servative treatment is therefore advised, whereas unimpacted—that is, dislocated—fractures need internal fixation—that is, surgery. Therefore, according to the principle of intention to treat, 22 patients with impacted fractures were excluded from the study, while 42 patients with dislocated fractures were included, although they were treated conservatively.

The reason for conservative treatment of the 42 patients was in many cases recent myocardial infarction, whose importance was the subject of our study. This, however, could be evaluated only by following the intention to treat principle.

The diagnostic criteria for myocardial infarction were those generally accepted.¹

It is not a previous myocardial infarction as such but a recent myocardial infarction—that is, within six months—that is generally held to be a serious risk factor.^{2,3} The relevant control group is composed of patients with similar surgical disease and a myocardial infarction more than six months before potential surgery.

We believe that our design is good for estimating the surgical risk of a recent myocardial infarction, being in many ways closely comparable with a randomised controlled clinical trial. A randomised controlled clinical trial would obviously be unacceptable for ethical reasons.

We agree that our mortality figures could have been put into perspective by comparison with a population matched for age and sex. We think, however, that mortalities between 9 and 16% a month speak for themselves.

It concerns us that Drs Herrema and Milligan are concerned about our conclusions. According to our results, complex scores such as the Goldman index⁴ no longer need to be used to estimate patients' surgical risk. The only information now necessary about patients with previous or recent myocardial infarction is left ventricular function.

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Anticoagulants in venous thromboembolism

SIR,—We do not wish your readers to be misled by the advice of Dr A Fennerty and colleagues that "intravenous infusion is the best way to give heparin" in managing venous thromboembolism.¹

Five randomised controlled trials have compared continuous intravenous heparin infusion with twice daily subcutaneous heparin in treating phlebographically confirmed deep vein thrombosis (table I). Two of these studies have shown subcutaneous heparin to be superior to intravenous heparin and two have shown no significant differences in efficacy. The only study showing an advantage for intravenous heparin⁴ was seriously flawed,⁵ and this has been widely acknowledged subsequently (J Hirsh, personal communication). Firstly, most recurrent thromboembolic events in this trial occurred well after the withdrawal of heparin, when patients were being treated with warfarin by their referring physicians. Secondly,

two thirds of the patients in the subcutaneous group received insufficient heparin to adjust their activated partial thromboplastin time adequately. Thirdly, the two groups were not matched for asymptomatic pulmonary embolic events on entry to the study, and subsequent symptoms may not necessarily have been related to fresh events.

TABLE I—Comparison of subcutaneous versus intravenous heparin in treatment of deep vein thrombosis in randomised controlled trials

Source	No of patients	Deep vein thrombosis (calf: proximal) (%)	Comparison subcutaneous v intravenous heparin
Bentley <i>et al</i> 1980 ²	100	100:0	Significantly better
Andersson <i>et al</i> 1982 ¹	141	17:83	No significant difference
Hull <i>et al</i> 1986 ⁴	115	0:100	Significantly worse
Doyle <i>et al</i> 1987 ⁵	103	32:68	No significant difference
Walker <i>et al</i> 1987 ³	96	44:56	Significantly better

We have recently completed another study comparing subcutaneous heparin and continuous intravenous heparin. Sixty five patients with phlebographically confirmed deep vein thromboses, 57% of which were proximal, were treated with heparin (250 U/kg/12 h) for six days by either twice daily subcutaneous injection or a continuous intravenous infusion, both adjusted to maintain the kaolin cephalin clotting time at 1.5-2.5 times the normal value. Patients were reassessed at the end of treatment by repeat phlebography. Subcutaneous heparin proved significantly better than intravenous heparin (table II), and the mean daily doses were almost identical (31 970 IU and 31 900 IU, respectively). No major haemorrhagic complications or clinical pulmonary emboli were noted. Six patients in the subcutaneous group experienced injection site bruising, and eight patients in the intravenous group suffered extravasation or thrombophlebitis.

TABLE II—Comparison of subcutaneous versus intravenous heparin in treatment of deep vein thrombosis

	Subcutaneous (n=32)	Intravenous (n=33)
Size of thrombus:		
Decreased	20	15
No change	11	10
Increased	1	8

$\chi^2=6.42$, $df=2$, $p<0.05$.

