private practice is done, other than to reduce the amount paid in proportion to notional sessional commitment. The first simple and most obviously equitable change would be to calculate the amount actually paid on the percentage of *earned* income coming from the NHS (or in the case of clinical academic staff the university). This would be patently fair if the money so released were ploughed back into providing more C awards in the same district or region. It would also be a real incentive for doctors to put their out of hours work into NHS related activities.

A second suggested change arises from the obvious anomalies facing many bright and highly productive young consultants including even some professors. They can be obstructed for years on the way to a C—the major hurdle—before then proceeding swiftly to higher things. Yet why try to mix chalk with cheese? Instead the equivalent of a C—£6760—should be sliced from the bottom of all the higher awards. This would create 6525 new C awards—that is, the existing total number of all awards—leaving in addition 1704 new B's (each worth £8580), 717 new A's (each worth £20 080), and 202 new A+'s (each worth £29 660)—9148 in all. The higher awards committees would award these as at present for academic and administrative excellence using the same criteria as they do at present (whatever those are). Really outstanding consultants would, of course, qualify for both a new C and a new higher award, and as dotage set in or interests shifted the burnt out academic might move from a new B to a new C without much loss of face or income.

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NHS review

SIR,—The consultants in this health district have been considering the way that the proposed changes in the government white paper *Working for Patients* may affect the care we can give our patients.

Though noting statements of some continuing increase in resource to the NHS, we think that they are incomplete without some acknowledgement that the proportion of the gross national product devoted to the health services remains among the lowest of all developed countries. We are gravely concerned at the extraordinarily limited time for considering the proposals for fundamental changes to one of the largest civil organisations in the world, particularly as they are wholly untested in practice.

Nevertheless, we welcome the support for initiatives to increase the awareness of all doctors in both the outcome and the costs of their clinical activities, and we have established a district audit advisory committee as recommended in the white paper. We also welcome the commitment to improve the extent and reliability of information about the costs of patient care and to transfer those costs rapidly between districts when patients have to cross district boundaries for their treatment. We are mindful, however, of the substantial resources that will be required to install adequate information systems; it will be months before the outcome of the evaluation even of pilot schemes will be available. We accept as a basis for discussion the proposals for reviewing consultant contracts and distinction awards, acknowledging that consultants must bear some accountability for the levels of resource they commit in the course of their clinical work.

If such changes were instituted and working effectively the results would meet nearly all the government's aspirations, including a degree of the managed competition it is so keen to introduce. We therefore greatly regret the further radical reorganisation entailed in the proposals for self governing hospitals and for the new funding and contractual arrangements for hospital services. Above all, the time scale proposed for implementing these wholly untried changes bears no relation to reality and seems to be dictated solely by political constraints. We are no less concerned by the notable absence of any consideration of how the proposals will affect the community health services and by the derisory consideration of the implications for undergraduate and postgraduate medical education.

The proposals seem to have been prompted largely by political concerns over waiting times for elective hospital procedures, which would be a fairly straightforward problem to resolve given minor additional resources and changes in working practices to permit the maximum use of hospital facilities. Without any indication even of modest further funding for clinical services we are gravely concerned that the effects of the white paper will be to enhance the acute hospital sector at the expense of some of our most vulnerable community health services, those for the elderly, the handicapped, and the chronically and mentally ill, and those for young children.

We are proud of the comprehensive services we have helped to develop, with our colleagues in management, for all our patients, often in the face of severe financial constraints, and of our role as a teaching district. With such limited available information about the detailed operation of the white paper's proposals, however, we have to regard them as potentially divisive and therefore oppose any proposals that individual units in this district should at this stage adopt self governing status.

I C S NORMAND

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SIR,—*Working for Patients* has raised a number of concerns. We would like to point out a fundamental flaw in the government's proposals as they apply to psychiatry, which derives from a failure to take a historical view.

The free market has never worked for psychiatry. Bethlem Hospital was the only institution for the mentally ill until private mad houses began to appear in the eighteenth century. The standards of these establishments were variable. Some, such as the Retreat, established in 1796 by William Tuke, were "conducted on humane principles," but the general pattern was one of inhuman and neglectful institutions.

In 1807 a select committee of the House of Commons recommended the establishment of public asylums. This was implemented by the "Wyn Act" of 1808, but few counties built asylums. A scandal in 1814 at York Asylum led to the establishment of another select committee in 1815. The committee heard how at Nottingham Asylum patients were categorised into either first and second class or paupers. The latter were "supported" by the parishes, whereas the former were wholly or partly paid for by relatives.[1] The miserable conditions of pauper lunatics was revealed further in evidence given to parliament in 1823 and 1827. So bad was their plight that the Earl of Shaftsbury introduced the 1845 act, which made provision of county and borough asylums mandatory. This was followed by such a proliferation of county asylums that the ratepayers objected strongly to the cost of providing for these.

