Epileptic drop attacks in partial epilepsy: clinical features, evolution, and prognosis

Paolo Tinuper, Angelina Cerullo, Carla Marini, Patrizia Avoni, Anna Rosati, Roberto Riva, Agostino Baruzzi, Elio Lugaresi

Abstract

Objectives—Sudden falls have been described in patients with partial epilepsy. However, no study has detailed the clinical, EEG, and evolutive features of partial epilepsies with drop attacks.

Methods—In a consecutive series of 222 patients with partial epilepsy admitted for uncontrolled seizures over a 10 year period, 31 patients presented with epileptic drop attacks during evolution of their illness. Twenty two patients had frontal, five temporal, and four multifocal or undefinable lobe epilepsy; 74% of the cases showed an EEG pattern of secondary bilateral synchrony during evolution. A statistical comparison of some clinical and EEG features between the patients with epileptic drop attacks and patients with partial epilepsy without drop attacks (control group of 191 patients) was carried out.

Results—Seventy four per cent of patients had a poor prognosis and 45% were mentally retarded; 52% of patients with epileptic drop attacks continued to have epileptic falls associated with partial seizures and mental deterioration at the end of the follow up. These characteristics of patients with epileptic drop attacks were significantly different from the control group.

Conclusion—Almost all literature reports concur that the physiopathogenetic substrate of epileptic drop attacks is a mechanism of secondary bilateral synchrony. A localised epileptic focus may lead to a process of secondary epileptogenesis involving the whole brain, causing a progressive cerebral disturbance with worsening of the epileptic seizures and higher cerebral functions.

(J Neurol Neurosurg Psychiatry 1998;64:231-237)

Keywords: partial epilepsy; epileptic drop attacks; secondary bilateral synchrony; prognosis

Falling may occur during an epileptic seizure and is typical of some epileptic syndromes—namely, epilepsies with grand mal seizures, myoclonic-astatic seizures, and atonic and tonic seizures in generalised cryptogenic or symptomatic epilepsies.¹ Falling is also possible in partial seizures, when secondary generalisation follows the partial onset or when the motor ictal pattern causes a loss of postural control leading to a fall. A sudden drop has been

described in patients with partial epilepsy and has been considered an ominous predictive sign.² Epileptic drop attacks are extremely disabling, both because of the continuous traumatic risk and for the need for patients to be accompanied or move only in a wheelchair or when wearing a helmet. There is no consensus on the site of origin of epileptic drop attacks. Geier *et al*,³ describing falls as the ictal clinical manifestation in 81% of patients with frontal lobe epilepsy, suggested several mechanisms to explain this kind of seizure. Falls may result from an atonia in postural muscles, or a clonic jerk, or a tonic contraction of axial and leg muscles leading to a loss of balance. Other authors⁴ described drop attacks followed by confusion and reactive automatism as of probable frontal lobe origin. Palmini *et al*⁵ found a high incidence of drop attacks in patients with neuronal migration disorders involving the central region, thus supporting the hypothesis of a spreading of the ictal discharge from mesial epileptic foci to frontal areas bilaterally through callosal pathways as the cause of falls. Epileptic drop attacks have also been described in temporal lobe epilepsy.^{6,7} The aim of the study was to detail clinical, EEG, and evolutive features of the partial epilepsies with epileptic drop attacks.

Method

We reviewed the clinical history of all adult patients with cryptogenic or symptomatic partial epilepsy admitted consecutively as inpatients to our Institute for uncontrolled seizures over a 10 year period. A total of 222 patients entered the study, of whom 31 experienced epileptic drop attacks during evolution of their illness. All patients were followed up for at least two years after admission to hospital. During stays in hospital and follow up different antiepileptic drug regimens were given to improve their clinical status. Diagnosis was made on the basis of anamnestic, clinical, radiological (CT and MRI), and electrophysiological data. The EEG machines used were two 17 channel Nihon Koden polygraphs. Nineteen electrodes were placed according to the 10-20 international system in anteroposterior and coronal montages. Sphenoidal leads were added in most patients. The EEG focus was defined as phase reversal in bipolar montage on scalp EEG. Secondary bilateral synchrony was defined as the coexistence in the same EEG of focal and bilateral synchronous discharges after the focal onset within 200 ms.⁸⁻¹² All patients had repeated and prolonged video-EEG recordings while stretched out in

