Head turning as a prominent motor symptom in status epilepticus

Gerhard Bauer, Gregor Broessner, Iris Unterberger, Gerald Walser, Bettina Pfausler, Eugen Trinka
Department of Neurology, Innsbruck Medical University, Innsbruck, Austria
Received November 5, 2007; Accepted February 20, 2008

ABSTRACT – Head and eye turning is frequently observed during seizures. Versions with tonic and/or clonic symptoms can be differentiated from smooth head deviations. Head turning as a prominent symptom of status epilepticus has not previously been reported. We present eight case reports, (7 women/1 man, mean age 41 years, median 41.5, range 10 to 74), of status epilepticus (SE), with head turning as a prominent motor symptom. Six were accompanied by continuous frontal, occipital and temporal ictal epileptiform discharges. Furthermore, two patients had absence status with rhythmic and clonic head versions. While the localizing significance of head turnings in SE is low, in our cases, the direction was away from the discharging hemisphere in all cases of focal SE regardless of whether the turning was classified as version (three cases) or deviation (three cases). In this small series of SE, the classical observation of a patient looking away from the discharging hemisphere is still valid.

Key words: status epilepticus, head turning, epileptic nystagmus, EEG, versive seizures

Turning of the head and eyes is frequently observed during epileptic seizures. The localizing and lateralizing value have been discussed controversially (Abou-Khalil and Fakhoury 1996, Ajmone-Marsan and Goldhammer 1973, Ochs et al. 1984, Wyllie et al. 1986a). Seizure-related head turning may present as forced unnatural tonic head version (“version”), frequently accompanied by ipsilateral turning of eyes and body (Abou-Khalil and Fakhoury 1996), or as smooth head turning resembling voluntary movements (“head deviation”) (Janz 1969, Kernan et al. 1993, Wyllie et al. 1986a). Furthermore, versions can occur as clonic jerks corresponding to focal motor seizures (Ajmone Marsan and Goldhammer 1973, Janz 1969). Tonic conjugate gaze deviations alone or combined with nystagmus-like eye movements, may represent the sole clinical seizure manifestation (Furman et al. 1990, Gire et al. 2002, Kaplan and Lesser 1989, Kaplan and Tusa 1993, Kompf and Klostermann 1996, Tusa et al. 1990). Turning of the head and body may occur in their most extreme form as complex gyratory or rotatory movements (Dobesberger et al. 2005). Besides the seizure semiology itself, the sequence of head turning during a seizure adds to their lateralizing and localizing value (Abou-Khalil and Fakhoury 1996). Head and eye turning have been observed in both, focal and generalized seizures. Focal seizures originating in the frontal (Bonelli et al. 2007, Rheims et al. 2005, Salanova et al. 1995), occipital (Boesebeck et al. 2002,
Ludwig and Marsan 1975, Schäffler and Karbowski 1988), and temporal lobe (Abou-Khalil and Fakhoury 1996, O’Dwyer et al. 2007) can be associated with head and eye turning. There may be a special preponderance of seizures originating in the temporo-occipital and parieto-occipital regions to present with head and eye turning (Dalmagro et al. 2005, Palmini et al. 1993). Turning of head and/or eyes have also been described in absence seizures or generalized tonic-clonic seizures (GTCS) in generalized idiopathic epilepsy syndromes (Aguglia et al. 1999, Chin and Miller 2004, Janz 1969, Watanabe et al. 1984).

Head turning can be observed as a transient phenomenon in several forms of status epilepticus (SE). A few reports describe oculoclonic SE (Gastaut and Roger 1954, Kanazawa et al. 1989). Ongoing nystagmus or gaze deviation was reported with periodic, lateralized epileptiform discharges (PLEDs) on the EEG (Kaplan 2005, Young et al. 1977). Thurston et al. (1985) reported a patient with consistently repeated gaze deviations and nystagmus associated with temporo-occipital ictal EEG activities. However, to our knowledge, there is no report on the clinical significance of head turning as the prominent motor symptom of SE.

Methods

Eight patients who presented with sustained or repetitive turning of the head as their prominent motor symptom during SE were identified in our outpatient seizure clinic and emergency unit between 1972 and 2003. As a standard procedure in our unit, EEG technicians are requested to call the doctor in charge to all patients who exhibit ongoing EEG activities that might cause them to suspect SE. Thus, all patients presented were seen by one of the authors (GB) personally. After the diagnosis of SE was made, all patients received intravenous antiepileptic treatment in the EEG laboratory of our unit. Corresponding clinical data and outcome after treatment are part of the EEG report. Further clinical and imaging data were retrospectively assessed from patient charts. Original EEG recordings and reports were available for reassessment in all patients.

