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The contribution of the EEG technologists in the diagnosis of Panayiotopoulos syndrome (susceptibility to early onset benign childhood autonomic seizures)

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Summary *Purpose*: To assess the contribution of the EEG technologists in the diagnosis of children with epileptic seizures. *Methods*: We analysed the clinical information obtained by the EEG technologists from children with epileptic seizures and their parents, and assessed its value for the generation of a clinically useful EEG report and a plausible electroclinical diagnosis. Interviews were based on a qualitative questionnaire, and were videotaped. We focused on Panayiotopoulos syndrome (PS) because it has a high rate of misdiagnosis, usually for encephalitis or other severe cerebral insults. *Results*: Between 1998 and 2001, 424 EEG were performed in 308 children aged 1—14 years, of whom 228 (74%) had one or more epileptic seizures. We diagnosed PS in 14 children (6.1%), mainly based on clinical information. Three other had symptomatic ictal vomiting. In 9 of the 14 children with PS, diagnosis was achieved by the information collected by the EEG technologist. Five of these children were being treated for encephalitis, and management was altered accordingly. In a further three children the diagnosis of PS was confirmed. *Conclusion*: These findings demonstrate that the contribution of the EEG technologists to the diagnosis of people with epilepsies can expand well beyond their established role of recording and describing an EEG. We propose that technologists should be actively involved in prospective electroclinical studies if carefully designed protocols are used.

Introduction

The EEG is the most important investigation in the diagnosis and management of epilepsies especially when it is performed by competent technicians, and carefully studied and interpreted in the context of a well-described clinical setting by physicians with clinical experience in epilepsies. Even

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under these conditions, however, there are factors that can limit the diagnostic yield of the EEG. Lack of obtainable clinical information is the most important. Up to 70—80% of the EEG referrals are for epileptic disorders, and commonly come from general paediatricians or general physicians who may not be familiar with the electroclinical expression of the various epileptic syndromes.[1] In addition, everyday practice in most EEG laboratories shows that a frequently significant and always frustrating proportion of request forms may be inadequately completed, and that clinical information is often poor or missing.

Recently, there has been a promising tendency for non-medical health professionals, such as epilepsy nurses and social workers, to become more involved in the management, including diagnosis of people with epilepsies.[2,3] The principal role of the EEG technologists is to obtain a high quality recording and an astute factual report. An EEG technologist spends 15—20 min preparing a patient for recording; this time may be valuably used to obtain additional clinical information, such as on the possibility of minor seizures, precipitating factors, circadian distribution and other aspects of the patient's symptomatology that will allow tailoring of the recording to the clinical needs and later, optimal EEG interpretation.

An example of how the contribution of the EEG technologists may be upgraded to better assist the diagnostic yield of the EEG in patients with epilepsies is illustrated by our approach to diagnosing benign focal epilepsies of childhood, and in particular Panayiotopoulos syndrome (PS). PS is a newly recognised condition that lies within the spectrum of benign childhood seizure susceptibility syndrome.[4] It is characterised by mainly autonomic focal seizures and functional spikes of multiple topography. Autonomic symptoms and signs (mainly vomiting) occur from the onset in 80% of seizures, half of which last more than 30 min amounting to status epilepticus. Other ictal autonomic changes may include pallor, mydriasis, cardiorespiratory, gastrointestinal and thermoregulatory alterations, incontinence, and hypersalivation. Syncope-like events may also occur during the ictal sequence. More conventional ictal manifestations include eye deviation, staring, and hemi or generalised convulsions. Speech arrest, hemifacial spasms, visual hallucinations and oropharyngolaryngeal movements occur less frequently, suggesting a maturation related continuum with Rolandic epilepsy.[5—10]

The diagnosis of PS may be easily missed. Most general practitioners and paediatricians are not familiar with the notion that prominent autonomic symptoms and signs may occur as epileptic seizure manifestations: in PS mild and brief ictal autonomic symptoms in the presence of clear consciousness would suggest trivial non-epileptic conditions such as atypical migraine, gastroenteritis or syncope, while prolonged and severe attacks may simulate life threatening insults such as encephalitis, for which many of these children are treated.[11] It may not be surprising that in the absence of adequate clinical information some clinical neurophysiologists, particularly if not experienced in paediatric electroencephalography and epilepsies, may interpret an EEG with multifocal spikes as suggestive of multifocal epilepsy with poor outcome,[12] thus contributing to misdiagnosis.

