pg/ml (compared with 2556 and 2888 pg/ml after infusion at 80 ng/kg/min in the neonate reported here). The same workers found ANP infusion to attenuate the pulmonary response to hypoxia in pigs.[1]

On the first day of infusion the infant showed a slight fall in Pa_{O_2} at the end of the infusion followed, 90 minutes later, by an appreciable rise that lasted for approximately two hours. The initial fall might be explained by increased perfusion of poorly ventilated lung units[3] but the late rise is difficult to explain, though the cGMP excretion rate did remain raised for two hours. A dose dependent relaxation of pulmonary arteries, induced by ANP, has been shown in bovine arterial rings[2] but in our case ANP concentration returned to baseline by around 23 minutes.

In this infant high dose ANP infusion was relatively well tolerated. Although we do not claim that this child's satisfactory outcome was related to treatment with ANP, we do believe that ANP should be added to the list of therapeutic options in the management of persistent pulmonary hypertension of the newborn that require further study.

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An indirect calorimetry system for ventilator dependent very low birthweight infants

SIR,—While the need for measurement of energy expenditure and nutrient utilisation in sick ventilator dependent infants is undisputed, we have several reservations about the indirect calorimetry system described by Forsyth and Crighton.[1] On p316 is $\dot{V}_E$ the inspired or expired minute ventilation? The equations given appear to take these to be equal, even though this amounts to assuming that the respiratory quotient (RQ) is 1. If instead the inspired and expired volumes of *inert gas* are assumed equal the oxygen consumption ($\dot{V}O_2$) can be found as

$$\dot{V}O_2 = \dot{V}_{inspired}\left\{\frac{FIO_2 - FEO_2 - FIO_2 . FECO_2}{1 - FEO_2 - FECO_2}\right\};$$

a similar expression exists for carbon dioxide production. When $FIO_2 = 0{\cdot}4$ the equations in the paper underestimate $\dot{V}O_2$ by 12% if RQ= 0·7 and overestimate it by 8% if RQ=1·2; calculated values of RQ will be biased towards 1,[2] with true RQs of 0·7 and 1·2 being computed as 0·80 and 1·11 respectively.

In the gas infusion studies reported in table 1 it is unclear how to assess the values calculated for $\dot{V}O_2$ when nitrogen is infused into the system. This part of the study calculates $\dot{V}O_2$ as if the results were from a patient; the first equation on p318 then allows the calculated $\dot{V}O_2$ to be checked in terms of nitrogen flow rate and FIO_2. If $\dot{V}O_2$ is calculated correctly then this check equation is

$$\dot{V}O_2 = \frac{\dot{W}N_2 . FIO_2}{1 - FIO_2};$$

if the equations on p316 are used then this becomes:

$$\dot{V}O_2 = \frac{\dot{V}_{inspired}\dot{W}N_2 . FIO_2}{\dot{V}_{inspired} + \dot{W}N_2} \cong \dot{W}N_2 . FIO_2.$$

For the nitrogen flow rates given in table 1 (10·10, 14·10 ml/min) and FIO_2 is 0·40, the expected values of $\dot{V}O_2$ would be, from the first equation, 6·73, 9·40 ml/min and from the approximate version of the second equation, 4·04, 5·64 ml/min. From the results for the higher nitrogen flow rate it would appear that the second equation has been used (as would be consistent with the earlier part of the paper) but the agreement is poor at the lower flow rate.

Finally, we were disappointed that gas infusion studies were performed using only one ventilator setting. Increasing the inspired oxygen concentration (while keeping $\dot{V}O_2$ constant) causes a reduction in the inspired–expired oxygen difference ($FIO_2 - FEO_2$). Thus as $FIO_2 - FEO_2$ decreases, the error sensitivity in the measurement of $\dot{V}O_2$ is magnified.[3] It is not uncommon for sick ventilated infants to need FIO_2 up to 1·00. The errors in the measurement of energy expenditure at high oxygen concentrations will be markedly increased and this must be acknowledged.

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1 Forsyth JS, Crighton A. An indirect calorimetry system for ventilator dependent very low birthweight infants. *Arch Dis Child* 1992;**67**: 315–9.
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Dr Forsyth comments:

Although the general principles of indirect calorimetry may apply to all systems and subjects, there are some features which require specific consideration depending upon the nature of the system and the size of the subject being studied. In our system for ventilated low birthweight infants,[1] there are at least two important aspects which differ from adult systems, first the effect of changes in RQ on expiratory flow volume ($\dot{V}E$) and second, the validity of excluding $FICO_2$ from the calorimetry calculations.

