the UK? In many countries the cost of alcohol-induced problems exceeds the revenue raised from alcohol taxation.[7]

Let not anyone, even members of Parliament, be surprised that if you give more of a drug you get a greater response. Whatever special aetiological factor—for example, genetic—there may be in alcoholism, this overall dose-response relationship with alcohol and its effects on society are established with a high degree of probability. I do not think that the experiment that the legislators seemed poised to carry out would pass any hospital ethical committee; it's been done before and the risk of a high incidence of adverse effects is too great.

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The North Sea

SIR,—I read with interest your special correspondent's article, "The North Sea" (20 March, p 705), and was impressed by the medical service operated by the eight general practitioners in Yarmouth. In the north-east of Scotland we have a similar set-up, which operates under the aegis of the Institute of Environmental Medicine of Aberdeen University. Unfortunately the institute is not yet as fully operational as it might be and there are still some individual arrangements being made, which tends to fragment and incoordinate medical care. This on occasion leads to duplication of effort and possibly variation in standards.

The NHS does not yet recognise a responsibility below the low-water line, but a recent court decision in Scotland (Crown Estates *v* Fairlie Boatyard) may help to change this situation. One of the problems of treating personnel on rigs and barges is the question of insurance, both accident and medicolegal, of doctors who supply services to off-shore installations, and this might be eased if the NHS were to become responsible for off-shore medical care, as indeed do police forces in their own fields.

I would agree that a national system of monitoring the health of divers should be instituted, especially as the number of divers is relatively small, and I hope that Admiral Rawlins's committee will take up this point. Funds should be made available, preferably from Government sources, for research into the long-term effects of compression, which would include hyperbaric facilities, computerisation, etc. A national register of divers is a must, as it is not unknown that a diver who has failed a medical with one company seeks to be employed by another company and may conceal relevant medical history.

In the article divers are described as "mavericks" and elsewhere as "prima donnas," but I feel that it should be the diver's own responsibility to ensure that his medical checks are up to date and that he holds a valid certificate of medical fitness. This responsibility already applies in the case of commercial airline pilots and heavy goods vehicle drivers. The medical records of the former are held and scrutinised centrally, and it takes the examining doctor only a few minutes by telephone to get a full breakdown on past medical history when required.

In the article it is stated that necropsies revealed some degree of right ventricular hypertrophy in otherwise healthy men. An impression I obtained, admittedly from the records of a small number of divers, was that the diastolic pressure was over 80 mm Hg more frequently than I would have expected in a similar number of men in the 20-35-year age group.

The profession should seize this opportunity of serving and investigating this new field of off-shore medicine.

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Renal lesions in a case of septicaemia

SIR,—In replying to the criticisms of Wing Commander T J Betteridge and Squadron Leader D J Rainford (28 February, p 522) I should like to make two preliminary points. Firstly, as many readers will be aware, the published accounts of clinicopathological conferences are edited versions of the proceedings. Some descriptive details are inevitably omitted, while the original presentation itself has to be a concise, didactic account of the pathological findings. Secondly, although histological diagnosis is a matter of subjective interpretation, our opinion is based on the examination of numerous tissue sections and not on two photographs.

The glomerular changes in the case presented were not focal. There was a diffuse increase in cellularity affecting all glomeruli apart from those hyalinised by previous ischaemic damage. While minor variations in the degree of hypercellularity were noted, there was little or no tendency for local or segmental variation within individual glomeruli. A measure of the degree of proliferation is given by total glomerular cell counts. The cells (excluding leucocytes) present in 20 midline sections of glomeruli in 1-μm sections stained by the periodic acid-Schiff method were counted. The mean cell count ($\pm$1 SD) was 194$\pm$18, which compares with a normal count of 122 for adult females.[1] The hypercellularity was principally a result of endothelial cell proliferation, but there was a definite increase in mesangial cells. Neutrophil polymorphs were present in excessive numbers throughout the glomeruli. Fibrin thrombi and foci of necrosis were not seen.

A diffuse, mild to moderate increase in mesangial matrix and a slight irregular thickening of tuft loops were present. While electron microscopy did not reveal convincing sub-epithelial "humps," it did confirm the presence of finely granular, electron-dense deposits of matrix or basement membrane-like material in the mesangium. It is unfortunate that tissue was not retained for immunofluorescence studies, but this deficiency does not preclude an accurate diagnosis; indeed, the results obtained from necropsy material may well be unsatisfactory.

