Monitoring end tidal CO2.

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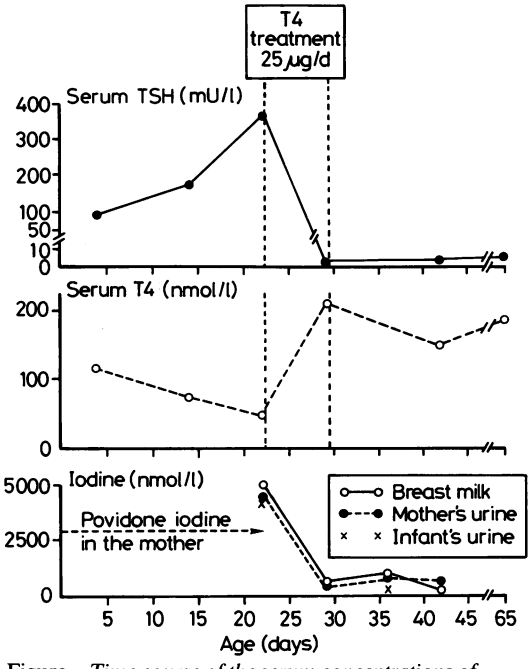

Figure *Time course of the serum concentrations of thyroid stimulating hormone (TSH) and thyroxine (T4) in the infant and the iodine content of the urine of mother and infant and the mother's breast milk.*

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, Pertzelan 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 CO_2

Sir,

Watkins and Weindling conclude that monitoring of end tidal CO_2 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 $PaCO_2$ and $PetCO_2$ 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 $PaCO_2$ and $PetCO_2$ 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 $PaCO_2$. 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 $PaCO_2$ from $PetCO_2$ 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 $PetCO_2$ accurately, as was assumed. End tidal CO_2 monitoring is useful during paediatric anaesthesia—it may yet be in neonatal intensive care.

References

[1] Watkins AMC, Weindling AM. Monitoring of end tidal CO_2 in neonatal intensive care. *Arch Dis Child* 1987;**62**:837–9.

[2] Badgewell JM, Heavner JE, May WS, Goldthorn JF, Lerman J. End tidal PCO_2 monitoring in infants and children ventilated

with either a partial rebreathing or a non-rebreathing circuit. *Anesthesiology* 1987;**66:**405–10.
[3] Badgewell JM, McLeod ME, Lerman J, Creighton RE. End tidal pCO_2 monitoring in infants and children during ventilation with the Air-Shields Ventimeter ventilator. *Anesthesiology* 1986;**65:**A418.

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Drs Watkins and Weindling comment:

We thank Dr Stow for highlighting a potential source of error in the monitoring of end tidal CO_2 in neonatal intensive care.

He is correct in surmising that we were using time cycled pressure limited ventilators with continuous gas flow (Vickers Neovent and SLE Newborn 250). It is the practice in this unit to use a flow rate of 7 l/minute in all cases unless high pressure and ventilator rates dictate a higher rate. All except two readings were at this rate and it is of interest that these (at 10 l/minute) were both inaccurate. This infant did, however, also have severe respiratory disease (alveolar/arterial oxygen difference 660 mm Hg) and so it is difficult to separate the two effects. We did find good correlations between end tidal CO_2 and $paCO_2$ measurements in some infants with milder respiratory disease despite the continuous gas flow, and so we felt that the effect of dilution was probably minimal. Our impression is that the effect of parenchymal lung disease far outweighs other effects in these infants.

The use of time cycled, pressure limited ventilators is almost universal in neonatal intensive care units. They are cheap and simple to use, and seem to be more effective in infants with hyaline membrane disease and its complications. Experience suggests that results are better than with volume cycled devices.[1]

Sampling of end tidal CO_2 from the tip of the endotracheal tube may well minimise any error due to gas flow. The majority of very low birthweight infants with hyaline membrane disease are ventilated using a 2·5 mm endotracheal tube. The luminal diameter is already critically small and considerably increases total respiratory resistance.[2] Any further impingement on the lumen should be avoided unless absolutely necessary.

We have already acknowledged[3] the important role of end tidal CO_2 monitoring in anaesthetics, when most of the patients have normal lungs. This is clearly not the case in infants ventilated in a neonatal intensive care unit, in whom we feel end tidal CO_2 monitoring to be inappropriate.

References

[1] Manginello FP, Grassi AE, Schechner S, Krauss AN, Auld PAM. Evaluation of methods of assisted ventilation in hyaline membrane disease. *Arch Dis Child* 1978;**53:**878–81.
[2] Le Souef PN, England SJ, Bryan CA. Total resistance of the respiratory system in preterm infants with and without endotracheal tubes. *J Pediatr* 1984;**104:**108.
[3] Watkins AMC, Weindling AM. Monitoring of end tidal CO_2 in neonatal intensive care. *Arch Dis Child* 1987;**62:**837–9.

Evaluation of nebulisers

Sir,

We were interested in the paper by Tsanakas *et al,*[1] and agree that it is important to have a nebuliser with the minimum variation in output when performing bronchial challenge tests. We were concerned, however, that the authors calculated the output of the nebuliser by weighing the device before and after nebulisation. This method does not give a true idea of the output of the nebuliser. As the nebulised cloud forms there is a huge cumulative surface area formed by the aerosol droplets. Most of these droplets are returned to the nebuliser solution by a series of baffles, allowing only the finer particles to escape. At the same time some evaporation takes place. The weight loss from the nebuliser, therefore, is due to dispersion of particles of the drug, such as histamine solution, but also to evaporation. Depending on which nebuliser is used, calculating the output by weighing the device before and after nebulisation may result in an overestimate of drug output of up to 50% (unpublished observations).

To measure the output of our nebulisers we used a multi stage liquid impinger which catches the nebuliser cloud as it emerges from the nebuliser. The impinger separates the cloud into fractions comprising particles of varying sizes and we then assayed the amount of drug in each fraction, so determining not only the actual amount of drug that leaves the nebuliser but also the amount of drug in particles that are likely to reach the lungs.

Reference

[1] Tsanakas JN, Wilson AJ, Boon AW. Evaluation of nebulisers for bronchial challenge tests. *Arch Dis Child* 1987;**62:**506–8.

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Sudden and unexpected death between 1 and 5 years

Sir,

The report by Southall *et al*[1] that one third of the deaths in their series of infants between 1 and 5 years were unexplained begs the question of why such a common phenomenon is not more widely recognised nor apparent from the Registrar General's annual mortality figures. I think that most pathologists would concede that unexplained deaths occur throughout childhood, adolescence, and adult life although not with the frequency seen during the first postnatal year.

When the sudden infant death syndrome was officially recognised by the Office of Population Censuses and Surveys (OPCS) as a distinct entity, the rise in deaths from this cause was matched by a decline in deaths from respiratory infection.[2] I can only suppose that the apparent rarity of unexplained deaths between 1 and 5 years has a

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