on scalp blood, such as measurement of lactate concentration, do not appear to improve diagnostic accuracy. Results of direct intrapartum monitoring of the neurological state of the fetus by electroencephalogram recordings^{18 19} are difficult to interpret and once again probably identify only severe fetal distress.

Against a background of slow technical progress there has been an increasing demand for more "natural" labour. Mothers often accept fetal monitoring,²⁰ especially when they have had previous obstetric problems. Radiotelemetry²¹ allows more mobility and although this may not confer any great obstetric advantage,²² it should make fetal heart rate monitoring more acceptable and it is surprising that it has not had a more enthusiastic reception.

There is not, and is never likely to be, a single simple test of fetal wellbeing in labour. It is impossible to predict with sufficient accuracy which fetuses will become distressed, and probably all labours should be monitored. At present this entails fetal heart rate recording and scalp blood analysis, for the evidence suggests that if continuous monitoring of the fetal heart rate is used scalp blood analysis is an essential adjunct. Far more attention should be given to analysis of fetal heart rate traces by computer, since if this is shown to be beneficial inexpensive microprocessors could be used on a wide scale. It is high time that we had a new generation of smaller and more intelligent fetal heart rate monitors which, together with telemetry might make fetal monitoring in labour more effective and less intrusive.

ROBERT S SAWERS

Senior Lecturer and Consultant Obstetrician and Gynaecologist,
Birmingham Maternity Hospital,
Birmingham B15 2TG

- ¹ Anonymous. Is fetal monitoring worthwhile? *Br Med J* 1971;i:515-6.
- ² Haverkamp AD, Thompson HE, McFee JG, Cetrulo C. The evaluation of continuous fetal heart rate monitoring in high-risk pregnancy. *Am J Obstet Gynecol* 1976;**125**:310-20.
- ³ Renou P, Chang A, Anderson I, Wood C. Controlled trial of fetal intensive care. *Am J Obstet Gynecol* 1976;**126**:470-6.
- ⁴ Haverkamp AD, Orleans M, Langendoerfer S, McFee J, Murphy J, Thompson HE. A controlled trial of the differential effects of intrapartum fetal monitoring. *Am J Obstet Gynecol* 1979;**134**:399-412.
- ⁵ Kelso IM, Parsons RJ, Lawrence GF, Arora SS, Edmonds DK, Cooke ID. An assessment of continuous fetal heart rate monitoring in labor. A randomized trial. *Am J Obstet Gynecol* 1978;**131**:526-32.
- ⁶ Wood C, Renou P, Oats J, Farrell E, Beischer N, Anderson I. A controlled trial of fetal heart rate monitoring in a low-risk obstetric population. *Am J Obstet Gynecol* 1981;**141**:527-34.
- ⁷ Edington PT, Sibanda J, Beard RW. Influence on clinical practice of routine intra-partum fetal monitoring. *Br Med J* 1975;iii:341-3.
- ⁸ Quilligan EJ, Paul RH. Fetal monitoring: is it worth it? *Obstet Gynecol* 1975;**45**:96-100.
- ⁹ Beard RW, Filshie GM, Knight CA, Roberts GM. The significance of the changes in the continuous fetal heart rate in the first stage of labour. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1971;**78**:865-81.
- ¹⁰ Pearson JF. Maternal and fetal acid-base balance. In: Beard RW, Nathanielsz PW, eds. *Fetal physiology and medicine*. London: W B Saunders, 1976:492-509.
- ¹¹ Wood EC. Scalp sampling, amniocentesis. In: Gluck L, ed. *Intrauterine asphyxia and the developing fetal brain*. London: Year Book Medical Publishers Inc, 1977:179-90.
- ¹² Pearce JM, Willson K. Fetal heart rate monitors—some clinical and technical aspects. *Br J Hosp Med* 1983;**30**:123-31.
- ¹³ Shelley T, Tipton RH. Dip area. A quantitative measure of fetal heart rate patterns. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1971;**78**:694-701.
- ¹⁴ Beguin F, Yeh SY, Forsythe A, Hon EH. A study of fetal heart rate deceleration areas. II. Correlation between deceleration areas and fetal pH during labor. *Obstet Gynecol* 1975;**45**:292-8.
- ¹⁵ Henry MJ, McColl DDF, Crawford JW, Patel N. Computing techniques for intrapartum physiological data reduction. II. Fetal heart rate. *J Perinat Med* 1979;**7**:215-28.
- ¹⁶ Hon EH, Koh KS. Electromechanical intervals of the fetal cardiac cycle. *Clin Obstet Gynaecol* 1979;**6**:215-21.
- ¹⁷ Stewart P, Hillan E, Calder AA, Nicol SM. A comparative assessment of an automated blood microprocessor for fetal blood pH measurements in the labour ward. *Br J Obstet Gynaecol* 1983;**90**:522-4.

