poration in Wellington. The information they review should properly have been presented in March 1988 to the International Rugby Football Board as part of a balanced review comparing the New Zealand law modifications (minimising the duration and mobility of the scrum) in their junior rugby with the law changes introduced world wide two seasons ago affecting the formation and posture of the front rows of the scrum. These latter changes in the laws apply to junior and senior New Zealand rugby. They do not state whether there have been any serious neck injuries in senior rugby in New Zealand during the period on which they report. The application of an Australian court judgment on rugby league to rugby union seems illogical, and we are in no position to comment on either rugby league or the law.

The International Rugby Football Board and its member unions are acutely aware of the problems of serious neck injuries and have, through their medical committees, collected detailed information on the frequency and cause of these injuries and have made recommendations about prevention. As a result law changes have been introduced and their benefits continue to be analysed and some of them have been reported by Drs Burry and Calcinaï. Detailed advice about the choice of players for the front row of the scrum has been issued to all rugby coaches in conjunction with specific advice on neck and shoulder girdle strengthening exercises.

The medical profession and medical advisory committees working with national rugby football unions and the International Rugby Football Board have made major advances in recognising, preventing, and managing serious injuries in rugby. This process will continue only if the messages from our profession to rugby administrators are presented in a clear and balanced manner, reviewing all relevant factors and eliminating extraneous material.

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AUTHORS' REPLY.—We wrote not as members of the Accident Compensation Corporation (which Mr Calcinaï is most certainly not), nor as members of the medical advisory committee of the New Zealand Rugby Football Union, but as concerned private individuals.

Messrs Macleod and Pattisson are well aware that these facts were presented at the 1986 medical advisory meeting of the International Rugby Football Board. The updated information has been published and we expect that the matter will be discussed again at the 1988 meeting. We can only hope that the response on that occasion will be more positive.

Since the article was written a senior New Zealand provincial player, while playing under current international laws, fractured his cervical spine as a scrum formed. At least one similar injury occurred in Australia in 1987. It is reasonable to conclude that the current international laws are still not adequate from a safety viewpoint. Despite your correspondents' assertion to the contrary, no reliable epidemiological data are available on cervical spinal cord injury other than the New Zealand survey to estimate the true magnitude of the problem. Our survey has detected only the most severe injuries, those of temporary or permanent cord damage. We have no knowledge of how many front row forwards damage their cervical spines without cord damage but none the

less suffer severe symptoms and disability in later life.

It is true that in some countries advice on choice and conditioning of front row players has been disseminated. This is laudable but is clearly insufficient to prevent injury if the activity is inherently unacceptably hazardous.

The appropriateness of extrapolating a legal principle and precedent from one sporting code to another seems so obvious that no further comment or justification is required.

Finally, it is difficult to escape the conclusion that Messrs Macleod and Pattisson believe that the editor of the *British Medical Journal* has elected to print a paper which contained unclear, unbalanced, and extraneous material. We can only feel grateful that the editor took a different view.

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Gas exocrine "failure" in the critically ill

SIR.—In their paper on gastric exocrine failure in the critically ill (16 January, p 155) Mr V A Stannard and others observed a high gastric pH in the absence of H_2 antagonists yet do not comment on the method used to measure pH.

We are investigating the relation between gastric pH and the bacterial content of gastric aspirate using a miniaturised pH probe. Near continuous recording—sampling at 16 second intervals—shows a fluctuation of up to 2.3 pH units from the baseline. This change in pH takes only a few minutes in some patients. From our experience pH in two hourly collections of gastric aspirate may correlate with mean probe pH during the entire period of fluid collection, but it is a poor reflection of instantaneous probe pH. Our impression is that gastrointestinal motility may help to explain both short and long term fluctuations in pH, a possibility which is now being explored.

Other authors have described an apparent association between the use of H_2 antagonists and subsequent colonisation of the ventilated airway,¹ where stomach contents may act as a bacterial reservoir. The substitution of H_2 antagonists with sucralfate, an antiulcer agent which has no significant effect on gastric pH, has been shown to reduce airway colonisation and ventilator associated pneumonia.²

If the role of hypotension is confirmed in the pathogenesis of "stress" ulcers the use of alternative prophylactic treatments that do not disrupt the antibacterial effect of gastric secretions may result in a decreased incidence of ventilator associated pneumonia.

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Cost of prophylaxis in joint replacement

SIR.—The article by Mr Ulf Persson and others (9 January, p 99) on the economics of prophylaxis against infection in total hip replacement indicated a rational financial basis on which measures may be selected and strengthens the case for the wider

adoption of surgical enclosures. Despite long-standing evidence for their benefits we found in a survey of orthopaedic surgeons in Scotland¹ that only 19% had access to ultraclean air facilities, which had been provided in only three centres.