Neurological Institute,
University of Bologna,
Bologna, Italy

P Tinuper
A Cerullo
C Marini
P Avoni
A Rosati
R Riva
A Baruzzi
E Lugaresi

Correspondence to:
Dr Paolo Tinuper,
Neurological Institute
University of Bologna, Via
Ugo Foscolo 7, 40123
Bologna, Italy. Telephone
0039 51 585158; fax 0039
51 6442165.

Received 2 April 1997 and in
revised form 22 July 1997
Accepted 21 August 1997

Table 1 Clinical features

Patient No	Type of epilepsy	Aetiology or neuroradiological lesion	Seizure semiology		Outcome	
1	F	Right hemisphere focal atrophy and gliosis	PA		Drop	DR
2	T	—	CP1	SGTC	Drop	DR
3	F	Left parietooccipital calcified lesion	PA		Drop	DR
4	F	—	PA		Drop	NDR
5	F	—	CP2	SGTC	Drop	NDR
6	F	—	PA	SGTC	Drop	NDR
7	TPO	—	CP2	SGTC	Drop	DR
8	F	—	PA	SGTC	Drop	DR
9	F	—	PA	CP2 SGTC	RSE	Drop
10	F	Pre and perinatal pathology	PA	CP2 SGTC	Drop	DR
11	F	—	PA	SGTC	Drop	DR
12	F	Left temporoparietal porencephalic lesion	CP2	SP SGTC	Drop	DR
13	F	—	PA	CP3	Drop	DR
14	F	—	PA	CP2 SGTC	Drop	DR
15	F	Right frontal focal atrophy and gliosis	PA	CP2 SGTC	Drop	NDR
16	F	Right occipital porencephalic lesion	PA	CP2 SGTC	RSE	Drop
17	TPO	Right occipital calcified lesion (coeliac disease)	CP2	SGTC RSE	Drop	NDR
18	F	Prenatal and perinatal pathology	CP2	SGTC	Drop	DR
19	I	Left frontal pachygyria	PA	CP2 SGTC	Drop	DR
20	F	—	CP2	SGTC RSE	Drop	DR
21	MF	Right hemisphere focal atrophy and gliosis	CP2	SGTC RSE	Drop	DR
22	F	Right anterior temporal porencephalic lesion	PA	SGTC	Drop	DR
23	F	Right temporal tumour	CP2	SGTC	Drop	NDR
24	T	Left hippocampal temporal mesial sclerosis	CP1	RSE	Drop	DR
25	T	Left deep temporal cortical calcified lesion	PA		Drop	DR
26	T	Left insula porencephalic lesion	PA	CP1+aura SGTC	Drop	NDR
27	T	Left polar temporal atrophy and gliosis	CP1+aura		Drop	NDR
28	F	—	PA	CP2 CP4	RSE	Drop
29	F	—	PA	CP3		Drop
30	F	—	PA			Drop
31	F	—	CP2			Drop

Type of epilepsy: F=frontal; T=temporal; TPO=temporoparietal-occipital; I=undefinable; MF=multifocal. Seizure semiology: PA, CP1, CP2, CP3, CP4: see text for explanation; SP=simple partial; SGTC=secondary generalised tonic-clonic; RSE=recurrent status epilepticus. Outcome: DR=drug resistant; NDR=not drug resistant. Interictal EEG focus: F=frontal; T=temporal; FT=frontotemporal; PO=parieto-occipital; MI=multiple independent; SBS=secondary bilateral synchrony; R=right; L=left; Bil=bilateral.

an armchair. Some patients had EEG for prolonged periods while standing up. In these cases patients wore a safety jacket secured to the ceiling with a cable to avoid traumatic injury in the case of a fall.

