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Status epilepticus on the paediatric intensive care unit—the role of EEG monitoring

CAROLINE ROSS*, ALISON BLAKE† & WILLIAM P. WHITEHOUSE*

**Department of Paediatric Neurology; †Department of Clinical Neurophysiology, Birmingham Children's Hospital NHS Trust, Birmingham B4 6NH, UK*

Correspondence to: Dr Caroline Ross, Birmingham Children's Hospital NHS Trust, Steelhouse Lane, Birmingham B4 6NH, UK

A pilot study was undertaken of the feasibility of continuous EEG monitoring of patients admitted to a Paediatric Intensive Care Unit (PICU) for management of status epilepticus or its immediate sequelae. Eight children were studied and seizure activity was recorded in four patients. Additional information influencing management was obtained: the bedside nurse considered decerebrate posturing in one patient to be a seizure: there were no epileptiform EEG changes. Another patient was considered to have seizures (clonic movements of both upper limbs) following cardiac arrest; the EEG showed electrocerebral silence, and thiopentone treatment was discontinued. In another patient, continuing epileptiform activity on EEG gave intensivists the confidence to use higher than usual doses of thiopentone. The problems encountered were delays in monitoring, once for a CT scan and once because of two admissions within hours of each other. We conclude that EEG monitoring on a PICU is feasible and provides clinically useful information.

Key words: status epilepticus; EEG monitoring; intensive care unit (ICU).

INTRODUCTION

Convulsive status epilepticus is a life-threatening medical emergency that demands prompt recognition and treatment. In the USA, it has been estimated to occur between 50 000 and 60 000 times per year in patients of all ages[1]. Twenty percent of cases occur in the first year of life and 60% in the first 5 years[2].

The two largest reported series, those of Aicardi and Chevrie[3] and Maytal *et al.* [4] show similar proportions of aetiologic agents. Febrile status accounted for 20–29% of all cases, chronic static central nervous system disease a further 14–23%, acute symptomatic 23–40% and idiopathic status 16–39%.

No general physician would consider treatment of cardiac arrest without real-time electrophysiological information about the nature of the arrest and the effects of his management, but most of us have to manage status epilepticus without accurate neurophysiological information. In adult practice, significant numbers of patients may be treated aggressively with dangerous drugs when they are not in status epilepticus and indeed have non-epileptic attack disorders. This is a smaller problem in children, but there are a number of other conditions which may be mistaken for convulsive status epilepticus, e.g. acute dystonic reaction, paroxysmal dyskinesia and the tonic extensor spasms seen in incipient tonsillar herniation.

No less dangerous is the occurrence of continued electrical status epilepticus after overt seizures have ceased. This study was prompted by a bad outome after a patient had been in just such a situation.

MATERIALS AND METHODS

Eight-channel EEG monitoring was performed on children admitted to a PICU for management of status epilepticus or its complications. When a suitable patient was admitted, the on-call neurophysiology technologist came in and glued on EEG electrodes, using the International 10/20 system of electrode placement. Eight-channel recordings were made on a Medelec Discovery. An initial EEG was recorded and portions of it faxed to the clinical neurophysiologist involved in the study (AB). Further 30-second printouts

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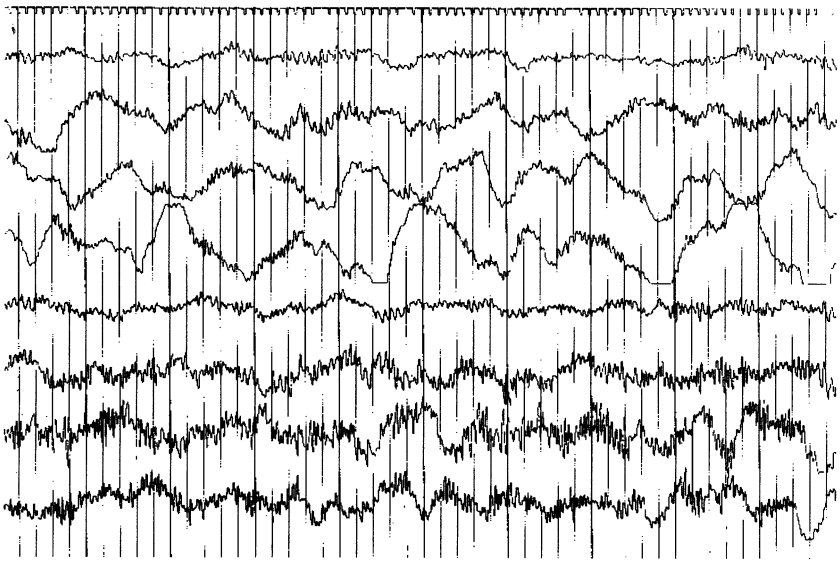

Fig. 1: EEG on patient 2 at the time of abnormal movements, showing muscle artefact and no epileptic discharges.

were made every 30 minutes for the first 2 hours and every 60 minutes thereafter. In addition to this, printouts were made when there were clinical events such as abnormal movements.