We defined versions as forced, unnatural, sustained tonic and/ or clonic head turning and head deviations as smooth head turning resembling voluntary movement of the head to one side. These symptoms had to last or – in cases of absence status - be regularly repeated for at least 30 minutes to be classified as SE. EEGs exhibited continuously or regularly recurring epileptiform activities time-locked to clinical symptoms in all patients. As the patient data were collected randomly over the years, the incidence of these symptoms during SE cannot be given. Definition of seizure-onset zone and diagnosis of SE was based on seizure semiology, ictal and interictal EEG and imaging findings (CT or MR).

Results

Clinical and demographic data of eight patients are summarized in table 1. Seizures originated in the temporal lobe in one patient (no. 6), in the frontal lobe in two (nos. 1, 2) and in the occipital lobe in three patients (nos. 3, 4, 5). Two patients had absence status with rhythmic and regularly repeated clonic head turning (nos. 7, 8). Detailed case reports are given below.

Case descriptions

Patient 1, FM, female

She was admitted for the first time to our department at the age of 58 years with a hypertensive intracerebral haemorrhage (ICH) in the left basal ganglia. She had right-sided hemiparesis and global aphasia. She improved markedly until she developed seizures with rhythmic clonic head versions two years later. Neurologically, she had a mild right-sided hemiparesis, some speech problems and hemianopia to the right. A CT scan showed a well demarked, hypodense defect in the left thalamic region due to the previous ICH. Repeated EEG records were abnormal, with a left-sided suppression of the basic rhythm and left anterior slow waves. We diagnosed symptomatic epilepsy secondary to the ICH and treated her with low doses of carbamazepine (CBZ) (serum level of 7.2 µg/mL). Her pre-existing renal insufficiency due to analgesic abuse worsened, and extracorporal dialysis has since become necessary. The seizure disorder was well controlled thereafter.

After missing a scheduled dialysis treatment, the patient was admitted to our department with repetitive clonic jerking of the head to the right. Several minutes after admission, these clonic versions were replaced by a continuous tonic head version to the right. No reaction to exogenous stimuli was noted. During this phase, the EEG was characterized by bi-frontal spikes, clearly accentuated over the left anterior region (figure 1). After treatment with 10 mg diazepam (DZP) intravenously (iv), the spike activities stopped (figure 2), the head version changed sides as well as the motor characteristics, with a smooth deviation of the head and the eyes to the left. After one hour, the patient was alert, reactive and no further motor seizure signs could be observed. She remained seizure-free for three years. She was lost to further follow-up.

Diagnosis: symptomatic focal epilepsy after ICH, SE provoked by metabolic disturbances due to non-compliance with the extracorporal dialysis regime, accompanied by clonic head versions followed by continuous tonic version to the right, with an EEG focus over the left anterior region.

Patient 2, SS, female

The patient started having simple and complex focal seizures with aphasic symptoms at the age of seven years.
Table 1. Demographic and clinical data, seizure semiology, EEG and imaging (CT/MR) findings in 8 patients with head turnings as a prominent symptom during status epilepticus.