Epileptic drop attacks were defined as a sudden fall without warning, accompanied or not by loss of consciousness.^{2,13}

For each patient we evaluated sex and age, family history of epilepsy, aetiology and presence of neuroradiological focal lesions, age at seizure onset, seizure frequency and semiology, neurological examination, ictal and interictal EEG, and clinical outcome.

We defined frontal or temporal or extratemporal lobe seizures on the basis of seizure semiology, interictal scalp EEG, and video-EEG recording of seizures.

We defined as drug resistant patients whose seizures, at the end of the follow up, recurred more than once a week despite multiple antiepileptic drug trials, with monotherapy or polytherapy, at the highest tolerable dose.

Statistical analysis was performed with student's *t* test or χ^2 test when appropriate.

Results

Our epileptic drop attacks group consisted of 20 men and 11 women aged from 20 to 55 (mean age 33 years, table 1). Mean duration of epilepsy was 22 years. We found a positive family history for epileptic disturbances in nine

patients (29%); 16 patients (52%) had symptomatic partial epilepsy: two had anamnestic prenatal and perinatal pathology, and 14 had neuroradiological focal lesions (four focal atrophy and gliosis, four porencephalic lesions, three cortical calcified lesions, one focal pachygyria, one hippocampal sclerosis, one brain tumour). Age at the onset of seizures was from 1 to 45 (mean 12 years) and was under 10 years in 16 (52%) cases. Age at onset of epileptic drop attacks was from 3 to 45 (mean 19 years). Drop attacks occurred in 26 patients (84%) from one to 34 years after onset of epilepsy. Seizures in the other five cases (patients 5, 8, 22, 25, and 27) were characterised from the beginning by both drop and partial attacks (three patients with frontal epilepsy and two patients with temporal lobe epilepsy). Seizure frequency during the first two years of illness was more than one a week in 18 (58%) patients.

Fourteen (45%) patients showed mental disability; three of these were mentally retarded and 11 showed a progressive intellectual deterioration.

Interictal EEG focus (table 2) was frontal or frontotemporal in 19 (61%) patients (frontal in 12, frontotemporal in seven), mid-temporal in two (6%), parietal or occipital in one (3%), and multiple independent in nine (29%); secondary bilateral synchrony were present in 23 patients (74%). Focal abnormalities in patients

Table 2 Interictal EEG and Ictal semiology of recorded seizures

Patient No	Interictal EEG focus	Ictal clinical and EEG features of recorded seizures
1	F+SBS	PA: diffuse spike and wave activity predominant on the anterior regions
2	MI+SBS	CP1: polyspike activity on the right temporal region
3	MI+SBS	PA: diffuse flattening of background activity
4	L F	PA: diffuse spike and wave activity predominant on the left frontal region
5	L FT+SBS	CP2: polyspike activity on the left frontotemporal region
6	L F+SBS	PA: diffuse slow spike activity predominant on the anterior regions
7	MI+SBS	CP2: low voltage fast activity on the left temporoparietal-occipital region
8	MI+SBS	PA: diffuse spike and wave activity
9	R F+SBS	PA: diffuse spike and wave activity
10	R F+SBS	CP2: diffuse spike and wave activity, Atonic drop: low voltage fast activity followed by diffuse spike and wave
11	R FT+SBS	PA: diffuse spike, polyspike and wave activity
12	L F+SBS	—
13	MI+SBS	CP3: diffuse flattening of background activity
14	LT+SBS	CP2: diffuse flattening of background activity followed by low voltage fast activity on the anterior regions
15	R F+SBS	—
16	MI+SBS	PA: diffuse spike and wave activity, Tonic drop: diffuse low voltage fast activity predominant on the right frontal region
17	R PO+SBS	CP2: diffuse flattening of background activity
18	R FT+SBS	CP2: diffuse slow spike activity
19	L FT+SBS	—
20	MI+SBS	CP2: diffuse flattening of background activity
21	R F	CP2: right frontal spike activity
22	R F+SBS	PA: diffuse spike and wave activity predominant on the left frontal region
23	R F	CP2: right frontal flattening
24	L FT	CP1: left temporal low voltage fast activity
25	L T	—
26	R FT	—
27	MI	—
28	RFT	—
29	L F+SBS	CP3: diffuse low voltage fast activity predominant on the left frontal region, Tonic drop: diffuse low voltage fast activity predominant on the left frontal region
30	Bil F+SBS	—
31	MI+SBS	CP2: diffuse low voltage fast activity predominant on the right anterior region, Tonic drop: diffuse low voltage fast activity predominant on the right anterior region

