Vibration during high frequency ventilation in neonates

Since there is no report quantifying vibration imposed upon neonates, we prospectively studied the vibration produced during high frequency ventilation (HFV) and compared it with that during conventional mechanical ventilation (CMV) of studied patients and weight matched controls (±250 g) receiving CMV or breathing spontaneously. A non-invasive accelerometer (Analogy Devices ADXL05, Norwood, MA, USA) was placed at the mid sternum or postauricular cranium to measure the linear vibration transmitted to the body and head, respectively (amplitude in time and frequency domains expressed in units of "g").

From April to October 1998 we enrolled seven neonates treated with HFV (mean (SD) weight 2210 (1680) g, gestation 32 (7) weeks) and 14 weight matched controls with that during conventional mechanical ventilation (CMV) of studied patients and weight matched controls (±250 g) receiving CMV or breathing spontaneously. A non-invasive accelerometer (Analogy Devices ADXL05, Norwood, MA, USA) was placed at the mid sternum or postauricular cranium to measure the linear vibration transmitted to the body and head, respectively (amplitude in time and frequency domains expressed in units of "g").

HFV treated neonates were higher than those of weight matched controls (fig 1, p<0.001), whereas no significant difference was found between the control groups. A higher amplitude of vibration at the chest was found in neonates with an adverse outcome than in normal survivors. Vibration at the chest exceeded the limit of whole body vibration in adults (0.05g at 12.5 Hz third octave band for 24 hours per ISO 2631).

The significance of our observations is not known. While cardiovascular instability is commonly observed in neonates during HFV and has been related to a high lung volume ventilation strategy, cardiovascular effects of vibration have been reported in animal and clinical studies. We speculate that the vibration during HFV may also contribute to the haemodynamic instability in neonates.
Furthermore, the effect of vibration on the developing brain is uncertain. We do not know whether the vibration will compromise the cerebral haemodynamic stability resulting in adverse neurological outcomes, especially in premature neonates who transmit vibration more efficaciously because of less body mass and fat compared with term neonates. Moreover, the combined effects of vibratory stress and environmental noise may contribute to hearing loss.  
Although no definitive vibration disease has been recognised in neonates, we have demonstrated the inadvertent exposure of neonates to excessive vibration. Research is required to examine the significance of HFV induced vibration and to reduce the vibration without compromising its effectiveness in critically ill neonates.

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National survey of detention and TB  
In England and Wales the power to detain individuals with tuberculosis who pose a threat to public health lies principally in sections 37 and 38 of the Public Health Act 1984. Section 37 authorises a local authority officer to remove an individual to a suitable hospital and section 38 authorises the “detention for a period specified in the order”. By way of the Public Health (Infectious Diseases) Regula-
tions 1988, in addition to the five notifiable diseases (cholera, plague, relapsing fever, smallpox, typhus), these sections currently apply to tuberculosis of the respiratory tract in an infectious state (although the term “infectious” is not defined by law).

Because central records are not kept, it has been unclear how many individuals are detained each year under legislation. No research has been conducted in Britain to determine trends in the use of detention as a public health tool in the control of tuberculo-
sis, although a survey conducted in the early 1990s of consultants in communicable disease control/medical officers of environmental health (CCDC/MOEH) reported the issuance of six orders, “equivalent to less than one use of the sections for every hundred years of CCDC/MOEH experience”.  

A brief structured postal questionnaire was sent to consultants in communicable disease control in all 99 health authorities in England and Wales in February 2000 requesting information on whether any detention orders (section 38 of the 1984 Public Health Act) for individuals with tuberculosis had been issued in the health authority to which the letter had been sent since 1993. Information was received from consultants in 97 of the health authorities.

Thirty detention orders were issued during the 6 years surveyed; the year of issuance was given for 29. There was no apparent clustering in any health authority although 13 orders (43%) were issued in London. The duration of the period of the detention orders varied from 3 days (n=1) to 6 months (n=6) with a median of 3 months.

The number of detention orders being issued for individuals with tuberculosis since 1994 has increased significantly (p<0.005). By 1999 0.2% of individuals notified with pulmonary tuberculosis were issued with detention orders. Regression analysis, taking into account of health authority response rates and notification rates for pulmonary tuberculosis, shows a significant increase in the issuance of detention orders since 1994 (b=1.8283, r²=0.9; fig 1).

The reasons for the increase in numbers of detention orders being issued are unclear. In an increase of tuberculosis outbreaks in healthcare settings and the scourge of drug resistant and multidrug resistant strains have, over the past decade, perhaps concentrated the minds of clinicians and public health physicians on ensuring that patients comply with treatment, and this may be playing a part. Elsewhere, notably in New York but also in Europe, consideration of the legal and ethical aspects of contemporary control measures has resulted in legislative amendments to public health laws which have enabled public health authorities to detain, for prolonged periods, patients with tuberculosis who will not or cannot comply with treatment.  

