

Surgical treatment of epilepsy due to cortical dysplasia: clinical and EEG findings

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Abstract

Seventeen patients with cortical dysplasia who had surgical resection for medically intractable partial epilepsy were studied. Compared with two groups of surgically treated patients with intractable epilepsy due to tumour (n = 20) and mesial temporal sclerosis (n = 40), patients with cortical dysplasia showed significantly more frequent extratemporal lesions, more frequent non-epileptiform EEG abnormalities and less favourable surgical outcome for seizure control. Patients with cortical dysplasia were younger at onset of seizures and had a lower detection rate of CT abnormalities compared with the tumour group, and lower IQ compared with the mesial temporal sclerosis group. MRI was abnormal in five of seven patients. Six patients became seizure-free or almost seizure-free but eight did not experience relief of seizures. Surgical outcome related to the extent of pathology but not to the histological abnormality. Lesions outside the temporal and frontal lobes were correlated with poor surgical outcome, as were generalised interictal EEG abnormalities, which may reflect extensive or multiple lesions. Ictal intracranial recordings were not useful for presurgical evaluation of cortical dysplasia.

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Cortical dysplasia (CD) is a disorder derived from a defect of neuronal migration with a disruption of the normal cortical lamination by abnormally large nerve cells and large astrocytes in the cerebral mantle as a prominent pathological feature. It is not necessarily confined to the cortex, but sometimes involves the underlying white matter and can be extensive.¹ The relation with the incomplete form of tuberous sclerosis has been a matter of discussion and some authors regard them as the same entity.² An association with other types of migration disorder such as lissencephaly, hemimegalencephaly or some neurocutaneous syndromes has also been recognised.^{2,3}

A relationship to epilepsy was first reported by Taylor *et al* in 1971.⁴ However, surprisingly few reports have been published since then, although CD was assumed to be found

in 2-5% of all surgically treated epileptic patients.¹ The significance of CD amongst the patients with intractable partial epilepsy and the surgical implications of this pathology remain to be clarified. We carried out this study to characterise the clinical and EEG features of CD compared with other pathological processes which also caused intractable epilepsy requiring surgical treatment, and to identify some factors correlating with surgical outcome. As the lesions of CD are often widespread or multiple, we predicted that this condition would be characterised by multiple or generalised EEG abnormalities and that these features would be associated with an unfavourable surgical outcome.

Material and methods

PATIENTS

Twenty patients with CD were found amongst the 297 who had surgical treatment in the Maudsley Hospital from January 1980 to December 1990 because of intractable epilepsy. Seventeen patients (9 men: 8 women) who have been followed up for more than two years (2-11 years, mean 5 years) are the subjects of this study. Two patients with hemimegalencephaly, two patients with neuronal heterotopias and one patient with the sebaceous linear naevus syndrome of Jadassohn were not included as CD.

The two pathologies most common in this surgical population were selected amongst the 162 who had surgery for epilepsy from January 1976 to December 1986 as the control groups to be compared with CD. Control group 1 consisted of 20 patients with tumour (TUM), most of which were dysembryoplastic neuroepithelial tumours.⁵ Control group 2 consisted of 40 patients with mesial temporal sclerosis (MTS), which included patients with hippocampal sclerosis or Ammon's horn sclerosis but excluded any cases with other concomitant pathologies or non-specific changes.

All patients received extensive evaluation within the half year before surgery at the Neurosurgical Unit of the Maudsley Hospital, including routine surface and usually sphenoid EEG, video-EEG telemetry usually with foramen ovale recordings, neuroradiology and psychometry. Intracranial recordings with subdural electrodes and/or stereotaxically inserted depth electrodes were performed when less invasive methods were considered

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not to have located the site of seizure onset. Carotid amygdala (Wada) testing was also carried out on candidates for temporal lobectomies if necessary.

At operation, all but one patient had acute electrocorticography (ECoG) to confirm the electrophysiological focus and the extent of resection. All resected material was taken *en bloc*, which enabled thorough pathological examination.

Regular follow up was carried out in all patients at six month intervals for at least the first two years after surgery and at one year intervals thereafter.