<table>
<thead>
<tr>
<th>No.</th>
<th>Initials</th>
<th>Age</th>
<th>Age at seizure onset</th>
<th>Preexisting seizure types</th>
<th>Semiology of SE</th>
<th>EEG during SE</th>
<th>Neuroimaging</th>
<th>Precipitating factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>FM</td>
<td>63/W</td>
<td>60</td>
<td>Focal motor</td>
<td>Clonic head version to R $\rightarrow$ tonic head version to R + unresponsiveness $\rightarrow$ after cessation of status head deviation to L + conjugate gaze deviation L</td>
<td>Continuous LF spikes</td>
<td>CT: residual cicatrix after ICH L hemisphere</td>
<td>Renal insufficiency with metabolic disturbances</td>
</tr>
<tr>
<td>2</td>
<td>SS</td>
<td>25/W</td>
<td>7</td>
<td>focal complex, focal aphasic, sGTCS</td>
<td>Non-fluent aphasia with good speech comprehension + head and eye deviation to R</td>
<td>LF continuous rhythmic slow activity with intermingled spikes</td>
<td>MRI: normal</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>LAM</td>
<td>74/W</td>
<td>74</td>
<td>none</td>
<td>Visual hallucinations $\rightarrow$ head deviation to L + nystagmus to L</td>
<td>Continuous spikes over R posterior</td>
<td>CT: ischaemic PCA infarction R</td>
<td>Cerebrovascular accident</td>
</tr>
<tr>
<td>4</td>
<td>LH</td>
<td>67/W</td>
<td>67</td>
<td>none</td>
<td>Comatose + continuous head version to L</td>
<td>Continuous spikes R posterior</td>
<td>CT: residual cicatrix after ICH R temporo-occipital</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>WS</td>
<td>58/M</td>
<td>58</td>
<td>none</td>
<td>Hallucinations, head deviation to L</td>
<td>Continuous R occipital spikes</td>
<td>Angiography, normal</td>
<td>Alcohol and drug withdrawal</td>
</tr>
<tr>
<td>6</td>
<td>SB</td>
<td>10/W</td>
<td>10</td>
<td>Simple focal vegetative, focal complex, sGTCS</td>
<td>GTCS $\rightarrow$ head version to L, dyscognitive symptoms</td>
<td>Slowing over R hemisphere, spikes over T6</td>
<td>MRI: R HS</td>
<td>Cryptogenic, HS</td>
</tr>
<tr>
<td>7</td>
<td>WE</td>
<td>15/W</td>
<td>10</td>
<td>Absence seizures, GTCS</td>
<td>Crescendo absences $\rightarrow$ unresponsiveness + rhythmic clonic versions of the head to L</td>
<td>Continuously repeated 3/sec saw</td>
<td>MRI: normal</td>
<td>AED withdrawal</td>
</tr>
<tr>
<td>8</td>
<td>WD</td>
<td>12/W</td>
<td>8</td>
<td>Absence seizures, GTCS</td>
<td>Crescendo absences $\rightarrow$ partial unresponsiveness + rhythmic clonic versions of the head to R</td>
<td>Continuously repeated 3/sec saw</td>
<td>Not performed</td>
<td>AED withdrawal</td>
</tr>
</tbody>
</table>

AED: anti-epileptic drugs; F: frontal; focal aphasic: simple focal aphasic seizure; focal complex: focal complex seizure; focal motor: focal motor seizure; focal vegetative: focal vegetative seizure; GTCS: generalized tonic-clonic seizure; HS: hippocampal sclerosis; ICH: intracerebral haemorrhage; L: left-sided; M: man, PCA: posterior cerebral artery; R: right-sided; sGTCS: secondary generalized tonic-clonic seizure; W: woman; 3/sec saw: 3/sec spikes and waves.
These seizures frequently evolved into GTCSs. After the GTCSs, the patient repeatedly remained in an aphasic state. Her language comprehension was not impaired, but she was not able to speak except for a few paraphatic syllables. Nevertheless, she was able to continue with her daily activities during these episodes. She acknowledged her deficits as epilepsy-related and came to our seizure clinic for help. During several episodes of seemingly postictal aphasia, an EEG was recorded. The ongoing, left anterior spike and wave activities led to the diagnosis of aphasic SE. During the first documented episode, aphasic SE was accompanied by continuous eye and head deviation to the right. The EEG (figure 3) exhibited 3/sec rhythmic activities over F3, with intermingled small spikes. After treatment with iv DZP, the clinical symptoms and EEG abnormalities cleared. With subsequent episodes of aphasic SE following GTCSs, (seven documented episodes until this time), no further head turning was observed.

Figure 1. Patient 1, FM, female, 62 years. tc 1.0, HF 70 Hz. EEG was recorded during continuous ictal head version to the right. Patient was unresponsive at this time. Ongoing ictal fast activities over left frontal region were accompanied by slow waves, intermittently forming 3/sec spikes and waves. Spread of 3/sec spikes and waves to the homologous, contra-lateral region. No basic rhythm over the posterior regions.

Figure 2. Patient 1, FM, female, 62 years. tc 1.0, HF 70 Hz. Identical record as for figure 1. After termination of SE. The end of iv injection of 10 mg diazepam is marked by “Ende”. At the same time, frontal spikes and head version stopped. Some 7/sec activities of μ-like shape over T5.
No definite etiological factors could be found. MRI was normal. The seizure disorder was refractory to anti-epileptic drug treatment. Presurgical evaluation, including EEG-video monitoring, led to the hypothesis of a seizure-onset zone in the left frontal Broca area on the basis of ictal Broca aphasia and left frontal ictal discharges (F3). Since resective surgery seemed to carry a high risk of permanent postoperative aphasia, a vagal nerve stimulator was implanted, resulting in a moderate improvement of seizure frequency.

