

improvement in survival was seen with oxygen until 500 days from the start of treatment. Thereafter the death rate in the oxygen group (12% a year) was less than half that of the control group (29% a year). From the outset the mortality of the control women was significantly greater than that of the treated women.

Only cautious conclusions can be drawn from these two different studies, but prolonged oxygen treatment does seem to improve survival with severe chronic obstructive lung disease if it is given for 15 or more hours a day, the benefit being most noticeable in patients with the greatest disability. The delay in the appearance of benefit from home oxygen among the men in the British study may perhaps indicate that more patients with very severe disability had been included and that these biased the initial results.

Furthermore, prolonging life by the use of long periods of oxygen each day may not necessarily have a dramatic effect on the quality of life of these patients. Necessarily the home oxygen system in current use in Britain seriously limits the patient's mobility, for he or she must trail tubing about in a limited radius from the cylinder. The two American liquid oxygen systems are more effective means for providing portable oxygen for exercise. Even so the weight of the portable equipment may abolish any gain in mobility unless the equipment is wheeled by the patient on a shopping trolley.¹⁴

The financial implications are also important. A survey in Britain in 1961 showed that 17% of men and 8% of women aged 40-64 years had chronic bronchitis and emphysema.¹⁸ The prevalence of the disease has declined with clean air legislation, and not all bronchitics have hypoxaemia; but nearly 30 million working days are lost each year in Britain from chronic bronchitis and emphysema.¹⁹ Possibly some 10 000-50 000 patients in Britain might benefit from long-term oxygen treatment, but more accurate figures are urgently required. If such long-term treatment is to be offered to hypoxaemic patients, clearly the most economical and effective means must be used.

The oxygen concentrator²⁰ seems to provide this, but at present oxygen cylinders—the most expensive way of delivering the oxygen—are the only means available through the NHS Drug Tariff. Continuous oxygen treatment for 15 or indeed 24 hours a day from the oxygen concentrator is five to seven times cheaper than the same treatment from oxygen cylinders.²¹ The oxygen concentrator is a device the size of a domestic refrigerator that extracts nitrogen from air, using an electric compressor. The concentrator has proved to be safe, reliable, and effective,¹⁷ and is extensively used already in North America, Dublin,²² and Birmingham.²⁰

Now that the clinical indications for long-term oxygen treatment are becoming more clear, the NHS should surely arrange to provide this treatment by the most economical method. A second priority should be a scientific investigation of the use of intermittent transient oxygen for the relief of breathlessness such as that following exercise. Quite possibly the total cost of home oxygen treatment could be reduced if cheap and effective treatment of hypoxic chronic bronchitis is substituted for the apparently haphazard use of home oxygen that appears to prevail at present.

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⁵ Scottish Health Services Council. *Uses and dangers of oxygen therapy*. Edinburgh: HMSO, 1969.

⁶ McNeill RS, Watson JM. Oxygen therapy in the home. *Br Med J* 1966;ii:331-3.

⁷ Howell JBL, Campbell EJM, eds. *Breathlessness*. Oxford: Blackwell Scientific Publications, 1966.

⁸ Porter R, ed. *Breathing: Hering Breuer centenary symposium*. London: J and A Churchill, 1970. (Ciba Foundation Symposium.)

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¹⁰ Trenchard D, Gardner D, Guz A. Role of pulmonary vagal afferent nerve fibres in the development of rapid shallow breathing in lung inflammation. *Clin Sci* 1972;42:251-63.

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¹³ Bradley BL, Garner AE, Billiu D, Mestas JM, Forman J. Oxygen-assisted exercise in chronic obstructive lung disease. The effect on exercise capacity and arterial blood gas tension. *Am Rev Respir Dis* 1978;118:239-43.

¹⁴ Leggett RJE, Flenley DC. Portable oxygen and exercise tolerance in patients with chronic hypoxic cor pulmonale. *Br Med J* 1977;iii:84-6.

¹⁵ Woodcock AA, Gross ER, Geddes DM. Oxygen relieves breathlessness in "pink puffers." *Lancet* 1981;ii:907-9.

¹⁶ Medical Research Council Working Party. Long-term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981;ii:681-6.