CLINICAL STUDIES

Age at onset of seizures, age at operation, type of seizures, frequency of seizures, IQ, neurological findings, neuroradiological findings, site of lesion, laterality of lesion, type of operation and surgical outcome were recorded in each patient.

Seizures were divided into three categories: complex partial seizures only, complex partial plus secondarily generalised tonic clonic seizures and mixed seizures if they had a third pattern, for example, versive or unilateral motor seizures in addition to complex partial, or complex partial seizures and generalised tonic-clonic. "Drop attacks" accompanying complex partial seizures were included in the complex partial category. Seizure frequency was also divided into three groups: "daily", "weekly" and "monthly", an average of at least one seizure per day, week or month respectively. IQ according to the Wechsler test was divided into: "normal": either verbal or performance IQ over 90; "retarded": both verbal and performance IQ under 69; and "low normal": the range between the former two. CT scans were performed on all patients and MRI studies in seven. Site of lesion was divided into three groups: temporal, frontal and others. Type of operation was also divided into three groups: anterior temporal lobectomy, subtotal frontal lobectomy and other types of resection. No hemispherectomies were included in this series. Two patients (16 and 17) had multiple subpial transection besides frontal resection. Surgical outcome was evaluated for seizure control at the point of two year follow up and graded in five groups according to a modification of Crandall's surgical therapeutic group classification⁶ by Hardiman *et al.*:⁷ 1) seizure-free; no seizures/ aura only/ few seizures but none for two years or more; 2) rare seizures; one per year/ long periods seizure-free/ only infrequent nocturnal seizures; 3) worthwhile improvement; one per month/ greater than 50% reduction in seizure frequency; 4) no worthwhile improvement; less than 50% reduction in seizure frequency; 5) no improvement; same or worse in seizure frequency or strength.

Findings in the CD group were compared with those of control group 1 and control group 2. The correlation between surgical outcome and remaining clinical variables was also examined.

ELECTROENCEPHALOGRAPHIC STUDIES

The EEGs were reviewed by a single assessor who was not blind to the diagnosis. The interictal surface EEG was assessed for ongoing activity over posterior regions, slowing, the topography of focal spikes and the presence of generalised paroxysmal discharges in each patient. The electrodes were applied according to the Maudsley system,⁸ which resembles the 10-20 system but provides more extensive coverage of the temporal regions.

Ongoing activity was classified as "normal", "asymmetrical" and "slow". Slowing was classified as "minimal", "localised" and "widespread" regionally or hemispherically. Focal spikes were divided into three categories: "localised" with a maximum confined to one electrode or to temporal electrodes only; "regional" with maxima not confined to one electrode but occurring also at two or three adjacent electrodes; and "other" with multifocal independent spikes or bilateral discharges. Generalised paroxysmal discharges included diffuse irregular spike-and-wave activity or multiple spikes-and-waves but excluded diffuse high voltage slow bursts.

Repeated EEGs had been taken in most patients. There were no patients who showed no abnormal focal discharges on serial surface EEGs. In the cases showing fluctuating or changing EEGs, the last record taken in our hospital before surgery was used for the purpose of our analysis.

Pre-resection ECoG findings were classified by the combination of the extent and the amount of discharges into four groups: "scanty": infrequent spikes; "localised": numerous but fairly localised spikes; "regional": numerous spikes confined to one lobe but multifocal or poorly localised; and "widespread": widespread discharges beyond the extent of the resection. Post-resection ECoG spike discharges were classified in comparison with pre-resection ECoG discharges into: considerably reduced or absent (I); reduced by at least 50% (II); or not so reduced or almost the same as those before resection (III).

These variables were compared with those in control group 1 or control group 2. Ictal findings obtained by surface EEG or intracranial recordings were also compared with interictal findings on the surface EEG. The correlation between these variables and surgical outcome was also examined.

PATHOLOGICAL STUDIES

All resected specimens were examined both macroscopically and histologically in the department of neuropathology of the Institute of Psychiatry, London. Specific immunohistochemical staining including that with antisera to glial fibrillary acidic protein (GFAP) and electromicroscopic studies were often performed in addition to the routine stains. The purpose of the operation and site of origin of the resected material were known to the pathologist, otherwise the assessment was carried out blind to clinical features.

