

Seizures presenting as apnoea

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SUMMARY Between the ages of 3 and 6 months a baby boy presented with repeated, non-specific episodes of cyanosis, apnoea, bradycardia, and abnormal movements of the limbs. The episodes were severe and required resuscitation and several admissions to hospital. Initial investigations showed only signs of oesophagitis. Despite treatment of the oesophagitis the symptoms recurred, and electroencephalography and polygraphy eventually showed evidence of minor seizures. Severe epilepsy with tonic-clonic seizures developed when he was 6 months old.

Convulsive disorders in infants can be difficult to diagnose. Initial symptoms sometimes mimic airway obstruction, vagal hyperactivity, or cardiac or digestive problems.

We report the case of an infant who had repeated, non-specific life threatening episodes of prolonged apnoea that were eventually diagnosed as seizures.

Case report

A baby boy was born at full term weighing 3950 g. He had an Apgar score of 8 at one minute, and 10 at five minutes. At the age of 3 months he had a sudden episode of respiratory obstruction with cyanosis, bradycardia, and hyperactive movements

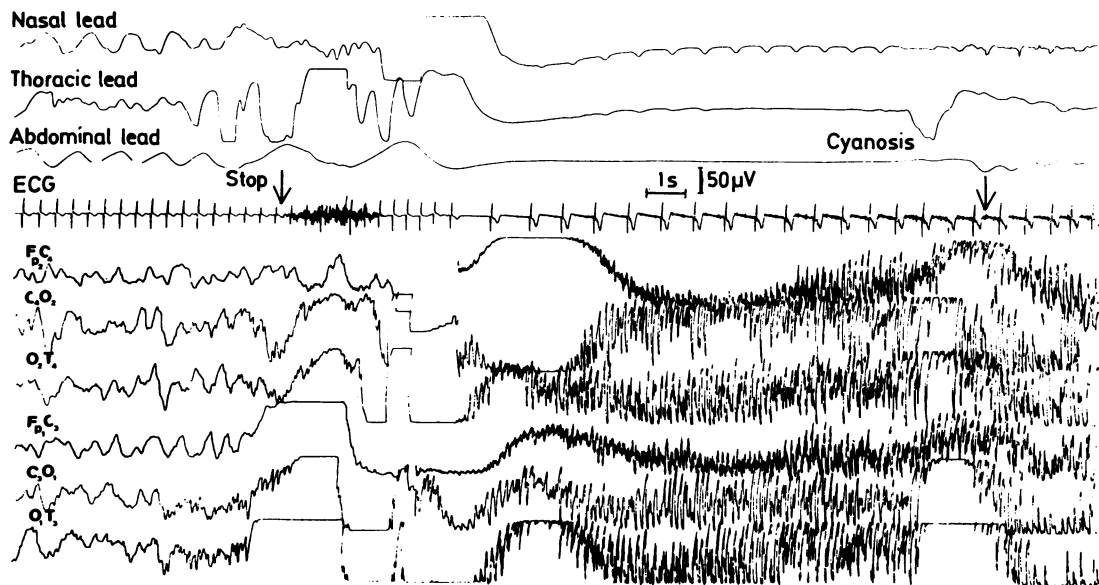


Fig 1 Seizure triggered by milk feed. Feeding was stopped at the first arrow. Oxygen was given by mask after the second arrow. Respiratory obstruction is recorded on the three respiratory leads (nasal, thoracic, and abdominal). Electroencephalographic discharge starts in the left occipital area with secondary generalisation. Short bradycardia is seen on the electrocardiogram lead.

of the upper limbs. During his hospital stay three identical episodes occurred that all required cardiopulmonary resuscitation. Neurological examination showed no abnormality. Examination of the cerebrospinal fluid, electroencephalography, 24 hour polygraphy, ultrasonography, and computed tomography were all normal. Hyperactive vagal reflexes were diagnosed; short episodes of bradycardia (heart rate 60/minute) followed ocular compression, and further episodes were recorded during continuous monitoring. Oesophagoscopy showed oesophagitis.

