Widal reaction negative; C-reactive protein ++; antibodies to influenza negative; serum proteins normal. Primary pulmonary tuberculosis was suspected so she received a trial of streptomycin, PAS, and isoniazid. There was only a slight improvement and the pyrexia persisted.

Three weeks later she complained of giddiness. No neurological signs were elicited but it was observed that neither radial pulse was palpable. The brachial pulses were absent and the axillary and carotid pulses very weak. The blood pressure could not be registered in either arm; the skin was cold but not gangrenous; she complained bitterly of pain in the arms and over the chest and back. The eyes were normal. A diagnosis was made of "pulseless disease," probably autoimmune in origin.

Streptomycin and chemotherapy were discontinued and replaced by steroid therapy (prednisolone 7.5 mg daily). Improvement was gradual. The pyrexia subsided and the chest x-ray cleared, but she continued to complain of pain in the arms, especially on movement, and arterial pulsation in the upper extremities remained absent for several months. In 1960, after three years' steroid treatment, all the arterial pulses in the upper extremities could be palpated easily and the carotid pulses were normal; the blood pressure was 120/80 mm in each arm. A systolic murmur was audible in the neck on both sides. Tests for antinuclear factor were now strongly positive. In 1963 an aortogram showed a normal arterial tree except for narrowing of the left subclavian distal to the first rib. After six years steroid treatment was reduced gradually over a period of four months. This was associated with recurrence of pain, especially in the arms, neck, and back, with Raynaud's phenomenon, and when the treatment was discontinued a rash appeared on the legs akin to erythema nodosum. Biopsy showed a lesion indistinguishable from lupus erythematosus; LE cells were now detected for the first and only time. The pulses remained normal. In March 1965 steroid treatment was started again. The skin lesions and pain gradually resolved. Any attempt to reduce the dose of prednisolone below 7.5 mg daily caused pain. For the past 12 years steroid treatment has been continued in a daily dose of 7.5 mg and she has remained reasonably well; the arterial pulses in the neck and upper extremities have remained full. Twenty years after the onset she continues to lead a full life as housewife and secretary.

In this case the aetiology seems to be systemic lupus. Not only has steroid treatment resolved the obstructive arterial lesions but it has probably prevented ocular complications.

F J FLINT

Northern General Hospital,
Sheffield

Acquired myopia and educational attainment

SIR,—There may be no real mystery about the apparent association between acquired myopia and educational attainment. In their careful report (26 February, p 542) Dr Catherine S Peckham and her colleagues rightly point out the genetic nature of this defect. It requires no Mendelian genius to deduce that a myopic trait without some other compensatory advantage would be a severe handicap to family expansion and support in a peasant society or in manual occupations. It is tempting to speculate that in Catholic societies even the clever myopic trait was denied breeding by feeding its possessors into the priesthood. At a guess it seems not unlikely that an epidemiological investigation at the other end of the scale, the multiply handicapped, would also show an apparent increased prevalence of myopia. In these cases transmission is unlikely.

As long as society does not introduce some

artificial restraint on the progress and breeding of individuals with some disadvantage such as excessive shortness, hirsutism, or skin pigmentation compensated gifted families with these traits will prosper. Unfortunately society is not usually so tolerant. Myopia gene carriers may well consider themselves fortunate to possess what would be a great handicap in a primitive society but which in the technological age finds not only occupation but benefits from an aura of intellectualism. Even in this age, however, the myopic conquerors must still be bright to benefit.

DEREK ROBINSON

Department of Tropical Community
Health,
Liverpool School of Tropical
Medicine,
Liverpool

Cardiomyopathy in uraemic patients on long-term haemodialysis

SIR,—Dr Tilman Drücke and others (5 February, p 350) have presented convincing haemodynamic evidence of a congestive cardiomyopathy developing in three patients undergoing regular haemodialysis.¹ Their data on two other patients with ventricular dilatation, decreased index of normalised rigidity, and normal ejection fractions suggested that the process initially affects cardiac rigidity. This change, however, may simply reflect a compensated chronic volume overloaded state. We have studied a dialysis patient with a severe cardiomyopathy in whom clinical and haemodynamic parameters improved following successful renal transplantation.