- ¹⁸ Wilson PC, Levy LF, Spies S, Chadwick J. The technique of fetal electroencephalography during labour. *Br J Obstet Gynaecol* 1979;**86**:266-8.
- ¹⁹ Maynard DE, Cigen RJ, Viniker DA. Intrapartum fetal monitoring with the cerebral function monitor. *Br J Obstet Gynaecol* 1979;**86**:941-7.
- ²⁰ Molfese V, Sunshine P, Bennett A. Reactions of women to intrapartum fetal monitoring. *Obstet Gynecol* 1982;**59**:705-9.
- ²¹ Flynn AM, Kelly J, Hollins G, Lynch PF. Ambulation in labour. *Br Med J* 1978;iii:591-3.
- ²² Haukkamaa M, Purhonen M, Teramo K. The monitoring of labor by telemetry. *J Perinat Med* 1982;**10**:17-22.

Changing attitudes to exercise induced asthma

Bronchoconstriction induced by exercise is a common feature in patients with both allergic and non-allergic asthma. In asthmatic children this symptom may be particularly troublesome and may lead to physical and emotional isolation by their peer groups. Over the past few years growing medical awareness of the problems associated with exercise induced asthma has led to changing attitudes and has encouraged investigation into its mechanism so that a more rational approach may be applied to its management.

The precipitating event in exercise induced bronchoconstriction is cooling of the airway mucosa during humidification of the inspired air.¹ In the laboratory this can be measured as respiratory heat exchange and depends on the rate and depth of ventilation² and the temperature and humidity of the inspired air.³ Exercise in a warm, humid atmosphere produces less bronchoconstriction than the same exercise performed under cold, dry conditions⁴—which explains (in part) why exercise induced asthma occurs more frequently in runners than in swimmers doing the same amount of work.

By itself exercise serves only to increase the tidal volume and respiratory frequency and is not essential to the bronchoconstriction. Indeed, sedentary hyperventilation under isocapnic conditions can induce the same degree of bronchoconstriction as with exercise provided the two stimuli are matched for respiratory heat exchange.^{5 6} Whether cooling of the airways or loss of water from the bronchial mucosa is the primary stimulus for bronchoconstriction has to be elucidated. Nor is it certain how a thermal stimulus to the large airways is propagated to the smaller airways during exercise induced bronchoconstriction. The close correlation between the ability of exercise or isocapnic hyperventilation to produce bronchoconstriction and the response of the airways to non-specific bronchial provocants such as methacholine or histamine suggests that exercise induced asthma is a reflection of non-specific bronchial hyperreactivity.⁷

