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Reflex anoxic seizures

Sir,

I am grateful to Dr Bower¹ for drawing attention to reflex anoxic seizures but I take strong issue with his preference for 'pallid syncope'. Of course there are many children who get pale and floppy and unresponsive, who may have vasovagal syncope, and for whom the term pallid syncope is perfectly acceptable. But what I mean by a reflex anoxic seizure is 'a particular type of fit which is neither epileptic nor due to breath-holding, but rather results from brief stopping of the heart through excess activity in the vagus nerve'. A typical example in the form of venepuncture fits has recently been published,² but it is important to recognise that the patients do not always go pale, nor do observers always notice the pallor. I believe I have seen more anoxic seizures after ocular compression than anyone else (over 300) and can assert that pallor is not constant, particularly is it not observed in children who are not reported as pale in the natural attacks.

An anoxic seizure is very much a seizure, as Gastaut has well described. It may be violent and dramatic.³ It is not sufficient to call such an event convulsive syncope for there is yet another type of seizure which must be distinguished, that is the anoxic-epileptic seizure⁴ in which the syncope is concluded by a train of spike and wave with clonic component. If anoxic seizures are not specifically recognised, they will be called epileptic, even, I suspect, in 1985.

Although I think precision in terminology is helpful in communication between doctors, different words may be more helpful with patients and parents. When I talk about reflex anoxic seizures I call them 'fainting fits and not epileptic fits'. I explain how the vagus makes the heart stop, but that it can only keep on telling the heart to slow down while it is getting enough blood so as soon as it does not the heart will speed up again and all will be well. I nowadays use ocular compression mainly as an aid to reassurance, and agree that in most cases a meticulous history will give the diagnosis. I do not think, however, that paediatricians need be worried about unsubstantiated

dangers of the technique when it has to be used for reassurance, investigation, or research.

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Sir,

I enjoyed reading Dr Bower's annotation.¹ He makes the point that most paediatricians would be reluctant to use the oculo-cardiac reflex to show increased vagal reactivity in children with the pallid form of breath holding. May I suggest a technique that I have found very useful when given a suggestive story of this disorder by the parents. Explaining what the disorder is about to the parents, one asks that the child be laid down on the examination couch without preamble and that the upper garments be removed. The doctor stands ready with stethoscope and, at the first sign of a yell which is certain to occur in response to this sudden 'rough' handling, one is easily able to detect the dramatic slowing of the heart which will accompany the cry. The doctor should prepare himself to be moderately frightened by how dramatic the pallid breath holding attack can be.

I presented a paper on this topic at the Second Rhodesian Medical Congress in 1972 which I entitled 'Breath-holding—an Adams-Stokes attack'.

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Neck radiographs in croup syndrome

Sir,

In recent correspondence, Dr Porter¹ implied that in Aberdeen the initial routine investigation for possible epiglottitis was lateral neck radiographs rather than laryngoscopy. We feel it would be unfortunate if that comment and its potential influence on the practice of others went without reply.

Since the mid 1970s The Royal Aberdeen Children's Hospital has practised a policy of protective intubation in cases of severe croup, an experience we are currently reviewing. Our routine in children with severe stridor is not to attempt clinical or radiological diagnosis with their potentially dangerous delays, but to proceed to laryngoscopy by experienced staff (ear nose and throat and anaesthetic). There have been no problems to date with

this programme. In the few cases where radiology was performed before direct inspection there was a poor correlation between its interpretation and laryngoscopic diagnosis. Similarly, we found that history and clinical examination were unreliable in distinguishing between epiglottitis and severe viral croup. Hence direct visualisation is important in children with severe acute stridor and requires the emergency availability of experienced staff in any centre admitting such patients.

References

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Exercise test for growth hormone deficiency

Sir,

I agree with many of Charles Brook's¹ comments on growth hormone tests but do not believe that in a busy district general hospital screening for growth hormone deficiency with the strenuous exercise test as described by Nicol² and Lazaro³ would be readily available, nor do I think that hospital admission for venous samples during deep sleep under electroencephalographic monitoring is an acceptable alternative. I am in no doubt that the kindest and most efficient screening test for growth hormone deficiency is referral to a growth centre. If this is done then generally no investigation is necessary and the half hour consultation will quickly allay the family's concern.

In my growth clinic in Bristol I see 150 to 200 short children a year and in the vast majority the diagnosis is self evident without any investigation. In the very few where growth hormone deficiency is a possibility I prefer to do a definitive test (sequential insulin and arginine) than a screening test. We do about 12 tests a year and diagnose eight to 10 cases of growth hormone deficiency annually which is no different from other regions of comparable size.

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Inhaled non-radio-opaque foreign bodies

Sir,

Most foreign bodies inhaled by children consist of parts of toys and organic materials (often foodstuffs such as peanuts) and these are unlikely to show up radiologically. Furthermore with laryngotracheal foreign bodies the radiographs of neck and chest are often normal but with bronchial foreign bodies obstructive emphysema may be present.

Recently, we have seen two infants presenting with croup syndrome who were later found to have laryngo-tracheal foreign bodies. Both infants had inhaled pieces of toys that did not show up radiologically. One infant had cardiorespiratory arrest and a piece of Lego was found in the trachea at necropsy. In the other infant there was a delay in making the correct diagnosis and therefore delay in removal of an Action Man from the larynx.

I suspect that paediatricians in other parts of the globe may have also encountered similar diagnostic dilemmas. Should we not therefore ask the toy manufacturers to consider seriously adding some radio-opaque substance to the toy material so that an earlier diagnosis and treatment can be instituted?

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