Adult calorimetry systems commonly use an inspiratory flow volume ($\dot{V}I$) in the region of 40 l/min, and for a 70 kg individual with an oxygen consumption ($\dot{V}O_2$) of 320 ml/min and RQ of 0·7, the difference between $\dot{V}I$ and $\dot{V}E$ will be 96 ml (0·24% of $\dot{V}I$). In our system $\dot{V}I$ is 6 l/min (inspiratory flow from the ventilator), and for a 1500 g ventilated infant with a $\dot{V}O_2$ of 9 ml/min and RQ of 0·7, the difference between $\dot{V}I$ and $\dot{V}E$ is 2·7 ml, that is 0·045% of $\dot{V}I$. In order to correct for this difference, Drs Matthews and Matthews offer a formula based on adult data,[2] and which involves the values of four different measurements. Not only is there a risk that the overall error of these measurements may exceed the error induced by the difference in the $\dot{V}I$ and $\dot{V}E$ flow volumes but their equation is not valid for our system. If it is applied to the 1500 g infant, the 'corrected' $\dot{V}O_2$ will be 7·79 ml/min compared with the actual $\dot{V}O_2$ of 9·0 ml/min (an underestimate of 13·3%). This underestimate is constant, and not limited to the margins of RQ. Using our simpler equation the calculated $\dot{V}O_2$ at RQ 0·7 is 7·9 ml/min (underestimate 12%), but this error reduces to 0% as the RQ approaches 1·0. The large persistent error with the Matthews' equation is due to the omission of $FICO_2$. In adults this is usually less than 1% of $FECO_2$ and commonly ignored, but for a low birthweight infant in our system the $FICO_2$ can account for up to 22% of $FECO_2$. The formula should therefore be corrected to

$$\dot{V}O_2 = \dot{V}I . \left\{\frac{FIO_2 - FEO_2 - FIO_2 . FECO_2 + FEO_2 . FICO_2}{1 - FEO_2 - FECO_2}\right\}$$

A simpler and potentially more accurate adjustment is the traditional Haldane correction, $\dot{V}I = \dot{V}E . F_{EN_2}/F_{IN_2}$, and as our system is continually measuring inspiratory and expiratory nitrogen this can be easily accommodated.

During the nitrogen infusion study the mean FIO_2 was 0·421 and $\dot{V}E$ 5·257 l/min. By using the measured values for $\dot{V}E$, FIO_2, and FEO_2 the predicted $\dot{W}N_2$ were compared with the actual $\dot{W}N_2$, and using our last equation the actual oxygen consumption as opposed to the simulated $\dot{V}O_2$ was confirmed to be zero. The $\dot{V}O_2$ data reported with the nitrogen infusions were calculated as for an infant study using the described software calculations and were included, albeit with the limitations as discussed above, to relate the infusion data to actual levels of $\dot{V}O_2$ which are seen in low birthweight infants. Unfortunately we did transcribe the wrong data point for the $\dot{V}O_2$ for the nitrogen infusion of 10·1 ml/min and this should have been 4·20 (0·22) ml/min. We are grateful for this being drawn to our attention.

Although we will shortly be providing clinical and technical data on the use of our system with higher levels of FIO_2, it is our experience that in the present surfactant era considerably fewer babies are requiring a very high FIO_2 for a long period of time. We realise that some babies do require an FIO_2 as high as 1·0, but we believe that for those babies there are more urgent priorities than a measurement of energy expenditure.

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Imposed upper airway obstruction and covert video surveillance

SIR,—Covert video surveillance (CVS) may be extremely useful and is often the only method to prove that some cases of apparent life threatening events (ALTE) are caused by imposed upper airway obstruction. Samuels

et al, recently reported 14 cases which they documented in this manner.[1]

Within a period of six months we have seen three babies with ALTE from three unrelated families. All three had been discovered limp, cyanotic, and apparently lifeless during their afternoon nap. Petechial haemorrhages were found on the face and neck of two of the babies on admission. No other episodes occurred during observation in hospital or the follow up period.

These would probably have remained unexplained unrelated cases were it not that when the third case was brought to the casualty department by the family doctor he was accompanied by the babysitter who had discovered the baby. She was recognised by one of the nursing staff as a regular attender at the casualty department with minor wounds that were suspected of being caused by automutilation and she had made several allegations of being attacked or raped.

In the ensuing discussions it came to light that this woman was the babysitter who had also been involved in the first two cases. She has since been investigated by the police. However, in spite of strong suspicions of imposed upper airway obstruction of the babies by her, there has been insufficient evidence to bring her to trial.

CVS as described by Southall *et al*[2] would have failed in these cases, because it is usually only the parents or very close relatives who are allowed to be continually present with the baby in hospital. Very thorough history taking especially concerning the surrounding circumstances remains extremely important in investigating every case of ALTE especially if imposed upper airway obstruction is suspected.

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1 Samuels MP, Mc Claughlin W, Jacobson RR, Poets CF, Southall DP. Fourteen cases of imposed upper airways obstruction. *Arch Dis Child* 1992;**67**:162–70.
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Anal abnormalities in childhood myotonic dystrophy

SIR,—The paper by Reardon and colleagues on abnormal anal signs seen in myotonic dystrophy is worrying on several counts.[1] The physical signs are described inadequately which makes interpretation of the paper difficult. The Royal College of Physicians report,[2] while referenced, is not heeded in the need for a consensus on definition and method of examination. Thus 'a reflex dilation of the anus was observed on parting the buttocks'. How was the child examined, for how long, and was this a dynamic sign and what degree of dilation was observed? It is noteworthy that only one child in six was said to demonstrate this sign but all had anal laxity.

