to follow treatment with hyperbaric oxygen.¹ A reperfusion injury mediated by these metabolites is thought to cause the reported side effects.

The delayed neurological complications of carbon monoxide poisoning may in fact be due to secondary oxidative damage which occurs during the resuscitation of patients with hyperbaric oxygen or high concentrations of inhaled oxygen.² Pretreatment with α tocopherol to protect against the effects of toxic oxygen metabolites has been shown to reduce the incidence of convulsions.² The role of xanthine oxidase in the mediation of anoxic reperfusion damage has led to the use of the xanthine oxidase inhibitor allopurinol in its prevention. Allopurinol may be a simple and effective treatment in the prevention of severe neuropsychiatric complications of carbon monoxide poisoning in combination with hyperbaric oxygen or with high concentrations of inspired oxygen at normal pressures.³

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AUTHORS' REPLY.—Dr Howard emphasises the potential hazards of treatment with hyperbaric oxygen and suggests that delayed neuropsychiatric sequelae after carbon monoxide poisoning may be due to oxygen toxicity incurred as a result of treatment. This is difficult to accept because these complications occur in the absence of oxygen treatment¹ and hyperbaric oxygen prevents their occurrence when used prophylactically and abolishes those that are present even when used up to 21 days after exposure to carbon monoxide.² Indeed, Dr Howard and colleagues recently stated that "the incidence of neuropsychiatric complications in carbon monoxide intoxication may be reduced by hyperbaric oxygen."³

Dr Howard suggests that xanthine oxidase may be involved in the development of neurological damage because in animals allopurinol and a sulphhydryl based free radical scavenger have been shown to ameliorate oxidative damage during hypoxic reperfusion injury.^{4,5} Dr Howard and colleagues have recently reported a case in which there was an apparent temporal relation between recovery from coma due to carbon monoxide toxicity and the use of allopurinol and N-acetylcysteine.³ If the proposed mechanism of toxicity is correct the same treatment should be effective in any form of postanoxic brain damage and undoubtedly more information is needed about this. However, we cannot recommend the use of allopurinol either alone or in conjunction with N-acetylcysteine for the treatment of carbon monoxide poisoning as a result of experience in one patient since full neurological recovery may occur after even prolonged coma due to carbon monoxide exposure.⁶

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SIR.—In their leading article on carbon monoxide poisoning (9 January, p 78) Drs Tim Meredith and Allister Vale underestimate the frequency of hospital admission with this diagnosis by failing to take into account victims of smoke inhalation.

The Hospital In-Patient Enquiry estimate of 475 admissions to and 10 deaths in hospital in 1985 due to carbon monoxide poisoning is misleading due to the nature of these statistics. The inquiry classifies cases under only their primary diagnosis and thus fire victims suffering concomitant carbon monoxide poisoning are often classified as suffering smoke inhalation or burns and not carbon monoxide poisoning. Fire victims who die from carbon monoxide poisoning will be correctly classified at necropsy since the unnatural mode of death requires a postmortem examination.

About half of early fire fatalities have lethal carboxyhaemoglobin concentrations,¹ while Clarke found that all admissions to hospital with major smoke inhalation had raised carboxyhaemoglobin concentrations.² The Hospital In-Patient Enquiry gives an estimate of 650 discharges and deaths in English hospitals for 1985 with a primary diagnosis of smoke inhalation, and 76 were recorded in Wales. While not all of these fire victims would require aggressive treatment for carbon monoxide poisoning, the degree of carbon monoxide poisoning, as judged by the carboxyhaemoglobin value on admission, has been shown to be an independent predictor of mortality in fire victims once age and percentage burns have been taken into account.³

The major adverse effects of smoke inhalation are often delayed for some hours after exposure⁴ and it is tempting to speculate how treatment for concomitant carbon monoxide poisoning with hyperbaric oxygen, as suggested in your leading article, would affect the prognosis. Indeed, there are many reports supporting this form of treatment but sadly these are all from abroad.⁵⁻⁸

Despite the recent plea by Dr J K Anand and others for independent studies in Britain to assess the cost effectiveness of hyperbaric treatment in carbon monoxide poisoning (6 February, p 432), it is noteworthy that to treat a patient with an intravenous infusion in situ or on ventilatory support requires a chamber capable of accommodating an attendant. No such NHS facility exists south of Newcastle upon Tyne.

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SIR.—The leading article by Drs Tim Meredith and Allister Vale (9 January, p 79) highlighted the mortality and morbidity associated with carbon monoxide poisoning.