Footnotes as for table 1.

with secondary bilateral synchrony were frontal or frontotemporal in 13, temporal in one, parietal or occipital in one, multiple independent in eight. Therefore there was no close correlation between the site of focal cerebral lesion, the EEG focus, and the presence of secondary bilateral synchrony.

All patients had other seizures besides drop attacks. In 23 patients we recorded 91 seizures during video-EEG monitoring (table 2). From a semiological standpoint we divided the recorded seizures into five groups: pseudoabsence attacks with very brief loss of contact with staring and

minimal automatisms sudden in onset and cessation (PA: 19 patients); loss of contact with complex oroalimentary automatisms with or without abdominal rising aura (CP1: four patients); vocalisation, tonic extension of head, neck, and trunk with impaired consciousness followed by oroalimentary automatisms (CP2: 16 patients); asymmetric tonic fit with impaired consciousness (CP3; two patients); nocturnal complex motor automatisms involving both legs, arms, and trunk, pelvic up-down movements; and bipedal movements similar to bicycling (CP4; one patient). Reviewing

Figure 1 Interictal EEG of patient 16. Focal spikes on the right temporal region spread bilaterally in diffuse spike and wave complexes.

Figure 2 Video-polygraphic recording of a drop seizure in patient 16. (A) Clinical sequence of the seizure. (1) The patient is standing up. He wears a safety jacket to prevent him hitting the floor. (2) A tonic contraction of the facial, axial, and upper limbs muscles appears. The patient bends his head and trunk slightly forwards. (3–5) One second later he begins to drop. The tonic posture is still evident. (6–7) The patient remains unresponsive, motionless, for 31 seconds. He is supported by the completely jacket. (8) He begins to get up without help. (9) The seizure is over. The seizure lasts 51 seconds. (B) Simultaneous polygraphic recording. The number indicates the sequence of the photographs in (A).

videopolygraphic documents and anamnestic data we hypothesised that PA, CP2, and CP3 seizure types could result, while standing up, in a sudden fall. We recorded 11 drop seizures while standing up in four patients (patients 10, 16, 29, and 31). Polygraphic features of falls

show that in one patient the drop was due to a brief atonia of antigravitational muscles (patient 10) and in three patients to an asymmetric tonic contraction of four limbs and trunk muscles (patients 16, 29, 31). Figure 1 shows the interictal EEG and figure 2 the clinical sequence (A)

B

Sba III Male 35 y No 21425

and the polygraphic recording (B) of a drop seizure in patient 16.

The electrical seizure begins with a burst of spike and wave activity, synchronous on both hemispheres but with a higher amplitude on the anterior regions. The clinical onset (2) coincides with the appearance of a high amplitude slow wave, with a superimposed high frequency activity and followed by a recruiting rhythm (3)

lasting seven seconds. Polygraphically the fast EEG activity of the beginning of the seizure is accompanied by a tonic activation of the axial and upper limb muscles. The contraction of the legs appears one second later and is asymmetric. Once he has dropped, the patient maintains a muscle tone in the upper limbs. The tone is absent in the legs. During the unresponsive state the EEG shows a continuous spike and wave