The purpose of this report is to demonstrate the contribution of our EEG technologists in the diagnosis of PS.

Methods

EEG methodology

All EEG recordings are performed with digital EEG machines (Nicolet-Voyageur) with montage reformatting capabilities. Electrodes are positioned according to the international 10—20 system. A standard EEG is a 15- to 20-min recording during alert wakefulness with sessions of overbreathing and photic stimulation. The EEG technologist however is expected to tailor the test according to the clinical hints that he or she might derive either from the referral form or the information acquired during patient's preparation for the test. The first pages of the EEG may also provide clues. In children with centro-temporal spikes from the outset of the recording, or in those with a history suggestive of Rolandic seizures, for example, we place additional C5 and C6 electrodes halfway between C3 and T3, and C4 and T4 respectively,[13] while we ask those with generalised discharges to count their breaths aloud during hyperventilation to detect possible ictal cognitive impairment on video-EEG.[14] We often recommend sleep recordings when routine EEGs are inconclusive, especially on patients with strong clinical evidence of epilepsy. However, not infrequently in such cases the responsible technologist decides to proceed with recording during natural sleep by allowing—if possible—the patient to dose. For sleep EEGs, patients are partially sleep deprived, and recordings are taken early in the afternoon during natural sleep. If idiopathic generalised epilepsy is suggested either clinically or by generalised spike-wave during sleep, overbreathing with breath counting and photic stimulation are performed on awakening.

Collection of clinical information by the EEG technologists

This is part of an on-going prospective study that started in January 1998, and includes all children aged 1—14 years referred for EEG to our department, irrespective of the reason for request and the provisional clinical diagnosis. All children and their carers are interrogated by the EEG technologist through a qualitative questionnaire (Appendix A) with particular emphasis on the circumstances of the attacks and the initial ictal symptoms including emetic and other autonomic symptoms and signs. Information is provided by witnesses, usually parents. When PS is suspected, the interview with parents and children is videotaped during the EEG session. Additional information—if needed—is obtained at a later stage, usually during follow-up EEG recordings or through telephone calls or letters to parents, witnesses and physicians. During the preparation of the factual report, the pertinent features of these interviews are summarised and stored as text files in our database.

EEG reporting

The EEG findings and the accompanying clinical evidence are then reviewed and correlated in joint sessions by physicians and technologists. The final interpretation includes, if possible, the possible electroclinical diagnosis, suggesting—if appropriate—further tests. For in-patients, direct communication with the referring physicians ensures fast and effective relay of important clinical information and electroclinical correlations. Follow-up sleep EEG recordings are performed with the agreement of the attending clinician.

Data evaluation and analysis

Re-evaluation of all children diagnosed with PS included review of the EEGs and the information from the video-recorded interviews, and also of the medical correspondence and the results of the laboratory tests, available from the medical notes.

Results

Between January 1998 and January 2001, 424 EEGs were performed in 308 children aged 1—14 years, of whom 228 (74%) had one or more epileptic seizures. We diagnosed PS in 14 children (6.1%), mainly based on clinical information (Table 1). There were 9 boys and 5 girls. The mean age at first seizure was 63 months (range 21—162), and the total number of seizures for each child ranged from 1 to 12 (mean 4). Brain MRI was performed in seven children (1—3 and 6—9) and was normal in all. Three other children (1.3% of all children with seizures, or 21% of those with autonomic seizures) had symptomatic or cryptogenic focal seizures with predominantly autonomic ictal features including vomiting. Eighteen children (8% of those with seizures) had benign Rolandic epilepsy.

EEG

Interictal EEG in those diagnosed with PS showed remarkable variation from normal to severely epileptiform (Table 2 and Figs. 1 and 2). The commonest abnormality consisted of multifocal independent spikes at various locations, mainly over the occipital centro-temporal and frontal areas. Overall 10 patients had at some stage occipital spikes, but only four had occipital paroxysms. The number of EEGs for each patient has so far ranged from 2 to 7; 10 children had three or more recordings.

