During the same year, 6584 cases of mumps were reported to the health authorities. Although one considers many unreported and subclinical infections, the occurrence of six cases of deafness due to mumps seems significantly higher than the assumed rate of 1/20 000 infections.¹ According to Sullivan *et al.*,² about 2.5% of patients with mumps may require treatment in hospital; considering this possible rate of admission the incidence of severe hearing loss associated with mumps may have been as high as 1/3400 cases of clinical mumps.

Two groups of patients with deafness related to infection with mumps may have been missed by the study of Hall and Richards: firstly, cases of mumps that were not apparent, which are considered to be in the range of 30–40% of all infections with mumps.³ In these cases, it is impossible to obtain a history of parotitis, but, as shown by our third patient, severe hearing loss may, none the less, occur. Secondly, there are rare cases of bilateral hearing loss caused by infection with mumps.⁴ Therefore, the importance of mumps as a cause of acquired deafness may be greater than usually estimated.

References

- Hall R, Richards H. Hearing loss due to mumps. *Arch Dis Child* 1987;**62**:189–91.
- Sullivan KM, Halpin TJ, Kim-Farley R, Marks JS. Mumps disease and its health impact: an outbreak report. *Pediatrics* 1985;**76**:533–6.
- Galbraith NS, Young SEJ, Pusey JJ, Crombie DL, Sparks JP. Mumps surveillance in England and Wales 1962–81. *Lancet* 1984;**i**:91–4.
- Linsay JR, Davey PR, Ward PH. Inner ear pathology in deafness due to mumps. *Annals of Otolaryngology* 1960;**69**: 918–35.

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Non-convulsive status epilepticus

Sir,

We have read Manning and Rosenbloom's interesting paper concerning non-convulsive status epilepticus.¹ They report 13 patients, five of whom presented with fluctuating neurological symptoms (ataxia, dysphasia, and unresponsiveness) and developmental deterioration coinciding with continuous paroxysmal activity on the electroencephalogram. The authors stress the 'epileptic' nature of such symptoms. They have not, however, reported whether the evidence of status epilepticus was also detected on an electroencephalogram taken during sleep.

We have observed five boys and five girls with a mean age of 6 years who have long standing 'electrical status epilepticus during slow sleep'. In six cases focal or

unilateral epileptiform activity occurred during more than 85% of non-rapid eye movement sleep, but electroencephalograms taken while they were awake disclosed only a frontal or frontal/central focus. The remaining patients presented with typical electrical status epilepticus during slow sleep characterised by a generalised epileptic pattern on electroencephalography during non-rapid eye movement sleep.² All patients had fluctuating neurological symptoms as well as disturbance of gait and motor coordination, speech impairment, behavioural changes associated with alteration in responsiveness, and developmental deterioration. All these children were suffering from epileptic seizures and were mentally retarded (six presented with congenital cerebral palsy). Remission of electrical status epilepticus during slow sleep was observed in two patients who fully recovered.

We believe that such fluctuating symptoms are not directly related to paroxysmal activity seen on electroencephalograms, but probably reflect a more complex brain disorder. In our cases a continuous epileptic activity was recorded on electroencephalograms only during sleep, while neurological symptoms were present when the children were awake. Moreover, our observations suggest that electroencephalography should be performed during spontaneous sleep in children with fluctuating neurological symptoms and mental deterioration, even if the recording made when awake did not show non-convulsive status epilepticus.

References

- Manning DJ, Rosenbloom L. Non-convulsive status epilepticus. *Arch Dis Child* 1987;**62**:37–40.
- Patry G, Lyagoubi S, Tassinari CA. Subclinical electrical status epilepticus induced by sleep in children. *Arch Neurol* 1971;**24**: 242–52.

