were present in 25 (56%) of 45 patients with MTLE. The occurrence of motor automatisms is given in Table 3. Bilateral motor automatisms were noted in 1 (2%) of the 45 patients with MTLE. Unilateral automatisms were ipsilateral to the epileptic focus in 21 patients with MTLE and in both patients with associated MTLE and NTLE. They were contralateral to the epileptic focus in 3 patients with MTLE and 2 patients with NTLE.

**Table 3. Occurrence of Motor Automatisms**

<table>
<thead>
<tr>
<th>Patient Description</th>
<th>Patients With Motor Automatisms, Total</th>
<th>Type of Motor Automatisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>With MTLE (n = 45)</td>
<td>25 (56)</td>
<td>21 (84) 3 (12) 1 (4)</td>
</tr>
<tr>
<td>With NTLE (n = 13)</td>
<td>2 (15)</td>
<td>0 2 (100) 0</td>
</tr>
<tr>
<td>With associated MTLE and NTLE (n = 2)</td>
<td>2 (100)</td>
<td>2 (100) 0 0</td>
</tr>
</tbody>
</table>

Data are given as the number (percentage) of patients. MTLE indicates medial temporal lobe epilepsy; NTLE, neocortical temporal lobe epilepsy.

**Association of Dystonic Posturing and Motor Automatisms**

The simultaneous or successive occurrence of dystonic posturing and motor automatisms was noted in 17 of 45 patients with MTLE, no patients with NTLE, and both patients with associated MTLE and NTLE (Table 4).

Dystonic posturing and motor automatisms were unilateral in both patients with associated MTLE and NTLE and in 16 patients with MTLE. All these patients, except for 2 patients with MTLE, exhibited dystonic posturing contralateral to the epileptic focus associated with motor automatisms ipsilateral to seizure onset. Of the 16 patients with MTLE who had unilateral signs, one presented with ipsilateral dystonic posturing associated with contralateral motor automatisms and another presented successively with contralateral motor automatisms and contralateral dystonic posturing.

One patient with MTLE exhibited bilateral motor automatisms followed by bilateral dystonic posturing.
CORRELATION BETWEEN CLINICAL SYMPTOMS AND INTRACRANIAL RECORDINGS

Three of the 17 patients with MTLE who exhibited dystonic posturing associated with motor automatisms underwent intracranial recordings because of bilateral scalp EEG abnormalities. In each case, the intracranial recordings showed bilateral ictal temporal abnormalities that were predominant on one side.

In 2 cases, dystonic posturing was always contralateral to the predominant ictal discharge and motor automatisms ipsilateral to it.

In 2 of the 3 patients, dystonia and motor automatisms occurred simultaneously 33 and 66 seconds, respectively, after seizure onset. In the third patient, motor automatisms started 31 seconds and dystonic posturing 46 seconds after seizure onset.

The first patient had right motor automatisms and left dystonic posturing during her complex partial seizures. Seizures beginning in the right temporal lobe were more frequent. In the few seizures beginning in the left temporal lobe, the discharge spread rapidly to the right temporal lobe, and it ceased in the left temporal lobe. In all seizures, the association of right motor automatisms and left dystonic posturing occurred in relation to a discharge beginning in or spreading to the right temporal lobe. The specific ictal behavior occurred approximately 66 seconds after right temporal lobe ictal onset and 57 seconds after left temporal lobe ictal onset. This patient underwent right temporal lobectomy and was still seizure free 3 years after surgery.

The second patient had seizures that began predominantly in the left temporal lobe. She exhibited left motor automatisms and right dystonic posturing exclusively when the ictal discharge spread to the left frontal lobe, approximately 60 seconds after the discharge onset. This patient failed the Wada test exploring right temporal lobe memory processes. She therefore underwent left temporal lobectomy sparing the hippocampal region. Four years after surgery, she still had worthwhile reduction of seizures (Engel classification IIIa).

The third patient had seizures that began in the right temporal lobe in 67% of the cases. The association of left motor automatisms and right dystonic posturing only occurred when the ictal discharge spread to the left temporal lobe. She underwent right temporal lobectomy despite the fact that the dystonic posturing was ipsilateral and the motor automatisms contralateral to the presumed ictal onset. The patient was not seizure free after surgery and died 3 years later during a status epilepticus.

CORRELATION WITH THE POSTOPERATIVE OUTCOME

There was no significant statistical correlation (Fisher exact test) between the occurrence of unilateral dystonic posturing and motor automatism and the postoperative outcome, although the presence of the association was more frequent in patients with Engel classification Ia than in patients with Engel classification II and III (43% and 27%, respectively) (Table 5).