Recording was continued until recovery or death or for a maximum of 3 days. All drug dosages and time of administration were recorded.

Patients

Eight patients were studied, seven of them male. Ages ranged from 3 days to 8 years. Four were admitted to a PICU for seizure management, one for airway management, one with septicaemia and one with an acute encephalopathy accompanied by status epilepticus. The last child was admitted after cardiorespiratory arrest and was thought to be having epileptic seizures.

Patient 1 was an 8-year-old boy who was admitted to a PICU for airway management after status epilepticus lasting 2.5 hours. There was parental delay in seeking medical help. His Glasgow Coma Scale score was four (of 15) and he had respiratory depression: respiratory rate of 12, pooled secretions in his mouth. An EEG showed diffuse slow delta activity. Seizures were controlled by propofol (8.3 mg/kg/hour) and alfentanyl (5.2 mg/kg/hour). Subsequently, he had no new deficit.

Patient 2 was a 19-month-old boy admitted with prolonged seizures in the context of a febrile illness (pneumonia). He was transferred to a PICU after failure of diazepam and phenytoin to control his seizures. Midazolam administered on the PICU was also unsuccessful, thiopentone did control them at a dose of 3.3 mg/kg/hour. The EEG was slow. Abnormal movements which were thought to be seizure by the bedside nurse, but to be decerebrate posturing by the paediatric neurologist (WW) did not have accompanying EEG discharges (Fig. 1). For several days after cessation of seizures he was ataxic, this may be related to the high dose of phenytoin he received on the PICU (16 mg/kg/24 hours). At the time of discharge home, he had no neurological deficit.

Patient 3 was a 12-week-old dysmorphic boy who had been admitted electively for hernia repair. He had started having seizures on the second day of life. On the first day of admission to the surgical ward he had 30 seizures and was not considered to be fit for surgery. Intravenous drugs including phenobarbitone, phenytoin and clonazepam failed to control his seizures and rendered him apnoeic. An EEG, whilst receiving a chlormethiazole infusion, showed frequent spikes but not status epilepticus. He had no new deficit after his admission. He was subsequently diagnosed to have pseudo-trisomy 18, that is, he had the clinical features of trisomy 18 but not the chromosomal abnormality.

Patient 4 was an 11-week-old boy who had been an unrestrained passenger in an automobile accident. A significant head injury was suspected when he started to have seizures. A CT brain scan showed bilateral contusions and large subdural collections. His seizures were initially treated with paraldehyde, diazepam, phenobarbitone and phenytoin. Initial response to midazolam was seen but when seizures broke through on this he was treated with thiopentone. An initial EEG showed prolonged generalized seizure with right-sided onset. Burst suppression was achieved with high doses of thiopentone (8.3 mg/kg/hour). Since this admission, he has developed spastic quadriplegia, microcephaly,

