recurrent episodes of intense cyanosis were due to seizure induced airway obstruction or a seizure induced prolonged absence of inspiratory efforts.¹ We accept that seizures may cause prolonged absence of breathing effort and hypoxaemia and have previously described one patient with this problem.² In our patient, however, the apnoea was present for more than 50 seconds before severe hypoxaemia became apparent. In situations of complete airway obstruction due to imposed apnoea the onset of sufficient hypoxaemia to produce cyanosis does not appear until after 60 seconds of absent airflow.³ In our opinion, it is more likely that the primary cause of the sudden onset and rapid progression of hypoxaemia was intrapulmonary shunting and prolonged expiratory apnoea.⁴ This may have triggered the seizure shown in the recording or may have resulted from the seizure. In order to answer this, it would be required to document the temporal association between the onset of hypoxaemia and development of seizure activity.

The onset of cyanotic episodes with meals or pain, the lack of response to antacids, metoclopramide, atropine, and phenobarbitone and the early onset and intensity of cyanosis would be typical of a sudden intrapulmonary shunt and prolonged expiratory apnoea.⁴ These latter episodes are induced in infants under 6 months typically by feeding or painful or stressful stimuli, and the resulting seizure is secondary to the hypoxaemia.

It is also possible that cerebral injury resulting from severe hypoxaemia early in life may have produced a focus for the subsequent development of epilepsy.

We suggest that all polygraphic monitoring during cyanotic episodes should include measurements of oxygenation, most reliably sensed by a pulse oximeter in a beat-to-beat mode.

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Range of neural tube defects in southern India

Sir,

The article on neural tube defects in southern India by Kulkarni *et al* mentions 'widespread prenatal screening and

termination of affected pregnancies' as contributing towards the downward trend of these defects in Western countries including the United States and Scotland.¹ However, the Report of the Chief Medical Officer for England and Wales in 1985 states that 'screening and subsequent elective abortion has not been a major cause of the decline'.² A similar decline has occurred in the Republic of Ireland, where abortion is not carried out.³

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Breath hydrogen excretion in infants with colic

Sir,

In some babies with troublesome crying the features of intense sympathetic activity after a prolonged screaming episode of, say, 30 minutes are very striking. These babies have a pronounced tachycardia, sweat, and become red in the face. Is it possible that the work of crying results in a shortened intestinal transit time with increased delivery of lactose to the colon reflected in high breath hydrogen excretion as Miller and colleagues have demonstrated?¹ Another observation is that babies with troublesome crying very often stop crying after hospital admission, often within 24-48 hours, with no alteration to their diet.² It would be very interesting to know if the hydrogen breath excretion dropped over this same short period. This may help to distinguish whether the abnormal results of Miller *et al* reflect a primary gut abnormality or a secondary phenomenon. A study to examine this may be easier to design than one to examine the effect of altering the lactose content of the diet.

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