In their use of systemic antibiotics, although most surgeons employed prophylactic regimens for primary arthroplasties which would be expected to be effective, the chosen duration and timing were often more appropriate to a treatment policy than to prophylaxis (34% started treatment before the day of surgery and 38% continued for longer than 24 hours postoperatively). Longer periods of administration are no more effective than short term cover and clearly increase the chances of side effects as well as being more costly.^{2,3} We found that "prophylactic" costs ranged from £5 to £22.50. Over two thirds of the systemic antibiotic regimens in use could be made more cost effective without loss of efficacy by more accurate timing and limiting the duration of administration.

In 1983 we found six revisions for deep infection and 18 for mechanical loosening in a year when 216 primary hip replacements were performed. We have also noted a progressive rise in the number of revision arthroplasties from 10 per year in 1970-4 to 22 per year in 1981-5. As the pool of primary arthroplasties enlarges the number coming to revision will inevitably increase.

A previous assessment of the cost of a primary hip arthroplasty indicated that 90% of the £2427 total was due to the cost of hospitalisation.⁴ The revision joint replacements in the same period entailed more than double the length of hospital stay and increased costs with, for example, systemic antibiotic costs from £8.70 to £80 per patient. Thus our estimated cost for a revision is of a similar magnitude to that of Mr Persson and colleagues, and their conclusions would seem as valid in our area as in Sweden.

The case for greater provision of surgical enclosures should be put to those responsible for deciding priorities in health spending, and surgeons should review their antibiotic policies.

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False negative colposcopic cervical biopsy

SIR.—We were surprised to read of the high number of false negative results of colposcopically directed cervical biopsies recorded by Dr Paul Byrne and others (16 January, p 172). Presumably all were from women with abnormal cytological findings and a colposcopically visible lesion. Although we are not given the total number of cases from which the 132 reported arise, it is assumed to be important enough to warrant reporting.

Several explanations for these results were considered. We suspect that another possibility is that the reporting pathologist either does not recognise or does not think it necessary to report the sometimes subtle changes of minimal change human papillomavirus infection.^{1,4} In our unit, with 3500 colposcopically directed cervical biopsies

performed each year, a review of the last 330 reports showed 34.9% with "human papillomavirus only" or "human papillomavirus plus immature squamous metaplasia" and another 6.7% with the physiologically normal "immature squamous metaplasia only." Only 4.6% were reported as entirely normal. It is important that these findings are reported as human papillomavirus infection alone can cause cytological atypia.

Furthermore, both human papillomavirus and immature squamous metaplasia can be visualised on colposcopy² and can be difficult to distinguish from cervical intraepithelial neoplasia. The colposcopist requires a histological explanation for the lesion he sees; without this he cannot make a proper decision on the further management of the patient. In our own unit if the cytological abnormality persists and worsens after a normal colposcopic biopsy then a laser cone biopsy is performed to provide a definitive diagnosis.

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- 1 Meisels A, Morin C, Casas-Cordero M. Human papilloma virus infection of the uterine cervix. *Int J Gynecol Pathol* 1982;1: 75-94.
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Providing donor organs for transplantation

SIR,—Now we—and the 7.4 million other people who watched the BBC's *That's Life* programme (16 January, p 202)—know why there are too few organs for transplantation; at last the accusing finger has been pointed at the intensive care unit. The excuses bandied about for years—not enough potential donors, too few carrying donor cards, relatives or coroners refusing permission—can be set aside. Failure occurred because the intensive care staff did not seek permission or asked too late.

From 1970 to 1983 I administered a donor system in a district general hospital, and the methods worked. An analysis of a four and a half year period showed that 34 kidneys were donated from a single intensive care unit.¹ If intensive care units in all the district general hospitals in the United Kingdom did the same then the result would be a surplus of donor organs. Our recipe for success depended on the admission of a potential donor to the intensive care unit at the right time. This was effected by admission policies covering all the conditions related to a general intensive care unit and by ensuring that these policies were strictly enforced. Hence patients with spontaneous subarachnoid haemorrhage aged 16 to 50 years were admitted immediately after a diagnosis whether or not they needed specialist treatment. The same applied to unconscious patients with head injury who did not require neurosurgical care. The potential donor was in the right place should apnoea ensue, when gas exchange and the circulation could readily be maintained.¹

This intensive care unit was administered clinically by one consultant physician, and later two, who, with the nurses and hospital chaplains, established a good relationship with relatives; refusal for organ donation was exceptional. The integration of intensive care with the other divisions was facilitated by the fact that the medical division was responsible for resuscitation, intensive care, and major trauma. The correct attitudes to organ donation were developed in 1968 and this was helped by the practice of dialysis, used to treat both acute and chronic renal failure. Responsibility for a few patients with end stage renal failure was a stimulus to contribute to the transplant team, and this was supported by systematic teaching and clinical research. Brain death, the care of the potential donor, and renal failure were all part of the teaching programme of the intensive care unit.²

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Communication of drug treatment to general practitioners

SIR,—There is a simple solution to the problems encountered by Drs J A Wilson and others in communicating with general practitioners about their patients' drug treatment on discharge (16 January, p 211), and that is to give the patient a sensible supply of drugs.