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Topical iodine, breastfeeding, and neonatal hypothyroidism

Sir,

The case of transient congenital hypothyroidism after topical iodine in pregnancy reported by Danziger *et al.*¹ confirms and extends our previous observation² that the cutaneous application of povidone-iodine (PVP-I) in mothers at the time of delivery results in iodine overload and in slight impairment of the thyroid function of their breastfed infants due to a Wolff Chaikoff effect.³ We recently observed severe transient congenital hypothyroidism in a breastfed infant born to a mother who had performed vaginal douching with PVP-I for gynaecological reasons twice a day since delivery. As shown in the figure the concentration of thyroid stimulating hormone (TSH) in serum was moderately raised at the time of systematic screening for congenital hypothyroidism, but thyroxine (T4) was normal. Control examinations performed on days 14 and 22 showed an appreciable increase in serum TSH

Figure Time course of the serum concentrations of thyroid stimulating hormone (TSH) and thyroxine (T4) in the infant and the iodine content of the urine of mother and infant and the mother's breast milk.

and decrease in serum T4; substitutive treatment (25 µg L-T4/day) was started on day 22. Physical examination of the child was normal. The figure shows that the iodine content of maternal milk and maternal and infant urines was extremely high during the vaginal douching period but reverted to normal within seven days after withdrawal of PVP-I treatment. Breastfeeding was not discontinued. Thyroxine treatment was interrupted after seven days and thyroid function remained normal during the next two months.

We agree that iodine overload should be systematically considered as a cause of neonatal transient hypothyroidism. We recommend that urinary iodine measurement is included in the control examinations of infants with abnormal screening tests even if the possibility of iodine overload is not suspected on the basis of the history of the infant and the mother. Our case indicates that early recognition and withdrawal of iodine causing the overload results in rapid and spontaneous resolution of the Wolff Chaikoff effect. As a consequence, longterm T4 treatment, with the unnecessary stress for the family, and the risk of iatrogenic hyperthyroidism as suggested by the data of Danziger *et al* could be avoided.

References

- 1 Danziger Y, Pertzalan A, Mimouni M. Transient congenital hypothyroidism after topical iodine in pregnancy and lactation. *Arch Dis Child* 1987;62:295-6.

- 2 Chanoine JP, Bourdoux P, Pardou A, Delange F. Iodinated skin disinfectants in mothers at delivery and impairment of thyroid function in their breastfed infants. In: Medeiros-Neto G, Gaitan E, eds. *Frontiers in thyroidology*. New York: Plenum Medical, 1986, Vol II: 1055-60.

- 3 Wolff G. Iodide goiter and the pharmacologic effects of excess iodide. *Am J Med* 1969;47:101-11.

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Monitoring end tidal CO₂

Sir,

Watkins and Weindling conclude that monitoring of end tidal CO₂ cannot be recommended for neonates with pulmonary disease, primarily because there are fluctuations in the alveolar arterial oxygen gradients associated with the clinical course of the disease.¹

This assumption may be correct. There are, however, other factors which may have accounted for the large PaCO₂ and PetCO₂ gradient that they found. Among these are the use of rapid ventilatory rates with low tidal volumes and high fresh gas flows, which are mentioned. Also of importance is the type of ventilator used. This is particularly so when end tidal gas is sampled proximally at the endotracheal tube connector. Infants weighing less than 8000 g and ventilated with a continuous flow, time cycled ventilator (for example, the Bourns) have large PaCO₂ and PetCO₂ gradients.² This may be partially due to the dilution of end tidal gas by the continuous flow of fresh gas past the sampling site. The capnograph waveform, therefore, either fails to reach a plateau or reaches a plateau that underestimates PaCO₂. In contrast, the use of a ventilator that automatically interrupts the flow of fresh gas at the completion of inspiration (for example, the Siemens Elema 'Servo' 900C) permits accurate prediction of PaCO₂ from PetCO₂ in infants.² The problems of end tidal sampling in children weighing less than 8000 g who are ventilated with a continuous flow ventilator can be partially overcome if sampling is performed distally, at the tip of the endotracheal tube.³

It would be of interest to know the type of ventilator used by Watkins and Weindling. It is possible that the capnometer was not measuring PetCO₂ accurately, as was assumed. End tidal CO₂ monitoring is useful during paediatric anaesthesia—it may yet be in neonatal intensive care.

References

- 1 Watkins AMC, Weindling AM. Monitoring of end tidal CO₂ in neonatal intensive care. *Arch Dis Child* 1987;62:837-9.
- 2 Badgewell JM, Heavner JE, May WS, Goldthorn JF, Lerman J. End tidal PCO₂ monitoring in infants and children ventilated