¹⁷ Nocturnal oxygen therapy trial group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease. A clinical trial. *Ann Intern Med* 1980;93:391-8.

¹⁸ Respiratory Diseases Study Group of the College of General Practitioners. Chronic bronchitis in Great Britain. *Br Med J* 1961;iii:973-9.

¹⁹ Royal College of Physicians. Report of the Committee on Thoracic Medicine. Disabling chest disease: prevention and care. *J R Coll Physicians Lond* 1981;15:3-20.

²⁰ Stark RD, Bishop JM. New method for oxygen therapy in the home using an oxygen concentrator. *Br Med J* 1973;ii:105-6.

²¹ Lowson K, Drummond MF, Bishop JM. Costing new services: long-term domiciliary oxygen therapy. *Lancet* 1981;ii:1146-9.

²² Callaghan B. Long-term low flow oxygen in the management of chronic pulmonary insufficiency. *Irish Medical Times* 1979; 28 September: 16-17, 19.

Obstetric training and general practitioners

Medical education has changed considerably in Britain over the past 20 years. It is now accepted that the undergraduate period is but the beginning, with an obligation to undertake further training before full registration. Even then the doctor will practise under supervision for a varying number of years depending on his chosen specialty. This broader, long-term view of training has led to appreciable pruning in the undergraduate years, with certain major and long-established disciplines cutting back their teaching programmes on the understanding that the young doctor will receive further instruction under supervision. In the sense that preregistration posts are predominantly in the medical or surgical wards there is the opportunity for physicians and surgeons to reinforce and amplify the teaching they have given in the clinical years of the undergraduate curriculum. This is not the case in obstetrics, which is widely held to be too specialised for inclusion in the preregistration year. Hence at the time of full registration most, if not all, young doctors have no more experience of obstetrics than was offered during the time they were medical students.

The recommendations of the General Medical Council about obstetrics in the undergraduate curriculum are that: "The teaching should include instruction in the principles of human reproduction and family planning and in the principles and practice of normal obstetrics. The teaching should emphasise antenatal and postnatal care, the management of normal labour and its complications, the impact of

pregnancy on general disease and of general disease on pregnancy." These recommendations are capable of different interpretation by individual medical schools, and the Maternity Services Subcommittee of the General Medical Services Committee has expressed concern that some students may receive inadequate training in practical obstetrics.¹ It would be of interest to know what the students think. Do they consider themselves competent, on leaving their medical schools, to supervise antenatal care, manage labour, and cope with the commoner complications that may arise before, during, and after delivery of the child? Some are sure to have doubts, yet they can pass through the preregistration year, complete a vocational training programme recognised by the Joint Committee for Postgraduate Training in General Practice, and in time become a principal in general practice without any further training in obstetrics. The point that most concerns the Maternity Services Subcommittee is that all principals in the National Health Service—whether or not they fulfil the criteria for their names to be on the obstetric list—are obliged by the Secretary of State under paragraph 13 of their terms of service to provide care in an obstetric emergency.

Inevitably there must be some comparison with the basic training given to midwives under the direction of the Central Midwives Board. That board has recently decided² that the training period for pupils who are already State-registered Nurses should be extended from 12 to 18 months and that the clinical component should include the antenatal examination of at least 100 women, the delivery of no fewer than 40 women, attendance at no fewer than 40 complicated labours, the examination of 100 mothers during the postnatal period, and the examination of 100 newborn babies, and that pupils should have experience in caring for ill babies and those of low birth weight. It is unlikely that were it to review its recommendations for the teaching of obstetrics in the undergraduate curriculum the General Medical Council would include such precise and extensive directions.