Several theories have been put forward to link airway reactivity with exercise induced asthma. One such mechanism, which may be loosely defined as the "leaky mast cell theory," is that increased local concentrations of inflammatory mediators in the airways may alter the threshold at which the smooth muscle or sensory nerves respond to a fall in temperature.⁸ Alternatively a reduction in the temperature or changes in the hydration of the bronchial mucosa may stimulate release of inflammatory mediators.⁹ After exercise plasma values of a high molecular weight neutrophil chemotactic activity and histamine both rise,¹⁰ two mediators which are associated with mast cells. Furthermore, the mast cell stabilising drug sodium cromoglycate inhibits both the mediator and airway responses to exercise.¹¹ These observations should, however, be interpreted with some caution since there is no definitive evidence

that high molecular weight neutrophil chemotactic activity is derived from mast cells, and increases in plasma histamine with exercise may originate from circulating basophils.^{6,12} Furthermore, sodium cromoglycate has several pharmacological effects other than mast cell stabilisation which may be relevant to its protective effect in exercise induced asthma.¹³

Several factors should be taken into account when assessing and treating patients, and especially children, with exercise induced bronchoconstriction.¹⁴ Asthmatics are often in poor physical condition and even small amounts of exercise may produce disproportionate hyperventilation and therefore bronchoconstriction. Nevertheless, they need regular physical activities to maintain their fitness and help them through difficult periods. Children with asthma are commonly exempted from physical exercise at school and they may avoid sports or exercise in their leisure time.¹⁵

These attitudes need to be reversed, and local activity groups are being or have been set up to encourage physical training and group activities. At least 70 such groups are operating in Britain and have proved a great success. As well as encouraging asthmatic children to be more competitive and sociable they promote the prophylactic use of drugs and the gradual build up of muscle strength. The groups' physical activities include a warming up period followed by games and organised physical training programmes. Several studies have shown definite psychological and social benefit from such programmes.¹⁵ The gains include an increase in aerobic fitness; an improvement in breathing mechanics, posture, body mass, cardiac output, muscle strength, and endurance; and also reductions in the frequency and severity of asthma attacks, absenteeism from school, and requirements for medication. Most training programmes are designed to require submaximal exercise, falling short of inducing exercise induced bronchoconstriction.¹⁶ One recent study, however, has shown that a distance running programme can be safely undertaken by asthmatic children—but with benefit only in relation to physical fitness and not in resting pulmonary function, exercise induced bronchoconstriction, or ventilatory muscle strength.¹⁷

Plainly the image of bronchial asthma is now changing—a fact reflected in the large number of asthmatic people who have achieved outstanding successes in a variety of international sports. How gratifying it is that these sporting celebrities have been so ready to allow their condition to be widely publicised for the benefit of others.

S T HOLGATE

Senior Lecturer in Medicine,
University of Southampton,
Southampton General Hospital,
Southampton SO9 4XY

¹ Deal EC Jr, McFadden EF Jr, Ingram RH Jr, Jaeger JJ. Hyperpnea and heat flux: initial reaction sequence in exercise-induced asthma. *J Appl Physiol* 1979;**46**:476-83.

² Kivity S, Souhrada JF. Hyperpnea: the common stimulus for bronchospasm in asthma during exercise and voluntary isocapnic hyperpnea. *Respiration* 1980;**40**:169-77.

³ Strauss RH, McFadden ER, Ingram RH Jr, Deal EC, Jaeger JJ, Stearns D. Influence of heat and humidity on the airway obstruction induced by exercise in asthma. *J Clin Invest* 1978;**61**:433-40.

⁴ Bar-Or O, Neuman I, Dotan R. Effects of dry and humid climates on exercise-induced asthma in children and preadolescents. *J Allergy Clin Immunol* 1977;**60**:163-8.

⁵ Deal EC Jr, McFadden ER Jr, Ingram RH Jr, Strauss RH, Jaeger JJ. Role of respiratory heat exchange in production of exercise-induced asthma. *J Appl Physiol* 1979;**46**:467-75.

⁶ Holgate ST, Church MK, Cushley MJ, Robinson C, Mann JS, Howarth PH. Pharmacological modulation of airway calibre and mediator release in human models of bronchial asthma. In: Kay AB, Lichtenstein LM, Austen KF, eds. *Asthma, physiology immunopharmacology and treatment*. New York and London: Academic Press (in press).