**Diagnosis:** cryptogenic, non-lesional, left frontal epilepsy with simple (aphasic) and complex focal seizures, occasionally evolving to GTCSs, and repeated aphasic SE following GTCSs, on one occasion accompanied by eye and head deviation to the right.

**Patient 3, LAM, female**

At the age of 74 years, she was admitted to a psychiatry unit in a delirious state with visual hallucinations, where she was treated with antipsychotics. She had a previous medical history of heart failure, pulmonary artery embolism and an ischaemic infarction in the area of the left medial cerebral artery. During her stay in the psychiatric ward, she became unresponsive, with head deviation and irregular nystagmus to the left evolving within minutes. She was transferred to our department, where treatment with 20 mg DZP iv was ineffective. The EEG exhibited continuous epileptiform activities over the right posterior quadrant (figure 4). After 250 mg phenytoin (PHE) iv, the head deviation and the nystagmus stopped, and the spikes became less prominent. The patient remained in a state of reduced vigilance, but no further motor seizure signs were observed. A CT scan revealed a hypodense area in the right posterior cerebral artery territory, indicating an ischaemic infarction. Five days later, the patient died from an acute pulmonary artery embolism.

**Diagnosis:** acute symptomatic SE with visual hallucinations and head deviation, as well as epileptic nystagmus to the left and a right occipital epileptogenic focus due to a recent cerebral infarction in the right posterior cerebral artery territory.

**Patient 4, LH, female**

At the age of 66 years, the patient suffered a severe, intracerebral haemorrhage, which led to admission to our department. The intracerebral haematoma was located within the right temporo-occipital region and was complicated by intraventricular blood accumulation. She was comatose on admission, and recovered slowly. She was transferred to a neurological rehabilitation unit. Two months later she was transferred to a nursing home, still hemiplegic on the left side and severely mentally impaired. After one year, she was readmitted with SE. She was unresponsive, with continuous forced head version to the left. The EEG (figure 5) exhibited rhythmical sharp and slow waves over the right posterior region. After 20 mg DZP iv, the SE stopped and the EEG showed slow waves over the entire right hemisphere (figure 6). After clearance of the DZP-induced sleep, the patient was in her habitual condition. AED treatment with gabapentin was started and the patient was discharged. No follow-up examination was available.

**Diagnosis:** remote symptomatic focal SE with head version after a severe, right posterior, ICH.
Patient 5, WS, male

The patient had been followed by the psychiatric unit for many years. He was diagnosed as having a personality disorder, with combined abuse of alcohol, sedatives and analgesics. He had no previous history of epilepsy. At the age of 58, he abruptly stopped all substance abuse of his own accord. A few days later, he slipped into a psychotic state, with visual hallucinations. After an intermittent GTCS, he exhibited ongoing head deviation to the left. He was hallucinating vividly during this period, and was referred to our institution for assessment. The EEG showed continuous, right occipital spikes and slow waves. The patient was treated with DZP and PHE iv, and his condition improved within two days. The EEG also normalized. A CT scan was not available at this time. Angiography and brain scintigraphy were normal, pneumencephalography indicated moderate, diffuse, cortical atrophy. The case report of this patient was published in detail earlier (Bauer 1975).

Diagnosis: acute symptomatic SE with visual hallucinations and head deviation to the left, due to an epileptogenic focus over the right occipital region brought about by alcohol and drug withdrawal.

Patient 6, SB, female

A 10-year-old girl, with no previous history of seizures, was admitted to our unit after a GTCS. She was disoriented...
and confabulating. A continuous, tonic head version to the left was observed. The EEG (figure 7) exhibited persistent rhythmical slow activities over the right hemisphere, with continuously intermingled spikes over T6. The EEG changes and clinical symptoms prompted the assumption of a focal SE following the termination of a GTCS, and the patient was treated with 10 mg DZP iv. The SE stopped and the EEG normalized. This episode represented the start of a chronic seizure disorder. She developed focal vegetative and focal complex seizures, both occasionally evolving to GTCSs. Developmental delay since the age of one year was reported, but no clear-cut etiological factors could be established. MRI revealed a right-sided hippocampal atrophy and sclerosis accompanied by subcortical white matter signal changes in T2, and FLAIR sequences within the right temporal lobe. The seizure disorder remained refractory to all major old and new antiepileptic drugs. After a long period of non-compliance, the patient was readmitted at the age of 40 years. Eight seizures were recorded during EEG-video-monitoring for presurgical evaluation.

