PostScript

LETTERS

The pressure is on! The danger of a broken blow off valve on a bag valve mask

We would like to draw the reader's attention to a potentially dangerous occurrence with bag valve mask systems when repair and reassembly is carried out incorrectly. Bag valve masks are in common use in neonatal and paediatric resuscitations^{1,2} and incorporate a pressure relief valve to prevent excessive pressures being delivered.

In this case, checking of a Laerdal paediatric (500 ml) resuscitator (catalogue number 86005033) before use revealed that the blow off valve was not working despite the initial cursory inspection failing to detect any problem (fig 1). Closer inspection revealed that the yellow over-ride button had been broken off and the device reassembled in such a way that the button was placed inside the valve the wrong way round (fig 1 inset). The resuscitator was taken out of use and subjected to further testing. The problem was then brought to the attention of the manufacturer.

As in other reports of problems with bag valve mask systems used in adults and children, the fault was not immediately apparent.^{3,4} In this case the reassembled device did not look so different from the normal functioning valve (fig 2 and inset), yet by connecting it to a manometer we could generate pressures of 70 cm H₂O even with gentle manual ventilation.

Although it seems likely that the blow off valve was reassembled by an inexperienced person, we feel that this could have been prevented had the button been too large to fit within the valve body. This design fault is not unique to Laerdal resuscitators and should merit a review by all manufacturers. This event also highlights to clinicians that equipment should always be checked before use and that visual inspection alone cannot be relied on to reveal any potential problems.

S B Ainsworth, R Humphreys, L Stewart

Neonatal Unit, Directorate of Women & Children's Health, Forth Park Hospital, Kirkcaldy KY2 5AH, Scotland, UK

Correspondence to: Dr Ainsworth; sean.ainsworth@faht.scot.nhs.uk

Figure 1 The "odd" appearance of the spring loaded blow off valve. Inset: the spring loaded pressure regulator assembly shown as it was reassembled with top of the yellow part reversed.

Figure 2 The spring loaded blow off valve of the Laerdal silicone paediatric resuscitator as it should normally appear. Inset: the spring loaded pressure regulator assembly, unscrewed to show the normal configuration.

doi: 10.1136/adc.2005.076018

Competing interests: none declared

References

- 1 O'Donnell CPF, Davis PG, Morley CJ. Positive pressure ventilation at neonatal resuscitation: review of equipment and international survey of practice. *Acta Paediatr* 2004;**93**:583-8.
- 2 Mackway-Jones K, Phillips B, Wieteska S, eds. *Advanced paediatric life support: the practical approach*, 4th ed. London: BMJ Books, 2004.
- 3 Smith G. Problems with mis-assembly of adult manual resuscitators. *Resuscitation* 2002;**53**:109-11.
- 4 Cushing P. Mis-assembly of adult and paediatric manual resuscitators. *Resuscitation* 2002;**55**:347-8.

Nasal nitric oxide to diagnose primary ciliary dyskinesia in newborns

Retrospective data suggest that approximately half of patients with primary ciliary dyskinesia (PCD) have symptoms of neonatal respiratory distress. Respiratory distress syndrome in a full term infant should therefore raise PCD as a potential underlying disease.^{1,2} The non-invasive measurement of nasal nitric oxide (NO) is of diagnostic value in adults and children with PCD,^{3,4} but similar information is not available for neonates with PCD.

A 3550 g male infant was delivered after uncomplicated pregnancy by caesarean section at 41 weeks of gestation. During the first hours of life, respiratory distress developed, and supplemental oxygen was required to maintain arterial oxygen saturation above 88%. Neonatal infection was suspected, and antibiotic treatment initiated. Chest radiographs and abdominal ultrasound scans showed complete situs inversus, compatible with the diagnosis of Kartagener's syndrome. C reactive protein increased to a maximum of 87 mg/l on day 2, blood cultures remained sterile, and throat swabs showed *Klebsiella pneumoniae*. Antibiotic therapy was continued for 10 days.

