

# Effects of 15% oxygen on breathing patterns and oxygenation in infants

Infants are probably safe in aircraft

Papers p 887

n article in this week's issue raises the spectre that exposing a young infant to the relative hypoxia of an airline flight may increase the risk of sudden infant death (p 887). The authors base this claim on their findings that exposing 3 month old infants to 15% oxygen for about six hours led to an increase in the time spent in periodic respiration and the number of episodes of mild desaturation. In addition, four of the 34 infants had more prolonged hypoxia, with transcutaneous arterial saturation values below 80% for more than one minute, for which they were given oxygen therapy. They could not predict which infants were likely to develop the more prolonged pattern of desaturation from their baseline cardiorespiratory monitoring. They conclude that further research is urgently needed into the effects of airline flights or holidays at high altitude on infants, particularly as they had contact with two families who had experienced sudden infant death syndrome within two days of intercontinental flights. Even more urgent is the need to decide the relevance of their findings and what advice we should give to parents about to set out on flights with their young infants.

We already have a considerable amount of information on the effects of hypoxia on infants. All aircraft used for medium and long flights are pressurised so that the pressure within the cabin is equivalent to that found at about 1700 m in Boeing 747s and 2500 m in the older DC9s.2 At 2500 m the atmospheric pressure is 0.75 bar compared with 100 bar at sea level. This has the effect of reducing the arterial oxygen tension from 12.7 to 7.5 kPa in healthy adults, and the arterial oxygen saturation from 97 to 89%.2 In early infancy the relatively high concentration of fetal haemoglobin will have a protective effect, so that newborn infants in Tibet at altitudes of over 3600 m have arterial saturations of 92% in quiet sleep compared with only 85% at 4 months of age.3

The physiological effects of breathing hypoxic gas mixtures have been well investigated in early infancy. Unlike adults, who have prolonged stimulation of ventilation in response to hypoxia, newborn infants have initial stimulation followed by suppression.<sup>4</sup> However, this secondary suppression is seen only in the first 10-14 days of life.<sup>5</sup> Babies born very preterm become apnoeic in response to hypoxia<sup>6</sup> and take a variable time to develop the adult response pattern.

There is evidence that viral respiratory tract infections cause these infants to revert to the immature pattern with episodes of prolonged apnoea. There have also been several studies on infants into the cardiorespiratory effects of life at high altitudes. The increase in periodic breathing noted in this study has previously been documented in infants living in Denver and Tibet. Of concern is the report that infants of Chinese parents who had moved to Tibet were at greater risk of dying from pulmonary hypertension, at a mean age of 2.3 months, than infants of Tibetan parents.

Before creating widespead concern about the possible risk to infants it is important to put these considerations into context. According to British Airways, who fly over 34 million passengers a year, no cases of sudden infant death have been recorded during flight (personal communication). They claim that if one had occurred there would certainly have been a full investigation and the results publicised. It is difficult to obtain information on how many of these 34 million are infants, but assuming it is 1 in 500, the number would be over 750 000 over 10 years.

The only relevant recommendation given by the Aerospace Medical Association and the Air Transport Committee is that, although physiologically newborn infants should be able to fly quite safely, it would be prudent to wait about a week after birth to be sure that the infant is healthy. Obviously sensible precautions need to be taken for babies who have reduced respiratory reserve—who may need oxygen for the flight. However, all the epidemiological evidence indicates that, whatever the effect of relative hypoxia on breathing patterns, flying appears to be safe for healthy children in the first year of life.

Anthony D Milner *Professor of neonatology*United Medical and Dental School, St Thomas's Hospital, London

SE1 7EH

BMJ 1998;316:873-4

Parkin KJ, Poets CF, O'Brien LM, Stebbens VA, Southall DP. Effects of exposure to 15% oxygen on breathing patterns and oxygen saturation in infants: interventional study. BMJ 1998;316:887-94.

<sup>2</sup> Parsons CJ, Bobechko WP. Aeromedical transport. Can Med Assoc J 1982;126:237-43.

<sup>3</sup> Neirmeyer S, Yang P, Drolkar S, Zhuang J, Moore LA. Arterial oxygen saturation in Tibetan and Han infants born in Lhasa, Tibet. N Engl J Med 1995;33:1248-52

<sup>4</sup> Cross KW, Oppe TE. The effect of inhalation of high and low concentrations of oxygen on the respiration of the premature infant. J Physiol (Lond) 1952;117:38-55.

- 5 Brady JP, Ceruti E. Chemoreceptor reflexes in the newborn infant: effects of varying degrees of hypoxia on heart rate and ventilation in a warm environment. J Physiol (Lond) 1966;184:631-45.
- 6 Alvaro R, Alvarez J, Kwiatkowski K, Cates D, Rigatto H. Small preterm infants (less than or equal to 1500g) have only a sustained decrease in ventilation in response to hyoxia. *Pediatr Res* 1992;32:431-5.
- ventilation in response to hypoxia. *Pediatr Res* 1992;32:431-5.

  7 Bruhn FW, Mokrohisky ST, McIntosh K. Apnea associated with
- respiratory syncytial virus infection in young infants. J  $\it Pediatr$  1977;3:382-6.
- Sui GJ, Liu YH, Cheng XS, Anand IS, Harris E, Harris P, et al. Subacute infantile mountain sickness. *J Pathol* 1988;155:161-70.
   Aerospace Medical Association, Air Transport Medicine Committee.
- 9 Aerospace Medical Association, Air Transport Medicine Committee Medical guidelines for air travel. Aviat Space Environ Med 1996;67: R1.16

#### Children in the mountains

High mountain trekking holidays are best avoided for the very young.

igh altitude areas of the world were once visited only by a privileged minority. Now the great mountain ranges have become popular tourist destinations. An increasing number of children accompany their parents on such journeys, but little consideration has been paid to the potential risks of exposing young children to high altitudes. We are aware of ascents in Nepal where infants have been carried to over 6000 m on mountains requiring technical snow mountaineering expertise.