Table 1 Clinicopathological findings

Case	Age onset	Type of seizure	Seizure frequency	IQ	Neurological findings	CT	MRI	Age op	Lat op	Type op	Pathological comments	Seizure outcome
1	8	Mixed	D	N	N	N		25	R	FR		I
2	4	CPS	D	N	A	N		15	R	TL	Lissencephaly	I
3	0	Mixed	M	R	N	C		1	L	FR	Localised lesion	II
4	0	Mixed	D	R	N	C + LDA		2	L	POR	Lissencephaly	IV
5	4	CPS + GTC	W	L	N	N		17	L	FR		III
6	0	Mixed	W	R	A	N		11	L	POR		V
7	1	CPS + GTC	M	L	N	N		10	R	FL		II
8	2	CPS	D	N	A	N		15	L	CR	Extensive lesion	IV
9	18	CPS + GTC	D	N	N	N	A	38	L	PrCR	Nerve cells only	V
10	9	Mixed	D	N	N	N	N	13	R	FL	Few nerve cells only	V
11	1	Mixed	D	L	N	N	A	19	R	FR	Extensive lesion	III
12	2	CPS + GTC	M	L	N	N		24	R	TL	Glial cells only + lissenceph.	I
13	7	CPS + GTC	D	N	N	N		23	L	TL	Glial cells only	III
14	1	Mixed	D	R	N	HDA	A	4	R	FL	Localised lesion	II
15	2	CPS + GTC	D	L	N	N	A	15	L	FL	Extensive lesion	V
16	0	Mixed	W	L	A	N	N	13	R	FR + MST	Nerve cells only	V
17	0	Mixed	D	N	N	N	A	19	R	FR + MST		IV

Key:
 Seizure frequency D: Daily; W: Weekly; M: Monthly;
 IQ N: Normal; L: Low normal; R: Retarded
 CT/MRI N: Normal; A: Abnormal; C: Calcification; LDA: Low density area; HDA: High density area
 Operation FR: Frontal resection; FL: Frontal lobectomy; TL: Temporal lobectomy; PrCR: Precentral resection; CR: Central resection; POR: Parieto-occipital resection; MST: Multiple subpial transection; R: Right; L: Left
 Outcome Classification (after Hardiman *et al*⁷): I: seizure-free; no seizures/ aura only/ few seizures but none for two years or more; II: rare seizures; one per year/ long periods seizure-free/ only infrequent nocturnal seizures; III: worthwhile improvement; one per month/ greater than 50% reduction in seizure frequency; IV: no worthwhile improvement; less than 50% reduction in seizure frequency; V: no improvement; same or worse in seizure frequency or strength.

When specific comments concerning the extent of the lesion and the histological abnormality were available in the reports, the relevance of these to clinical and EEG findings was also examined.

Results

CLINICAL FINDINGS

Clinical profiles of each patient with CD are summarised in table 1. Age at onset of seizures ranged from birth to 18 years (mean 3.5 years of age), and age at operation was from one to 38 years (mean 15.5 years of age). The seizures were most commonly of mixed type, predominantly with versive features, seen in eight patients, seven of whom had frontal lesions. CT scan revealed abnormal findings in three patients: a microcalcification in the frontal lobe (patient 3); a lucent area with some calcification in the parieto-occipital region (4); and a high density area in the frontal lobe (14). MRI showed abnormal findings in five of seven patients studied. Common features of MRI findings were: 1) irregular, sometimes multiple, high intensity areas in the white matter, and 2) blurred demarcations between the grey and white matter. T2-weighted images revealed the former more clearly and T1-weighted images the latter. Postoperatively, six patients became seizure-free (I) or almost seizure-free (II) and three experienced worthwhile improvement (III), but eight showed no worthwhile (IV) or no (V) improvement. Among those with no improvement, no patients showed a progressive deterioration.

Comparisons between CD and two control groups for each clinical variable are shown in table 2. Compared with control group 1 (TUM), the patients with CD were significantly different in age at onset, type of seizures, site of lesion, type of operation, the presence of CT abnormalities and surgical outcome. Compared with control group 2 (MTS), they were significantly different in

type of seizures, IQ and surgical outcome in addition to the difference in site of lesion and type of operation. Extratemporal lesions, mixed seizures and poor surgical outcome were therefore the distinguishing clinical features of the patients with CD compared with both TUM and MTS groups.