The first EEG was recorded a few hours to 7 years after the first seizure, but within a month in nine children. In six children (1, 2, 3, 6, 9 and 10, Table 2), the first recording was taken within 2—3 days after the first seizure, during their admission for possible encephalitis. The first EEG was normal in three children (two of whom were in-patients treated for encephalitis), showed brief generalised discharges only in two other children, and typical multifocal spikes in nine children (five with and four without occipital spikes).

Background activity and sleep patterns were usually normal but some post-ictal EEGs showed focal slowing (Fig. 2).

Diagnosis on referral and contribution of the EEG technologists

In nine children (1—5 and 9—12, Table 2) with various other provisional diagnoses at the time of the EEG referral, PS was identified mainly by the information collected by the EEG technologist (Tables 1 and 2). Five of these children (1—3, 9 and 10, Table 2) were in-patients, treated for encephalitis, and the report of the EEG contributed substantially in changing their management. In children 1 and 10 in particular, the diagnosis of PS relied exclusively on the clinical information as their first EEGs during their admission were normal.

In a further three children (7, 13 and 14, Table 2) the diagnosis of PS was already suspected on

Table 1 Clinical features of the 14 children with Panayiotopoulos syndrome.

	Gender	Age at onset (months)	Time between first seizure and EEG	Number of seizures	Number of EEGs	Ictal manifestations[a]
1	F	39	1 day	10	3	Two seizures with vomiting, eye/head deviation to left, also four seizures with heavy breathing, eye/head deviation to right
2	F	67	2 days	12	5	One seizure with nausea and sickness, retching, possibly coloured vision, unresponsiveness and generalised convulsions, and 11 other with mixed PS and rolandic features (nausea, retching, hypersalivation, speech arrest, convulsions)
3	M	108	1 month	3	3	One seizure with headache, sickness, disorientation; then became floppy like ''rag doll'', staring with mydriasis, double incontinence and vomiting. Two Rolandic followed
4	M	21	5 months	3	3	Repeated vomiting, became subdued and gradually unresponsive and flaccid, brief bilateral convulsions 15 min from onset
5	M	42	84 months	4	4	Felt unwell, vomiting, eye deviation to left, pale, unresponsive and floppy
6	F	40	2 days	1	3	Breathing abnormalities-spitting and vomiting-eye deviation to left—left hemifacial spasms-GTCS
7	F	162	18 days[b]	1	2	Woke up confused, wandering and looking up as if at something, mydriasis, eyes to left, floppy shaking
8	M	37	1 month	3	7	Floppy and unresponsive, asystole in the first attack. Became pale and incontinent with mydriasis twice later, ending with convulsions
9	M	53	3 months	1	2	Woke feeling unwell, sick, left convulsion of body, hypersalivation
10	M	50	5 months	2	4	Sudden vomiting, staring, ash grey, double incontinent
11	M	48	42 months	7	2	Felt unwell, vomited, eye deviation, unresponsive, doubly incontinent
12	M	65	1 month	2	2	Felt sick, eyes to right,-small jerking body arms and legs for 1 min-unresponsive ''like sleep'' waking up to vomit and back down again, no recollection
13	F	65	2 days	4	3	Wakes unwell and pale, vomits, eye deviation, floppy and unresponsive
14	M	84	2 days[b]	2	4	Vomits, then becomes unresponsive and flaccid with short and superficial breaths, vacant eyes and double incontinence. Then, eyes/head deviation to right, right-sided twitching

[a] Mostly derived from technologists' notes.
[b] First EEG recorded elsewhere. His first EEG in our department was recorded 1 month after his second and last seizure.

referral, and was confirmed despite the fact that the EEG was normal in one (child 13). Child 14 had been treated elsewhere for encephalitis after the first seizure, but PS was suspected and he was sent to us for confirmation.

Diagnosis was not possible in two children. In child 6 (Table 2), the initial ictal symptoms and their sequence were not known at the time of the first EEG, which was taken a few hours after admission. The diagnosis was also missed in child 8 because of

Table 2 Suggested diagnosis on referral for first EEG and change after the interview by the technologist—subsequent EEG findings.