Initially their standards were good. Medical officers, such as John Connolly, advocated non-restraint, and lunatics were rehabilitated through employment in local farms or light industry. With growing urbanisation during the industrial revolution and an increase in the population, however, asylums became lunatic colonies harbouring up to 1000 inhabitants, with the result that standards had declined again by the end of the century. The advent of the NHS and the recent renaissance in the care of the mentally ill has led to an improvement again.

Thus over 200 years a transition has occurred from private funding to public provision for the insane, which was precipitated by an outcry over the inadequate facilities available in private institutions. A state system of care for psychiatric patients was necessary 150 years ago—simply because there was no other reliable source of provision of care for this disadvantaged population. In contrast, mandatory public expenditure for the provision of acute hospital services did not occur until 1948.

In the sphere of mental illness our Victorian forbears pioneered state provision of health care. The failure of non-state provision of other medical services led inexorably to the extension of state provision to these services. A government which claims inspiration from Victorian values should take care that it knows what those values were.

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Drug Points

Erythromelalgia induced by nicardipine (inverse Raynaud's phenomenon?)

Drs H LEVESQUE, N MOORE, L M WOLFE, and H COURTOIS (Hôpital de Boisguillaume, BP 100, 76233 Boisguillaume, France) write: Calcium antagonists such as nifedipine, nicardipine, and diltiazem have become drugs of choice for Raynaud's phenomenon.[1] Erythromelalgia affects the hands and feet with paroxysmal, intense red discoloration, raised skin temperature, and burning pain. Though its mechanism is probably different, it can be loosely described as inverse Raynaud's phenomenon. It would therefore seem logical that it could be caused by calcium antagonists. We observed such a case with nicardipine.

A 67 year old woman was treated for about a year with nicardipine (60 mg/day) for hypertension. Three to four weeks after the onset of treatment she started complaining of intermittent redness, heating, and pain of the fingers, occurring more often in the evening after she went to bed and when the weather was warm. The symptoms were relieved when she put her hands in cold water. The diagnosis and relation with nicardipine were not immediately recognised, and she was referred after almost a year for diagnosis: blood and platelet counts were normal, there were no antinuclear antibodies, no rheumatoid factor, and capillaroscopy showed non-specific dilatation compatible with acrocyanosis. Nicardipine was withdrawn, and all symptoms subsided rapidly.

Erythromelalgia is often idiopathic or secondary to systemic or myeloproliferative disorders.[2] Three cases have been described with nifedipine,[3-5] some with bromocriptine,[6] and none with nicardipine. In our patient the temporal relation with nicardipine and the absence of other causes make this drug the most probable cause. The exact mechanism of erythromelalgia is not well known: platelets, serotonin, and prostaglandins have been incriminated, but the impact of the calcium antagonists on these is unclear.[7,8] The French national adverse drug reaction monitoring system's data bank contains a few cases which may be similar,[9] but the manufacturer (Sandoz) knows of no other cases.

Erythromelalgia due to calcium antagonists may be more frequent than reported. It is probably often reported as flushing or paraesthesia of the hands and feet. Doctors prescribing these drugs should be aware of this inverse Raynaud's phenomenon and withdraw the drug(s) before sending their patients for extensive investigations.

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Tamoxifen-warfarin interaction: the Aberdeen hospitals drug file

Drs LEWIS D RITCHIE AND SANDRA M T GRANT (Medicines Evaluation and Monitoring Group, Aberdeen Royal Infirmary, Aberdeen AB9 2ZB)

write: In Aberdeen for the past 17 years a computerised file of the drug history of each patient during hospital admission has been maintained.[1] The drug information is linked to personal and morbidity data, routinely collected for the Scottish Morbidity Return (SMR1). At the end of 1988 the file contained 932 000 admissions with over 2·5 million drug prescriptions.

Following the report by Dr P Tenni and others on life threatening interactions between tamoxifen and warfarin,[2] we examined all admissions to the Aberdeen general hospitals from 1980 to 1988 in which the two drugs had been prescribed together. Twenty nine such patients were identified and their case notes scrutinised. Seven were excluded (initial treatment elsewhere, or the two drugs were not given concurrently). In the remaining 22 cases no problems had been noted in 17. In the five other cases two patients had grossly raised British comparative ratios on introduction of warfarin but no episode of bleeding; one had no noted problem with control but experienced a subconjunctival haemorrhage; one had difficulty with control and developed a haematoma of the thigh; and one, who was taking long term warfarin, had problems with intraocular haemorrhage and haemorrhagic rashes after tamoxifen was introduced. Our findings confirm those of Dr Tenni and colleagues and we echo their concerns about this life threatening interaction.

The Aberdeen hospitals drug file offers the opportunity for studying potential drug interactions. We would welcome further inquiries about specific uses for this facility.

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Reversal of chloral hydrate overdose with flumazenil

Drs K L DONOVAN and D J FISHER (Cardiff Royal Infirmary, Cardiff CF2 1SZ)

write: Dr J G Whitwam says that flumazenil has been used in many conditions other than to reverse sedation induced by benzodiazepines.[1] We observed an interesting interaction between flumazenil and chloral hydrate taken in overdose.