To give a three day supply combines the worst of nearly all worlds. It ensures that all patients discharged on Wednesdays and Thursdays run out of their treatment over a weekend and that those discharged on Fridays will present in a panic for prescriptions on Monday, a day when even general practitioners are a little busy. The instruction to the patient to contact his general practitioner "as soon as possible" can only compound the number of pseudo-urgent doctor-patient contacts, often leading to unnecessary visits in the elderly.

In east Dorset it is generally accepted that a two week supply of drugs is a reasonable compromise between cost, compliance, and communication and that to expect a patient to visit a doctor (or vice versa) soon after discharge merely to obtain prescriptions is expensive and inefficient, as well as plain silly. This policy was endorsed by Baroness Trumpington when she was junior health minister.

The whole problem is merely another side effect of the current muddle over money in the NHS, and we should not allow artificial budgetary barriers to deflect us from our prime function, which is to treat patients in the most reasonable way possible.

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New year message

SIR,—Three cheers for Mrs Sonia Alexander (16 January, p 209). That a health authority chairman has to highlight the intellectual bankruptcy of the NHS is an indictment of a medical profession dedicated (as always) to maintaining the status quo.

Setting up a health commission, as suggested by Dr Tony Smith (2 January, p 1), seems to me a gesture of despair; it will take several years to report and there is no guarantee that its recommendations will be acted on. For example, none of the proposals made by the four commissions

between 1965 and 1980 into restructuring medical training have been adopted.

Much needed radical solutions, of which there are many, will not be popular. One in particular should be mentioned because I suspect Mrs Alexander may have been inhibited from doing so. Doctors' salaries are an expensive item in the hospital budget; additional costs are generated by what doctors do. Urgent answers are needed to several questions. How much do the many demands made on consultants, especially as they become more senior—teaching, committees, examining and other work for their professional bodies, conferences, giving expert advice, etc—compete with direct care of patients? If this was the only criterion (and I am not necessarily suggesting that it should be) there might well be too many doctors.

How much clinical work that is currently done by hospital doctors could be transferred to other health professionals? It is acknowledged that many hospital patients could be looked after in general practice, and progressive family doctors would welcome this. The pressure on hospital outpatient departments might be substantially relieved if consultants could be persuaded to look at present practice. Liaison sisters, nurse practitioners, and specialist nurses are slowly taking on jobs traditionally the province of doctors: they could be rewarded with proper status and salaries and still save the NHS money. Similarly, the employment of properly paid secretaries in every unit would rapidly increase efficiency—and probably reduce the number of doctors needed. We should not be frightened to look also at the use of technical staff in traditional medical areas.

The profession rightly believes that it should be allowed to regulate its own clinical practices. It should demonstrate by action that it is capable of doing so; otherwise it has only itself to blame if external pressure forces the pace for change.

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Hyperbaric oxygen, carbon monoxide, and cerebral oedema

SIR,—The leading article by Drs Tim Meredith and Allister Vale (9 January, p 77) is a timely reminder of the mortality from and morbidity of carbon monoxide poisoning and smoke inhalation. In 1931 Putnam *et al* showed that carbon monoxide (in common with microembolism and tetanus toxin) damages the blood-brain barrier,¹ especially in the cerebral white matter, where the venous drainage predisposes to focal oedema.² This may lead to a leucoencephalopathy, necrosis of the basal ganglia, or, in less severe cases, demyelinating plaque formation.³ These are probably the cause of the neuropsychiatric sequelae of carbon monoxide poisoning.

Delayed deterioration is well documented and occurs when the carboxyhaemoglobin concentrations are negligible. It may follow severe hypoxia from any cause⁴ and is likely to be due to the development of oedema from the blood-brain barrier damage, because if the dementia was due to neuronal death it would be evident immediately. In our clinical experience hyperbaric oxygen therapy may need to be repeated, suggesting that the oedema recurs. One patient, who inhaled gas directly from an exhaust pipe, needed several prolonged sessions of hyperbaric oxygen therapy before he regained consciousness and further treatment over two weeks because he continued to relapse after therapy. He finally made a remarkable recovery. At the other end of the