When the correlation between surgical outcome and other clinical variables was tested (Mann-Whitney test), lesions located in other than temporal and frontal lobes ($U = 10.0$, $p = 0.1$) showed a trend towards having a poor surgical outcome. Infrequent seizures with a monthly interval preoperatively were seen only in patients with favourable (I + II) outcome.

ELECTROENCEPHALOGRAPHIC FINDINGS

EEG and ECoG data in each patient are shown in table 3. Ictal recordings were obtained in 10 patients (7 with surface EEG, 3 with intracranial recordings). Concerning the correlation between interictal and ictal recordings, 7 patients showed ictal localisation almost identical to or more discrete than that in interictal recordings. One patient (4) showed a discrepancy between interictal and ictal findings. Another two patients (11, 15) showed no clear focal onset on ictal EEG. There was no difference for surgical outcome between the former group and the latter two groups. Intracranial recordings were carried out in three patients whose epileptogenic areas were not clearly determined by surface EEG. These patients all had a poor surgical outcome, although the epileptogenic areas disclosed by ictal intracranial recordings were all within the resected areas.

No significant correlation was found between "localised" focal spikes on surface EEG and relatively confined ("scanty" plus "localised") discharges on pre-resection ECoG. However, no patients with "localised" focal spikes on surface EEG showed "widespread" ECoG discharges.

Comparisons between CD and other two

Table 2 Comparisons of patients with cortical dysplasia and two control groups with benign tumour or mesial temporal sclerosis

Variables	Cortical dysplasia (n = 17)	Tumour (n = 20)	Mesial temporal sclerosis (n = 40)
Age at onset	3.5 + 4.7 (0-18)	8.4 + 6.0* (0-21)	6.6 + 4.2 (0-15)
Age at op	15.5 + 8.9 (1-38)	16.5 + 8.2 (3-33)	22.4 + 7.6 (8-39)
Seizure type			
CPS	2	9	20
CPS + GTC	6	9	20
Mixed	9	2	0
Seizure frequency			
Monthly	3	2	8
Weekly	3	6	18
Daily	11	9	11
IQ			
Normal	7	15	33
Low normal	6	3	6
Retarded	4	2	0
Neuro findings			
Negative	12	13	38
Positive	5	7	2
CT findings			
Normal	14	1	15
Abnormal	3	14	3
Site of lesion			
Temporal	3	15	40
Frontal	10	3	0
Others	4	2	0
Operation			
TL	3	14	39
FL	5	1	0
Other	9	5	0
Side			
Left	8	10	25
Right	9	10	15
Surgical outcome			
I	3	13	25
II	3	4	4
III	3	3	7
IV	3	0	4
V	5	0	0
EEG: Background			
Normal	3	18	27
Asymmetrical	10	1	6
Slow	4	1	7
EEG: Slowing			
Minimal	4	12	29
Localised	4	4	5
Widespread	9	4	6
EEG: Focal spikes			
Localised	7	5	24
Regional	7	6	11
Other	3	9	3
EEG: General dis			
Absent	11	17	35
Present	6	3	4

*p < 0.02; **p < 0.01; ***p < 0.001, with respect to cortical dysplasia group.

control groups of each interictal surface EEG variable are shown in table 2. Compared with control group 1 (TUM), patients with CD were significantly different for ongoing activ-

ity. Compared with control group 2 (MTS), they were significantly different for ongoing activity and slowing.

When the correlation between surgical outcome and the interictal surface EEG and ECoG variables was tested, as predicted, "widespread" slowing ($U = 17.0$, $p < .05$), non-"localised" focal spikes ($U = 4.0$, $p < .002$) and the existence of generalised paroxysmal discharges ($U = 15.0$, $p < .05$; one-tailed) on surface EEG were significantly correlated with poor surgical outcome. All patients with "normal" ongoing activity showed a good or moderately favourable (I-III) surgical outcome, but this trend was not significant. ECoG findings, either before or after resection, showed no significant relationship to outcome, but all patients with "widespread" discharges on pre-resection ECoG and without reduction of spikes (III) on post-resection ECoG had a poor surgical result. Neither of the two patients with "scanty" spikes on pre-resection ECoG had a good result.