About half the adult tourists on the popular trekking routes in Nepal develop acute mountain sickness, 1-3 a disorder characterised by headache, nausea, vomiting, anorexia, fatigue, dizziness, and sleep disturbance that is particularly common above 2500 m, especially when ascent is rapid.4 Although acute mountain sickness is generally benign, it may progress to life threatening high altitude cerebral or pulmonary oedema. Little information exists about altitude illness in children. Wu studied 464 children travelling across the Tibetan plateau at 4550 m and found incidences of acute mountain sickness and high altitude pulmonary oedema of 34% and 1.5% respectively on the basis of symptoms, physical examination, chest radiography, and relief of symptoms after oxygen therapy.<sup>5</sup> These were almost identical to the corresponding incidences for 5355 adults who were also studied.

Similarly, Theis et al surveyed 558 children aged 9-14 years at 2835 m in Colorado and found that 28% reported symptoms of acute mountain sickness.<sup>6</sup> However, it is unclear whether these symptoms were related to altitude or to travel itself, as 21% of a control group at sea level also reported similar symptoms. Yaron et al recently attempted to examine the incidence of acute mountain sickness in children aged under 37 using a behavioural score based on the standard adult mountain sickness symptom questionnaire.8 In 14 children aged 3-36 months ascending from 1609 m to 3488 m there was an incidence of mountain sickness of 22% (compared with 20% in 45 adults on the conventional scoring system). Pulmonary oedema seems to develop more often in children who have had recent upper respiratory tract infections.9

While this limited information suggests that children are no more likely to develop acute mountain sickness than adults, it is harder to recognise the condition in children. Headache, nausea, fatigue, anorexia, dizziness, and sleep disturbance are rarely reported by children under 5 years, in whom non-specific symptoms such as lethargy, food refusal, irritability, and excessive crying may be the only indication of the condition. Unfortunately, these symptoms can be attrib-

uted to changes in routine or diet associated with remote travel, or to intercurrent illness.

Since acute mountain sickness and the onset of high altitude pulmonary or cerebral oedema can be easily overlooked in young children, the diagnosis should always be assumed when a child becomes unwell above 2500 m and descent should start immediately. Rapid descent will usually relieve the symptoms of acute mountain sickness, may be lifesaving when high altitude pulmonary and cerebral oedema are present, and is the only definitive treatment for all forms of altitude illness. There is no place for a "wait and see" approach when children have acute mountain sickness.

Pharmacological treatment for altitude illness has not been studied in children, but in life threatening situations paediatric doses of drugs proved effective in adults should be used.<sup>4</sup> When cerebral oedema or severe acute mountain sickness is suspected oxygen and dexamethasone (0.15 mg/kg/dose 4 hourly) should be given in combination with immediate descent. Pulmonary oedema also responds well to descent and oxygen therapy and, although systemic hypotension could be a problem, nifedipine (0.5 mg/kg/dose 8 hourly, maximum 20 mg for capsules and 40 mg for tablets) will lower pulmonary arterial pressure and relieve symptoms.

Prolonged exposure to high altitude should be avoided in infants aged under 1 year because of the risk of subacute infantile mountain sickness. <sup>10</sup> This condition is characterised by pulmonary hypertension and consequent fatal right heart failure and occurs in up to 1% of infants of lowland parents who are born at 3000-5000 m or arrive there shortly after birth. It was first described in Tibet, where it almost exclusively affects infants of Han Chinese origin who have recently migrated from low altitude areas.

Travelling with children can be a valuable experience, but these valuable insights must always be balanced against the risks of serious illness and death from exposure to environmental hazards such as hypoxia and cold. In most cases there is little justification in taking young children to high altitude. Most positive outdoor experiences can be gained at modest elevations, where there is plenty of oxygen and more warmth. Slow ascent is recommended, regardless of the altitude. Additional caution is required when the child has recently had a respiratory tract infection, as this increases the risk of pulmonary oedema.<sup>9</sup>

Until further information suggests otherwise, when trekking in a remote setting a conservative approach would be to sleep no higher than 2000 m for children aged under 2 and no higher than 3000 m for children

Editorial p 873

 $BMJ\ 1998;\!316:\!874\!-\!5$ 

aged 2-10 years. High treks are no place for little children.

Andrew J Pollard Action Research fellow in paediatrics Department of Paediatrics Infectious Diseases, St Mary's Hospital, London W2 1NY

David R Murdoch Senior registrar in infectious diseases Christchurch Hospital, Christchurch, New Zealand

Peter Bärtsch *Professor of sports medicine* University Clinic of Medicine, Heidelberg, Germany