Table 3 Statistical comparison between patients with epileptic drop attacks (EDA group) and those without (control group)

	EDA group (n=31)	Control group (n=191)	p Value
Sex	20M 11F	106M 85F	NS
Age (y, mean (SD))	33 (14)	33 (14)	NS
Mean duration of epilepsy (y, mean (SD))	22 (12)	20 (12)	NS
Family history of epilepsy	9	49	NS
Lesional aetiology	16	88	NS
Age at seizure onset <10 y	16	77	NS
Seizure frequency during the first 2 years of illness > one a week	18	127	NS
Mental deterioration	14	26	< 0.001
Secondarily generalised seizures	20	115	NS
Changes in seizure type during evolution	22	75	< 0.002
Secondary bilateral synchrony	23	25	< 0.001
Drug resistance	23	63	< 0.001

activity more evident over the anterior regions. After the drop bradycardia occurs provoked, in our opinion, by the Valsalva manoeuvre secondary to the tonic contraction and the squatting position.

According to ictal semeiology and EEG features we defined 18 patients with frontal lobe epilepsy, two with temporal lobe epilepsy, and three with multifocal or undefinable onset epilepsy. We were unable to record seizures in eight patients (patients 12, 15, 19, 25, 26, 27, 28, and 30); we hypothesised a frontal lobe onset in four patients, temporal in three, and undefinable onset in one patient according to the anamnestic semeiological features and the interictal EEG, so we had 22 patients with frontal lobe epilepsy, five with temporal lobe epilepsy, and four with multifocal or undefinable epilepsy (table 1).

The clinical outcome (table 1) was unfavourable (drug resistant epilepsy) in 23 patients (74%) of whom 17 had frontal, three had temporal, and three had multifocal or undefinable lobe epilepsy; 19 out of the 23 drug resistant patients showed an EEG pattern of secondary bilateral synchrony. At the end of follow up 16 patients (52%) still had epileptic drop attacks (12 with frontal, two with temporal, and two with multifocal or undefinable lobe epilepsy): they were all drug resistant, 10 had mental deterioration (eight with frontal, one with temporal lobe epilepsy, and one with undefinable epilepsy), whereas among the 15 patients without epileptic drop attacks (10 with frontal, three with temporal, and two with temporoparietal-occipital lobe epilepsy) only seven were drug resistant and three were mentally retarded.

We made statistical comparisons of some clinical and EEG features between our patients with epileptic drop attacks and those without (control group; table 3). Among the variables analysed those strongly linked to epileptic drop attacks ($p < 0.002$) were mental deterioration, changes in seizure type during evolution of illness (more than two types of seizures), drug resistance, and presence of secondary bilateral synchrony on interictal EEG.

Discussion

From the general clinical features of our population with epileptic drop attacks we did not find any predominant characteristic; family predisposition was not relevant, age at onset of epilepsy ranged from 1 to 45 years, and lesional

factors were documented in about half of our patients. Only five of our patients began having epileptic drop attacks at the beginning of their epilepsy, always associated with other kinds of seizures. The large majority of our series added epileptic drop attacks during evolution of their illness with a delay of one to 34 years.

Intractable epileptic drop attacks are a disabling seizure type characteristic of patients with Lennox-Gastaut syndrome. In this condition falling is due to tonic, atonic, or myoclonic-astatic seizures.¹⁴ Epileptic falls, however, have been described in partial epilepsies, especially of frontal lobe origin.^{2-5 8 9} Other authors^{6 7 15} hypothesised a temporal lobe origin. In our series 71% of the patients had a frontal lobe epilepsy. As recently reported by other authors,¹⁶ we found that the fall in our patients is due to a sudden, asymmetric tonic contraction of axial and limb muscles or, in other patients, to a sudden atonia involving the postural muscles. Irrespective of the site of origin of the ictal discharge, almost all literature reports concur that the physiopathogenetic substrate of the epileptic drop attacks is a mechanism of secondary bilateral synchrony. This consists of a rapid spread of the focal ictal discharge via the corpus callosum and the hippocampal commissure. For other investigators,^{7 17} the focal discharge via the corticoreticular pathways involves the pontine reticular formation, producing a motor inhibition such as in cataplectic attacks.¹⁸ Neurosurgical series confirm the efficacy of callosotomy¹⁹⁻²⁴ and patients with an EEG pattern of secondary bilateral synchrony, drop attacks, and partial seizures had a better outcome than patients with generalised epilepsy. Seventy four per cent of our patients showed a typical EEG pattern of secondary bilateral synchrony during evolution. This pattern has been considered a negative EEG prognostic factor in partial epilepsy.^{4 9}