In two seizures, a version to the left, accompanied by dystonic abnormalities in the left arm, were observed. Right-sided temporal lobe epilepsy was diagnosed and modified right temporal lobe resection was performed. After epilepsy surgery, the patient has now been seizure-free for two years.

**Diagnosis:** symptomatic, right-sided, temporal lobe epilepsy with hippocampal sclerosis, focal SE with psychic symptoms and continuous tonic head version to the left.

**Patient 7, WE, female**

The patient started having pyknoleptic absences at the age of 10 years. Later, occasional GTCSs occurred. No head turning was reported with single absence seizures. Although there was mild mental retardation, the diagnosis was still compatible with childhood absence epilepsy (CAE). The EEG showed generalized 3/sec spikes and waves, and a normal background activity. Family history was uneventful. Later on, a daughter of the patient also developed CAE.

---

**Figure 6.** Patient 4, LH, female, 67 years. tc 0.3, HF 35 Hz. Identical record as for figure 5 after 10 mg diazepam iv. Marked attenuation of sharp waves and slow activities over the right hemisphere. Cessation of clinical seizure symptoms.
At the age of 15 years, the patient stopped taking her antiepileptic drug treatment, which consisted of a combination of ethosuximide and phenobarbital, the treatment of choice in Austria at this time (1972). The next day she developed SE. Absence seizures were repetitive, and accompanied by rhythmic clonic versions of the head and eyes to the left. The jerks were repeated with a frequency of 3/sec. The EEG was characterized by trains of generalized 3/sec spikes and waves relapsing every 5 to 10 sec. In the intervals, a normal alpha rhythm appeared. During the spikes and waves, no reaction to exogenous stimuli was noted. In the intervals, a fragmentary reaction could be elicited. However, most attempts to communicate were followed by an absence seizure. After 10 mg DZP iv, the SE stopped immediately. The patient was followed until the age of 51 years at our institution. A high resolution MRI at this age was normal. Absence seizures and GTCSs became infrequent after implementation of valproic acid (VPA). At the last follow-up she had been seizure-free for seven years. No further episodes of absence status had occurred. Diagnosis: CAE with absence seizures and GTCSs, absence status with clonic head and eye versions to the left provoked by abrupt withdrawal of AEDs.

**Patient 8, WD, female**

At the age of eight years, the patient started to have frequent, typical, daily, absence seizures. Her neurological and mental statuses were normal. Some unusual personality traits, combined with family problems, resulted in difficulties with her epilepsy treatment. After a period of non-compliance, the patient was admitted in an absence status. Absence seizures occurred every 10 to 20 seconds and were always accompanied by clonic head versions to the right. The EEG (figure 8) showed typical generalized 3/sec spikes and waves in trains of up to 30 sec without any focal characteristics. During the intervals in between the absence seizures, a normal alpha rhythm was registered, and the patient reacted adequately. Absence status was treated successfully by DZP 10 mg iv. After treatment with VPA, the pattern of occurrence of the absence seizures changed to a random repetition rate, with several seizure-free days a week. Two years after the reported absence status, we lost track of the patient. Diagnosis: CAE with absence seizures, absence status with clonic head versions to the right provoked by non-compliance to AEDs.

**Discussion**

Data from the eight reported patients were collected over several years by one of the authors. Although a precise incidence of SE, with head turning as the prominent motor symptom can not be given, it seems to be an exceptionally rare phenomenon. We found a poor localizing value for this symptom: it occurred in SE originating in the frontal lobes (patients 1 and 2), occipital lobes (patients 3, 4, and 5), and temporal lobes (patient 6), as well as in idiopathic generalized epilepsies (patients 7 and 8). Associated symptoms in patients with a frontal seizure-onset were unresponsiveness (patient 1), or aphasia (patient 2). In one patient (no. 1), the initial clonic head version evolved into fixed tonic version during status. After the successful interruption of the SE, the tonic head version changed into a smooth head deviation towards the contra-lateral side of the initial fixed tonic version. This conjugate eye and head version...
deviation indicates a selective dysfunction of cortical areas involved in the control of eye movements (Singer et al. 2006). When a cortical lesion is associated with a loss of function, the direction of the conjugate eye and head deviation is directed towards the lesion, which is well known from general clinical neurology (Tijssen et al. 1991). In our patient (no. 1), the direction was initially away from the epileptogenic lesion, compatible with a contra-lateral head version (Abou-Khalil and Fakhoury 1996, Wyllie et al. 1986a), and changed after successful treatment into an ipsilateral head deviation towards the lesion, resembling a postictal deficit.

