tive to treating crying is to mobilise services to help parents to cope. The need for such support is highlighted both by the lack of reliable treatments and by evidence that referred infants do in fact cry for long periods (I St James-Roberts et al, unpublished observations) and continue to have behavioural problems at 3 years of age.14

Considering the impact of crying on families and the cost of referral to the health services, the lack of standard tested methods of surveillance and support is striking. Potential goals for such services include providing parents with information about the normal range of crying and its development, counselling them about their feelings of frustration and inadequacy, and helping to prevent their relationship with their infants from deteriorating. Diaries and questionnaires for measuring crying are available together with normative information and may provide reassurance and a framework for planning.<sup>1 15 16</sup> Studies establishing the most cost effective means of delivering such services are an obvious priority.

If viable treatments are to be achieved improvements in measurement and the design of research are needed. In particular, parental measure of "crying problems," though important, need to be distinguished from measures of crying, since the two only loosely correspond.1+16 Standardised parental diaries are sufficiently accurate measures of crying for most purposes.<sup>5 10 15</sup> Where parents cannot keep such diaries or the intensity of crying needs distinguishing from its inconsolability audio or video recording is appropriate.<sup>16</sup> Day to day variabilities in crying are substantial, making it difficult to decide whether a genuine improvement has occurred. Barr has calculated that six days of diary data provide an optimum measure.17

Guidelines have been drawn up for the design and control of studies of dietary intervention<sup>57</sup> but not for studies of parental behaviour. These should include measures of the

## Nocturnal asthma

What happens to the airways at night?

Waking at night struggling for breath, wheezing, and coughing can be a terrifying experience. To have doctors write off these scaring episodes as unimportant has annoyed generations of people suffering from asthma. But now we are realising that the patients were right after all.

Nocturnal asthma is both common and serious. Mortality from asthma rises at night. A survey of general practitioners has shown that nocturnal wheeze or cough wakes three quarters of asthmatic patients occasionally; two fifths wake every night.1 Nocturnal wheezing often impairs daytime cognitive performance.<sup>2</sup> Unless we specifically ask every asthmatic patient about nocturnal symptoms we will miss this important cause of impaired quality of life.

Why should nocturnal symptoms be so common? Like most biological variables normal lung function has a circadian rhythm, with peaks at 1600 and troughs at about 0400. In people with asthma the fluctuations are much more pronounced: peak expiratory flow may vary by about 50%, compared with 8% in unaffected people.3

Several factors trigger bronchoconstriction at night.45 Sleep seems to synchronise the circadian rhythm: in shift workers peak expiratory flow varies with the time of sleeping rather than time of day. Techniques that enable continuous monitoring of airway resistance during sleep show that sleep increases airway resistance, partly by reducing the hyper-

target parental behaviour before and after treatment and control groups so that relations between parental behaviour and crying can be established. Lastly, as the pattern of crying varies with age (describing an inverted U) and is increasingly subject to learning as infants develop,<sup>18</sup> the age of the infants being studied needs to be taken into account.

## IAN ST JAMES-ROBERTS

Senior Lecturer in Child Development, University of London, London WC1H 0AL

- St James Roberts I, Halil T. Infant crying patterns in the first year: normative and clinical findings. *J Child Psychol Psychiatry* 1991;32:951-68.
   Illingworth RS.Infantile colic revisited. Arch Dis Child 1985;60:981-5.
- Hwang CP, Danielsson B. Dicyclomine hydrochloride in infantile colic. BMJ 1985;291:1014.
- Danielsson B, Hwang CP. Treatment of infantile colic with surface active substance (simethicone). Acta Paediatr Scand 1985;74:446-50.
   Forsyth BWC. Colic and the effect of changing formulas: a double-blind, multiple-crossover study. J Pediatr 1989;115:521-6.
- 6 Lothe L, Lindberg T. Cow's milk whey protein elicits symptons of infantile colic in colicky formula-fed infants: a double blind crossover study. *Pediatrics* 1989;83:262-6.
- 7 Sampson HA. Infantile colic and food allergy: fact or fiction? 7 Pediatr 1989:115:538-4
- 8 Hubbard FOA, Van Ijzendoorn MH. Maternal unresponsiveness and infant crying: critical replication of the Bell and Ainsworth study. In: Tavecchio LWC, Van Ijzendoorn MH, eds. Attachment in social networks. North Holland: Elsevier, 1987:339-75
- 9 Hunziker UA, Barr RG. Increased carrying reduces infant crying: a randomised control trial. Pediatrics 1986;77:641-8. 10 Barr RG, McMullen SJ, Spiess H, Leduc DG, Uaremko J, Barfield R, et al. Carrying and colic
- 'therapy': a randomised controlled trial. *Pediatrics* 1991;87:623-30. 11 Taubman B. Clinical trial of the treatment of colic by modification of parent-child interaction.
- 7 Pediatr 1984;74:998-1003.
- 12 Taubman B. Parental counselling compared with elimination of cow's milk or soy milk protein for the treatment of infant colic syndrome: a randomised trial. *J Pediatr* 1988 81:756-61. 13 McKenzie S. Troublesome crying in infants: the effect of advice to reduce stimulation. *Arch Dis*
- Child 1991;66:1416-20.
- 14 Forsyth BWC, Canny P. Perceptions of vulnerability 31/2 years after problems of feeding and crying behaviour in early infants. J Pediatr 1991;88:757-63. 15 Barr RG, Kramer MS, Boisjoly C, McVey-White L, Pless IB. Parental diary of infant cry and fuss
- behaviour. Arch Dis Child 1988;63:380-7.
- 16 St James-Roberts I. Measuring infant crying and its social perception and impact. Association of Child Psychology and Psychiatry Newsletter (in press). 17 Barr RG. The normal crying curve: hoops and hurdles. In: Lester B, Newman J, Pederson F, eds.
- Biological and social aspects of infant crying. New York: Plenum Press (in press). 18 Gustafson GE, Green JA. Developmental coordination of cry sounds with visual regard and
- gestures. Infant Behavior and Development 1991;14:51-7.