	In-/out-patient	Diagnosis on referral (alternatives)	Findings of first EEG (subsequent EEGs)	Diagnosis after interview and EEG findings
1	In-P	Encephalitis[a]?	Normal (O1 and P4 spikes)	PS suggested despite normal EEG
2	In-P	Encephalitis[a]?	O2-O1-C4 spikes (O2-O1 spikes and brief generalised discharges)	PS suggested
3	In-P	Encephalitis[a]? (Atypical migraine? Raised intracranial pressure?)	Cz spikes (C3-C4-C6-O2 spikes)	PS suggested
4	Out-P	Two atypical febrile seizures	C4 and positive occipital spikes (multifocal spikes)	PS suggested
5	Out-P	Three afebrile and one febrile seizures	Brief generalised discharges with phantom absences (F4-Cz-C3 spikes)	PS suggested despite inconsistent EEG findings
6	In-P	Encephalitis? (First seizure-rolandic?)	Focal slow and small positive occipital spikes (C3-C4-P4 spikes)	None—ictal vomiting and other autonomic manifestations though present were not fully known. Also, confusing postictal EEG with focal slowing. PS was diagnosed after the second EEG where a more accurate history was achieved
7	Out-P	Possible PS	Repetitive multifocal spike-wave complexes (multifocal spikes; normal)	PS confirmed
8	Out-P	Cardiopulmonary arrest of unknown aetiology? (First seizure? Encephalitis?)	Brief generalised discharges (occipital paroxysms)	None—the diagnosis was made after the second seizure with typical autonomic features and focal spikes in the EEG
9	In-P	Encephalitis[a]? (Atypical migraine? Space occupying lesion?)	P4 and Pz spikes (multifocal spikes)	PS suggested
10	In-P	Encephalitis[a]? (Choking attack? Febrile seizure? Gastroenteritis?)	Normal (T4-O2-F4)	PS suggested despite normal EEG
11	Out-P	Epilepsy? (Faints? Gastroenteritis?)	O2-O1-Pz-F3-T4 and brief generalised discharges (multifocal spikes)	PS suggested
12	Out-P	First seizure? (Acute de-hydration?)	Left occipital spikes (C4-C3-O1-F3 spikes)	PS suggested
13	Out-P	Possible PS	Normal (repetitive multifocal spike-wave complexes)	PS confirmed despite normal EEG
14	Out-P	Possible PS (previous episode diagnosed as encephalitis for which he was treated)	Repetitive multifocal spike-wave complexes	PS confirmed

[a] Treated for encephalitis until EEG was performed; PS: Panayiotopoulos syndrome.

