ward and was not keen to go home. When it was noticed that the mother had a small scratch on the left thumb the possibility of false illness was considered. The amount of blood initially available for analysis was not sufficient to clarify the source of the blood, and after discussions with the local social services department the child was discharged and followed up in the outpatients clinic.

Some eight weeks after the first presentation the child was readmitted with an apparent major haemorrhage from the mouth as evidenced by an area of blood 4 cm in diameter on the baby's cot blanket. There was no evidence of any bleeding from the child's mouth on examination. The baby was therefore readmitted (with the mother) pending further investigations, and we confirmed that the blood on the cot blanket was of the same group as the mother's.

Subsequently, the parents were confronted with the forensic evidence. The mother initially denied the possibility, but eventually admitted that she had simulated all the bleeding from the child's mouth. Two case conferences were held, and after the second the child was allowed to go home. The child's name was placed on the local child abuse register, arrangements were made for the child to be followed up, and the family was referred to the local child and adolescent psychiatrist, an adult psychiatrist having found no evidence of psychiatric illness in the mother.

Comment

Ounsted, in his original description of "open warning," commented that people in authority were often unaware of the signs of child abuse. Further experience has indicated the importance of injuries to the head, mouth, and pharyngeal structures as indicators of child abuse.³ In this particular case numerous clinical examinations and an examination under anaesthesia failed to establish a site of bleeding. False illness became a definite possibility and was subsequently proved. Certain features favoured this, including the age of the mother, her relationships with her own mother, and the caring behaviour and attention she gave to her child when in hospital.

Thus an open warning, even if it is not an indication of non-accidental trauma, may still point to another area of child abuse. As the presentation initially may be to clinic staff or a general practitioner it is clearly important that such an important symptom as bleeding from the mouth in young babies is not dismissed or ignored.

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Blowing hot and cold: "near miss" sudden infant death and episodic hypothermia

We report a previously unrecorded association between "near miss" sudden infant death syndrome and the subsequent development of episodic hypothermia.

Case report

A previously normal boy was admitted at the age of 9 weeks after being found apnoeic and apparently dead during sleep and resuscitated by vigorous shaking. There was no family history of sudden infant death syndrome or hypothermia. Clinical examination and investigations (including chest radiography, lumbar puncture, and electroencephalography) yielded normal results. Cardiorespiratory monitoring showed frequent episodes of sleep related apnoea and associated bradycardia. He was diagnosed as having had a "near miss" sudden infant death and discharged home with an apnoea mattress. Multiple further episodes (requiring mouth to mouth resuscitation) were treated with oral theophylline and, later, caffeine with limited success. Throughout these episodes his parents

did not notice that he felt cold. During an admission when aged 1 he had two episodes of severe apnoea during sleep associated with rectal temperatures of <35°C. His daytime temperatures were normal.

At 17 months the near miss episodes stopped but he continued regularly to become hypothermic during sleep, particularly in cold weather. Home rectal temperature recordings (Model 272-Vickers Medical) confirmed that his temperature fell to 32°C over 10 minutes in quiet sleep. He felt "deathly cold" without shivering or sweating. Vigorous reheating restored his temperature to normal in about one hour. Various measures failed to prevent these episodes. His development was completely normal.

Investigations, including full blood count, creatine phosphokinase, autoantibody screen, metabolic screen, and measurements of free fatty acids, 24 hour vanillylmandelic acid, and serum thyroxine and thyroid stimulating hormone, gave normal results. An insulin stress test showed normal growth hormone and cortisol responses. Responses to thyroid releasing hormone were normal, as were gonadotrophin responses. Serum prolactin concentration (non-stressed) was normal and cortisol values showed a normal diurnal variation. An early morning urine sample showed normal concentrating ability. No infection was found, and the electrocardiogram, electroencephalogram, and brain stem auditory evoked responses were normal. Maximum end tidal carbon dioxide tension in quiet sleep was 5.1 kPa (38 mm Hg). A computed tomograph of the head was normal, with the corpus callosum present.

Although the parents did not wish formal tests of temperature control, during an upper respiratory tract infection he became feverish (38°C rectal) and sweated.

Comment

Episodic hypothermia is rare. Although first postulated as "diencephalic epilepsy," Summers *et al* recognised it as a distinct condition with characteristic sweating attacks often leading to stupor or coma and absence of the corpus callosum in seven of the nine cases that were adequately investigated.¹

Our patient had episodic hypothermia without sweating, a normal corpus callosum, and no evidence of hypothalamic dysfunction. He also had recurrent severe apnoea requiring vigorous resuscitation for which no cause could be found. This is the first reported association.

In the absence of temperature studies and control data the cause of his thermoregulatory defect is unknown, but the absence of sweating during an attack makes increased heat loss^{2,3} unlikely. The most likely explanation for his apnoea and hypothermia is a hypothalamic defect. The hypothalamus is important in temperature control³ and may play a part in respiratory regulation.⁴ Hypothermia and apnoea have been associated with agenesis of the corpus callosum.⁵ Hypothalamic endocrine dysfunction has been noted in the absence of structural abnormality.² Structural abnormality has also been seen at necropsy despite normal endocrine values during life.³ Our negative results do not therefore completely exclude a hypothalamic lesion.

Although more sudden infant deaths occur in winter, hyperthermia is more favoured than hypothermia as a possible cause. Our patient maintains the interest in the relation between temperature and sudden infant death.

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Correction

Cigarette smoking and prolactin in women

We regret that an error occurred in this article by Dr John A Baron and others (23 August, p 482). In the table prolactin values were given in SI units (nmol/l); the conversion at the foot of the table should have read: 1 nmol/l ≈ 25 µg/l, and not as stated, 0.04 µg/l.