A 34-year-old, non-hypertensive, non-alcoholic White male developed end-stage renal disease secondary to complications of cystinuria. He began haemodialysis in 1973 and experienced multiple bouts of congestive heart failure starting early in 1974. Treatment consisted of ultrafiltration dialysis for 4 hours 5 days a week and digitalis. In April 1975 he underwent pericardial stripping after developing tamponade. The myocardium was described by the surgeon as contracting poorly. The patient underwent cardiac catheterisation in October 1975 because of continued severe exertional dyspnoea, fatigue, and paroxysmal nocturnal dyspnoea.

Left ventricular hypertrophy was noted from the ECG voltage and chest radiographs showed four-chamber cardiac enlargement with increased pulmonary vascularity. Physical examination revealed 1+ oedema, hepatomegaly, jugular venous distension without Kussmaul's sign, biventricular enlargement, increased pulmonary second sound, and loud S₂ and S₃ gallops. Left and right heart catheterisation revealed: normal blood pressure, reduced cardiac index (2.4 l/min/m² by dye dilution when haematocrit was 30%), reduced stroke index (28 ml/beat/m²), elevated left ventricular end-diastolic pressure (24 mm Hg), reduced ejection fraction (0.23), and no haemodynamic evidence of constrictive disease. There was marked ventricular dilatation and diffuse hypokinesis with mild mitral regurgitation. A 50% stenosis of the left main coronary artery and another 50-60% lesion at its bifurcation were noted.

A non-living donor renal transplant was performed in November 1975. In April 1976 the patient underwent a limited haemodynamic assessment. His haematocrit was 40.5% and serum creatinine 168 µmol/l (1.9 mg/100 ml). Medications included azathioprine 50 mg, cyclophosphamide 75 mg, and prednisone 10 mg daily; diuretics were stopped five days before the study. Heart size had markedly decreased, exercise tolerance was much improved, and his dry weight had increased by about 15 kg. Blood volume was elevated at the time of study, intra-arterial blood pressure was 137/60 mm Hg, and cardiac and stroke indices were 4.34 l/min/m² and 46 ml/beat/m² respectively.

Our patient's clinical course before transplantation differs from that of Bailey's group of severely uraemic patients who improved after initiation of dialysis.¹ One cannot rule out a favourable effect of corticosteroids on myocardial function. However, good function persisted despite tapering of steroids. If this cardiomyopathy is indeed a uraemic complication of uniform origin and evolution its reversal with transplantation suggests a toxic aetiology. Although serial multiplanar echocardiographic studies of our patient's cardiac dimensions and myocardial function were not performed, such studies might be a non-invasive method of determining the prevalence and evolution of this "uraemic cardiomyopathy."

JOSEPH H BREZIN
PATRICIA LYONS

Hahnemann Medical College Hospital,
Philadelphia, Pennsylvania

JEFFREY BELL

Crozier Chester Medical Center,
Upland, Pennsylvania

¹ Bailey, G L, *et al*, *Transactions. American Society for Artificial Internal Organs*, 1967, **13**, 263.

Wire as suture material

SIR,—I was very interested in your leading article on burst abdomen (26 February, p 534), my particular interest being in the statement that "wire is not the easiest of sutures to use." This statement I would strongly contest. I have used wire as interrupted suture material in this hospital for 23 years with every registrar under my supervision following suit and I do not think there is the slightest difficulty in tying wire sutures. A running wire suture is another matter, but I presume that this is not what is meant in the article. It is also much easier to tie than monofilament polyethylene and in contrast to this material, which occasionally causes a sinus, wire *never* does, I should be interested to have your further comments.

A J M MATHIESON

Bridge of Earn Hospital,
Bridge of Earn, Perth

* * *What is important is to use a monofilament non-absorbable suture and we are sure that interrupted wire sutures are the equal of interrupted polyethylene. On the other hand, as Mr Mathieson says, although continuous wire is a very good suture material, it is not the easiest of sutures to use.—Ed, *BMJ*.

Need for maintenance diuretic treatment

SIR,—We read with great interest the article by Dr Sylvia M Dobbs and others (19 March, p 749) reporting a controlled trial in which maintenance digoxin was stopped in a group of outpatients. They comment that "a study evaluating long-term diuretic treatment in a similar group of patients would seem to be indicated."