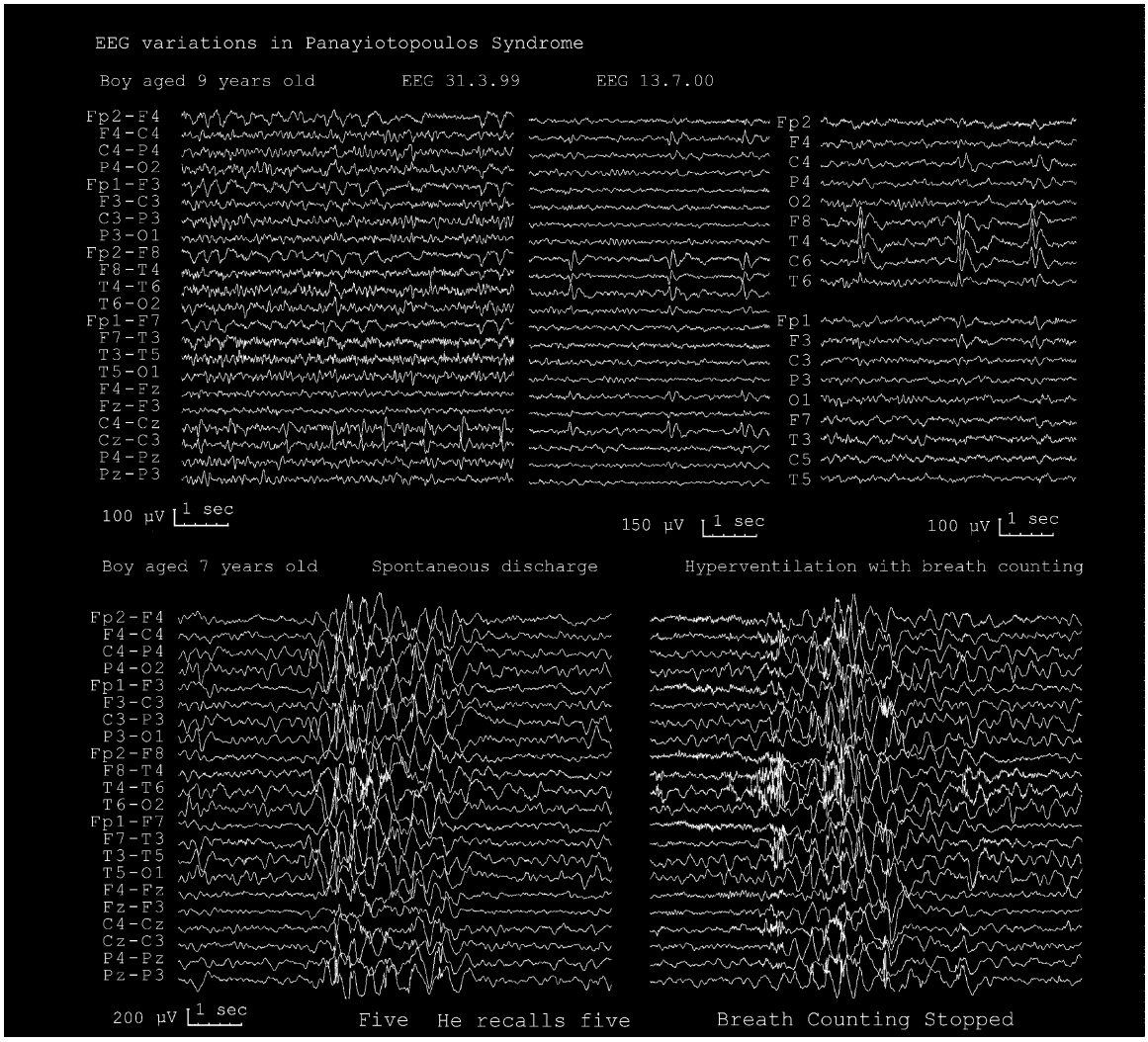

Figure 1 Top row: video-EEG of case 3. Left trace: first EEG with spikes at the vertex (Cz). Middle and right traces: subsequent EEG showed giant right centro-temporal spikes with maximum voltage at T4 electrode. Note the additional C5 and C6 electrodes for exact localisation. Bottom row: video-EEG of case 5. Left trace: spontaneous brief generalised discharge. The patient was able to recall what the technologist told him during the discharge. There were no apparent clinical manifestations on video-EEG. Right trace: hyperventilation with breath counting showed that generalised discharges were associated with clinical manifestations (the boy stopped counting). Such subtle behavioural changes would have passed unnoticed without breath counting and video-EEG.

the atypical clinical and electrographic presentation (Table 2).

Again, encephalitis was the most common diagnosis, suspected in eight children, six of whom received relevant treatment at some stage. Other diagnoses were atypical migraine, faints, gastro-enteritis, dehydration, atypical febrile seizures or seizures.

Discussion

In this paper we demonstrated that EEG technologists can play a significant role in the diagnosis of epilepsies. Correct syndromic diagnosis was possible in 12 of the 14 children with PS, thanks to the clinical information collected by the technologists during the preparation of the children for EEG. Detecting key symptoms that are firmly associated with PS (emesis and other autonomic manifestations) may prevent erroneous diagnoses, and shorten the length of unnecessary treatments (e.g., for encephalitis) even if the EEG is normal or not typical as in cases 5 and 8 (Table 2). In addition, early recognition of PS can provide firm reassurance to families in situations that can be very alarming.