No significant relationship was found between site of lesion and any of the EEG and ECoG variables.

PATHOLOGICAL FINDINGS

Specific comments concerning the extent of pathological lesions and the histological abnormality are also listed in table 1. Three patients had extensive lesions which spread to the edge of the specimen and two patients had localised lesions. In relation to surgical outcome, no patients with extensive lesions showed favourable outcome, whereas both patients with localised lesions showed a favourable one. No specific correlation was found between the histological abnormality and surgical outcome. The details of the pathological findings have been published elsewhere.¹

Discussion

Some characteristic features of CD have emerged in comparison with the two other

Table 3 Electrophysiological findings

Case	Surface EEG					Intracranial SEEG			
	Ongoing activity	Slowing	Focal sp	General dis	Interictal focus	Ictal focus	Ictal focus	Pre ECoG	Post ECoG
1	N	L	L	-	r:sup fr	r:mesial fr		R	I
2	A	L	L	-	r:temp + sphenoid			R	II
3	S	W	L	-	l:sylvian			R	N.D.
4	A	M	R	-	l:pari-occip	l:cent-pari		R	I
5	N	L	L	-	l:sup fr-mid fr			S	I
6	A	L	M	-	l:pari-occip	l:parietal		W	III
7	N	M	L	-	r:sup fr			L	II
8	A	W	R	+	l:cent-vertex	l:central		W	I
9	A	W	R	+	l:fr-cent-temp		l:central	R	III
10	S	W	BiF	+	bifr (r > 1)		r:orbital fr	S	I
11	S	M	R	-	r:pre fr-inf fr	no focal onset		L	II
12	A	M	L	-	r:mid temp			R	II
13	A	W	L	-	l:mid temp			R	III
14	A	W	R	+	r:fr-cent			L	I
15	S	W	R	-	l:pre fr-sup fr	no focal onset		R	I
16	A	W	BiF	+	bifr (r > 1)		r:fr-cent	W	II
17	A	W	R	+	r:fr-cent	r:fr-cent		W	III

Ongoing activity; N: normal; A: asymmetrical; S: slow. Slowing; M: minimal; L: localised; W: widespread.

Focal spikes; L: localised; R: regional; M: multiple; BiF: bi-frontal. Interictal and Ictal focus; fr: frontal; temp: temporal; pari: parietal; cent: central; occip: occipital; sphenoid: sphenoidal electrodes; sup: superior; inf: inferior.

Pre (resection)-ECoG; S: scanty; L: localised; R: regional; W: widespread; ND: not done.

Post (resection)-ECoG; I: discharges markedly reduced or absent; II: reduced by at least 50%; III: not so substantially reduced; ND: not done.

major pathological entities which caused medically intractable partial epilepsy leading to resective surgery. These are: an earlier age at onset of seizures (compared with TUM), lower IQ (compared with MTS), nonepileptiform EEG abnormalities, extratemporal preponderance of lesions, low prevalence of abnormal CT scan (compared with TUM) and poor surgical outcome.

Early onset of seizures is supposed to be due to the congenital nature of CD. As the migration of neurons takes place between seven and 16 weeks gestational age,³ it is likely that the abnormal tissues can develop epileptogenicity in early life. Low IQ and nonepileptiform EEG abnormalities probably indicate more widespread cerebral pathology compared with other groups, which may eventually cause poor surgical outcome. Concerning the site of lesion, 14 of 17 patients showed extratemporal lesions and in particular 10 patients had frontal lobe involvement. The original report by Taylor *et al*⁸ described extratemporal lesions in five of 10 patients including frontal lesions in three.