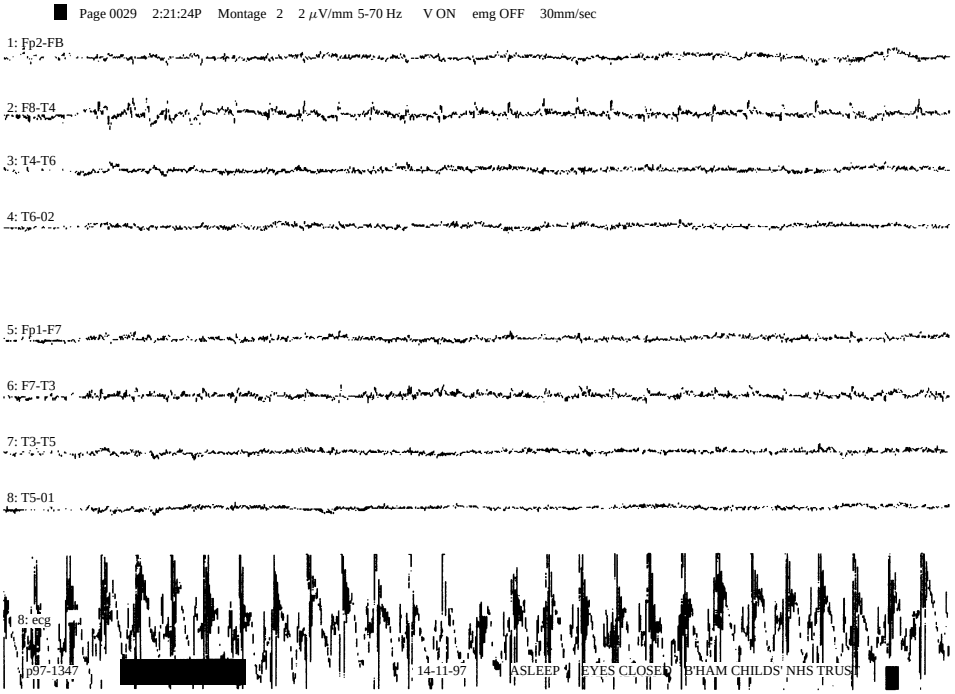

Fig. 2: EEG from patient 8 showing electrocerebral silence. Note the high gain used (2 μV/mm).

Table 1: Summary of patient characteristics and outcome.

	Age	Sex	Reason admission	Outcome	Diagnosis
1	8.3 years	M	Protect always	No new deficit	Non Hodgkins Lymphoma
2	19 months	M	Seizures	Ataxia	Febrile illness
3	12 weeks	M	Seizures and apnoeas	No new deficit	Pseudo-trisomy 18
4	11 weeks	M	Seizures	Cerebral palsy	RTA
5	8 months	M	Seizures	Increased deficit	Propionic aciduria
6	10 days	M	Septicaemia	No deficit	Gp B strep septicaemia
7	11 weeks	M	Encephalopathy	Hypotonia	H Simplex encephalitis
8	3 days	F	Post arrest	Died	Cardiac arrest

visual impairment and intractable epilepsy.

Patient 5 was an 8-month-old boy with intractable epilepsy as part of his underlying metabolic defect (propionic aciduria). Diazepam, phenobarbitone and phenytoin did not control his seizures, thiopentone at doses of up to 8 mg/kg/hour did. An initial EEG showed burst suppression, later EEGs were dominated by slow delta rhythms with no seizure discharges. Following this episode of status epilepticus he was shown to have an increased neurological deficit (generally hypertonic with no awareness and probably no vision).

Patient 6 was a 10-day-old boy admitted with a presumed septicaemia who then started convulsing. His seizures did not respond to phenobarbitone or midazolam and thiopentone (infusion of 2.7 mg/kg/hour) was needed to control them. An EEG showed burst suppression. Group B Streptococcus was later isolated from blood cultures. Somewhat surprisingly, given the usually devastating consequences of this infection in neonates, at the time of discharge home he was thought to be neurologically intact.

Patient 7 was an 11-week-old boy who was admitted with an encephalopathic illness and focal seizures (left-sided jerking, upper limbs more than face and lower limbs). An initial EEG showed asynchronous low amplitude slow components and right-sided spikes. Phenytoin, phenobarbitone and midazolam were unsuccessful at controlling seizures and thiopentone was required. EEG whilst receiving thiopentone at 3 mg/kg/hour showed asymmetrical suppression burst with epileptiform activity predominantly on the right. Herpes simplex infection was subsequently demonstrated. He was markedly hypotonic at discharge from hospital. Fourteen months later, he was described as severely handicapped with spastic quadriplegia.

Patient 8 was a 3-day-old girl who had had a cardiorespiratory arrest whilst in her mother's arms and

feeding at home. Cardiac output returned during resuscitation at her local hospital. Her mother later gave a history that she had not fed well from birth. After admission to the PICU she was noted to be hypertonic in both upper limbs and also had tonic–clonic movements of these limbs. This had been interpreted as a seizure and thiopentone treatment was started at a dose of 2.5 mg/kg/hour, rising to 7.5 mg/kg/hour. An EEG showed electrocerebral silence (Fig. 2). Thiopentone was discontinued. Serial EEGs were unchanged from the initial recording and intensive care was withdrawn. She then died and post-mortem examination revealed intraventricular haemorrhages.

Clinical details of these patients are summarized in Table 1.

DISCUSSION

Additional information obtained from the EEG that led to changes in management

The bedside nurse of patient 2 considered decerebrate posturing to be seizures—the EEG confirmed that these events were episodes of decerebrate posturing. The clonic movements of patient 8 were considered to be seizures—the EEG showing electrocerebral silence allowed thiopentone therapy to be discontinued. Continuing seizure activity on EEG in patient 4 enabled intensivists to feel confident about using high doses of thiopentone.

Problems encountered

There were delays in monitoring in two patients, once for a CT brain scan (electrodes not compatible with scanner) and once because two patients were admitted within hours of each other (patients 4 and 5). Sometimes it was not possible to ascertain the exact time of administration of bolus drugs.

CONCLUSION

Eight-channel EEG monitoring on a PICU is feasible and provides clinically useful information.

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