inflation seen in asthmatic patients.6 This effect hardly varies according to the stage of sleep, but snoring may exacerbate the problem.<sup>7</sup> Nasal continuous positive airways pressure is effective in patients with obstructive sleep apnoea and asthma.8 Lying down may exacerbate wheezing slightly; an adverse effect from gastro-oesophageal reflux is less certain.9

More severe daytime disease is associated with more nocturnal bronchoconstriction. Airways become more twitchy at night, with higher reactivity to inhaled bronchoconstrictors and aerosol allergens. Although the late asthmatic response is enhanced at night,10 allergens in bedding are not the cause. Hypoxia increases bronchial reactivity slightly, and lower body temperature during sleep may cause bronchoconstriction. Although airway mucus is cleared more slowly at night, its importance in nocturnal asthma is uncertain.

All these factors exacerbate nocturnal wheezing slightly on their own; we are only just beginning to understand how their interactions may contribute substantially to the problem. (So far no factors improving nocturnal asthma have been identified.)

How do these factors trigger bronchoconstriction? The autonomic nervous system, hormones, and inflammation are possible candidates. The autonomic nerves responsible for bronchial smooth muscle tone are mainly under parasympathetic control<sup>11</sup>—atropine substantially reduces nocturnal bronchoconstriction.<sup>12</sup> Non-adrenergic, non-cholinergic nerves also directly innervate bronchial smooth muscle. Capsaicin, a non-adrenergic non-cholinergic stimulant, is less effective at night<sup>13</sup>; nocturnal inhibition of non-adrenergic, non-cholinergic nerves seems to contribute modestly to bronchoconstriction.

Corticosteroids, adrenaline, and histamine have substantial effects in asthma. Could their plasma concentrations, which exhibit circadian rhythm, be important in nocturnal asthma? Abolishing plasma cortisol's circadian rhythm does not affect the nocturnal fall in peak flow: changes in concentration may represent the body's defensive response to nocturnal asthma rather than be part of its cause.<sup>14</sup> Catecholamines seem of little relevance to nocturnal asthma. Although plasma catecholamine concentrations fall at night and the sensitivity of  $\beta$  adrenoceptors varies throughout the day,<sup>15</sup> infusing adrenaline does not improve nocturnal asthma,<sup>16</sup> and adrenalectomy has no effect. The plasma concentration of histamine is raised at night, but its role is unclear: antihistamines are ineffective.<sup>5</sup>

Asthma is being increasingly regarded as an inflammatory disease; could the inflammation of airways vary diurnally? Martin and colleagues suggest that it does.<sup>17</sup> Fluid obtained by bronchoalveolar lavage from patients with nocturnal asthma contained significantly more neutrophils, eosinophils, lymphocytes, and epithelial cells at 0400 than at 1600. This difference was not found in asthmatic patients without nocturnal asthma. Inflammatory cells could therefore flow into and out of the bronchial tree, producing cyclical epithelial damage. This might explain how increased bronchial reactivity, enhanced late asthmatic response, and reduced mucociliary clearance occur in nocturnal asthma.

Despite all its idiosyncracies nocturnal asthma may represent a cyclical flare up in the activity of asthma. As with asthma itself we are progressively discovering how nocturnal asthma happens. The challenge now is to understand why.