- 3 Murdoch DR. Symptoms of infection and altitude illness among hikers in the Mount Everest region of Nepal. Aviat Space Environ Med 1995;66:148-51
- 4 Pollard AJ, Murdoch DR. The high altitude medicine handbook. Oxford: Radcliffe Medical, 1997.
- 5 Wu T. Children on the Tibetan plateau. Newsletter of the International Society for Mountain Medicine 1994;4:5-7.
- 6 Theis MK, Honigman B, Yip R, McBride D, Houston CS, Moore LG. Acute mountain sickness in children at 2835 meters. Am J Dis Child 1993:147:143-5.
- 7 Yaron M, Waldman N, Niermeyer S, Nicholas R, Honigman B. The diagnosis of acute mountain sickness in pre-verbal children. In: Houston CS, Coates G, eds. *Hypoxia: women at altitude*. Burlington, VT: Queen City Printers, 1997:107-8.
- 8 Roach RC, Bärtsch P, Hackett PH, Oelz O, the Lake Louise AMS scoring consensus committee. The Lake Louise acute mountain sickness scoring system. In: Sutton JR, Houston CS, Coates G, eds. Hypoxia and molecular medicine. Burlington, VT: Queen City Printers, 1993:272-4.
- 9 Durmowicz AĞ, Noordeweir E, Nicholas R, Reeves JT. Inflammatory processes may predispose children to high altitude pulmonary edema. J Pediatr 1997;130:838-40.
- 10 Sui GJ, Liu YH, Cheng XS, Anand IS, Harris E, Harris P, et al. Subacute infantile mountain sickness. *J Pathol* 1988;155:161-70.

## Why doesn't audit work?

Attempts are being made to revitalise audit

Then representatives from 10 British hospital trusts met last September to tackle the issue of why clinical audit has failed to bring about change, the NHS white paper and the term "clinical governance" had not been born. It is now clear, however, that the Action on Clinical Audit project, which brought these trusts together, was conceived in the same camp—and with the same aim, to improve clinical services.

Action on Clinical Audit is a two year project, funded by the NHS Executive, that is devised to unravel the complex relationships that seem to render audit unworkable.\* On paper, clinical audit takes the form of a neat cycle of events, leading to harmonious improvement in the activity under scrutiny.¹ In the gritty world of doctors, patients, and managers, the cycle can all too easily lose its shape, stop short, or simply vanish. The promised improvement never materialises.

The founders of Action on Clinical Audit began with the premise that we hold unreasonable expectations of audit, and that discrepancies exist between theory and reality. The project's aim is not to perform audit as an end in itself, but by encouraging each of the trusts to conduct an "action inquiry"—a systematic analysis of the stages of the audit process—to examine the processes which underpin audit. The action inquiry approach involves asking at every step why things are being done, rather than just doing them as normal without particularly questioning them. By asking questions, the participants themselves identify the barriers that are defeating their end product.<sup>2</sup>

Over the two years teams from 22 trusts will work together on the barriers that they identify and other topics related to clinical audit, learning from each other's experiences. For example, some teams have identified that a significant obstacle to achieving change in their own trusts is the lack of priority given to audit by the trust board. Indeed one team has stated that audit is not achieving change in clinical practice in their trust—despite the huge regard paid to

academic achievement—because the chief executive simply does not see audit as important: audit is something that clinicians should be doing, but he does not see its relevance to the trust as a whole. Some of the teams are therefore exploring ways of raising the profile of audit and getting it on to the boardroom agenda. To this end the NHS white paper has given them a leg up and boosted their credence with their chief executives. Clinical governance is no longer an added luxury: trust strategies must now be clinically driven and not simply financially accountable. Audit, the systematic examination and improvement of clinical activity, is an important component of clinical governance.

Another common theme identified by several trusts is that audit tends to be managerially commissioned in response to political pressure to "do something." The values of the clinicians and the issues they are interested in studying, however, may well differ from those of managers. Clinicians, for example, may want to audit their management of patients with acute myocardial infarction, while managers are interested in the bed occupancy of the coronary care unit. For these trusts the tensions thrown up by such ideological mismatch have made it virtually impossible to achieve any real change with audit.

While Action on Clinical Audit is claiming no predetermined outcomes, there is clearly at least one pay off for the trusts taking part. When clinicians and managers are asked why something is being done they often come up with different answers. Patients should certainly benefit if the clinicians, managers, and audit staff learn to appreciate different vantage points and motivations and to understand each other's language. The *BMJ* will be following the project and reporting on the progress of individual trusts.

Action on Clinical Audit was born of a desire to turn audit into a more useful process than it often is now. For its participants it has already crushed two assumptions: firstly, that good audit always leads to a better quality of patient care, and, secondly, that

BMJ 1998;316:875-6

Hackett PH, Rennie D, Levine HD. The incidence, importance, and prophylaxis of acute mountain sickness. *Lancet* 1976;ii:1149-54.

<sup>2</sup> Kayser B. Acute mountain sickness in western tourists around the Thorong pass (5400 m) in Nepal. J Wilderness Med 1991;2:110-7.

clinicians and managers work well together. It remains to be seen if the action inquiry approach that has produced the insights so far can revitalise the whole activity of audit—not only for its participants but in the wider NHS.

Abi Berger Associate editor, BMJ

\*Action on Clinical Audit was commissioned by the NHS Executive and is being run by the Royal College of Physicians, Anglia Polytechnic University, the University of Manchester Health Services Management Unit, and the Centre for Social and Organisational Learning and Reanimation, Nene University College, Northampton.

- 1 Dixon N. Good practice in clinical audit. A summary of selected literature to support criteria for clinical audit. London: National Centre for Clinical Audit. 1996.
- 2 Berwick DM. A primer on leading the improvement of systems. BMJ 1996;312:619-22.

## Collapse reactions after whole cell pertussis vaccination

Pertussis remains a bigger risk than collapse after vaccination

"To child should be denied immunisation without serious thought as to the consequences both for the individual and the community." This powerful statement prefaces the chapter on contraindications in the new edition of the United Kingdom's *Handbook on Immunisation against Infectious Disease.* In the 1970s a barricade of contraindications to pertussis vaccine was erected in response to undue concerns about safety. As the evidence has been reassessed, this barricade has now been largely demolished.