If doctors, especially general practitioners, are to be trained to a standard that will allow them with confidence to step in and help a midwife faced by some emergency then additional and suitable training must be offered in the postgraduate period. It is unrealistic to expect that the preregistration year should always include worthwhile obstetric experience; this would very likely be impossible to arrange and not all doctors would want the experience anyhow. Much better would be agreement about some module of obstetric training, probably coupled with family planning and diseases of women, to be included in all training programmes submitted for recognition to the Joint Committee for Postgraduate Training in General Practice. This at least would ensure that all future principals in general practice had an accepted, basic level of training in obstetrics and closely related subjects. Such an innovation might well affect posts already recognised for training by the Royal College of Obstetricians and Gynaecologists and the implications would need to be discussed with the college and other interested bodies. Whether this component of the vocational training programme should vary depending on whether the doctor wishes to provide a full maternity service or simply undertake antenatal and postnatal care is a point of detail that could well be discussed after the basic principles have been agreed. It is timely that this whole subject should be raised and discussed fully for the present state of affairs is unsatisfactory, embarrassing in some ways, and potentially dangerous.

¹ Anonymous. Strong views on obstetrics. *Br Med J* 1981;282:1169-72.

² Central Midwives Board. Approved training syllabus, September 1980.

Paroxysmal nocturnal haemoglobinuria

Paroxysmal nocturnal haemoglobinuria is a rare, acquired haemolytic anaemia in which the circulating red blood cells are abnormally sensitive to lysis by activated serum complement. As the name implies, excessive destruction of red cells takes place mainly during the night so that haemoglobinuria is seen only or principally in the early morning. The platelets and granulocytes in paroxysmal nocturnal haemoglobinuria are also abnormally sensitive to complement, and thrombocytopenia or granulocytopenia or both are often seen during the disease.

Paroxysmal nocturnal haemoglobinuria may be diagnosed simply and reliably by the acidified serum lysis (Ham's) test; haemosiderinuria is a valuable sign of chronic intravascular haemolysis since iron-containing granules will be present in the urinary sediment even if there is no haemoglobinuria at the time. Haemolysis in paroxysmal nocturnal haemoglobinuria is due to a defect in the erythrocyte membrane. Nevertheless, not all circulating erythrocytes are equally sensitive to activated complement since a considerable population of abnormal cells usually coexist with moderately or normally sensitive red cells. The proportions of normal and abnormal cells in a given patient can best be measured by analysing the curves obtained from complement lysis sensitivity tests.¹ Thus the severity of haemolysis will depend on both the relative proportions of normal and abnormal cells and the degree of the abnormality. Platelets and granulocytes also interact abnormally with complement,² and, as with the red cells, only some of the granulocytes are defective.^{3,4} These reactions may partly be responsible for the venous thromboses which occur in patients with paroxysmal nocturnal haemoglobinuria as well as for the chemotactic defect in the granulocytes incubated with activated complement.⁵ Recently Tumen *et al*⁶ have shown that erythroid and myeloid precursor cells also have an increased sensitivity to complement.

Paroxysmal nocturnal haemoglobinuria is a chronic disease, and, if threats such as haemolytic crises and thromboses can be avoided, patients may live for many years.⁷ Venous thrombosis is a frequent problem in patients with paroxysmal nocturnal haemoglobinuria and can cause headache and abdominal pain.^{8,9} Progressive diffuse hepatic venous thrombosis is one of the most serious complications and a common cause of death. Indeed, abdominal pain in patients with paroxysmal nocturnal haemoglobinuria should be considered secondary to intra-abdominal thrombosis, particularly hepatic venous thrombosis, unless proved otherwise.⁹ Recently Clarke *et al*¹⁰ have presented evidence of widespread renal lesions in paroxysmal nocturnal haemoglobinuria, which they attributed to repeated microvascular thrombosis, and no fewer than seven of the 104 patients reviewed by Polli *et al*¹¹ died from renal failure. Vascular accidents in the brain caused the deaths of three of the eight patients reviewed by Clarke *et al*¹⁰ and 12 of the 104 reviewed by Polli *et al*.¹¹ Neutropenia in paroxysmal nocturnal haemoglobinuria results in increased susceptibility to infection, which may be exacerbated by other factors such as saturation of the reticuloendothelial system as a result of haemolysis¹² or abnormal granulocyte function.²

There is good evidence that the abnormal cells in paroxysmal nocturnal haemoglobinuria arise from a defective haemopoietic stem cell, and that normal and abnormal cells coexist in the patient's bone marrow. The clonal origin of the abnormal red cells in a patient with paroxysmal nocturnal haemo-