Failures in tuberculosis control allied to insufficient resources to facilitate patients’ adherence to treatment, particularly in London, may also be contributing to the use of more restrictive measures by the authorities. An alternative explanation may be that, because of drug resistance and associated HIV infection, the treatment of tuberculosis is becoming increasingly complex, demanding greater commitment from patients and clinicians. This survey suggests that there is a need to monitor formally, in an ongoing fashion, trends in the issuance of detention orders for individuals with tuberculosis.

I thank the consultants in communicable disease control in England and Wales for assisting with this research and the Communicable Disease Surveillance Centre for providing the data on pulmonary tuberculosis notifications.

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Churg-Strauss syndrome  
We read the review of Churg-Strauss syndrome (CSS) by Conron and Beynon with interest. The studies they quote are all reported from tertiary or national referral centres with inevitable referral bias. We have published our experience of 23 patients with CSS—the only study from a district general hospital which may be more representative of the natural history of this systemic vasculitis in
Acute rib fracture pain in CF

Recent papers in Thorax have described the high prevalence of low bone mineral density (BMD) in individuals with cystic fibrosis;1–4 these patients are at increased risk of fractures.1,4 Rib fracture pain can often be difficult to treat, despite standard analgesia such as non-steroidal anti-inflammatory drugs and opiates. Rib pain can impair sputum clearance and lead to an exacerbation of CF pulmonary disease. There are reports that calcitonin can relieve bone pain for patients with osteolytic metastases5 and osteoporotic vertebral fractures.3 Recently, we have successfully used subcutaneous calcitonin for the treatment of rib fracture pain in two patients with CF.

A 25-year-old woman fractured two ribs when she was crushed in the crowd at a rock concert and a 28 year old man fractured ribs following a bout of coughing. Both patients had CF related low BMD with DXA Z scores at the lumbar spine of –2.5 and –3.6, respectively. They had been taking long term oral prednisolone at a dose of 10 mg per day. Both patients had continuous uncontrolled pain from their fracture sites despite regular oral analgesics. The female patient was taking paracetamol 1 g qds and morphine sulphate modified release 30 mg bd: the male patient was taking paracetamol 1 g qds and ibuprofen 600 mg tds. Both patients were given courses of antibiotics as the pain was leading to an exacerbation of their CF lung disease. Subcutaneous calcitonin (salcatonin) was given in a dose of 50 units once daily. The pain completely resolved within 48 hours in both cases, and the patients were able to mobilise, perform sputum clearance, the other analgesics were withdrawn, and the chest exacerbations resolved. The calcitonin injections were continued for a total of 7 days, then stopped without recurrence of any pain. Neither patient experienced any side effects from the calcitonin.

Although calcitonin is involved in the regulation of bone turnover, the mechanism of its analgesic action is unknown. It reduces bone resorption and bone turnover in animal models but may also have central analgesic effects. Conversely, intravenous bisphosphonates, given to improve bone density, were associated with severe bone pain in individuals with CF.7 Pain control is essential in patients with CF and rib fractures if adequate sputum clearance is to be achieved and an acute deterioration in lung disease avoided. Calcitonin should be considered as an analgesic in this situation. Setting and sitting up may reduce morbidity and mortality associated with rib fractures in this group of patients.

Postscript

We read with interest the article by Roland et al on endothelin-1 (ET) levels in exacerbations of chronic obstructive pulmonary disease (COPD). The authors found increased sputum levels of ET-1 in patients with COPD during an exacerbation and this was reflected by a smaller rise in venous ET-1 levels.7 We have recently performed a study to evaluate pulmonary and renal ET levels in nine consecutive COPD patients during an acute exacerbation.6 ET was detected by radioimmunoassay in venous and arterial blood as well as in a timed urine specimen. For each subject the ratio of systemic arterial/venous mixed venous ET-1 levels (ir-ETart/ir-ETven) was evaluated as an index of pulmonary clearance/production of the peptide.1 The ir-ETart/ir-ETven ratio was calculated in patients with COPD examined both during an exacerbation and at recovery (0.75 (0.12) and 0.77 (0.13), respectively). Otherwise, the calculated ir-ETart/ir-ETven ratio in patients with COPD was significantly lower than that calculated in patients with acute pulmonary embolism, a condition in which pulmonary endothelial dysfunction is likely to occur.2 A significant difference was found with respect to a 24 hour ir-ET urinary excretion between COPD patients during an exacerbation and at recovery (142.1 (12.8) ng/24 hours and 89.0 (15.1) ng/24 hours, respectively). Furthermore, renal clearance of ET was higher in COPD patients during an exacerbation (29.2 (5.2) ml/min) than at recovery (17.5 (3.9) ml/min), suggesting an increase in renal ET production in patients with COPD during an exacerbation. These findings are in contrast to the reported significant changes in ET-1 circulating levels.

We therefore think that increased sputum ET-1 levels found in patients with COPD during an acute exacerbation could represent a true increase in local ET production with a firm relationship between venous and sputum ET-1 levels could not be established.