It so happens that we have just published the results of a controlled trial of withdrawing long-term diuretics in 106 elderly people.¹ Our patients differed from those of Dr Dobbs and her colleagues in that they were geriatric inpatients and did not all have a history of cardiac failure. The chief effect of discontinuing diuretics was a tendency for ankle oedema to increase in some patients, but this was usually

slight and the majority did not require diuretic therapy to be resumed within 12 weeks. We concluded that old people receiving long-term diuretic therapy without obvious current indication should have the drugs withdrawn under careful supervision so that those needing them can be identified.

M L BURR
S KING

MRC Epidemiology Unit,
Cardiff

H E F DAVIES

Gwent Geriatric Services,
Newport

M S PATHY

University Hospital of Wales,
Cardiff

¹ Burr, M L, *et al*, *Age and Ageing*, 1977, 6, 38.

Hazards of non-practolol beta-blockers

SIR,—Relating to your leading article on the side-effects of beta-blocking drugs (26 February, p 529) I would like to ask some questions.

For 8½ months I took propranolol in doses of 30 mg a day, subsequently rising to 45 mg a day for the control of paroxysmal tachycardia. In January of this year I had to discontinue this treatment because of quite disabling side effects, most of which are listed in your article.

I am a person, I consider, of strong adrenergic drive and my questions are: (1) How does one recognise, as a doctor, the patient with a strong adrenergic drive and thus avoid prescribing beta-blocking drugs? (2) Why are these drugs contraindicated for such people? (3) What is the basic cause for problems arising from beta-blocking medication in such patients? (4) Are there any other drugs contraindicated for patients with a strong adrenergic drive?

ANNE S BARBER

Henley-on-Thames,
Oxon

Caesarean section and respiratory distress syndrome

SIR,—In your leading article on this subject (24 April 1976, p 978) several mechanisms are suggested which might be responsible for the higher incidence of the respiratory distress syndrome (RDS) in newborns delivered by caesarean section.

On the basis of certain indirect evidence we have developed a new hypothesis postulating that prostaglandins may play a central part in the pathogenesis of RDS in such infants. Our theory was suggested by the death from RDS of two near-term neonates whose mothers had been unsuccessfully treated with indomethacin according to the protocol of Zuckerman¹ in an attempt to prevent premature delivery.

In support of this hypothesis the following findings should be taken into account:

(1) It has been demonstrated that labour delivery after vigorous uterine activity² is associated with a higher rate of prostaglandin production than delivery by caesarean section.³

(2) Maternal treatment with prostaglandin antagonists is known to increase pulmonary vascular resistance in the neonate, resulting in right-to-left shunting of blood at the foramen ovale and/or ductus arteriosus and in subsequent pulmonary hypoperfusion.^{4,5} The pulmonary ischemia would damage the alveolar lining cells that produce surfactant and increase alveolar-wall permeability, leading to membrane formation.⁶

(3) Prostaglandins are known to enhance adrenal steroid production.⁷ It therefore seems to be relevant to assume that endogenous prostaglandin production could have a considerable effect on the fetal serum concentration of cortisol. Increased cortisol output by the fetal adrenals may be one mechanism by which normal maturation of pulmonary surfactant occurs.^{8,9}

(4) Augmentation of adenosine 3,5-monophosphate concentration in fetal lung has been associated with accelerated pulmonary maturation.¹⁰ Prostaglandins have been reported to increase the tissue cyclic AMP concentration through enhancing the adenylate cyclase activity.^{11,12} Consequently prostaglandins may be supposed to contribute to the acceleration of fetal lung maturation.

In conclusion, prostaglandins are intimately involved in normal cardiopulmonary adaptation to extrauterine life. Therefore decreased endogenous prostaglandin production, as observed after delivery by elective caesarean section and after maternal treatment with indomethacin, may be regarded as one of the factors responsible for the development of RDS. However, further studies are needed to provide more direct information as to the role of prostaglandins in the pathogenesis of RDS.

I F CSABA
E SULYOK
J HADNAGY

Department of Obstetrics and Gynaecology,
University of Pécs,
Pécs, Hungary

¹ Zuckerman, H, Reiss, U, and Rubinstein, I, *Obstetrics and Gynecology*, 1974, 44, 787.

² Batra, S, and Bengtsson, L P, *Lancet*, 1976, 1, 1164.