The child was discharged taking antacids and metoclopramide, and his mother was advised to thicken his feeds and keep him sitting upright as much as possible. He was also given atropine because of the severity of the repeated episodes of bradycardia, and phenobarbitone because of the possibility of epilepsy.

He was readmitted to hospital at the age of 5 months with identical symptoms that were often associated with meals or pain (for example, venepuncture). The hyperactive vagal reflexes and oesophagitis were still present. Gastro-oesophageal reflux was seen on barium meal examination. Monitoring of the oesophageal pH, however, showed that it was less than 5 during only about a third of the first hour after feeding, and this was interpreted as being within normal limits. Twenty four hour polygraphy showed minor seizures, the

first one occurring while he was drinking milk (figs 1, 2, and 3). All six seizures were identical (table). Electroencephalographic discharges started in the left occipital area and then became generalised. Electroencephalography between the seizures showed either localised spike waves in the left occipital area, or generalised spike wave patterns. Thereafter he developed severe epilepsy with generalised tonic-clonic seizures.

At the age of 2½ years the neurological examination was normal, as was his development assessed by the Denver developmental screening test and the Brunet-Lezine test.

Discussion

Gastro-oesophageal reflux is common in infants of less than 15 months old¹ and may cause both oesophagitis and apnoea.² The most prominent feature in our case was the early onset of epilepsy with symptoms associated with gastro-oesophageal reflux and apnoea. This syndrome has not been widely described. In six of 59 infants at high risk of the sudden infant death syndrome (SIDS), Jeffery *et al*³ reported seizures associated with gastro-oesophageal reflux diagnosed by barium meal examination and monitoring of oesophageal pH. They suggested that reflux could be a part of the autonomic dysfunction that accompanies temporal lobe seizures; this could be compared with 'eating

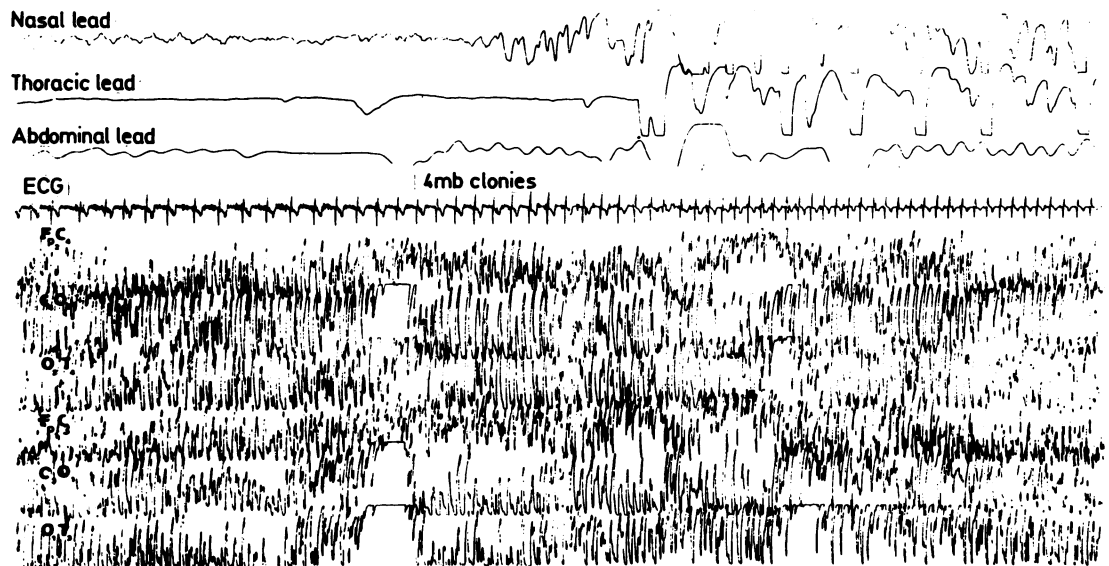


Fig 2 The same seizure continued. The whole period of respiratory obstruction lasted 37 seconds. The term '4 mb clonies' = clonic jerks of the four limbs.