Measurements of nasal NO revealed concentrations below 5 ppb on day 4, while the patient was treated for neonatal infection. Low nasal NO was confirmed after antibiotic therapy was discontinued and the infant was without clinical symptoms on day 34 of life

(9.4 ppb). Mean nasal NO in six healthy term newborns with a median age of 14 days (range 2-24) was 171.2 ppb (range 100-232).

The diagnosis of PCD was subsequently confirmed by nasal brush biopsy at 6 weeks of age.

PCD is a rare cause of neonatal respiratory distress but is under-diagnosed, as many infants recover spontaneously. Delayed diagnosis is accompanied by poorer outcome, as sufficient treatment maintains lung function or at least delays disease progression in the lower airways.^{1,4} Therefore there is a need for early diagnosis and treatment. This case illustrates that nasal NO measurements may be used to diagnose PCD in the newborn.

NO is thought to play an important role in postnatal adaptation of the pulmonary circulation. Decreased nasal NO in the neonate may have a negative impact on physiology of the lower airways, as newborns usually inhale high levels of NO which form in the upper airways. It can therefore be speculated that decreased nasal NO in newborns may contribute to the clinical signs of neonatal respiratory distress.

F Stehling, C Roll, F Ratjen, H Grasemann

Children's Hospital, University of Duisburg-Essen, Essen, Germany

Correspondence to: Dr Grasemann, The Hospital for Sick Children, Division of Respiratory Medicine, 555 University Ave, Toronto, Ontario, Canada M5G 1X4; hartmut.grasemann@sickkids.ca

doi: 10.1136/adc.2005.086702

Competing interests: none declared

References

- 1 Meeks M, Bush A. Primary ciliary dyskinesia (PCD). *Pediatr Pulmonol* 2002;**29**:307-16.
- 2 Holzmann D, Felix H. Neonatal respiratory distress syndrome: a sign of primary ciliary dyskinesia? *Eur J Pediatr* 2000;**159**:857-60.
- 3 Baraldi E, Pasquale MF, Canginiotti AM, et al. Nasal nitric oxide is low early in life: case study of two infants with primary ciliary dyskinesia. *Eur Respir J* 2004;**24**:881-3.
- 4 Coren ME, Meeks M, Morrison I, et al. Primary ciliary dyskinesia: age at diagnosis and symptom history. *Acta Paediatr* 2002;**91**:667-9.

Pooling of trials is not appropriate in the case of heterogeneity

We read with great interest the systematic review by Thome *et al*¹ on the elective use of high frequency ventilation compared with conventional mechanical ventilation in preterm infants. Thome *et al* included 17 randomised trials and stated that, unlike previous meta-analyses, they did not find significant benefits in pulmonary outcome. They also referred to our published cumulative meta-analysis.² However, we would like to point to the fact that, in our meta-analysis, we reported the same finding, but we restricted pooling to subgroups according to ventilation strategies used for both high frequency ventilation and conventional mechanical ventilation. The reason we did not pool all studies was because there was considerable heterogeneity between studies. The use of random effect models to overcome the problem of heterogeneity is

debatable.³ Thome *et al* showed an odds ratio of 0.87 with a 95% confidence interval of 0.75 to 1.00. Strictly speaking, the effect of high frequency ventilation compared with conventional mechanical ventilation was not statistically significant. However, these results would leave ample room for debate whether or not high frequency ventilation confers favourable pulmonary outcome. Instead, comparing high frequency ventilation with a high lung volume strategy with optimal conventional mechanical ventilation with a lung protective strategy resulted in our study in a relative risk of 0.95 with a 95% confidence interval of 0.85 to 1.07. Thome *et al* used the same technique of recursive meta-analysis as we used in our publication to quantify the relative change in effect size between trials over time and attributed these changes to differences in ventilation strategies used in trials. To establish a causal relation between differences between trials and reported effect estimates is problematic. Such an analysis would be observational with trial as unit of measurement. As in all observational research, associations could be confounded by covariates. For example, the fact that Thome *et al* did not find an association between time lag of enrolment and outcome could be confounded by the covariate ventilation strategy used in conventional mechanical ventilation.