In 1981, without scientific substantiation, pertussis vaccine was contraindicated for a wide range of conditions, including cerebral irritation in the neonatal period and a family history of epilepsy or other diseases of the central nervous system. Interpretation of these recommendations was left to the vaccinating doctor or health visitor, resulting in considerable variation in practice and a large fall in pertussis immunisation rates. In contrast, the latest guidelines now recommend pertussis vaccine for children with a personal history of epilepsy and for those who have had a convulsion after a previous dose. The guidelines also give a comprehensive list of conditions that are not contraindications to vaccination.

Collapse after pertussis vaccine, the so called hypotonic-hyporesponsive episode, remains a contraindication to further doses as so far there has been no information on the outcome when such children are revaccinated. The pathogenesis of this condition, which typically occurs after a first dose and has a benign outcome,3 is still unclear and estimates of its incidence differ widely. In a recent efficacy trial in Sweden, the risk after a first dose of a British whole cell diphtheria, tetanus, and pertussis vaccine was around 1 per 1000 doses,4 although a prospective study in England found only one possible case after 6000 first doses of a similar vaccine.5 Whatever the cause and incidence, without information on the risk of revaccination doctors have been understandably reluctant to give further doses of pertussis vaccine to children experiencing such an episode. The paper in this week's issue by Vermeer-de Bondt et al provides this (p 902).6

They describe 101 children who experienced a hypotonic-hyporesponsive episode, of whom 84 subsequently received further doses of pertussis vaccine: none experienced a recurrence or other adverse event. In contrast, one of the 17 children who remained

unvaccinated had severe pertussis. In an earlier study in America, one of 14 children denied further vaccine because of a hyptonic-hyporesponsive episode or convulsion after a previous dose later developed pertussis, which lasted for three months and was transmitted to both her parents. Interestingly, one case of encephalitis (190 days after vaccination and therefore unrelated) occurred among the 82 892 children who received pertussis vaccine in the Swedish trial; three cases occurred among the 17 607 children who did not participate—all three resulted from a confirmed pertussis infection. Such observations highlight the real risks and benefits of vaccination.

How can the findings of the Dutch workers be translated into policy and practice? Their paper should be considered by the UK Joint Committee on Vaccination and Immunisation and similar advisory bodies in other countries and its message translated into a clear policy statement. Those giving the vaccine need to be reassured that their actions accord with national policy based on expert independent review of evidence, particularly when the manufacturers' data sheets may list a variety of contraindications drawn up with a view to protecting the vaccine rather than the child.2 With the current media interest in Britain on the alleged risks of measles, mumps, and rubella vaccine, the Dutch paper is a timely reminder that a sound, evidence based assessment of the risks of withholding, as well as giving, a vaccine is the basis on which national policy should be made.

#### Elizabeth Miller Head

Immunisation Division, Public Health Laboratory Service Communicable Disease Surveillance Centre, London NW9 5EQ Papers p 902

BMJ 1998;316:876

Department of Health, Welsh Office, Scottish Office Department of Health, Department of Health and Social Services (Northern Ireland). Immunisation against infectious disease. London: HMSO, 1996.

Hull D. Interpretation of the contraindications to whooping cough vaccination. BMJ 1981;283:1231-3.

<sup>3</sup> Baraff LJ, Shields WD, Beckwith L, Strome G, Marcy SM, Cherry JD, et al. Infants and children with convulsions and hypotonic-hyporesponsive episodes following diphtheria-tetanus-pertussis immunisation: follow-up evaluation. *Pediatrics* 1988;81:789-94.

<sup>4</sup> Olin P, Gustafsson L, Rasmussen F, Hallander H, Heijbel H, Gottfarb P. Efficacy trial of acellular pertussis vaccines. Technical report trial II with preplamed analysis of efficacy, immunogenicity and safety. Stockholm: Swedish Institute for Infectious Disease Control, 1997:1-65.

<sup>5</sup> Pollock TM, Miller E, Mortimer JY, Smith G. Symptoms after primary immunisation with DTP and DT vaccine. *Lancet* 1984;ii:146-9.

<sup>6</sup> Vermeer-de Bondt PE, Labadie J, Rümke HC. Rate of recurrent collapse after vaccination with whole cell pertussis vaccine: follow up study. BMJ 1998;316:902-3.

## Simian virus 40 and human malignancy

Contamination of early polio vaccine may be linked to rare tumours

The introduction of the Salk parenteral vaccine in the mid-1950s led to a dramatic decline in the incidence of poliomyelitis. By 1961, the majority of young adults in Britain and America had been immunised and the numbers of reported cases of poliomyelitis had fallen from 8000 a year to 100 a year.1 At that point in the mass immunisation programme, a contaminating virus was identified in the rhesus monkey kidney cells that were used to culture the poliovirus. It was named simian virus 40 (SV40). It was more resistant than poliovirus to chemical denaturation and survived into some vaccine samples. There are no reliable data about the proportion of batches that were contaminated with live SV40, and estimates range up to 30%.2 Early worries that the contaminant might be implicated in the development of human cancers have recently resurfaced.

SV40 was characterised as a double stranded DNA virus belonging to the group of papovaviruses. They share with adenoviruses (another DNA virus) a potent ability to induce tumours in species that are not their natural hosts. SV40 itself was found to be highly oncogenic in hamsters shortly after it was identified, and epidemiological surveillance of immunised cohorts was begun.<sup>3</sup> Except for one study, which reported an increased incidence of neural tumours in children of mothers vaccinated during pregnancy, all studies were essentially negative.<sup>4</sup> Occasional cases were reported of SV40 infection in association with tumours, but until recently the view was that SV40 has no role in the pathogenesis of human malignancy.