Our diagnosis of diffuse proliferative glomerulonephritis in a patient with staphylococcal septicaemia and acute bacterial endocarditis of short duration is not "most unusual." Powell found this form of glomerulonephritis in 16 of 40 patients dying from staphylococcal septicaemia,[2] and its association with coagulase-negative staphylococcal bacteriaemia complicating the insertion of ventriculoatrial shunts for hydrocephalus has been clearly established.[3-5]

Most authorities recognise two forms of glomerulonephritis in bacterial endocarditis. Firstly, a focal type ("embolic" nephritis), characterised by segmental fibrinoid necrosis with local proliferation of cells infrequently associated with impaired renal function; and, secondly, a diffuse proliferative lesion, in which elevation of blood urea nitrogen and creatinine levels is common. Heptinstall claims that the focal form is very uncommon in cases of short duration irrespective of the organism.[6] Diffuse proliferative glomerulonephritis complicating coagulase-positive staphylococcal endocarditis has been well documented[6-8] and has been substantiated by animal experiments.[9]

At a clinicopathological conference published in 1960[10] a 52-year-old man was presented who developed staphylococcal septicaemia following a laminectomy and died in renal failure. Post-mortem examination revealed acute bacterial endocarditis involving the tricuspid valve and a diffuse proliferative glomerulonephritis. Our case represents a further example of this interesting but not infrequent complication.

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Sociological realities

SIR,—In your leading article under this heading (3 April, p 790) you ask, "Why then is so little being done to halt the decay and decline of the links between the health and social services?" At a recent half-day meeting in this division between social workers and general practitioners it was striking that there was a much more positive wish to do this among the former than among the latter. It came over strongly that mutual expectations were in conflict and sometimes unreal. The GPs felt that the ideal social worker should be more active and of practical help (like GPs) and the social workers felt the ideal GP should find time to be understanding, tolerant, and willing to listen (characteristics which they value highly in themselves).

This contrast in attitudes is borne out in your article, which fails to recognize the complexity of the roles of the social services in the community. These roles are still in the process of definition, and if doctors wish to enter the debate they will have to find time to sit around and discuss with social workers their differing roles and attitudes as well as the practicalities

of the job in hand. Perhaps, if this is done, true links will be forged so that the inevitable changes we are facing can be tackled in a more positive way.

Incidentally, we do not think that any writer in the *BMJ* should scoff at any other publication for using "language at times incomprehensible to the reader."

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SIR,—The hostility of your leading article (3 April, p 790) to the discussion paper on social work training (3 April, p 790) is likely to widen the gulf between our two professions rather than narrow it. A main aim of social work, as the paper makes clear, is the "sustaining of personality," which needs a longer-term perspective on clients and their problems than undergraduate medical education has ever bothered to conceptualise, let alone teach, any more than it has ever taught "the disciplined use of the self" (a concept whose significance you evidently fail to grasp).

Social workers can get little job satisfaction out of arranging case conferences when doctors involved in the case either do not attend, come late and leave early, or merely state their own viewpoint without listening adequately to the contributions of other people present. Like doctors, they are prone to find it easier to do work than to talk about it with others involved in the same task, but (unlike doctors) they are at least trained to realise that it is not always better to do so. In medicine we no longer consult about patients; we merely refer them to our colleagues.

Where social workers are "strangers to their medical colleagues" it is more the fault of the latter than the former, since it is medical men who claim always to be the leaders of caring teams, primary or other. In the present climate it is more important for the social work profession to confront the medical profession and stand its ground than to seek to co-operate with it; for that could only mean capitulation to its authoritarian but myopic insistence on the eradication of short-term problems while ignoring clients' needs for the long-term fostering of whatever personality strengths they may possess.

Finally, for a medical journal such as yours to criticise the discussion paper for the incomprehensibility of its language implies so gross a case of the pot calling the kettle black as to leave me (almost) speechless.