We focused on Panayiotopoulos syndrome because it is a relatively frequent condition within

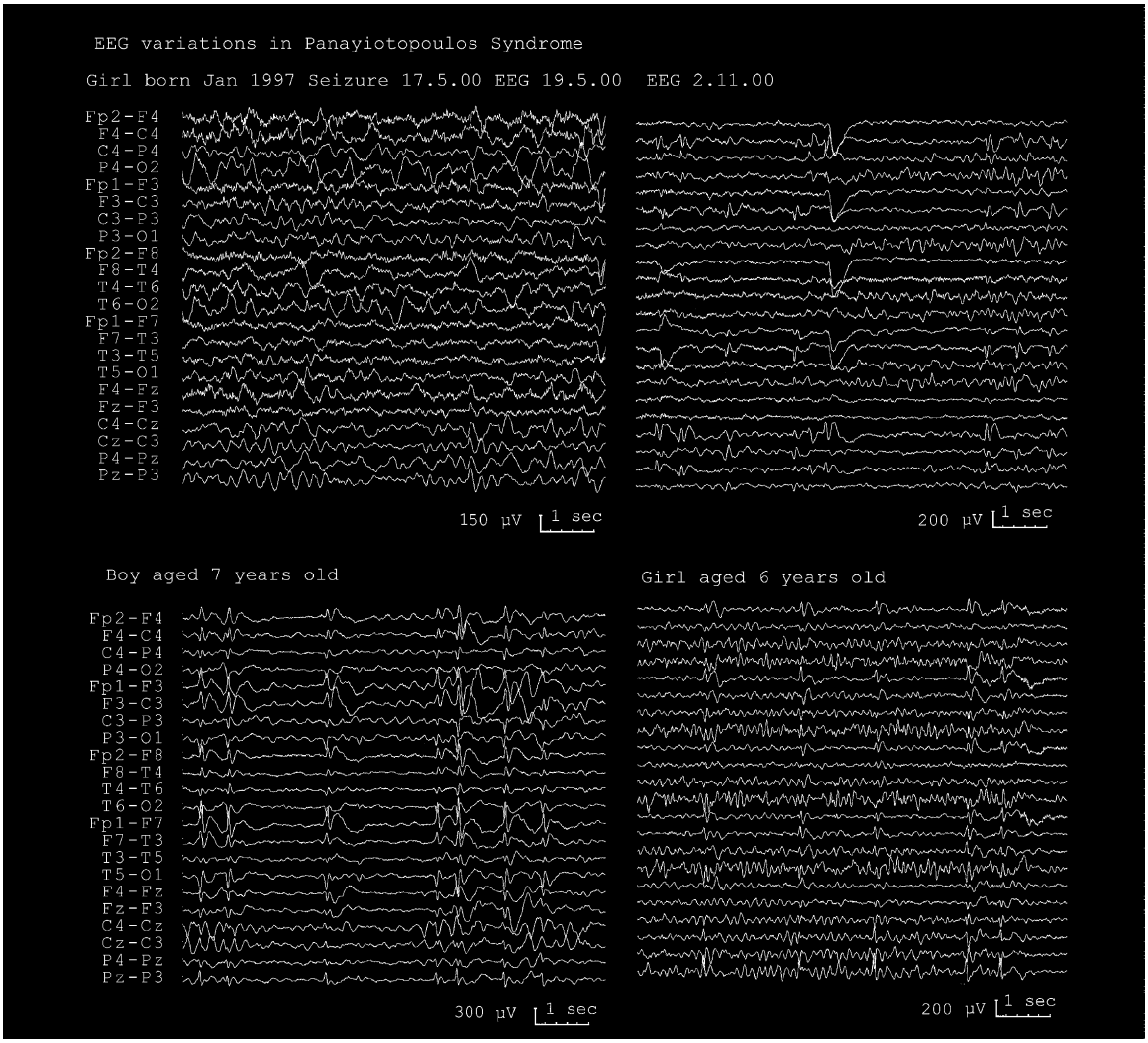

Figure 2 Top row video-EEG of case 6. Left trace: first recording 2 days after a prolonged seizure. Note the significant EEG abnormality in the right posterior quarter with marked slow waves and relevant poverty of alpha rhythm. The diagnosis of PS was then missed because the clinical details were not known to the recording technologist, and the EEG findings were consistent not only with postictal suppression but also with encephalitis and posterior lesion. Right trace: EEG of the same child 6 months later showed cloned-like repetitive multifocal spike-wave complexes, typical of PS. Bottom row: cloned-like repetitive multifocal spike-wave complexes of two other cases with typical clinical manifestations of PS. These are otherwise normal children with excellent scholastic performance.