In an attempt to identify biological features associated with depressive illness we have been carrying out necropsy studies on brain tissue from suicide victims with a history of depression. Five of the suicide victims died by carbon monoxide poisoning as a result of intentionally inhaling vehicle exhaust fumes (blood carboxyhaemoglobin saturations of 50-80% measured at necropsy). Activity of glutamic acid decarboxylase, the enzyme which catalyses the synthesis of γ -aminobutyric acid (GABA, the major inhibitory neurotransmitter in the mammalian central nervous system), was about 50% lower in the frontal and temporal cortex of these subjects than in that of 16 suicide victims dying by other methods and 20 subjects dying suddenly from accidents or natural causes. The last groups did not differ significantly in glutamic acid decarboxylase activity from each other. A further subject who died by accidental carbon monoxide poisoning as a result of a faulty bathroom water heater also had much reduced glutamic acid decarboxylase activity.

The mechanism of the loss of glutamic acid decarboxylase activity is unclear but was not related to the time between death and tissue storage or to the duration of tissue storage. Terminal illness associated with hypoxia—for example, bronchopneumonia—has been shown to lower glutamic acid decarboxylase activity measured after death.^{1,2} It is uncertain, however, if the brief but profound hypoxia associated with acute carbon monoxide poisoning would produce the same effects. A direct effect of carbon monoxide on glutamic acid decarboxylase activity is a possibility, although we have no direct evidence for this.

Although the present results relate to subjects who died by acute carbon monoxide poisoning, they suggest that sublethal poisoning may result in some loss of glutamic acid decarboxylase activity and hence reduced ability to synthesise γ -aminobutyric acid. Since abnormalities in neurotransmission mediated by γ -aminobutyric acid have been implicated in a number of neurological and psychiatric disorders, reduced synthesis of γ -aminobutyric acid might contribute to the delayed neurological and psychiatric sequelae associated with sublethal carbon monoxide poisoning.

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Newly registered elderly patients

SIR.—The paper by Drs Helen Graham and Brian Livesley on the delay in transferring the records of newly registered elderly patients (30 January, p 340) tells a tale that must be familiar to all general practitioners. It must ring particularly true to any

who look after those in nursing or residential homes for the elderly.

Typically patients will be admitted to such homes at times of crisis, when the availability of medical information may be critically important. There is an additional problem when people are not certain that they will settle in a particular home and prefer to register temporarily with a new doctor at first. When they register permanently the transfer of records is delayed by the time spent as a temporarily registered patient. Meanwhile hospital reports and discharge summaries continue to be sent to the previous doctor.

I care for 21 patients in a nearby nursing home. One was previously registered with my practice, one is temporarily registered (two months), and I am waiting for the records of five (mean 4.2 months so far). For the remainder, who are permanently registered with me, there was a mean interval between registration and receipt of records of 5.1 months (range 2-8 months) with an additional temporary registration period in half of them of 3.7 months. Thus eight months or more can be spent caring for an elderly and often ill patient with no knowledge of his or her medical past. This is despite excellent liaison with the local family practitioner committee. I issue a questionnaire to all new patients, but the information gained is often vague and unhelpful. One patient mentioned problems with her neck and her blood, but not until her notes finally arrived did I appreciate her multiple myeloma. The last note by her previous general practitioner read, "Admit for terminal care."

What can be done about this heroic but dangerous business of treating patients blindfold? The idea of a summary card held by the patient is attractive but probably impractical. Who should have one? When does one become elderly? Why just the elderly anyway? If all patients have one why do they not simply transfer the entire record? While we wait for electronic "credit cards" containing medical information in a decipherable but secure form a simple solution suggests itself. When it is imperative to know about a patient's history a telephone call to the previous practitioner (why are doctors' telephone numbers not on medical cards?) could easily result in the summary card from the patient's notes or at worst a brief résumé of the history being sent directly to the new doctor. This would involve a minimum of time and effort but would at least provide some peace of mind to the legions of blindfolded doctors.

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Psychiatry: private and public provision

SIR,—Dr Greg Wilkinson (9 January, p 79) has failed to appreciate the implications of his own analysis. This may explain the bland tone of his homilies on the "mix" between NHS and private psychiatric care, which he seems to believe can be satisfactorily resolved "by market forces and empirical means."

He is doubtless correct in stating that "people with mental illness descend down the social scale," that insurance risks are so high that only the well off can afford to insure for genuinely comprehensive psychiatric treatment, and that private psychiatry is concentrated on "the outpatient treatment of patients with neurosis." A transfer of consultant commitment towards private practice in psychological medicine must, therefore, mean a shift of medical effort towards the higher social

classes and those with less severe illness. This is "the inverse care law" squared. In this scenario severe psychotic illness would inevitably be neglected in favour of the "walking worried." Furthermore, history teaches us of the risk of an expanded private psychiatric sector bringing with it a burgeoning of dubious and unsupervised therapies. Because of the particular vulnerability of the mentally ill and their families there seems to be an unanswerable case for a divorce between treatment and personal profit.