It is not yet clear which physiopathological mechanisms support the appearance of secondary bilateral synchrony but we think, as previously suggested,^{8 9 25} that a progressive cerebral disturbance from a localised epileptic focus leads to a process of secondary epileptogenesis involving the whole brain.²⁶ We hypothesise that a focal cortical discharge, mainly frontal, abruptly spreads to both hemispheres via the corpus callosum (secondary bilateral synchrony mechanism) subsequently acting on structures

controlling the postural tone in an activatory (tonic) or inhibitory (atonic) manner.

Seventy four per cent of our patients had a poor prognosis and 45% were mentally retarded. Mental retardation significantly differs for patients with epileptic drop attacks and the control group. In our opinion therefore, the mental deterioration depends on the poor outcome, the frequent drops, and the process of secondary epileptogenesis.

Fifty two per cent of patients with epileptic drop attacks continued to present drops more than once a week at the end of the follow up and all of them were drug resistant. These characteristics, together with the presence of secondary bilateral synchrony on EEG and the variability of seizures during evolution of the illness, are statistically different from our control group. The high seizure frequency added to the continuous danger of a sudden fall makes the daily life of these patients extremely difficult.

Among patients with partial epilepsies prospective studies must distinguish the population at risk of developing epileptic drop attacks during evolution. Furthermore, videopolygraphic studies are needed to analyse the different motor sequences leading to the falls depending on the different site of origin and diffusion of the ictal discharges. The possibility of documenting the way each patient falls will be useful to teach patients and their families the effect of the drops and therefore improve preventive strategies as features of different helmets, knee-pads, and other protective devices.

We thank Ms Elena Zoni for technical assistance in preparing the manuscript and the EEG technicians of our EEG laboratory (F Bisquoli, C Fiore, R Monfreda, A Pallotti) for their skill and cooperation.