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Uterine hypertonus after induction of labour with prostaglandin E_2 tablets

SIR,—Dr J E Felmingham and his colleagues (6 March, p 586), commenting upon a case of uterine hypertonus (not defined by the authors) witnessed during labour induction with oral prostaglandin E_2 (PGE_2), may give the impression that this complication had not been reported previously. Admittedly, when PGE_2 is given by mouth, even in relatively high doses (up to 3 mg) at quite short intervals (30-120 min), the risk of hyperstimulating the myometrium is smaller than when the drug is infused intravenously. Vomiting generally supervenes as soon as effective doses are exceeded, and this seems to provide the oral route with a built-in safety valve.[1] This view is further strengthened by intrauterine pressure data (unpublished) collated during elective induction of labour at term; with oral PGE_2 mean uterine activity was comparable to that of spontaneous and surgically induced labour and was significantly less than when PGE_2 was infused by vein.

Nevertheless, investigators using intrauterine tocography have reported an overall incidence of uterine hypertonus during oral PGE_2 administration of somewhat less than 1%.[2] It must be stressed that authors have used divergent definitions for hypertonus and that this figure must therefore be taken as an approximation. In two of our own cases (2%) a transient elevation of basal uterine tone to more than 12 mm Hg was registered after a single dose of 0·5mg PGE_2 had been given. One of these episodes caused fetal bradycardia.[3] Even severe hypertonus (760 mm Hg) with marked fetal bradycardia (60/min) can follow the oral administration of small doses of PGE_2 as shown by Fraser.[4] Lauersen and Wilson,[5] having observed myometrial hyperstimulation after 1 mg PGE_2 was given orally, went so far as to recommend the use of an initial dose of 0·25 mg in selected patients to allow proper assessment of uterine sensitivity to the drug.

In other words, although relatively uncommon, uterine hyperstimulation during induction of labour with oral PGE_2 has been observed before. Hypertonus is a potential complication of parturition whether an oxytocic is used or not. It follows that, whenever feasible, labour should be monitored by electronic means.

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Effect of posture on dental anaesthetic mortality

SIR,—On a previous occasion we commented on the difficulty of drawing valid conclusions from incomplete data of dental anaesthetic mortality.[1] In spite of this difficulty we have been studying for some years the details of deaths associated with dental treatment with the help of the Registrar General's office and many coroners. At the moment our studies are incomplete and thus not ready for publication, but one point has emerged which seems of sufficient topical importance to justify an early comment. Its significance will be obvious from the conflicting and in some respects ignorant views expressed in the press and reported in the *British Dental Journal*[2] following a recent death in the dental chair. The point may also release practitioners from unjustifiable pressures to use the supine position for general anaesthesia irrespective of the dictates of their own clinical judgment in any particular situation.

In 1974, the latest year for which the Registrar General's returns are available, there were 17 deaths associated with dental treatment and 13 of them were classified as connected with anaesthesia. Of these, nine were in ambulant patients; six of the nine were in dental surgeries, one was in a dental centre, and the other two were hospital outpatients. The posture of the patients during the anaesthetic was reported to the coroners as follows:

	Supine	Sitting	Not stated
Dental surgery ..	5	1	—
Dental centre ..	—	—	1
Hospital outpatients	1	—	1

The relationship of six supine to one sitting is a complete reversal of previous observations. For instance, in 1971, of eight ambulant dental anaesthetic deaths, one patient was anaesthetised in the supine position, five were sitting, and in two cases the posture was not stated. The 1974 findings may have three possible interpretations. Firstly, the observation may be an isolated "freak" of no special significance. Secondly, practitioners may have become sensitive to criticism and failed to report the facts accurately. This seems unlikely, since the campaign to make it obligatory to use the supine posture has been conducted for many years[3] and there was to our knowledge no special change of circumstances to account for a sudden change of attitude or accuracy of reporting in 1974. Finally, there is the possibility that the supine campaign is being successful and that the dramatic reversal of the postural mortality trend represents a genuine tendency for more practitioners to anaesthetise their patients in the supine position. If indeed this is, even in part, the true explanation then there will continue to be most deaths in that posture which is most commonly employed. Thus Tomlin's comment in 1974[4] that the posture is seldom if ever causally related to the anaesthetic death would appear to be vindicated and the 1974 data may indicate a conversion of dental anaesthetists to the use of the supine position for their patients.

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Pseudomonas aeruginosa in hospital pharmacies

SIR,—The study by Dr Rosamund M Baird and others (28 February, p 511) demonstrates that hospital pharmacies are often contaminated with *Pseudomonas aeruginosa*, but no attempt is made to relate this to individual cases of hospital infection. I would like to report briefly an outbreak of *Ps aeruginosa*