The low detection rate of abnormalities by CT scan was a feature of CD. Though there is a case report demonstrating the discrete lesions with CT,⁹ it seems the rule for CD not to show definite CT abnormalities. MRI was available for only seven recent cases, but it demonstrated some abnormalities in five patients. A recent report concerning the MRI findings of CD with temporal lobe involvement is encouraging for the presurgical diagnosis.¹⁰ Whether it can detect variants of CD and whether it can accurately indicate the extent of lesions remains to be determined. It was also reported that PET could detect localised hypometabolism due to CD in cryptogenic cases of infantile spasms with no definite MRI abnormalities.¹¹

Previous studies of surgical outcome in CD,^{4 10 12 13} report widely differing results. Thus Bruton¹² found no benefit from surgery in any of his series of eight patients, whereas Kuzniecky *et al*¹⁰ claimed complete or 80% relief of seizures in nine out of 10 patients, it should be noted, all with temporal lesions. No previous studies to our knowledge specifically compare outcome in CD with that in patients with other pathological conditions from the same centre. Our findings suggests a relationship between outcome and site of resection. Temporal lesions showed the most favourable outcome, followed by those frontal lesions amenable to a restricted resection. Extensive frontal lesions, and those outside the frontal and temporal lobes gave consistently poor results. A multicentre survey concerning outcome of epilepsy surgery for seizures also indicates a poorer outcome of extratemporal resection compared with that of anterior temporal lobectomy, irrespective of pathology.¹⁴ Relatively poor outcome in our series seems partly to reflect the inclusion of many cases with extratemporal lesions, particularly outside the temporal and frontal lobes. Though the type of operation did not show a significant correlation with outcome

in our study, it is possible that the resection of lesions outside the temporal and frontal lobes tends to be incomplete because of a concern to avoid unacceptable neurological complications.

The pathological correlate of poor surgical outcome of CD seems to be the extent of the lesions. Two patients with localised lesions had a favourable outcome whereas results in all three patients with extensive lesions were poor. As extensive lesions are difficult to remove completely, a poor result might be expected. This is consistent with a previous report that the extent of lesion removal was the factor most strongly correlating with surgical outcome of CD.¹³ By contrast, the histological abnormalities did not appear to influence the result.

In view of the early age of presentation and the mean interval of 12 years which elapsed between seizure onset and surgery, consideration must be given to the possibility that earlier treatment would have led to a better outcome. The present material offers little evidence of this; admittedly a good result was obtained in two of the three youngest subjects, but both had localised pathology. Overall, the outcome in children and adolescents was not better than in those treated as adults.

A significant predictive value was found in interictal surface EEG, at variance with the report by Palmieri *et al*.¹³ Widespread slowing, non-localised distribution of focal spikes and the coexistence of generalised paroxysmal discharges which were correlated with poor surgical outcome are considered to indicate underlying extensive or multifocal lesions. Some controversies exist concerning the significance of interictal EEG for presurgical evaluation.¹⁵ However, it is suggested that interictal epileptiform activity provides a more useful definition of the optimal extent of epileptogenic lesion to be removed, while ictal recording provides the indispensable information to confirm that the ictal onset zone is indeed within the regions considered for resection.¹⁶

Widespread discharges in the pre-resection ECoG and poor reduction of them in post-resection ECoG implied unfavourable surgical outcome, though no statistical significance was found. Persistence of 50% or more of pre-resective epileptiform activity in post-resection ECoG correlated with poor outcome in a study of temporal resections.¹⁷ It is possible that our ECoG findings also reflect the extensive or multifocal pathology of CD.

On the contrary, ictal EEG, even with intracranial recordings, did not improve surgical outcome, which was poor in all the patients who had intracranial recordings. In our practice only a selected 20% of patients undergo invasive recordings, and it is possibly because of underlying extensive or multiple lesions that these patients failed to show definite or consistent findings on surface EEG, which led to the decision to insert intracranial electrodes. The ictal onset zone which ictal recordings can demonstrate does not neces-

sarily indicate the whole epileptogenic lesion or zone in a structural or pathophysiological sense.¹⁸

In summary, localised focal spikes or slowing on surface EEG possibly suggests an underlying epileptogenic lesion, small or confined enough to be removed successfully. Conversely, widespread, non-localised changes suggest a lesion too extensive or multiple to be removed completely even if intracranial recording can apparently detect an ictal onset zone. It seems important therefore for presurgical determination of the extent of resective areas or the type of operation to consider not only ictal EEG findings but also epileptiform and non-epileptiform features on the interictal surface EEG.

