

contributed to the development of anti-mouse antibody.

The development of human-derived monoclonal antibodies may prevent or at least minimise this problem in the future.

A C NEWLAND

Department of Haematology,
London Hospital Medical College,
London E1 1BB

D C LINCH

Department of Haematology,
University College School of Medicine,
London WC1

¹ Cosimi AB, Calvin R, Burton RL, *et al.* *N Engl J Med* 1981;**305**:308-14.

² Miller RA, Maloney DG, McKillop J, Levy R. *Blood* 1981;**58**:78-86.

³ Linch DC, Beverley PCL, Newland AC, Turnbull AL. *Clin Exp Immunol* (in press).

⁴ Dresser DW, Gowland G. *Nature* 1964;**203**:733-6.

Obstructive sleep apnoea syndrome

SIR,—In your leading article on obstructive sleep apnoea syndrome (21 August, p 528) Dr J R Stradling made no reference to mandibular size when discussing the possible aetiological factors involved. During the past two years, I have seen three small boys who had experienced numerous episodes of obstructive sleep apnoea. In each case the mandible was abnormally small.

The first patient, aged two years, had an asymmetrical micrognathia due to ankylosis of the left temporomandibular joint. The ankylosis was excised, and the joint was reconstructed using a costochondral graft. Eighteen months later there is good mandibular opening, facial growth appears to be proceeding normally, and there have been no further apnoeic attacks.

The second patient was five years old, and had Treacher Collins syndrome. Surgical rotation and advancement of the very small mandible has resulted in an improved facial profile and the cessation of episodes of nocturnal apnoea.

The most recent patient, aged two, exhibited the features of bilateral hemifacial microsomia. Persistent obstructive sleep apnoea had necessitated a tracheostomy at the age of three months. Mandibular reconstruction six months ago has improved the appearance, but it has not been possible to remove the tracheostomy tube. This is probably due to an element of pharyngeal hypoplasia, an occasional feature of hemifacial microsomia.¹

Recognition of micrognathia as a cause of obstructive sleep apnoea, and appropriate surgical correction, may obviate the need for a long-term tracheostomy.

D R JAMES

The Hospital for Sick Children,
London WC1N 3JH

¹ Shprintzen RJ, Croft CB, Berkman MD, Rakoff SJ. *Cleft Palate J* 1980;**17**:132-7.

SIR,—I found your leading article by Dr John R Stradling (21 August, p 528) both interesting and educative. I would like to bring to the attention of your readers another cause of the syndrome and its management.

Kuo *et al*¹ reported in 1979 three patients who were suffering from the syndrome, and the next year Bear and Priest² reported another case. All four patients suffered from retruded mandibles. The management in all four cases was by a pull-forward mandibular osteotomy. This increased the pharyngeal air space and

allowed normal sleep pattern. This operation prevented a permanent tracheostomy with all its disadvantages.

Recently I have been involved in the management of a girl with the syndrome who has benefited from a bimaxillary pull-forward osteotomy. She was born with a Pierre Robin syndrome but did not develop the sleep apnoea syndrome till she was 16 years of age. Early postoperative results have been good, but there is always the danger of relapse, hence the long-term results of the treatment are not yet available. Bimaxillary osteotomies allow patients with normal dental occlusion to benefit from the advantages of mandibular pull forward but still retain a normal dental occlusion.

Patients who have the syndrome might benefit if oral surgeons are added to the team who are involved in the management of these patients. Not only is the syndrome helped with an osteotomy but the patient might also benefit from an improved facial appearance, an improved dental occlusion, or both.

JAMES WALLACE

Department of Oral Surgery,
University of Edinburgh,
Edinburgh EH1 1EE

¹ Kuo PC, West RA, Bloomquist DS, McNeil RW. *Oral Surg* 1979;**48**:385-92.

² Bear SE, Priest JH. *J Oral Surg* 1980;**38**:543-9.