SV40 has now re-emerged as a potentially oncogenic virus. In 1992 Bergsagel et al used polymerase chain reaction techniques to search for DNA from human polyomaviruses, which are usually asymptomatic, in childhood ependymomas and choroid plexus tumours. They identified DNA which more closely matched that of SV40.<sup>5</sup> Since then SV40-like DNA has been identified in other human tumours, particularly osteosarcomas and malignant mesotheliomas though not in adenocarcinomas.<sup>6 7</sup> These findings mirror the range of tumours induced by SV40 in animals: injection of SV40 into hamsters results in lymphoid tumours and osteosarcomas, SV40 transgenic mice develop choroid plexus tumours, and intrapleural SV40 seems more potent than asbestos in inducing mesotheliomas.

DNA viruses such as SV40 carry only a limited amount of genetic information, and in order to reproduce they must subvert normal cellular DNA replication. This process is facilitated by viral proteins that inactivate products from cellular tumour suppressor genes. These products normally have inhibitory effects on DNA replication, and if their function is impaired this can contribute to the escape from replicative control that is an important step in the development of malignancy. When viruses enter cells which do not support their replication their DNA can become incorporated into the host genome, allowing inhibitors of tumour suppressor genes to be produced. The SV40-like DNA found in human tumours codes for the

large T antigen, which inactivates the products of tumour suppressor genes. The T antigen is structurally similar to the e7 and e8 antigens of the papillomaviruses, which are now recognised as important in the aetiology of cervical cancer.

The identification of virus-like DNA in tumours, the studies in animals, and the molecular actions of SV40 all suggest that it might have a role in some human malignancies. Epidemiological studies make it unlikely that the virus plays an important part in the aetiology of common cancers, but there are few other examples of known human oncogenic viruses and if the findings are confirmed they would be of considerable importance. For the present, however, we must remain cautious. The polymerase chain reaction techniques used to identify the viral DNA from fixed specimens are poorly standardised, and SV40 is a commonly used laboratory virus which might contaminate assay systems. No large scale studies have been undertaken, control tissue has often been inadequate, and the findings have not been replicated in all laboratories.10

Even if the identity of the DNA is confirmed as viral in origin, its source would remain unclear as SV40-like DNA has been identified in tumours from those who are far too young to have been immunised with contaminated vaccines. If this cannot be explained by artefact or misidentification then it implies either some other source of human SV40 infection or vertical transmission from immunised subjects. It thus remains possible that a late adverse effect of the polio vaccination programme is emerging, although any risk of cancer is likely to be more than outweighed by the benefit of vaccination to the postwar generation. Indeed, if it leads to an improved understanding of tumour biology it might even result in a treatment for tumours such as mesotheliomas, which to date have proved depressingly resistant to treatment.

#### S C Stenton Senior lecturer and consultant physician

Department of Respiratory Medicine and Regional Unit for Occupational Lung Disease, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP

BMJ 1998;316:877

Salk J, Salk D. Vaccination against poliomyelitis. In: Voller A, Freedman H, eds. New trends and developments in vaccines. Lancaster: MTP Press, 1978:138-9

<sup>2</sup> Shah K, Nathanson N. Human exposure to SV40: review and comment. Am I Epidemiol 1976:103:1-212.

<sup>3</sup> Eddy BE, Borman GS, Grubbs GE, Young RD. Identification of the oncogenic substance in rhesus monkey kidney cell cultures as SV40. Virology 1962;17:65-75.

<sup>4</sup> Heinonen OP, Shapiro S, Monson R, Hartz C, Rosenberg L, Slone D. Immunization during pregnancy against poliomyelitis and influenza in relation to childhood malignancy. Int J Epidemiol 1973:2:229-35.

<sup>5</sup> Bergsagel DJ, Finegold FJ, Butel JS, Kupsky WJ, Garcea RL. DNA sequences similar to those of simian virus 40 in ependynomas and chestical plant tumours of childhood. N End LM J 1009;396:058, 33

choroid plexus tumours of childhood. N Engl J Med 1992;326:988-93.
 Carbone M, Rizzo P, Procopio A. SV40-like sequences in human bone tumours. Oncogene 1996;13:527-35.

<sup>7</sup> Carbone M, Pass H, Rizzo, Marinetti MR, DiMuzio MD, Mews DJY, et al. Simian virus 40-like DNA sequences in human pleural mesothelioma. *Oncogene* 1994;9:1781-90.

<sup>8</sup> Carbone M, Rizzo P, Grimley PM, Procopio A, Mew DJY, Shridhar V, et al. Simian virus-40 large T antigen binds p53 in human mesotheliomas. Nature Med 1977;3:908-12.

<sup>9</sup> Tannock IF, Hill RP. The basic science of oncology. New York: McGraw-Hill, 1996.

<sup>10</sup> Strickler HD, Goeden JJ, Fleming M. Simian virus 40 and pleural mesothelioma in humans. Cancer Epidemiol Biomarkers Prev 1996;5:473-5.

## Training in advanced trauma life support

Senior house officers should be trained before working in accident and emergency

dvanced trauma life support offers clear protocols for managing major trauma and is now regarded as a common international language of trauma care. 1-3 In 1993 Teanby et al highlighted the generally poor care given to trauma victims in Britain and recommended that protocols for advanced trauma life support should be instituted in prehospital care as well as accident and emergency departments.4 Most, if not all, major British accident departments now use the protocols in their resuscitation rooms. Similarly, the number of advanced trauma life support courses have expanded since the first one in Britain in 1988 (Royal College of Surgeons, personal communication). But is this training being provided to those who need it most?