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motor automatisms that are frequently bilateral. This may explain why motor automatisms have no reliable lateralizing value, in contrast to dystonic posturing, which is usually contralateral to the seizure focus.1,2 Distinctive characteristics of MTLE and NTLE are lacking.3,4 As both syndromes may have common clinical features, Saygi et al5 showed that ipsilateral motor automatisms and contralateral dystonic posturing were significantly less frequent in NTLE than in MTLE. In contrast, in a recent study, Gil-Nagel and Risinger6 suggested that early contralateral dystonic posturing was more frequent in NTLE.

These findings demonstrate that isolated unilateral dystonia can be present in MTLE and NTLE, but that the association of contralateral dystonic posturing and ipsilateral motor automatisms predominantly exists in MTLE. None of the patients with NTLE exhibited this association, whereas it was observed in both patients with associated MTLE and NTLE. This suggests that the association of contralateral dystonic posturing and ipsilateral motor automatism is highly related to mesial temporal lobe seizure onset. In this study, 31% of the patients with MTLE exhibited dystonic posturing contralateral to the seizure focus and motor automatisms ipsilateral to the seizure focus; there was only one false lateralization and one case of bilateral motor signs. In the patient with a false lateralization, the overall lateralizing value of the association may well have been correct but incorrectly assessed. It may be hypothesized that the main epileptogenic zone was in the contralateral temporal lobe based on her intracranial recordings and the postoperative outcome.

This study demonstrates that the association of contralateral dystonic posturing and ipsilateral motor automatism is a reliable sign to differentiate between MTLE and NTLE. The occurrence of dystonic posturing and motor automatisms reflects a specific primary spread of the ictal discharge and not the seizure onset itself.1,2,18 In this study, intracranial recordings demonstrated that the occurrence of dystonic posturing and motor automatisms was delayed with respect to the onset, and was seen during the primary spread of the ictal discharge at least 30 seconds after the ictal onset on intracranial recording. The exact anatomic location of this specific spread is still unknown. Symptomatic focal dystonia of the upper limb is usually attributed to lesions of the striatopallidal complex and the thalamic-subthalamic area.19,20 Precise anatomic-clinical correlations of dystonia have been established.20 Lesions associated with tonic spasms are preferentially located in the striatopallidal complex, including the posterior putamen and the dorsolateral part of the caudate nucleus contralateral to the dystonia; and lesions associated with myoclonic dystonia are located predominantly in the thalamus contralateral to the dystonia. The dystonic posturing reported in complex partial seizures is similar to that described in lesions within the striatopallidal complex. There is anatomic and experimental evidence for projections from the amygdala and hippocampus to the basal ganglia structures in humans.21,22 Among the hippocampal efferents, the precommissural fornix fibers originating from the subiculum complex are distributed to the accumbens nucleus, which massively projects to the ventral pallidum.23 Furthermore, Newton et al,24 in an ictal single photon emission computed tomography study in patients with TLE, demonstrated that the occurrence of ictal dystonia was significantly associated with a relative increase in perfusion of the basal ganglia contralateral to the dystonic limb. Thus, it seems likely that ictal dystonic posturing may be pathophysiologically linked to the basal ganglia and, therefore, that the spread of the ictal discharge involves the striatopallidal complex. The pathophysiologic characteristics of motor automatisms remain unclear, however. Many researchers8,25 report simple gestural activities to be more characteristic of frontal seizures than of temporal seizures. Although simple motor automatisms often occur in TLE, they could reflect the spread of the ictal discharge into the frontal lobe. The role of the cingulate gyrus in determining this type of motor automatism has been widely discussed in the literature.26,27 According to the findings, motor automatisms occurred either just before or simultaneously with dystonic posturing. It can be thus hypothesized that the ictal discharge either spread initially from the medial temporal structures to the medial frontal cortex and then to the basal ganglia or spread simultaneously to the medial frontal cortex and the striatopallidal complex. Anatomic relationships between the hippocampal structures and the medial frontal cortex and the basal ganglia suggest this hypothesis. Hippocampal efferents (precommissural fornix fibers originating from the subicular complex) are distributed to the medial parts of the frontal cortex.28 Since such afferent and efferent systems are specific to the medial temporal structures, this may explain the specificity of the association of dystonic posturing and motor automatisms in MTLE.

The association of contralateral unilateral dystonic posturing and ipsilateral motor automatisms may help to differentiate MTLE from NTLE and, thus, provide reliable information on the location of the seizure onset. This association is only present in patients with initial involvement of the hippocampal structures, ie, patients with MTLE and patients with associated MTLE and NTLE.

Accepted for publication September 14, 1998.

We thank Marie Vidalhiet, MD, PhD, for her helpful assistance.

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REFERENCES