³ Jonsson, C E, Tuvemo, T, and Hamberg, M, *Biology of the Neonate*, 1976, 29, 162.

⁴ Tyler, T L, *et al*, *Federation Proceedings*, 1975, 34, 319.

⁵ Manchester, D, Margolis, H S, and Sheldon, R E, *American Journal of Obstetrics and Gynecology*, 1976, 126, 467.

⁶ Chu, J, *et al*, *Pediatrics*, 1965, 35, 733.

⁷ Flack, J D, Jessup, R, and Ramwell, P W, *Science*, 1969, 163, 691.

⁸ Farrell, P M, and Avery, M E, *American Review of Respiratory Diseases*, 1975, 111, 657.

⁹ Fencel, M de M, and Tulchinsky, D, *New England Journal of Medicine*, 1975, 292, 133.

¹⁰ Barrett, C T, *et al*, *Pediatric Research*, 1976, 10, 621.

¹¹ Butcher, R W, and Baird, C E, *Journal of Biological Chemistry*, 1968, 243, 1713.

¹² Hinman, J W, *American Review of Biochemistry*, 1972, 41, 161.

Dysphagia

SIR,—Drs R Ferguson and M Atkinson (12 March, p 714) appear to have misread my letter (5 February, p 382) regarding dysphagia. I did not write that the only hope for oesophageal stricture was surgical bypass. However, I wish to make the point that when a stricture does not respond satisfactorily to dilatation surgery is necessary and that this is perhaps more often the case in the elderly.

As regards the operation of oesophageal bypass, this is not a difficult procedure in the hands of a surgeon with oesophageal experience, and the mortality is certainly not greater than that quoted for dilatation in inoperable disease. In my opinion, the relief afforded is better than that obtained by dilatation and an in-dwelling tube.

DOUGLAS PARK

Oldchurch Hospital,
Romford, Essex

Administrative staff in the NHS

SIR,—As one of the doctors that Mr N H Harris feels should be dispensed with (26

March, p 842), I find myself fearing that he might be right. May I be permitted to make two personal offers to him? I will seriously consider returning to clinical medicine if he will seriously consider leaving London to work in an under-doctor part of the country. Alternatively, would he allow me the opportunity to see if I could save the cost of my salary by investigating whether there are any ways in which he or his colleagues might be working more efficiently?

K R WOODCOCK

Kensington and Chelsea and
Westminster Area Health Authority (Teaching),
London W2

Certification

SIR,—The article in "Briefing" (12 March, p 726) still, I am convinced, does not see the complete nonsense in the administrative reasoning for doctors having to certify incapacity for work by having to write out National Insurance certificates.

I could go into great detail, but let me just say that if it is felt that such certificates are necessary, then surely this should be done by someone who is quite unbiased. I am certainly not unbiased when someone claims he is unfit because of minor ailments when I could be "suffering" similarly. There is no question of my having time off work for trivialities. Further, should I have the luxury of a few days off work but feel fit to start work, for example, on a Friday, then I start on a Friday and not, as is almost always the case with the patient, on a Monday. The article states that we are often asked to sign certificates because of the status in society of doctors. I don't think that we have much status when, as happens, employers or their representatives often tell employees, "go to your doctor and tell him to give you a certificate." Home visits are frequently requested simply for supply of certificates. Refusal to give certificates not infrequently results in arguments, unpleasantness, and tensions of which the next patient, who could well be feeling ill, usually bears the brunt. Also if, as the article suggests, patients feel unable to attend their job because of the very nature of their work, then what has that got to do with the doctor? It certainly isn't illness. As there is often no objective evidence to back up the patient's account of the symptoms, then why can't the employer take his employee's word for his feeling ill? Why must it be the doctor? As happens abroad, persistent claimers will soon be found out.

Lastly, under "Objections to abolishing certification" (hospital staff must also provide certificates where necessary) I cannot see an ill patient not consulting his doctor if the need for getting a certificate was removed. The abolition of certification would greatly reduce the number of patients seeing their doctor for minor ailments. The people really feeling unwell would be given much more time and better attention, and as a bonus I am sure that the drug bill would be greatly reduced because the sufferers from minor ailments would not be there to get what most of us unfortunately do—that is, write out a script for something simply to keep them happy and let us get on with seeing the people who really need our attention.

JOHN M ANDERSON

Dundee