A young man with a history of drug overdose was brought to the casualty department unconscious with respiratory depression and hypotension. Both pupils were constricted and his breath smelt sweet. Intravenous naloxone (0·4 mg×4 doses) produced no improvement, but flumazenil 200 μg, followed at one minute intervals by three further 100 μg doses, produced a dramatic response with increased level of consciousness, verbalisation, and pupillary dilatation; both respiratory rate and blood pressure returned to normal. The sludge obtained by gastric lavage was shown to be chloral hydrate and he later admitted to having taken a total of 10 g of the drug in isolation.

Although flumazenil is a specific competitive benzodiazepine receptor antagonist, it has been shown to have a dramatic but inconsistent effect in ethanol intoxication,[2-4] possibly by modifying the response of the γ aminobutyric acid-benzodiazepine ionophore receptor complex to the effects of ethanol.[5] The sedative mode of action of chloral hydrate, similar to that seen with benzodiazepines and ethanol, is poorly understood. The rapid reversal of recognised effects of chloral hydrate overdose seen in this case may therefore provide further information about the modes of action of both flumazenil and chloral hydrate. So far as we and the manufacturers are aware this has not been reported before, though attempted reversal of benzodiazepine-chloral hydrate induced sedation with flumazenil precipitated ventricular arrhythmias.[6] The mechanism is unclear, but it may be that while flumazenil antagonised the benzodiazepine element it failed to protect against chloral hydrate arrythmogenesis, a well recognised side effect.

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Cross sensitivity to antithyroid drugs

Drs A SMITH, R F GLEDHILL, and P JENKINS (East Surrey Hospital, Redhill, Surrey RH1 5RH)

write: Medical practitioners have come to rely on the *British National Formulary* for sound prescribing advice. The section on antithyroid drugs states, "Propylthiouracil may be used in patients who suffer sensitivity reactions to carbimazole as sensitivity is rarely displayed to both drugs."[1] This statement runs counter to views expressed elsewhere,[2-5] a standpoint which we highlight through the following case report.

A 66 year old woman presented to the accident and emergency department in June 1988 with a 24 hour history of pain in the wrists, elbows, shoulders, and interscapular area. Four months earlier she had had a myocardial infarction complicated by cardiac arrest and consequent thoracic spinal cord infarction. A flaccid, hyporeflexic paraplegia with diminution of pain sensation distal to either ankle had remained. She had been readmitted three months later in left ventricular failure with atrial fibrillation. At that time the serum free thyroxine concentration was 37 pmol/l (normal 10·4-24·2) and the thyroid stimulating hormone value 0·16 mU/l (0·44-3·16). Carbimazole 15 mg three times daily had been started on 23 May. This had been replaced on 13 June by propylthiouracil 100 mg three times daily after the appearance (one week earlier) of a pruritic macular eruption. Daily concurrent medication comprised nifedipine (60 mg), digoxin (250 μg), and amiloride hydrochloride 5 mg plus frusemide 40 mg (Frumil; 2 tablets).

On admission her temperature was 38·4°C and pulse 84 beats/min with a regular rhythm. The rash had disappeared. Both arms were held immobile and in a disordered posture, the slightest displacement evoking an anguished response. No joint swelling or erythema was evident. A full blood count and standard biochemical profile gave normal results. The erythrocyte sedimentation rate (Westergren) was 30 mm in the first hour. Tests for rheumatoid factor and antinuclear antibody gave negative results. The antibody titre to thyroglobulin was 1/1700 and to thyroid microsomes 1/110 000. Propylthiouracil was withdrawn on 17 June and dispersible aspirin 600 mg four times daily given. The temperature settled and less opioid analgesic was required for pain relief. Two days later the patient suffered a fatal cardiac arrest.

Arthralgia without joint inflammation was the second most common side effect in a recent survey of 500 patients with thyrotoxicosis given antithyroid drugs.[6] Of the five patients affected, four displayed sensitivity also to the alternative agent. Such events are not limited to rheumatic symptoms, cross sensitivity having been described in agranulocytosis,[7,8] marrow aplasia,[9] and hepatic injury.[10] The reaction to a second agent may also be more severe than that to the first.[7,9] Extensive immunological cross reactivity to all three antithyroid drugs was shown in patients who had developed agranulocytosis during treatment with either propylthiouracil or carbimazole.[5] The thioamide group, common to each compound, may either have been the antigen or have become antigenic by forming a complex with a neutrophil protein. Cross sensitivity might also arise on the basis of close chemical analogy.

Our patient's rheumatic symptoms developed 24 hours after both the introduction of propylthiouracil and the withdrawal of carbimazole. Although the carbimazole rash had already resolved, this drug, rather than propylthiouracil, might have been responsible. At all events, some authors, having regard to the potential for cross sensitivity, advocate using alternate treatments for patients who manifest an adverse reaction to one antithyroid drug and who need further treatment for hyperthyroidism.[2,3] The prudence of this policy merits an amendment to the *British National Formulary*.

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