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 accelerometric sensor (Analog Devices ADXL05, Norwood, MA, USA) was placed at the mid sternum and 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 (CMV group: n=7, 2100 (1730) g, 31 (8) weeks; spontaneous breathing group: n=7, 2150 (1520) g, 32 (7) weeks). The groups were not different with regard to body weight, length, and head circumference. Neonates received HFV at a frequency of 12 Hz, mean airway pressure of 14 (2) cm H2O, amplitude of 39 (10) cm H2O, and back up CMV at 6 breaths/min. Higher amplitudes of vibration were detected during HFV than during CMV (0.098 (0.026) y v 0.017 (0.006) g at the chest and 0.011 (0.003) y v 0.007 (0.001) g at the cranium, p<0.05) in six HFV treated neonates. One HFV treated neonate did not tolerate the switch to CMV. The vibrations at the chest and postauricular cranium in seven 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 (0.136 (0.014)g v 0.087 (0.024)g, respectively), while demographic data and the duration and amplitude of HFV were not different. Interestingly, the 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.

Figure 1 Section 38 detention orders controlled by pulmonary tuberculosis notification rate and health authority response rate in England and Wales, 1994–9.

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) Regulations 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 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
the general population. The mean age of onset was 57 years, significantly older than the 40 years reported in patients from tertiary centres and quoted by Conron and Beynon. We found there was a slight male preponderance (65%), also shown in many previous studies. The commonest sites of extraspinal involvement were, in descending frequency, the nervous system, joints and muscles, kidneys, skin, heart, and gastrointestinal tract. Involvement of the gastrointestinal tract occurred in only 30% and abdominal pain was uncommon. Cardiac involvement occurred in 44% and was the cause of death in two. Renal involvement, although occurring in nearly half the patients, was usually mild and none had severe renal failure.

The mean age of onset of asthma was 50 years and, although not reported, the asthma was not usually troublesome or severe when the systemic vasculitis developed. Indeed, very few patients were on oral steroids for their asthma at that time. Very rarely, patients present with CSS who do not have asthma. In our series there was one such patient who satisfied the original histological features of Churg and Strauss.

We found that the criteria developed by Lanham et al. were the most useful, particularly an eosinophil count of >1.5 × 10⁹/l. While this is arbitrary, only one of our patients had a level below this and that was 1.4 × 10⁹/l.

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: these patients are at increased risk of fractures. 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 metastases and osteoporotic vertebral fractures. 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 DEXA 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 controlled uncontinuous 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 50,000 IU was given every 48 hours 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 fluid flow but may also have central analgesic effects. Conversely, intravenous bispophonates, given to improve bone density, were associated with severe bone pain in individuals with CF.

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. Side effects are rare but may reduce morbidity and mortality associated with rib fractures in this group of patients.

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**Endothelin in acute exacerbations of COPD**

We read with interest the article by Roland et al. on endothelin-1 (ET-1) 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. We have recently performed a study to evaluate pulmonary and renal ET levels in nine consecutive COPD patients during an acute exacerbation. 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 ET-1 levels (ir-ETAr/ir-ETVen) was evaluated as an index of pulmonary clearance/production of the peptide. The ir-ETAr/ir-ETVen ratio was significantly lower 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-ETAr/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. A significant difference was found with respect to non 24 hr 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. This may also reflect the 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.
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 *Pseudomonas 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 cephalexin trial have indicated that its results will be published soon (Eliezer Nussbaum, personal communication). However, until there is published evidence from at least one properly designed randomised controlled trial, the proposal that prophylaxis encourages pulmonary infection with *P aeruginosa* remains entirely speculative.

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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.

**Letters to the editor**

The HMF Fund has been able to fund two medical student projects in the last 18 months, one on TB in Malaysia and one on TB in Ghana, and has awarded three travel grants to study the following aspects of respiratory disease: Dr Veronica White (London) to study 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

**CORRECTION**

In the “Statement on Malignant Mesothelioma in the United Kingdom” by the British Thoracic Society Standards of Care Committee which appeared in the April issue of *Thorax* (2001;56:250–65), the telephone number given for the National Mesothelioma Helpline on page 264 is incorrect. The correct number is 0113 206 6466. The email address is maviro@ulth.northy.nhs.uk