Intermittent subcutaneous heparin is far easier to administer than continuous intravenous heparin; is not complicated by the problems of mechanical pump failure, extravasation, or thrombophlebitis; and reduces the burden on nursing staff as the ambulant patient is not restricted by the pump. Furthermore, as Fennerty *et al* have previously shown, outside clinical trials continuous intravenous heparin infusion is poorly controlled by resident medical staff.⁶ Subcutaneous heparin therefore is simpler and at least as effective as continuous intravenous heparin in treating deep vein thrombosis.

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AUTHORS' REPLY.—Although stating that intravenous infusion is the best way to give heparin, particularly in comparison with an intermittent intravenous bolus dose, we did agree that subcutaneous administration may prove to be a satisfactory alternative. So far, however, no ideal comparisons have been made between subcutaneous and intravenous administration. In the study of Doyle *et al* significantly better anticoagulation was achieved in the group receiving subcutaneous heparin than in that receiving intravenous heparin, the discrepancy being greatest in the crucial first three days of treatment.¹ Most patients receiving intravenous heparin by infusion did not reach the authors' lowest acceptable activated partial thromboplastin time until the third or fourth day.² Similar criticisms have been made about the work of Walker *et al*.^{3,4}

The data of Dr Stringer and his colleagues are encouraging, although they do not indicate whether anticoagulant control (as distinct from heparin dose) was equal in the two groups. Our view is that an adequate dose of heparin is crucial, particularly in the first few days after a thromboembolic episode, whatever the route of administration. The choice of intravenous infusion or intermittent subcutaneous administration may depend on the clinical situation, but making sure enough is given—that is, by regular monitoring—is probably what matters most.

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Hours of work, disasters, and safety

SIR,—There have recently been three major tragedies in public transport. In each case the government has rightly praised the work of the emergency services, including hospital staff.

Last year the working party on the management of patients with major injuries commissioned a retrospective study of 1000 deaths from injury.¹ They concluded that 170 of these were from preventable causes, many of them occurring be-

cause of deficiencies of treatment in the "golden hour"—the first hour in hospital.²

We start 1989 reading an article that confirms scientifically what most doctors know from personal experience: "Continuous working may adversely affect the cognitive function and mood to the detriment of the welfare of doctors and their patients."³

The latest disaster occurred on a Sunday night, when the junior medical staff in the receiving hospitals had been on duty continuously for 37 hours (or 61 hours if they worked a three day weekend)—longer than the period in the study by Drs Orton and Gruzelier of performance of house officers after night duty. These staff would bear the brunt of the responsibility for management in the golden hour.

When the government is deciding its course of action on Lord Rea's bill to reduce doctors' hours it should perhaps consider whether there is any relation between these two studies. But if hours are reduced this should be followed by increased staffing levels, not by more cross cover between specialties, resulting in fewer doctors on site at night and weekends. This would remove the safety net providing the immediate availability of junior medical staff in case of a major disaster.

Two weeks ago the pilot of the aircraft missed a village because of a combination of skill, quick clear thinking, and bravery. He is not allowed to work more than 18 hours continuously. The doctors treating the injured all had to make crucial decisions beyond this time limit.

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Dentistry and total oral health

SIR,—In his Personal View Dr H A Dewar considers that dental examination should not be necessary on a regular basis.¹ We suggest that this does need to be performed regularly. Dentistry has moved on from the days of treating just caries and periodontal disease to participation in total oral health, including the oral manifestations of systemic disease—as indeed is evident from various papers in the *BMJ*.^{2,3}

To teach medical students to examine the mouth and associated tissues correctly would surely add more burden to the already heavy programme of undergraduate medical training, and as a consequence other aspects of training could suffer.

Nevertheless, we do agree that medical students could and should be taught more about oral

examination and disease; indeed, at some schools they receive no teaching in this. Comparatively few medical graduates have any experience in oral diagnosis and this is particularly highlighted when the amount of material on oral diagnosis in medical textbooks is considered (table). Some texts contain no material at all on oral or dental matters, while in those readily available texts surveyed the maximum percentage component was only 1.6%. Although oral carcinoma is one of the most prevalent tumours world wide and is now increasing in Western societies,⁴ this is not reflected in these authoritative texts and neither are many of the vast array of other diseases that can affect the oral cavity.⁶

It is also suggested that doctors would specialise in dentistry. Many dentists do already become qualified in medicine, returning back to dentistry⁷ in one form or another as a dental specialist. So perhaps this might not change as radically as Dr Dewar envisages.