the benign focal epilepsies of childhood, but with a high rate of misdiagnosis due to the unusual clinical and EEG characteristics. Ictal symptoms may be disguised as non-epileptic manifestations of other diseases or conditions such as migraine, gastroenteritis or syncope, and even mimic serious cerebral insults like encephalitis, for which many of these children are treated. From the EEG viewpoint, the main factors for misdiagnosis are the presence of multifocal spikes that may suggest severe multifocal epilepsy with poor prognosis,[12] and the absence of occipital spikes or paroxysms that are still erroneously accepted as diagnostic prerequisite for PS.[15]

We demonstrated significant EEG variability with multifocal spikes, and showed that occipital paroxysms and even occipital spikes might not occur in otherwise typical clinical presentations. Our findings are consistent with those of the original study of Panayiotopoulos:[16] of 21 children with normal development and seizures with ictal vomiting 12 had occipital spikes, five had extra-occipital spikes, three had normal EEG and only one had infrequent and asymptomatic brief generalised spike-wave discharges. In the ensuing years attention focused on the sub-group of children with occipital spikes and paroxysms, resulting in the inaccurate terms ''early onset benign childhood epilepsy with

occipital spikes'' or ''early onset benign childhood occipital epilepsy (Panayiotopoulos type)''.[17] It has been recently appreciated that the clinical manifestations are the same irrespective of the topography of spikes in the interictal EEG,[7,18] and it has been known for a long time that occipital spikes are non-specific abnormalities occurring in children with or without seizures.[19]

The need for more active involvement of the EEG technologists in the process of generating clinically helpful EEG reports has been officially acknowledged in the UK, and currently the National Standards for Clinical Neurophysiology (basic level) require the technologist to gather details of clinical events and any relevant medical history. Many technologists do not do this. The databases provided with most modern digital EEG machines do not automatically require details of the last attack, or patients' ictal symptoms. Time may also be limited and a number of EEG technologists may be reluctant to accept more responsibilities without extra reward. At best, extraction of clinical information is casually performed and data is orally relayed to the reporting Consultant when and if a joint reporting session is held, but is seldom written down. Such information may be invaluable in interpreting subsequent EEGs in the series. Specifically designed clinical protocols and guidelines may be missing in many EEG departments.

Our experience at St Thomas' EEG department shows that apart from contributing to a clinically useful EEG report, the information collected by the technologists is invaluable for conducting prospective clinical-EEG research, provided that a structured but easy-to-use questionnaire is used, adaptable to new evidence. Other examples of the possible contribution of the EEG technologists to good clinical practice and research may be the reflex seizures and epilepsies, such as those associated with high cognitive functions, praxis and reading or other facets of language function. The data collected can be reviewed during the joint reporting sessions of physicians and technologists, and the filtered information can be used to suggest a plausible electroclinical diagnosis and possibly protective measures, but can be also stored for research purposes. For EEG technologists recognition of this additional role should act as a strong professional motivation.

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Appendix A. Questionnaire for Panayiotopoulos syndrome

1. Number of attacks chronologically
2. Details of each attack
   (a) Was the child febrile before, during or after?
   (b) Precipitating factors
   (c) Did the seizure occur during awake or sleep (daytime nap/nocturnal)

Symptoms before the attack

Was the child unusually irritable or quiet? Description of other behavioural changes, if any
Did the child complain of sickness or headache?
How were cognition and speech?

Symptoms at onset and during the attack sequentially

(a) Autonomic symptoms

- Emetic (nausea, retching, vomiting)
- Coughing or breathing abnormalities
- Incontinence of urine or faeces?
- Colour of the child's face (pallor, cyanosis or flushing)
- Hypersalivation
- Size of the pupils (mydriasis, miosis or normal)
- Pulse rate

(b) Non-autonomic symptoms

- State of eyes (deviation to one side or other)
- Head deviation
- Muscle tone (stiff or flaccid)
- Convulsions and their distribution and duration
- Speech arrest
- Responsiveness
- Visual symptoms (hallucinations or illusions, other)

(c) Syncope-like episodes[#1]

- Occurrence during the seizure or independently?
- Triggers?
- Body position (lying or upright?)
- Duration

(d) Duration of the seizure

- Recovery period and postictal symptoms/signs

[#1] Combination of hypotonia (floppiness) and unresponsiveness.

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