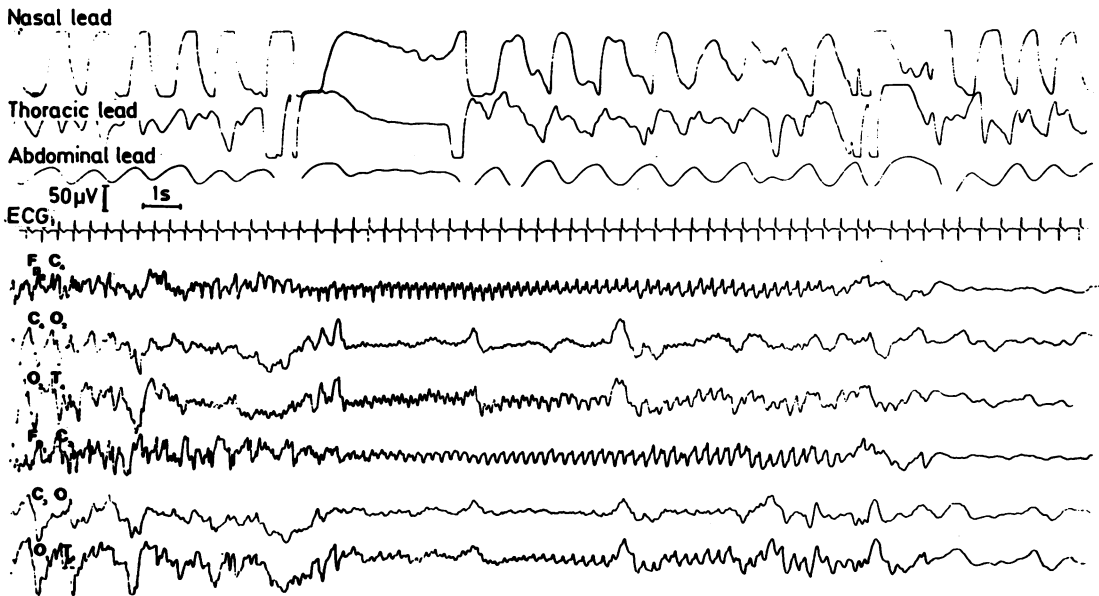


Fig 3 The end of the same seizure. There was a short (5 s) central respiratory pause without bradycardia or clinical symptoms.

Table Summary of the main features of the six recorded seizures

Electroencephalographic discharge:	Alpha-like left occipital onset Generalisation in 2-3 s Prolonged slowing after the seizure
Bradycardia:	Heart rate 60/minute 7-13 s delay after onset of discharge
Prolonged central apnoea:	Same delay as bradycardia 30-40 s duration
Duration of seizure:	30-60 s
Clinical signs:	Initial hypertonia Intense cyanosis Respiratory obstruction Ocular revulsion Few clonic movements of the limbs

epilepsy',⁴ in which apparently spontaneous attacks are triggered by meals. Monod *et al*⁵ reported a case similar to ours; antral dyskinesia was treated with antireflux drugs but symptoms recurred and seizures were diagnosed. After starting antiepileptic drugs the patient remained free of symptoms.

Our patient had repeated life threatening episodes of prolonged apnoea and bradycardia, and this led us to wonder if there was an association between this syndrome and SIDS. Beal⁶ reported six families in which a sibling developed severe atypical or fatal seizures at the same age as a child had died of SIDS.

When a child presents with atypical repeated episodes of apnoea, repeated electroencephalography and polygraphy with stimulation may lead to the diagnosis of epilepsy and appropriate treatment can be started.

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Accepted 18 July 1988