The illustration shows a degree of anal laxity we have never seen, is this laxity or dilation? Was this degree of gaping achieved on merely parting the buttocks? Was the child constipated and demonstrating the 'visibly relaxed anus' of Clayden?[3] This child was 15 years old and had had a lifetime of soiling, what had earlier examinations revealed and to what treatments and manoeuvres had she been subjected?

Single physical signs are rarely diagnostic and in making a diagnosis of child sexual abuse the jigsaw must be carefully constructed.[4] To begin an investigation on the basis of a child with a known bowel dysfunction and anal laxity is clearly problematic but in the context of a girl who is alleging abuse it is, in child protection terms, quite proper. Advice to doctors stated 'It is important to take what the child says very seriously, and to spend time listening to what the child has to say.'[5] It is also evident that children with special needs are at risk of abuse and to recognise abuse in children with communication problems requires particular skill.[6] Children do need protection from the trauma of wrongful diagnosis but similarly physicians have until recently failed their patients by their inability to recognise maltreatment.

Finally, what proportion of children with myotonic dystrophy have bowel disorders or an abnormal anus on examination? What proportion have no abnormality? The association of two conditions does occur.

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1 Reardon W, Hughes HE, Green SH, Lloyd Woolley V, Harper PS. Anal abnormalities in childhood myotonic dystrophy—a possible source of confusion in child sexual abuse. *Arch Dis Child* 1992;**67**:527–8.
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Drs Reardon, Hughes, and Green and Professor Harper comment:

The comments of Drs Wynne and Hobbs reflect the difficulties with which clinicians are obliged to grapple in attempting to confirm or refute a suspected diagnosis of child sexual abuse. Foremost among these is the non-specificity of single clinical signs. However, in many instances, abnormalities of anal physiology do contribute significantly to decisions with far reaching effects for patients and families. It is precisely because we absolutely agree with Drs Wynne and Hobbs that 'children need protection from the trauma of wrongful diagnosis' and because of the central role which anal abnormalities and interpretation thereof often has in reaching diagnoses of sexual abuse that we felt prompted to submit our report.

The report did not pretend to be a scientific treatise of anal sphincter dysfunction in myotonic dystrophy. Our hope in sharing these non-specific clinical observations is to make colleagues aware of added potential pitfalls which attend the diagnosis of child sexual abuse in myotonic dystrophy. We feel that shared clinical experiences such as ours are of value to clinicians in avoiding inappropriate diagnoses of child sexual abuse.

Prostacyclin concentrations in haemolytic uraemic syndrome after acute shigellosis

SIR,—Although the epidemiology of certain forms of the haemolytic uraemic syndrome (HUS) has become clearer in recent times, pathogenic details remain largely unresolved. The role, if any, of prostacyclin (PGI2) in the diarrhoea-associated forms of HUS (D+HUS) cannot be deduced from previous reports and the study by Alam *et al* on patients with shigella induced HUS adds to the confusion.[1] In the following we acknowledge, however, that in past European and American studies the aetiology of D+HUS is likely to be verocytotoxin producing *Escherichia coli*, which although similar is not homologous with shigella induced HUS.

Prostacyclin is probably not a circulating hormone in man: its plasma half life is brief and intact molecules cannot be measured directly in clinical practice. Thus the term 'prostacyclin concentration' is at best an extrapolation. Two main approaches have been used to identify abnormalities of PGI2 metabolism in disease states. The more direct of these is to measure stable degradation products such as 6-keto PGF-1α in biological fluids. The commonly used radioimmunoassay kits give plasma estimates which exceed those of the more specific gas chromatography and mass spectrometry. Blood sampling in itself induces artifacts, and although the urinary excretion of degradation products may be more consistent in health, this is unlikely to be a reliable method when renal function is rapidly changing. Both Stuart *et al*[2] and Turi *et al*[3] found plasma concentrations of prostacyclin metabolite to be increased in D+HUS at onset.

The second approach has been to determine whether plasma or serum stimulates generation of prostacyclin by endothelial tissue in vitro. Prostacyclin release into the supernatant medium is measured indirectly either by bioassay or as 6-keto PGF-1α. Published results are conflicting; Schlegel *et al*[4] and Levin[5] found that prostacyclin generation was promoted by plasma, whereas Turi *et al*[2] and Siegler *et al*[6] showed reduced production. Methodological differences may explain these inconsistent findings, but abnormalities of plasma stimulating factors or inhibitors have in any case not been shown to reflect disturbances of prostacyclin metabolism in vivo.

Unfortunately Alam *et al* did not make clear which method they used. The result section and abstract refer to plasma concentrations of 6-keto PGF-1α suggesting direct measurement. However, their method refers to radioimmunoassay for 'prostacyclin', but also to generation of 6-keto PGF-1α by rabbit aortic rings in response to patients' plasma.

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