Despite the maturation of accident and emergency medicine as a specialty and the formation of trauma teams in many hospitals, a senior house officer is often still the first doctor to assess and provide initial care for multiply injured patients.<sup>5</sup> As front line providers of trauma care, they should have received training in advanced trauma life support.6 Yet a survey we conducted in January 1996 of all accident and emergency departments in England seeing more than 30 000 patients a year (95% response rate) showed only 16.7% of senior house officers had been trained in advanced trauma life support and a further 26.8% were on a course waiting list. Only 38% of all respondent hospitals had a dedicated trauma team.

These nationwide data confirmed a trend previously seen in the South West region.<sup>7</sup> As well as highlighting the low proportion of senior house officers with advanced trauma life support certification, they clearly show that most have not even applied for a course. This probably reflects difficulty in gaining access to training. Firstly, waiting lists for advanced trauma life support courses are long8 and many senior house officers will have finished their accident and emergency attachments before being trained. Secondly, doctors are expected to use study leave time and money to attain this basic training requirement. The advanced trauma life support course therefore competes with other courses that demand the trainee's attention. Our experience is that many senior house officers defer training in trauma life support until they have completed their accident and emergency attachment. Thirdly, places on courses are still preferentially offered to middle and higher level trainees.9 Initially this was necessary to create a core of senior doctors who were familiar with the principles and to increase the number of instructors. In 1996, however, the records of the Royal College of Surgeons of England show that almost half the doctors gaining certification in advanced trauma life support were more senior than senior house officers, despite their smaller numbers, and among the senior house officers only a third were actually working in accident and emergency departments (personal communication). Whatever the reasons, the result is that many doctors in accident and emergency medicine work without being trained in advanced trauma life support.

Certification in advanced trauma life support should be a prerequisite for appointment to a post as a senior house officer in accident and emergency or be incorporated into accident department induction courses, as occurs in some units. As a substitute for a full course, the teaching of advanced trauma life support principles is a useful stopgap but has been shown to be inferior.10 Even better if doctors were exposed to the principles at an earlier stage as medical students or very junior doctors, as was originally envisaged. In America, the birthplace of advanced trauma life support, the course is increasingly taught in the undergraduate curriculum.11 It can be argued that this is too early to teach such skills and that the preregistration year is probably a better time.

We believe that the principles of advanced trauma life support, along with those of advanced life support and advanced paediatric life support, should be introduced to undergraduates, perhaps as a generic resuscitation course. This should be followed by a formal course in the preregistration year and refresher courses at the start of senior house officer posts, similar to refresher cardiac life support updates. The multispecialty culture of advanced trauma life support will thus be introduced to all doctors at an early stage. Undergraduate deans and the General Medical Council will then have a responsibility to ensure that the training is completed. This may not solve the financial issues but may help unblock some of the staffing and resource difficulties of study leave. Individual patient care in the "golden hour" will improve and the numbers of avoidable trauma deaths will continue to fall.12

#### Andrew Price Specialist registrar

Department of Orthopaedic Surgery, Wexham Park Hospital, Slough SL2 4HL

#### Geoff Hughes Clinical director

Emergency and Trauma Services, Wellington Hospital, Wellington, New Zealand

BMI 1998:316:878

American College of Surgeons. Advanced trauma life support manual. Chicago: American College of Surgeons, 1994.
Collicott MD. Advanced trauma life support (ATLS): past, present, future.

J Trauma 1992;33:749-53.

Anderson ID, Anderson IWR, Clifford P, Law LH, Ryan J, Stoneham J. Advanced trauma life support in the UK: 8 years on. *Br J Hosp Med* 

Teanby DN, Gorman DF, Boot DA. Regional audit of pedestrian accident care. Injury 1993;2:435-7

Yates DW, Woodford M, Hollis S. Preliminary analysis of the care of the injured patients in 33 British hospitals; first report of the United Kingdom major trauma outcome study. BMJ 1992;305:737-40.

Skinner DV. Advanced trauma life support. Injury 1993;24:147-8.

Price AJ, Hughes G. Accident and emergency doctors lack proper training in trauma. *BMJ* 1995;311:1644.

Graham CA, Sinclair MT. A survey of advanced trauma life support train-

ing for trainees in acute surgical specialties. *Injury* 1996;27:631-4. Gwinnutt CL, Driscoll PA. Advanced trauma life support. *Eur J Anaesth* 

<sup>10</sup> Gautam V, Heyworth J. A method to measure the value of formal training in trauma management: comparison between ATLS and induction courses. *Injury* 1995;26:253-5.

<sup>11</sup> Ali J, Cohen R, Reznick R. Demonstration of acquisition of trauma management skills by senior medical students completing the ATLS program. Trauma 1995;38:687-91.

<sup>12</sup> Royal College of Surgeons. The management of patients with major injuries. Report of the Working Party of the Royal College of Surgeons. London: Royal College of Surgeons, 1988.

## Tackling violence

#### Interagency procedures and injury surveillance are urgently needed

Tiolence is now on the public health agenda in Britain largely because of increases in injury and homicide rates, particularly among men, and an increase in reports of domestic violence. In the absence of a national system of violence surveillance in accident and emergency departments, surveys such as the British Crime Survey are a starting point for understanding the circumstances and extent of assault, and one lesson that emerges is that health professionals need to do more to help prevent violence.

