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concluded that old people receiving long-term diuretic therapy without obvious current indication should have the drugs withdrawn under careful supervision so that those needing them can be identified.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

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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 paroxymal 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 Zuckerman[1] 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 activity[2] 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

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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-doctored 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