To improve preoperative assessment and identification of the minority of patients with cortical dysplasia who can benefit from surgery, future studies are likely to focus on enhanced imaging techniques, particularly MRI and PET, and the relationship between morphological and EEG findings.

- 1 Janota I, Polkey CE. Cortical dysplasia in epilepsy. In: Pedley TA, Meldrum BS, eds. *Advances in epilepsy* 5. Edinburgh: Churchill Livingstone, 1992:37-49.
- 2 Andermann F, Olivier A, Melanson D, Robitaille Y. Epilepsy due to focal cortical dysplasia and the forme fruste of tuberous sclerosis: a study of 15 patients. In: Wolf P, Dam M, Janz D, Dreifuss E, eds. *Advances in epileptology*, vol 16. New York: Raven Press, 1987:35-8.
- 3 Barth PG. Disorders of neuronal migration. *Can J Neurol Sci* 1987;14:1-16.
- 4 Taylor DC, Falconer MA, Bruton CJ, Corsellis JAN. Focal dysplasia of the cerebral cortex in epilepsy. *J Neurol Neurosurg Psychiatry* 1971;34:369-87.
- 5 Daumas-Duport C, Scheithauer BW, Chodkiewicz J-P, Laws ER, Vedrenne C. Dysembryoplastic neuroepithelial tumor: A surgically curable tumor of young patients with intractable partial seizures. *Neurosurgery* 1988;23:545-56.
- 6 Crandall PH. Postoperative management and criteria for evaluation. In: Purpura DP, Penry JK, Walter RD, eds. *Advances in neurology*, vol 8. New York: Raven Press 1975:265-79.
- 7 Hardiman O, Burke T, Phillips J, Murphy S, O'Moore B, Staunton H, Farrell MA. Microdysgenesis in resected temporal neocortex: Incidence and clinical significance in focal epilepsy. *Neurology* 1988;38:1041-7.
- 8 Margerison JH, Binnie CD, McCaul IR. Electroencephalographic signs employed in the location of ruptured intracranial arterial aneurysms. *Electroencephalogr Clin Neurophysiol* 1970;28:296-306.
- 9 Moreland DB, Glasauer FE, Egnatchik JG, Heffner RR, Alker GJ Jr. Focal cortical dysplasia. Case report. *J Neurosurg* 1988;68:487-90.
- 10 Kuzniecky R, Garcia JH, Faught E, Morawetz RB. Cortical dysplasia in temporal lobe epilepsy: Magnetic resonance imaging correlations. *Ann Neurol* 1991;29:293-8.
- 11 Chugani HT, Shields WD, Shewmon DA, Olson DM, Phelps ME, Peacock WJ. Infantile spasms: I. PET identifies focal cortical dysgenesis in cryptogenic cases for surgical treatment. *Ann Neurol* 1990;27:406-13.
- 12 Armstrong DD, Bruton CJ. Postscript: What terminology is appropriate for tissue pathology? How does it predict outcome? In: Engel J Jr, ed. *Surgical treatment of the epilepsies*. New York: Raven Press, 1987:541-52.
- 13 Palmieri A, Andermann F, Olivier A, Tampieri D, Robitaille Y. Surgical treatment of patients with intractable seizures due to focal or lateralized neuronal migration disorders. *Epilepsia* 1991;32(suppl 1):93-4.
- 14 Engel J Jr. Outcome with respect to epileptic seizures. In: Engel J Jr, ed. *Surgical treatment of the epilepsies*. New York: Raven press, 1987:553-71.
- 15 Engel J Jr. Approaches to localization of the epileptogenic lesion. In: Engel J Jr, ed. *Surgical treatment of the epilepsies*. New York: Raven Press, 1987:75-95.
- 16 Wyllie E, Luders H, Morris HH III, et al. Clinical outcome after complete or partial cortical resection for intractable epilepsy. *Neurology* 1987;37:1634-41.
- 17 McBride MC, Binnie CD, Janota I, Polkey CE. Predictive value of intraoperative electrocorticograms in resective epilepsy surgery. *Ann Neurol* 1991;30:526-32.
- 18 Gloor P. Commentary: Approaches to localization of the epileptogenic lesion. In: Engel J Jr, ed. *Surgical treatment of the epilepsies*. New York: Raven Press, 1987:97-100.