In 1995 the British Crime Survey data showed that the most common type of violence was that by acquaintances (40% of violent offences), followed by domestic violence (25%), violence by strangers (25%) and during robberies (10%) and that men were most at risk (6.7% of men; 3.8% of women). Importantly in a medical context, repeat victimisation is more common for violence than for other crimes: 30% of victims in the 1995 survey (and 43% in a 1989 accident and emergency department survey²) had been assaulted before. Fear of reprisals and a continuing relationship with the assailant prevents men as well as women from reporting offences.³ The incidence of all categories of violence has increased since 1981, particularly domestic violence (240%) and that by acquaintances (120%).

Overall, England and Wales has risen in the crime prevalence league table from 6th out of 15 in 1989 to 2nd out of 11 in 1995. Victimisation rates for violence are now highest in England and Wales (3.6% adults in 1995) and the United States (3.5%) but lower in Scotland (2.7%) and much lower in Northern Ireland (1.5%). These international comparisons accentuate the effectiveness of firearm control in Britain: 30 000 Americans have been killed with guns every year in the past 30 years.<sup>5</sup>

Since its inception in 1985 the British Crime Survey has consistently shown substantial amounts of crime which are not recorded by the police. In 1995, for example, only 30% of domestic violence, 60% of robberies, 40% of violence by strangers, and 37% of violence by acquaintances was reported. Nevertheless, crime surveys also underestimate domestic violence and male violence in bars. Despite more proactive community policing, it is now widely acknowledged that the police cannot fight crime alone. Following research in accident and emergency departments on unrecorded violence the Home Office has recently identified health authorities as potential partners.

How can doctors contribute to this new interagency approach? Putting violence on the public health agenda has undoubtedly begun to pay dividends in America: homicide rates have fallen, legislation such as the Brady bill has made it harder to buy firearms, laws have been enacted to protect children in the home by requiring that firearms are kept in locked cupboards. In Britain a medical perspective on violence has shown that many more of those injured in violence have criminal records than those injured in accidents. Clearly, delinquent behaviour increases the risk of injury by assault, and primary prevention should focus on preschool education

and early family support. A key starting point, however, is the establishment of violence surveillance in accident and emergency departments, both to provide national data on morbidity and to inform local community violence prevention.

Recent research has highlighted the need for the injured to have increased access both to the police and victim support services immediately after injury and to screening for alcohol and substance misuse and the acute stress reactions which predict serious psychological sequelae. Collaboration between licensing authorities and accident and emergency departments would help to make drinks licensing sensitive to local injury rates as well as to unreliable crime rates. It is too early to assess the effectiveness of these initiatives but the principles which have emerged include importance of dealing with the many offenders not currently being investigated, giving all those injured by violence an opportunity to report offences, and integrating accident and emergency departments into community policing without dissuading those who may be on the edge of the law from seeking treatment.

Although all assaults causing injury are criminal offences, there is more to providing a comprehensive service to the injured than finding out if the police are involved. Other agencies also have an important role, including victim support schemes, community alcohol teams, and women's refuges. Ethical issues relating to confidentiality and data protection are important: clear interagency procedures agreed by doctors, the police, mental health and support services, and local authorities are as necessary in dealing with crime directed against adults as they are for child protection. Drawing on existing guidance and educational material produced by many groups, they would help provide not just better services for patients but also increased security and staff protection from one of the scourges of our time.

Jonathan P Shepherd Professor of oral and maxillofacial surgery

Department of Oral Surgery, Medicine, and Pathology, University of Wales College of Medicine, Cardiff CF4 4XY

BMJ 1998;316:879

Mirrlees-Black C, Mayhew P, Percy A. The 1996 British Crime Survey. Home Office Statistical Bulletin 1996;19:27-36.

<sup>2</sup> Shepherd JP. Violent crime: an accident and emergency perspective. Br J Criminol 1990;30:280-305.

<sup>3</sup> Clarkson C, Cretney A, Davis G, Shepherd JP. Assault: the relationship between seriousness, criminalisation and punishment. *Criminal Law Review* 1994 Jan 4: 4-20.

<sup>4</sup> Mayhew P, White P. The 1996 international crime victimisation survey. London: Home Office, 1996.

<sup>5</sup> Baker SP, O'Neill B, Ginsburg MJ, Li G. The injury fact book. 2nd ed. New York: Oxford University Press, 1992.

<sup>6</sup> Home Office. Getting to grips with crime: a new framework for local action.
London: Home Office, 1997.
7 Champings P. Creenten, D.C. Pivers, EP. Konsoell, TD. State, gap, sefer.

<sup>7</sup> Cummings P, Grossman DC, Rivara FP, Koepsell TD. State gun safe storage laws and childhood mortality due to firearms. JAMA 1997;278:1084-6.

Rivara FP, Shepherd JP, Farrington DP, Richmond PW, Cannon P. Victim as offender in youth violence. *Ann Emer Med* 1995;26:609-14.
 Yoshikawa H. Prevention as cumulative protection: effects of early family

<sup>9</sup> Yoshikawa H. Prevention as cumulative protection: effects of early family support and education on chronic delinquency and its risks. *Psychol Bull* 1994;115:28-54.

 $<sup>10\,</sup>$  Shepherd JP, Farrington DP. Assault as a public health problem. J R Soc Med 1993;86:89-92.

<sup>11</sup> Victim Support. Treating victims of crime: guidelines for health professionals. London: Victim Support, 1996: 15.

