thidine, and mecamlamine, the patient was given 10 mg of propranolol twice, six hours apart. She had no apparent response to the first dose; however, within two hours of the second dose her pulse rate fell from 120 to 60/min and her blood pressure from 240/120 to 90/50 mm Hg, and she became unresponsive. An intravenous infusion of isoprenaline was required for several hours to restore and maintain pulse rate and blood pressure at normal levels. Within a day thereafter, without additional medication, the patient's tachycardia and hypertension returned. She had no symptomatic or objective response of visceral or peripheral neuropathy to propranolol. Several days later the patient developed bleeding due to "stress" ulceration of the gastric mucosa. While receiving fluid and blood replacement she developed acute congestive heart failure and pulmonary oedema at a time when her blood pressure was low and clinically it was felt her intravascular volume depletion had not been corrected entirely.

We interpret these events as indicating that our patient was compensating haemodynamically for elevated blood pressure (before and during her acute attack) by β -adrenergic stimulation of cardiac function. This increase in β -adrenergic activities may have resulted from physiological reflex mechanisms and/or from an influence of acute intermittent porphyria on the sympathetic nervous system. In any event the positive chronotropic and inotropic effects of endogenous catecholamines appeared essential to cardiovascular homeostasis, which was adversely and severely affected even by a small amount of propranolol.

While propranolol is of benefit in some patients in porphyric relapse, it is not without risk in patients with incipient cardiac failure. It may also be of risk in the pronounced hypovolaemic states which have been observed in patients with acute intermittent porphyria⁶ in the absence of apparent excessive volume loss. Beta-adrenergic stimulation in some episodes of porphyria may provide haemodynamic compensation rather than being simply a manifestation of acute relapse.—We are, etc.,

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Idiosyncrasy to Phenothiazines in Phenylbutazone-associated Aplastic Anaemia

SIR,—The interesting paper by Dr. J. L. Cunningham and his colleagues (3 August, p. 313) suggested that poor paraoxidation of phenylbutazone may be a factor in the aplastic anaemia associated with phenylbutazone therapy. We have observed an idiosyncrasy to phenothiazines in a patient with phenylbutazone-associated aplastic anaemia.

A 27-year-old man developed aplastic anaemia as a result of 2½ years of phenylbutazone therapy for ankylosing spondylitis. He was admitted for bone marrow transplantation and, prior to this, received a buffy-coat transfusion followed by high-dose intravenous cyclophosphamide. The vomiting and nausea caused by the cyclophosphamide was treated with three 25-mg intramuscular doses of chlorpromazine over a two-day period and four 12.5-mg doses of prochlorperazine over the ensuing two-day period. This resulted in a dyskinesia manifested by oculogyric movements. Further phenothiazines were withheld and the dyskinesia passed off spontaneously in three hours.

Dyskinesias are a well-recognized side effect of the phenothiazines. They are not dose-related and can occur in the first few days of treatment, suggesting an individual predisposition.¹ It is of interest that the phenothiazines, while being metabolized in many different ways, undergo aromatic

oxidation² as one of their major pathways in a similar manner to phenylbutazone.³ We therefore wonder whether the two drug idiosyncrasies of this patient may indicate a defect in a common oxidation pathway, and would welcome reports of any similar interactions.—We are, etc.,

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Doctors' Pay

SIR,—We would like to voice our support for Dr. J. F. Nunn (31 August, p. 579).

The plight of young consultants is being overlooked by many of the older members of the profession who negotiate on our behalf. Many newly appointed consultants are in very difficult financial circumstances. They spend 8-10 years in junior hospital grades, frequently subsidizing their income by working for a deputizing service or undertaking general practice locums during their holidays. On appointment as a consultant they find themselves with an income barely sufficient to raise a mortgage and the prospect of 10 years until they reach the top of the consultant salary scale. To raise the maximum mortgage many start with full-time contracts, thus losing allowances for telephones and motor car depreciation, etc.

Is it surprising that many continue to scan the back pages of the *B.M.J.* to find employment in countries where the population is more appreciative of their talents? —We are, etc.,

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SIR,—Dr. S. Mehta and his colleagues (14 September, p. 690) are to be commended for directing attention to the question of consultant relativities, though these are not entirely new.

Clinicians are in clinical charge of patients after a relatively long and rigorously selective training. This includes a grounding in the complexities of dealing with the great British public wider than that of the more ancillary and subcontractual specialties and appropriate to those whose lot it is to stop most of the medical bucks going.

Lengths of apprenticeship in different specialties vary, which in turn affects total life earnings. If applicable, domiciliary fees and the differing incidence of merit awards compensate somewhat for longer pre-consultant training. Perhaps such things as work in operating theatres, anaesthetic or operative, and an incessant heavy load of emergency work deserve more recognition than at present. All graduates appear to

start with equal opportunities of seeking a consultancy in any specialty, without compulsion to enter a particular one. Applied consultant egalitarianism might prevent the future manning in full of the more exacting specialties.—I am, etc.,

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Trade Union Tactics

SIR,—Dr. P. H. Woodcock has expressed so aptly what most of us must feel about the present state of remuneration of the profession (21 September, p. 744).

We are told by the Secretary of State for Social Services that our recent small awards must stand for 12 months (17 August, p. 479), notwithstanding the recognition by the Review Body that it still leaves us behind in the financial stakes. If only other Ministers would dare say the same thing to those militant unions who are finding reasons why they should be exempted—unions whose members are among the higher paid workers.

Likewise, most of us will agree with what Dr. P. E. Baldry has said on the same subject (21 September, p. 744), but I would take issue with his suggestion that we should employ the services of an experienced specialist in trade unionism to help us protect our interests. We have our own able and experienced negotiators, the essential difference being that theirs have "teeth"—namely, the threat of industrial action—which they do not hesitate to use to achieve their aims without any hint of compromise. Are we prepared to give ours the same "teeth"? —I am, etc.,

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Dynamism and Inflation

SIR,—The latest issue of *B.M.A. News* (August/September 1974) contains the statement that "the new dynamising formula introduced for general practitioners in 1972 protects them against inflation over their careers in the build up of their pensions" (p. 37). This is not true.

The Review Body's Fourth Report reveals an anomaly which "results from the decision to use the increase in general practitioners' earnings as the 'dynamising factor' in the