A further and larger objection lies in the main thrust of psychiatric intervention today. This is at the community level and demands the participation of experienced consultants in assessing patients, supporting their clinical teams, liaising with general practitioners (who are increasingly asked to carry psychiatric responsibilities beyond their formal training), and coping with the complexities of the Mental Health Act 1983. Nor should one forget teaching, research, planning of services, and defending services against the deprivations of budgetary cuts. Even the most ambivalent of psychiatrists is not yet capable of being in two places at the same time. A growing proportion of the working week spent in private clinics must reduce a consultant's availability on NHS wards and risk a decline in the clinical standards on such wards.

We are not enthusiasts for the expansion of private practice in any specialty. The fairness, efficiency, and public acceptance of a properly financed National Health Service seems self evidently preferable. A return to market place psychiatry could be envisaged only by those not engaged in a clinical commitment to society's most underprivileged victims, the mentally ill.

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Viral infection, human papillomavirus DNA, and cervical neoplasia

SIR,—I was surprised that Dr J B Murdoch and others (6 February, p 381) infer from their data a good correlation between histological or cytological diagnosis of wart virus infection and the presence of human papillomavirus deoxyribonucleic acid (DNA) sequences.

Of 34 samples taken from women with either histological or cytological evidence of wart virus infection, 10 had no human papillomavirus DNA, whereas nine of 20 normal samples did have such DNA. That the correlation between histological or cytological diagnosis and the results of DNA hybridisation was better when the samples were taken from colposcopically abnormal areas reflects mainly on the correlation between colposcopic and histological abnormalities.

The increasing frequency with which human papillomavirus 16 DNA is found in normal tissues must surely suggest that the presence of human papillomavirus 16 DNA does not indicate a high risk of subsequent malignancy.^{1,2} Indeed, the data from the matched internal controls in this Glasgow study suggest that the relation between human papillomavirus 16 infection and cervical malignancy is association rather than causation.

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Variability in biothesiometry: an error of technique?

SIR,—Dr Gareth Williams and others (23 January, p 233) may underestimate the reliability of biothesiometry. They show wide variability between both contralateral sites and ipsilateral sites in 25% of non-diabetic and 24% of diabetic patients. It would be interesting to see the variability and scatter for the first readings at each site because, as in many other forms of sensory testing, the "fatigue" of the patient's sensorium is an important factor which may have clouded their results. Dr Williams and his colleagues discard the first 12 readings (three at each site) and then proceed to an additional two cycles of three measurements at each site—another 24. After 36 measurements it is not surprising that such variability is shown.

My own practice is to use one measurement of threshold starting from zero volts at each site, after an initial reference point—remote from the test sites—for example, clavicles or sternum—to familiarise the patient with the sensation and its initial perception. A comparison of standard deviations with this technique and with the multiple tests used by the authors might prove the point. Biothesiometry remains a valuable way of quantifying threshold, but neurologists have long been familiar with the dangers of using a single sensory modality as a "screen" for neuropathies, since impairment may result from root or posterior column lesions. It is a useful way of monitoring change.

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AUTHORS' REPLY.—We share Dr Pearce's concern about the need to strike a compromise between familiarity and "fatigue." We tried to develop a protocol for measuring vibration perception threshold which would allow the subjects to become fully accustomed to an unfamiliar stimulus without becoming bored and losing interest. We performed vibration perception threshold measurements in groups of three at each of the four sites in rotation, with a total of nine determinations at each site. Although this is a relatively large number of observations, the whole procedure took only four to six minutes and would be much less vulnerable to boredom and distraction than the three hour testing procedures used at some centres.¹

We do not think that fatigue was an important contributory factor to the wide variability in vibration perception threshold among different sites, as the values at any given site were confined to a narrow range which showed no tendency to increase as more readings were taken. At the right big toe, for example, the first and last readings differed by >5 voltage units in only six of the 64 non-diabetic and 10 of the 110 diabetic subjects, and in each of these cases the last six values all fell within a range of five units. This does not suggest increasing variability, which would be expected with fatigue or failing concentration; indeed, it highlights the need for several estimations as the first may be unrepresentative. In most cases the final stable value was lower than the first, presumably because some time was necessary to "tune in" to the stimulus, whereas those subjects reporting a lower