## Cancer in parents: telling children

Sensitive communication can reduce psychological problems

hildren of cancer patients "represent a hidden, high risk group whose problems are minimised by overwhelmed parents and unknown to the medical staff who seldom see them." A recent editorial in the BMJ highlighted the difficulties doctors have in communicating the news of a cancer diagnosis to their patients.<sup>2</sup> How much harder must it be for parents with newly diagnosed cancer to tell their children while coming to terms with the implications themselves? Cancer in a parent is an issue that confronts many families. For example, breast cancer affects one in 12 women in Britain,3 30% of whom are likely to be diagnosed while they have children still living at home.4

Good doctor-patient communication about the diagnosis and shared decision making over treatment are crucial and have a protective effect on patients' psychological adjustment,5 and useful guidelines on how to impart the diagnosis are available.<sup>6</sup> Little attention has been paid to what, or how, children should be told about the diagnosis, however; the responsibility seems to be left largely to the unaided parents. Information and guidelines for doctors are urgently needed so that they can help families with this difficult task.

The few studies on this issue have tended to be small and retrospective, but some themes are emerging. The balance of evidence suggests that children of parents with cancer are at increased risk of developing psychological disturbance.<sup>7-9</sup> It is becoming clear that children's levels of anxiety are related to whether they are told about the illness and to the quality of the communication with their parents.8-10 In one study of parents with advanced cancer, the anxiety levels of children who were told the diagnosis by their parents were lower than those of children who were not told.8 In some families the parents had discussed the illness with older children but not with younger ones, providing an opportunity to examine whether anxiety was associated with a child's knowledge or with particular family characteristics. Analyses revealed that anxiety was specifically related to a child's knowledge, or lack of it, and not to the particular family. These findings were supported in a second study, which found that high anxiety scores among adolescent children of parents with cancer were linked to an inability to discuss the illness with the parents.9 In a third small study of adolescent girls whose mothers had undergone a mastectomy, most felt that they were not given enough explanation about the disease and complained of being ignored.10 Importantly, all three studies assessed children directly and did not rely on parental reports.

The importance of communication between parents and children was confirmed by a large, well conducted study from America, which found that parents underestimate the impact of the illness on their children.<sup>7</sup> This study found a marked discrepancy between children's and parents' reports of children's emotional and behavioural adjustment in the months after cancer was diagnosed. Parents reported little or no evidence of emotional distress or disruptive behav-

iour in their children, while the children themselves reported greatly increased psychological symptoms. A further study of children whose mothers had breast cancer found that the quality of their peer relationships was related to the amount of communication with the well parent.11 These studies all underline the importance of communication and suggest that doctors need to discuss with parents at an early stage how and what their children should be told. Parents may need considerable support when dealing with their children's feelings and reactions.

Most difficult is how to tell a child that a parent might die. Little specific work exists to guide clinicians, but Kane has elucidated the developmental stages that children go through in their understanding of the meaning and permanence of death.12 Knowledge of these stages is helpful in tailoring information and language to children's developmental age and experience of death.12

Research is needed to identify what information should be given to children at what stage and in what manner while taking account of parents' views and feelings. Some useful booklets are available to guide clinicians and parents when talking to children about a parent's diagnosis of cancer,13 but properly developed and evaluated guidelines and training packages are urgently required.

Leanda Kroll Senior registrar in child psychiatry Tavistock Centre, London NW3 5BA

Jacqueline Barnes Senior lecturer in psychology Alison L Jones Consultant medical oncologist Royal Free Hampstead NHS Trust, London NW3 2QG

Alan Stein Professor

Leopold Muller University Department of Child and Family Mental Health, Royal Free Hospital School of Medicine, London NW3 2PF

BMI 1998:316:880

<sup>1</sup> Rait D, Lederberg M. The family of the cancer patient. In: Holland JC, Rowland JH, eds. Handbook of psychooncology. Oxford: Oxford University Press, 1990:589.

Buckman R. Talking to patients about cancer. No excuse now for not doing it. *BMJ* 1996;313:699-700.

The Cancer Research Campaign Fact Sheet 6. London: Cancer Research Campaign, 1996.

Bloom JR, Kessler L. Risk and timing of counselling and support interventions for younger women with breast cancer. J Natl Cancer Inst Monogr 1994;16:199-206.

Fallowfield LJ, Hall A, Maguire P, Baum M, A'Hern RP. Psychological effects of being offered choice of surgery for breast cancer. BMJ 1994:309:448.

Maguire P. ABC of breast diseases: psychological aspects. BMJ 1994;309:1649-52.

Welch AS, Wadsworth ME, Compas BE. Adjustment of children and adolescents to parental cancer. Cancer 1996;77:1409-18.

Rosenheim E, Reicher R. Informing children about a parent's terminal

illness. J Child Psychol Psychiatry 1985;26:995-8.

Nelson E, Sloper P, Charlton A, While D. Children who have a parent with cancer. A pilot study. J Cancer Educ 1994;9:30-6.

<sup>10</sup> Rosenfeld A, Caplan G, Yaroslavsky A, Jacobowitz J, Yuval Y, LeBow H. Adaptation of children of parents suffering from cancer: a preliminary study of a new field for primary prevention research. J Primary Prev

<sup>11</sup> Lewis FM, Hammond MA, Woods NF. The family's functioning with newly diagnosed breast cancer in the mother: the development of an explanatory model. J Behav Med 1993;16:351-70.

<sup>12</sup> Kane B. Children's concepts of death. J Genet Psychol 1979;134:141-53.

<sup>13</sup> Couldrick A. When your mum or dad has cancer. Oxford: Sobell Publications, 1991.