JOHN B MACDONALD

Consultant Physician, Crosshouse Hospital, Kilmarnock, Ayrshire KA2 0BE

- Turner-Warwick M. Epidemiology of nocturnal asthma. Am J Med 1988;85 (suppl 1B):6-8.
   Fitzpatrick MF, Engleman H, Whyte KF, Deary IJ, Shapiro CM, Douglas NJ. Morbidity in
- Htzpatrick MF, Engleman H, Whyte KF, Deary JJ, Snapiro CM, Douglas NJ. Morolatty in nocturnal asthma: sleep quality and daytime cognitive performance. *Thorax* 1991;46:569-73.
   Hetzel MR, Clark TJH. Comparison of normal and asthmatic circadian rhythms in peak expiratory flow rate. *Thorax* 1980;35:732-8.
- 4 Douglas NJ. Nocturnal asthma. QJ Med 1989;71:279-89.
- 5 Barnes PJ. Inflammatory mechanisms and nocturnal asthma. Am J Med 1988;85 (suppl 1B):64-70. 6 Ballard RD, Irvin CG, Martin RJ, Pak J, Pandey R, White DP. Influence of sleep on lung volume in artificial antimicial and normal publicity. J Acad Burger 1990;69:2024 41
- asthmatic patients and normal subjects. J Appl Physiol 1990;68:2034-41. 7 Douglas NJ, Flenley DC. State of the art: breathing during sleep in patients with obstructive lung disease. Am Rev Respir Dis 1990:141:1055-70.
- disease. Am Rev Respir Dis 1990;141:1055-70.
  8 Chan CS, Woolcock AJ, Sullivan CE. Nocturnal asthma: role of snoring and obstructive sleep apnea. Am Rev Respir Dis 1988;137:1502-4.
- 9 Pack AL. Acid: a nocturnal bronchoconstrictor? Am Rev Respir Dis 1990;141:1391-2.
- 10 Mohiuddin AA, Martin RJ. Circadian basis of the late asthmatic response. Am Rev Respir Dis 1990;142:1153-7.
- Barnes PJ. State of art: neural control of human airways in health and disease. Am Rev Respir Dis 1986;134:1289-1314.
   Morrison JF, Pearson SB, Dean HG. Parasympathetic nervous system in nocturnal asthma. BMJ
- 1983;296:1427-9. 13 Mackay TW, Fitzpatrick MF, Douglas NJ. Non-adrenergic, non-cholinergic nervous system and
- overnight airway calibre in asthmatic and normal subjects. Lancet 1991;338:1289-92.
   Haen E, Hauck R, Emslander HP, Langenmayer I, Liebl B, Schopohl J, et al. Nocturnal asthma.
- Adverse of the second se
- IS Szefler SJ, Ando R, Cicutto LC, Surs W, Hill MR, Martin KJ. Plasma histamine, epinephrine, cortisol, and leukocyte β-adrenergic receptors in nocturnal asthma. *Clin Pharmacol Ther* 1991;49:59-68.
- Morrison JFJ, Teale C, Pearson SB, Marshall P, Dwyer NM, Jones S, et al. Adrenaline and nocturnal asthma. BMJ 1990;301:473-6.
   Martin RJ, Cicutto LC, Smith HR, Ballard RD, Szefler SJ. Airways inflammation in nocturnal
- 7 Martin RJ, Cicutto LC, Smith HR, Ballard RD, Szefler SJ. Airways inflammation in nocturnal asthma. Am Rev Respir Dis 1991;143:351-7.

## **Duplicate publication**

## If in doubt ask the editor

Readers, researchers, and editors frown on the publication of the same scientific information in different journals. It clogs up the already congested scientific literature without adding new information and may keep someone else's work out of print. It complicates the process of retrieving information and wastes time and money. An average scientific paper published in the BMJ will have been read by eight people, taken up about eight hours of a technical editor's time, and cost around £1300 to process, print, and distribute.

On p 1029 Waldron shows that duplicate publication is common.<sup>1</sup> Doctors are now under such pressure to publish to impress appointments and grants committees—that the temptation to milk as many publications as possible out of a piece of research is strong. Often this leads to salami publication (submitting small slices of the work as separate papers), but sometimes authors try to get substantially the same paper published more than once. Authors may be ignorant of the rules, even though the main principle is enshrined in the instructions to authors of this and most other journals.

Editors have no desire to stifle scientific communication. Their aim is to ensure that information first appears publicly in a form that has undergone scrutiny so that as many ambiguities and inconsistencies as possible have been ironed out. Rather than keeping knowledge from the public this scrutiny reduces the risk of misleading information causing unnecessary alarm and ensures that the full story is available. This was why the  $BM\mathcal{J}$  refused to consider further a paper that had been issued to newspapers suggesting that toxic

fumes from mattresses might contribute to cot deaths.<sup>2</sup>

The  $BM\mathcal{J}$ , like many other journals, subscribes to the rules on duplicate publication set out by the Vancouver group of medical journal editors.<sup>3</sup> Essentially these say that articles cannot be considered for publication, either in print or in electronic form, if they are under consideration or have been published elsewhere.<sup>4</sup>

Some exceptions allow for the normal processes of scientific communication. Full details may be reported after publication of a preliminary report, usually an abstract. Information presented at a meeting may be published later so long as a full report has not appeared in the proceedings. Journalists at a meeting can't be prevented from reporting the findings, but speakers should not hand over written copies of their data or amplify them afterwards. The Vancouver guidelines also give clear advice on secondary publication in another language.<sup>3</sup>

An increasing number of cases do not, however, fall neatly into one of the Vancouver categories. The annual reports of directors of public health, for example, often describe work that the investigators later want to write up for a professional journal. Some large funding bodies produce annual reports that are widely circulated. And in some countries government researchers are required to publish their reports as official documents.

The  $BM\mathcal{J}$  has decided that difficult cases will be settled by two criteria: how close the previous version is to the paper submitted to us and how retrievable it is. Many briefing reports written for government departments, for example,