In conclusion, we fully agree with Thome *et al* that ventilation strategies used in both high frequency ventilation and conventional mechanical ventilation seemed to play an important role in determining outcome. However, pooling of all studies in spite of considerable heterogeneity was not appropriate. The exact causal relations of differences between trials in ventilation strategies, time of enrolment, and population characteristics with effect sizes have yet to be established.

C W Bollen, C S P M Uiterwaal, A J van Vught
University Medical Centre, Lundlaan 6, Utrecht, the Netherlands; c.w.bollen@umcutrecht.nl

Competing interests: none declared

References

- 1 Thome UH, Carlo WA, Pohlandt F. Ventilation strategies and outcome in randomised trials of high frequency ventilation. *Arch Dis Child Fetal Neonatal Ed* 2005;**90**:466–73.
- 2 Bollen CW, Uiterwaal CS, van Vught AJ. Cumulative metaanalysis of high-frequency versus conventional ventilation in premature neonates. *Am J Respir Crit Care Med* 2003;**168**:1150–5.
- 3 Egger M, Smith G, Altman D. *Systematic reviews in health care*, 2nd ed. Blackwell: BMJ Books, 2001.

Asphyxia or just hypoxia?

Dr Stenson claims that the article of Fugelseth *et al*¹ showed that “The use of 100% oxygen offered no benefit in terms of restoration of cardiac output or normalisation of pulmonary artery pressure after severe asphyxia”.² However, if we still take asphyxia

to mean “without pulse”, the pigs were not asphyxiated, they were just rendered hypoxic.

Fugelseth *et al* largely avoid the term asphyxia in their article, but cite another study from Oslo³ which they state involved asphyxiated pigs (although actually that also involved hypoxia alone).

My dictionary does not help much here. It states that asphyxia used to mean “without pulse”, but now just means the same as suffocation. It clearly does not. Perhaps it's time for us medics to reclaim this useful, and quite descriptive word.

T H M Fenton

London Road, Croydon CR7 7YE, Surrey, UK;
theo.fenton@mayday.nhs.uk

Competing interests: none declared

References

- 1 Fugelseth D, Børke WB, Lenes K, *et al*. *Arch Dis Child Fetal Neonatal Ed* 2005;**90**:229–34.
- 2 Stenson B. Fantoms. Air or oxygen? *Arch Dis Child Fetal Neonatal Ed* 2005;**90**:F189.
- 3 Børke WB, Munkeby BH, Morkrid L, *et al*. Resuscitation with 100% O₂ does not protect the myocardium in hypoxic newborn piglets. *Arch Dis Child Fetal Neonatal Ed* 2004;**89**:F156–60.

Not all osmolality is created equal

The article “Increased osmolality of breast milk with therapeutic additives” by Srinivasan *et al*¹ reports the osmolality of supplements, drugs, and fortifiers in breast milk for preterm infants based on the assumption that laboratory measured osmolality presents an increased risk of necrotising enterocolitis (NEC) for the preterm infant. When the association between hyperosmolar feeds and NEC is considered, there may be an important differentiation of osmolar substances into two groups: some substances create an osmotic gradient in vivo whereas others do not.² There is a difference between the osmolality that is measured in a laboratory and the effective osmolality in vivo.³ Molecules that cross semipermeable membranes, such as alcohol, do not present an osmotic load in vivo, but do contribute to osmolality measured by an osmometer.³ Molecules that do not set up an osmotic gradient are not likely to increase the risk of NEC by their osmolality.

Drugs as well as vitamin supplements often contain carrier molecules that can diffuse across membranes.² On the other hand, nutrients in feeds, such as sugars, amino acids, salts, minerals, and the constituents of breast milk fortifiers do not readily diffuse across membranes. Therefore hyperosmolar feeds are more likely to pose an osmolar risk when the source of the hyperosmolality is nutrients, but may not pose an osmolar risk when the source of the hyperosmolality is drugs and supplements.