Problems with perinatal pathology

SIR,—Like Dr H Kohler (5 June, p 1709), I also applaud the efforts of Dr A J Barson and others (27 March, p 973) in highlighting the serious lack of paediatric pathologists.

It may seem strange for a psychiatrist to enter this debate to plead for more (and better trained) paediatric pathologists, but in my work with bereaved families following the loss of a baby from "cot death" or sudden infant death syndrome I share the view of my paediatric and other colleagues that this diagnosis at a coroner's necropsy (usually totally appropriate in the circumstances as I have explained elsewhere¹) depends entirely on the sophistication and expertise of the pathologist involved.

The definition, of course, of a sudden infant death turns on the event occurring in an apparently healthy baby. Professor John Emery² and others over the past few years have explained how a detailed necropsy may, in the hands of an experienced paediatric pathologist, reveal abnormalities in some of these hitherto unexplained deaths. For a proportion of parents, therefore, there may be a pathological explanation which may help them to bear the loss of their child and help those supporting them to relieve the guilt and self-blame which they so commonly experience.

Even where no clear pathological evidence of disease is forthcoming, we are aware that it is extremely helpful and constructive for the parents to know that the investigation was carried out in detail by a pathologist with training and experience in the field.

It would be wrong, however, to suppose that only these parents of babies dying of sudden infant death syndrome experience bewilderment and confusion following the death of their baby. Others with whom I have been involved clinically (including a study of 100 families following infant deaths) are parents whose baby has been stillborn or has died in the neonatal period. Both from the very important

point of genetic counselling made by Dr Kohler and that of bereavement counselling and support, adequate information as to the cause of death and likelihood of recurrence are vital.

It is now widely understood and accepted that physical, psychiatric, and psychosocial sequelae following bereavement do occur and that preventive intervention is important and effective.³ The prevention and mitigation of family distress and marital pathology is surely a worthwhile objective, and in this context any improvement in the quality of necropsy information available is most desirable.

KERRY BUGGLASS

St Christopher's Hospice,
London SE26 6DZ

¹ Buglass K. *J Child Psychol Psychiatry* 1981;**22**:411-21.

² Emery JL, McWeeney P. *Arch Dis Child* 1975;**50**:191-96.

³ Parkes CM. *Br Med J* 1980;**281**:3-6.

A resurgent evil

SIR,—I agree with all of Dr J P Lester's principles (21 August, p 548) except the one advocating parity in three years. Maximum earnings at about age 30 years, in only the fourth year of a career of about 38 years, is quite imbalanced and unlike the conditions obtaining in any other profession.

I would advocate a much longer "incremental" scale starting at 60% of the maximum and rising by 16 steps of 2½% annually to reach parity. Extra increments would be awarded for postgraduate experience additional to vocational training, one increment for each year, and for postgraduate degrees and diplomas. One extra increment might be appropriate for the diploma in child health while membership of the Royal College of General Practitioners would command five. (All partners with membership of the Royal College of General Practitioners should also take the multiple choice questionnaire paper every five years and would lose five increments (12½%) on failure. The college should make provision for such multiple choice questionnaire only tests.)

Thus the applicant who qualified at 24 and has done vocational training, passed the diploma in child health and membership of the Royal College of General Practitioners, and eventually joins a practice at 34 would start with 12 increments (90%) and reach parity in four years; the vocationally trained but without further qualifications or experience would have 16 years to parity (reduced by five as soon as he passes membership of the Royal College of General Practitioners).

JOHN C ANDERSON

Southminster CM0 7T

SIR,—I was pleased to read Dr J P Lester's article "A resurgent evil" (21 August, p 548). The fact that it was written by a course organiser was very encouraging as it would appear that the importance of the "business" of general practice is now being more widely taught. The GMSC is aware of problems in this field, and the report of a working party is awaited. In Scotland for many years the SGMSC has published advice on partnership agreements and offered an advisory service on proposed agreements submitted to it. This has proved valuable for the whole profession in