Finally, we are sure he could imagine the public outcry if the medical profession was to discourage visits to a general practitioner for cervical screening tests and possible detection of early dysplastic lesions. Indeed, there is already an inordinate delay in diagnosis of oral cancer.⁹ The medical profession should encourage the public to attend for regular oral examination by a person trained in oral diagnosis in the hope of early detection of carcinomas. Or could it be that it is much more politically acceptable to provide finances for more "important" or "glamorous" aspects of medical care than care of the mouth and associated tissues?

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Thrombolytic drugs in myocardial infarction

SIR,—In the discussion of their paper on the European Cooperative Study Group's trial of tissue

plasminogen activator in acute myocardial infarction Drs Frans Van de Werf and Alfred E Arnold state: "Mortality after treatment with plasminogen activator in this trial . . . is the lowest reported so far in large thrombolysis trials."¹

Although correct in its context, this statement is liable to be seriously misinterpreted to imply that plasminogen activator will save more lives than other thrombolytic drugs. This cannot have been the authors' intended meaning as their study did not compare thrombolytic agents, and the fewer deaths in the group treated with plasminogen activator scarcely reached significance relative to the group receiving no thrombolytic treatment at all (0.1>p>0.05 at 14 days, 0.2>p>0.1 at three months). Because the statement quoted has already been widely cited by proponents of plas-

Comparison of effect of thrombolytic agents on mortality from myocardial infarction. Point estimates of effect and 95% confidence intervals for most comparable published analysis in each trial: (1) streptokinase v placebo given 0-4 hours after onset of symptoms, 35 day survival; (2) streptokinase and aspirin v placebo given 0-4 hours after onset, 35 day survival; (3) anistreplase v placebo given within six hours after onset, one month survival; (4) tissue plasminogen activator v placebo given within five hours after onset, one month survival; (5) tissue plasminogen activator v placebo given within five hours after onset, three month survival.

minogen activator it should be explicitly stated that there is currently no evidence from trials that any thrombolytic agent is superior in reducing mortality after myocardial infarction.

Drug and therapeutics committees are now being asked to formulate policies on the use of thrombolytic agents; it is therefore timely to compare the best available mortality data on streptokinase, tissue plasminogen activator, and anistreplase (figure). Each of these drugs improves survival.

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Amount of material on oral diagnosis in selected medical textbooks

Book	Author or editor	Publisher	Year	Edition	Total No of pages of text	No of pages on oral or dental matters	Oral component (% of whole)
<i>Modern Medicine</i>	Read A, Barritt DW, Langton Hewer R	Churchill Livingstone	1984	3rd	689	0	0
<i>Oxford Handbook of Clinical Medicine</i>	Hope RA, Longmore JM	Oxford University Press	1987	1st	698	0	0
<i>Essential Clinical Medicine</i>	Salper RH	John Wright	1984	1st	509	0	0
<i>Short Textbook of Medicine</i>	Houston JC, Joiner CL, Trounce JR	Hodder and Stoughton	1985	8th	784	1	0.1
<i>The Principles and Practice of Medicine</i>	Harvey AM, et al	Appleton-Century-Crofts	1984	21st	1531	2	0.1
<i>Cecil Textbook of Medicine</i>	Wyngaarden JB, Smith LH	W B Saunders	1988	18th	2404	6	0.3
<i>Harrison's Principles of Internal Medicine</i>	Thorn GW, et al	McGraw-Hill Blakeston	1977	8th	2088	6	0.3
<i>Davidson's Principles and Practice of Medicine</i>	Macleod J, et al	Churchill Livingstone	1987	15th	820	3	0.4
<i>Oxford Textbook of Medicine</i>	Weatherall DJ, Ledingham JGG, Warrell DA	Oxford University Press	1983	1st	2739	14	0.5
<i>Current Medical Diagnosis and Treatment 1987</i>	Krupp M, Schroeder S, Tierney LM	Appleton and Large	1987		1114	6	0.5
<i>Essential Medicine</i>	Brackenridge RG	MTP Press	1979	2nd	422	3	0.7
<i>A Primer of Medicine</i>	Papworth MH	Butterworth	1984	5th	271	6	1.6