The association between NEC and osmolality in preterm infants has been studied, inadvertently, in only one randomised clinical trial.⁴ The remainder of the evidence that

associates NEC with high osmolality feeds is from one quasi-experimental trial,⁵ three case reports,^{6–8} and some animal studies.^{9–14} In all of these studies, the source of hyperosmolality associated with damage to the intestine was substances that presented an osmolar load to the intestine.

The only substances that need to be considered risky in terms of osmolality for potential inducement of NEC are those that create an osmotic gradient across the intestinal membrane in vivo. Some of the measured osmolality from drugs and vitamin supplements is not likely to present a risk from their osmolar load.

T R Fenton

Department of Community Health Sciences, Faculty of Medicine, University of Calgary, 3330 Hospital Drive NW, Calgary, Alberta, Canada T2N 4N1, Canada;
tanisfenton@shaw.ca

Competing interests: none declared

References

- 1 Srinivasan L, Bokincic R, King C, *et al*. Increased osmolality of breast milk with therapeutic additives. *Arch Dis Child Fetal Neonatal Ed* 2004;**89**:F514–17.
- 2 Newmark HL, Johnson L, Etches PC, *et al*. Osmolality: limits of physical tests. *Dev Pharmacol Ther* 1988;**11**:313–16.
- 3 Gennari FJ. Current concepts. Serum osmolality. Uses and limitations. *N Engl J Med* 1984;**310**:102–5.
- 4 Book LS, Herbst JJ, Atherton SO, *et al*. Necrotizing enterocolitis in low-birth-weight infants fed an elemental formula. *J Pediatr* 1975;**87**:602–5.
- 5 Willis DM, Chabot J, Radde IC, *et al*. Unsuspected hyperosmolality of oral solutions contributing to necrotizing enterocolitis in very-low-birth-weight infants. *Pediatrics* 1977;**60**:535–8.
- 6 White KC, Harkavy KL. Hypertonic formula resulting from added oral medications. *Am J Dis Child* 1982;**136**:931–3.
- 7 Tuladhar R, Daftary A, Patole SK, *et al*. Oral gastrografin in neonates: a note of caution. *Int J Clin Pract* 1999;**53**:565.
- 8 Wilcox DT, Fiorello AB, Glick PL. Hypovolemic shock and intestinal ischemia: a preventable complication of incomplete formula labeling. *J Pediatr* 1993;**122**:103–4.
- 9 Norris HT. Response of the small intestine to the application of a hypertonic solution. *Am J Pathol* 1973;**73**:747–64.
- 10 Stordahl A, Laerum F, Lunde OC, *et al*. The effects of water-soluble contrast media on luminal distension and blood flow in closed loops of small bowel in minipigs. *Scand J Gastroenterol* 1988;**23**:991–9.
- 11 Kameda H, Abei T, Nasrallah S, *et al*. Functional and histological injury to intestinal mucosa produced by hypertonicity. *Am J Physiol* 1968;**214**:1090–5.
- 12 Torma MJ, Flannery MJ, French HG, *et al*. Intestinal mucosal exfoliative response to graded hypertonic loads in neonatal and premature primates. *Surg Forum* 1978;**29**:410–13.
- 13 Teichberg S, Lifshitz F, Pergolizzi R, *et al*. Response of rat intestine to a hyperosmotic feeding. *Pediatr Res* 1978;**12**:720–5.
- 14 Stordahl A, Haider T, Laerum F. Acute lethality after enteral administration of contrast media in anaesthetized rats with intestinal ischaemia. *Acta Radiol* 1989;**30**:213–16.

Book reviews in ADC this month

The following book reviews are published in this month's *Archives of Disease in Childhood*:

- Set free childhood—coping with computers and TV
- The treatment of epilepsy, 2nd edition
- Practical paediatric problems, a textbook for the MRCPCH
- Towards MRCPCH Part II theory examination
- Get through MRCPCH Part 2: 125 questions on clinical photographs