In those patients with an occipital origin of the status, vivid visual hallucinations (patients 3 and 5), accompanied by epileptic nystagmus (patient 3), were observed. Both patients were initially diagnosed with psychosis and were treated for this, before the epileptic character of the hallucinations was recognized and antiepileptic treatment started. One patient (no. 6), presented as dyscognitive SE accompanied by head version, which originated in the right temporo-parietal lobe.

In three of our patients (nos. 2, 5, and 6), prolonged head turning occurred after a GTCS. In these patients, the EEG was crucial for the diagnosis of SE. Without an EEG recording in the acute setting a clear differentiation between ongoing seizure activity and postictal, or lesional deficit symptoms is almost impossible (Bauer et al. 1982).

Head and/or eye turning has been described in patients with single absence seizures or non-focal GTCSs (Aguglia et al. 1999, Chin and Miller 2004, Janz 1969, Watanabe et al. 1984), but not with absence status. In our series, patients 7 and 8, both had classical CAE. During SE, the absences occurred in series, with head jerks to one side with a frequency of 3/sec. Symmetric, rhythmic eye lid fluttering and retropulsive head movements were frequently observed in typical absence seizures. One might speculate that these symptoms are mediated by discharges affecting both frontal eye fields. Clonic head versions in single absence seizures, as well as in absence status, add to the focal features observed in idiopathic generalized epilepsy syndromes and might be explained by asymmetries of the epileptogenic network involved, with a frontal preponderance (Craiu et al. 2006). However, these focal characteristics are not necessarily indicative of a localized structural abnormality.

The lateralizing value of turning symptoms is the subject of a ongoing discussion and with conflicting results. Head turning has neither localizing nor lateralizing significance, if it is looked at as an isolated and uniform phenomenon (Ajmone Marsan and Goldhammer 1973, Ochs et al. 1984, Robillard et al. 1983). Newton et al. (1992) found this sign to be without lateralizing value, even when only tonic versions were analyzed. The lateralizing value of ictal head turning is different according to the pattern of head movements and the muscles involved (Jayakar et al. 1992). Furthermore, the side of the cerebral hemisphere controlling the muscle movements seems to be not entirely clear (Marcus 1989). The lateralizing significance might be enhanced by taking into account a refined semiology of the turning movements, whether they were remembered or the patient was amnesic of them, the differentiation between ictal and postictal phenomena, the type of seizures, the point of time where turning occurs during a seizure, and the lobular localization of the seizure-onset

Figure 8. Patient 8, WD, female, 12 years. tc 0.6, HF 30 HZ. Reduced paper speed 1.5cm/s. Ictal 3/sec spikes and waves for 17 seconds, accompanied by adversive absence seizures with head and eye deviation to the right. The versive movements are marked as “Blick n. re oben...”. These seizures occurred every 10 to 20 seconds, merging into an enduring epileptic condition.

In focal SE with head turning as the prominent motor symptom, the direction was away from the suspected seizure focus in all reported cases regardless of whether the turning was classified as version (cases 1, 4, 6) or deviation (cases 2, 3, 5). An ipsilateral turning was never observed with SE. With single seizures, this symptom was seen as a late version after secondary generalization (Wyllie et al. 1986b), or as a smooth head deviation (Rheims et al. 2005). Furthermore, ipsilateral turning occurs more frequently with frontal lobe epilepsies (Rheims et al. 2005) and at the beginning of a seizure (Fakhoury and Abou-Khalil 1995, Rheims et al. 2005). Different brain mechanisms might be involved in versions and deviations. However, in this small series of SE, the classical statement is valid: the patient looks away from the discharging hemisphere. After termination of SE, the head deviation might change side, and the patient looks towards the lesioned hemisphere (patient 1).

To summarize, in focal SE, prolonged head turning per se has good lateralizing, but no localizing value. Only additional seizure symptoms allow the correct localization of seizure origin.

References


Craiu D, Magureanu S, van Emde BW. Are absences truly generalized seizures or partial seizures originating from or predominantly involving the pre-motor areas? Some clinical and theoretical observations and their implications for seizure classification. Epilepsia Res 2006; 70 (Suppl. 1): S141-S555.