⁷ Chatham M, Bleecker ER, Smith PL, Rosenthal RR, Mason P, Norman PS. A comparison of histamine, methacholine and exercise airway

reactivity in normal and asthmatic subjects. *Am Rev Respir Dis* 1982;**126**:235-40.

⁸ Souhrada M, Souhrada JF. The direct effect of temperature on airway smooth muscle. *Respir Physiol* 1981;**44**:311-23.

⁹ Lee TH, Assoufi BK, Kay AB. The link between exercise, respiratory heat exchange, and the mast cell in bronchial asthma. *Lancet* 1983;**ii**:520-2.

¹⁰ Lee TH, Brown MJ, Nagy L, Causon R, Walport MJ, Kay AB. Exercise-induced release of histamine and neutrophil chemotactic factor in atopic asthmatics. *J Allergy Clin Immunol* 1982;**70**:73-81.

¹¹ Lee TH, Nagy L, Nagakura T, Walport MJ, Kay AB. Identification and partial characterization of an exercise-induced neutrophil chemotactic factor in bronchial asthma. *J Clin Invest* 1982;**69**:889-99.

¹² Morgan DJR, Phillips MJ, Moodley I, Elliott EV, Davies RJ. Histamine, neutrophil chemotactic factor and circulating basophil levels following exercise in asthmatic and control subjects. *Clin Allergy* 1982;**12**, suppl: 29-37.

¹³ Bernstein IL. Cromolyn sodium in the treatment of asthma: changing concepts. *J Allergy Clin Immunol* 1981;**68**:247-53.

¹⁴ Osseid S. Physical activity as part of a comprehensive rehabilitation programme in asthmatic children. In: Osseid S, Edwards AM, eds. *The asthmatic child in sport and play*. London: Pitman Press, 1983:237-45.

¹⁵ Fitch K. Sport, physical activity and the asthmatic. In: Osseid S, Edwards AM, eds. *The asthmatic child in sport and play*. London: Pitman Press, 1983:246-80.

¹⁶ Keens TG. Exercise training programs for pediatric patients with chronic lung disease. *Pediatr Clin North Am* 1979;**26**:517-24.

¹⁷ Nickerson BG, Batista DB, Namey MA, Richards W, Keens TG. Distance running improves fitness in asthmatic children without pulmonary complications or changes in exercise-induced bronchospasm. *Pediatrics* 1983;**71**:147-52.

Antibiotic resistance in *Serratia marcescens*

The Gram negative bacillus now called *Serratia marcescens* and classified as a member of the Enterobacteriaceae was formerly known by a variety of names, including *Chromobacterium prodigiosum*. The bacillus is ubiquitous, and it characteristically produces a red pigment, prodigiosin. There are reports of it being responsible for the discoloration of food,¹ and romantic stories from the middle ages of communion bread mystically dripping blood are explained by its growth on a starchy substrate incubated in moist conditions at ambient temperatures.² For many years bacteriologists considered *Serratia marcescens* to be non-pathogenic and used it as a biological marker because of its easily recognised red colonies. Studies in man were carried out as late as 1960, all apparently without ill effect. In one notable British study *Serratia marcescens* was isolated from the blood of nearly half of the patients whose gums had been painted with a culture of the organism before dental extraction.³

In hospitals the organism is now regarded as an important pathogen, a reputation which started in the 1950s when infections due to *Serratia* were first reported.⁴ Most of the strains that cause infection fail to form pigment, so their colourless colonies are difficult to distinguish from those of other coliform organisms.⁵ They are usually identified by a series of biochemical tests which were not routinely performed before the introduction of commercially prepared kits (such as the API 20 E system). Isolates of *Serratia marcescens* may have been reported as "paracolon bacilli," and possibly many infections went unrecognised.⁶ Although this may be true, two reports from Glasgow Royal Infirmary, separated by a decade, suggest that *Serratia* infections are on the increase in Britain.^{7,8}

A genuine increase is not surprising because the organism causes opportunistic infections in patients ill from some other cause and further predisposed to infection by recent treatment with antimicrobial drugs (often cephalosporins) to which