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CF and antistaphylococcal prophylaxis

Dr Robinson’s review of cystic fibrosis (CF) touches on the use of continuous antistaphylococcal antibiotic prophylaxis.1 Dr Robinson reiterates the oft repeated assertion that there is an association between the use of prophylactic antibiotics and the early acquisition of pulmonary infection with Pseudomonas aeruginosa in patients with CF. This association has entered CF folklore but is not supported by any published evidence. The paper cited by Robinson describes a pilot study of cephalixin which lasted 2 months.2 A subsequent multicentre, randomised, placebo-controlled trial of cephalixin, commenced in children under 2 years, has been undertaken and its methodology described.3 However, no results have so far appeared in the published literature.

We have recently published an updated systematic review of randomised controlled trials of prophylactic antibiotics in CF.4 This describes data from three studies involving 185 children. We found that fewer children...
who received prophylaxis from birth had one or more isolates of Staphylococcus aureus over a 3 year period. There was also evidence at 2 years that less time was spent in hospital in the prophylaxis group. The number of children receiving prophylaxis who had one or more isolates of P. aeruginosa over a 3 year period was half that of the control group who had intermittent antibiotic treatment only. This was not, however, statistically significant (Peto odds ratio 0.54, 95% confidence interval 0.23 to 1.26).

The steering group of the North American cephalaxin trial have indicated that its results have been interpreted as providing only weak evidence for or against prophylaxis, and that further studies are needed to confirm the results. This was not, however, statistically significant (Peto odds ratio 0.23 to 1.26).

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AUTHOR'S REPLY
I thank Drs Smyth and Walters for their comments concerning the issue of whether antistaphyloccocal prophylaxis leads to a higher risk of colonisation with Pseudomonas aeruginosa in patients with cystic fibrosis. I share their concern as to the lack of definitive data supporting this notion and, indeed, tried to illustrate this in my article by stating “There is some evidence that it may be associated ...” I would suggest, however, that the authors’ evidence of a lack of association is equally thin—to quote a multicentre trial whose methodology was presented as an abstract some 9 years ago but whose results do not appear to have ever been published in a peer reviewed journal is certainly not basing one’s evidence on hard evidence based facts. I did not mention the review by Smyth and Walters in my own paper as I submitted my review some 18 months before theirs had been published; however, the authors did not include in their own letter discussion of the recent paper by Ratjen et al using data from the German CF database which included 639 patients, all under 18 years of age and P. aeruginosa negative prior to entry in the study. 48.2% of the patients received continuous antistaphyloccocal treatment, 40.4% received intermittent antibiotic treatment, and 11.4% received no antibiotic treatment. While the rate at which patients acquired positive respiratory cultures for Staphylococcus aureus was significantly lower in the group receiving continuous antistaphyloccocal antibiotic treatment than in those receiving no such treatment, patients receiving continuous antistaphyloccocal antibiotic treatment had a significantly higher rate of P. aeruginosa acquisition than patients receiving only intermittent or no antibiotic treatment. This difference was especially apparent for children under the age of 6 years. The authors concluded that “continuous therapy with antistaphyloccocal antibiotics directed against Staphylococcus aureus increases the risk of colonisation with P. aeruginosa”. This interesting study I believe again supports my original statement that “there is some evidence that it (continuous antistaphyloccocal antibiotic therapy) may be associated with earlier acquisition of P. aeruginosa”.

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NOTICES

The Dr H M (Bill) Foreman Memorial Fund

The Trustees of the above fund invite applications for grants relating to study in respiratory disease and allied fields. Limited funds are available for registered medical practitioners to assist in travelling to countries other than their own to study respiratory disease, and also for support for clinical research abroad.

The HMF Fund has been able to fund two medical student projects in the last 18 months, one on TB in Bangladesh; Dr R T Jagoe (Newcastle upon Tyne) to study the ATP-ubiquitin-proteasome proteolytic system in Boston, USA; Dr J S Parmar (Cambridge) to study cell motility in Toronto; and a grant to Dr Anne Chang (Brisbane) to study the relationship between cough and asthma.

Intending applicants should provide further details to Dr Brian H Davies, Llandough Hospital, Penarth, Vale of Glamorgan CF64 2AA, UK.

PHARMACOLOGY OF ASTHMA

A course on the “Pharmacology of Asthma” organised by Professor Peter Barnes will be held at the Imperial College School of Medicine at the National Heart & Lung Institute in collaboration with the Royal Brompton Hospital, Dovehouse Street, London SW3 6LY, UK on 26–29 November 2001. The course is suitable for physicians or scientists with an interest in the pharmacology and therapeutics of asthma. For further information please contact the Postgraduate Education Centre, Imperial College School of Medicine at the National Heart & Lung Institute, Dovehouse Street, London SW3 6LY. Telephone: 020 7351 8172. Fax: 020 7351 8246. Email: shortcourses.nhli@ic.ac.uk

RESPIRATORY MEDICINE

A conference on Respiratory Medicine will be held at the Royal College of Physicians of Edinburgh on 26 October 2001. For further information contact Ms Eileen Strawn, Symposium Coordinator. Telephone 0131 225 7324. Fax 0131 220 4593. Email: e.strawn@rcpe.ac.uk. Website: www.rcpe.ac.uk.