- 1 Commission on classification and terminology of the international league against epilepsy. Proposal for revised classification of the epilepsies and epileptic syndromes. *Epilepsia* 1989;30:389-99.
- 2 Pazzaglia P, D' Alessandro R, Ambrosetto G, et al. Drop attacks: an ominous change in the evolution of partial epilepsy. *Neurology* 1985;35:1725-30.
- 3 Geier S, Bancaud J, Talairach J, et al. The seizures of frontal lobe epilepsy. A study of clinical manifestations. *Neurology* 1977;27:951-8.
- 4 Delgado-Escueta AV, Swartz BE, Maldonado HM, et al. Complex partial seizures of frontal lobe origin. In: Wieser HG, Elger CE, eds. *Presurgical evaluation of epileptics*. Berlin: Springer-Verlag, 1987:267-99.
- 5 Palmini A, Andermann F, Olivier A, et al. Focal neuronal migration disorders and intractable partial epilepsy: a study of 30 patients. *Ann Neurol* 1991;30:741-9.
- 6 Jacome DE. Temporal lobe syncope: clinical variants. *Clin Electroencephalogr* 1989;20:58-65.
- 7 Gambardella A, Reutens DC, Andermann F, et al. Late onset drop attacks in temporal lobe epilepsy: a reevaluation of the concept of temporal lobe syncope. *Neurology* 1994;44:1074-8.
- 8 Gastaut H, Mouren P, Paillas J. A propos de la "bisynchronie secondaire" en électroencéphalographie: paroxysmes bilatéraux, synchrones et symétriques révélateurs d'absence temporal. *Rev Neurol* 1968;119:295-8.
- 9 Gastaut H, Zifkin B, Maguadda A, et al. Symptomatic partial epilepsies with secondary bilateral synchrony: differentiation from symptomatic generalized epilepsies of the Lennox-Gastaut type. In: Wieser HG, Elger CE, eds. *Presurgical evaluation of epileptics*. Berlin: Springer-Verlag, 1987:308-16.
- 10 Gabor AJ, Ajmone-Marsan C. Co-existence of focal and bilateral diffuse paroxysmal discharges in epileptics. *Epilepsia* 1969;10:453-72.
- 11 Niedermeyer E. The occurrence of generalized (centrencephalic) and focal seizure patterns in the same patients. *John Hopkins Med J* 1968;112:11-25.
- 12 Niedermeyer E, Laws ER, Walker AE. Depth EEG findings in epileptics with generalized spike-wave complexes. *Arch Neurol* 1969;21:51-8.
- 13 Ikono T, Shigematsu H, Miyakoshi M, et al. An analytic study of epileptic falls. *Epilepsia* 1985;26:612-21.
- 14 Tassinari CA, Ambrosetto G. Tonic seizures in the Lennox-Gastaut syndrome: semiology and differential diagnosis. In: Niedermeyer E, Degen R, eds. *The Lennox-Gastaut syndrome. Neurology and neurobiology*. Vol 45. New York: Allan R Liss, 1988:109-24.
- 15 Holmes GL. Partial complex seizures in children: an analysis of 69 seizures in 24 patients using EEG FM radiotelemetry and videotape recording. *Electroencephalogr Clin Neurophysiol* 1984;57:13-20.
- 16 Tassinari CA, Rubboli G, Meletti S, et al. Epileptic falling seizures in standing patients [abstract]. *Epilepsia* 1996;37(suppl 5):31.
- 17 Smith T. Cataplexy in association with meningiomas. *Acta Neurol Scand* 1983;94(suppl):45-7.
- 18 Siegel JM, Nienhuis R, Fahringer HM, et al. Neuronal activity in narcolepsy: identification of cataplexy-related cells in the medial medulla. *Science* 1991;252:1315-8.
- 19 Wilson DH, Reeves AG, Gazzaniga MS. Central commissurotomy for intractable generalized epilepsy: series two. *Neurology* 1982;32:687-97.
- 20 Gates JR, Leppik IL, Yap J, et al. Corpus callosotomy: clinical and electroencephalographic effects. *Epilepsia* 1984;25:308-16.
- 21 Spencer SS, Spencer DD, Williamson PD, et al. Effects of corpus callosum section on secondary bilaterally synchronous interictal EEG discharges. *Neurology* 1985;35:1689-94.
- 22 Gates JR, Rosenfeld WE, Maxwell RE, Lyons RE. Response of multiple seizure types to corpus callosum section. *Epilepsia* 1987;28:28-34.
- 23 Spencer SS. Corpus callosum section and other disconnection procedures for medically intractable epilepsy. *Epilepsia* 1988;29(suppl 2):S85-99.
- 24 Oguni H, Olivier A, Andermann F, et al. Anterior callosotomy in the treatment of medically intractable epilepsies: a study of 43 patients with a mean follow-up of 39 months. *Ann Neurol* 1991;30:357-64.
- 25 Tinuper P, Cerullo A, Riva R, et al. Clinical and EEG features of partial epilepsy with secondary bilateral synchrony. *J Epilepsy* 1995;8:210-4.
- 26 Gloor P. Generalized cortico-reticular epilepsies. Some considerations on the pathophysiology of generalized bilaterally synchronous spike and wave discharge. *Epilepsia* 1968;9:249-63.