they were analysed separately. Although making a difference in the control group, in the iv heparin group giving sc heparin (eight patients) or not (13 patients) made no difference and therefore the results of these two groups were combined in the table. The results on drip life were analysed by the Student's t test, and the comparisons between rates of spontaneous stoppage and elective removal by χ^2 with Yates's correction.

Both sc and iv heparin prolonged the life of a drip (see table), there being significant differences between the control group and both the heparin groups, and also between the two heparin groups themselves. In the iv heparin group there was a significant increase in elective removals, with a parallel decrease in spontaneous stoppages as compared with control patients.

Comment

This study showed that small doses of heparin had a significant effect on the life of a drip. This was almost entirely due to reducing spontaneous stoppage and is presumably related to prevention of clotting in or around the canula tip. No significant reduction in thrombophlebitis was found. In patients with poor peripheral veins this simple intravenous heparin regimen usefully prolongs drip life without increasing the incidence of thrombophlebitis.

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Bacterial growth in raw and pasteurised human milk

Human milk is generally agreed to be the food of choice for most newborn babies, but there is controversy over the safest form of breast milk for sick or premature neonates when normal breast feeding is impossible. Pooled raw human breast milk, recommended by some,[1] affords greater protection against neonatal necrotising enterocolitis than pasteurised breast milk.[2] Others, however, emphasise the risks of bacterial contamination in untreated breast milk and suggest that pasteurisation preserves some desirable antimicrobial properties in human milk.[3] We have tested one effect of heating breast milk by its ability to support bacterial growth before and after pasteurisation.

Methods and results

Paired samples of fresh breast milk were either pasteurised at 62·5°C for 30 minutes or untreated. Twelve paired samples were then inoculated at room temperature with an enteropathogenic *Escherichia coli* O125 inoculum containing 10^5 organisms/ml. A further six paired samples were inoculated with the Oxford strain of *Staphylococcus aureus*. Bacterial growth was compared in raw and pasteurised milk by Miles and Misra counts after 14 hours' incubation at 37°C.

All pasteurised samples were sterile before inoculation while raw milk samples invariably contained a variety of bacterial contaminants. To eliminate the effect of incidental bacterial contamination on growth of the inoculum raw milk was passed through a Seitz E K filter and shown to be bacteria free. Six paired samples treated in this way were then tested by the described method, an enterotoxigenic strain of *Staph aureus* (NCTC 10657) being used as the inoculated organism. Bacterial counts in raw and pasteurised samples were compared by paired t tests (table). Highly significant differences in bacterial growth were found with *E coli* and *Staph aureus*. Similar results were obtained with filtered milk.

Bacterial counts (Miles and Misra) in raw and pasteurised milk after inoculation with test organism and incubation for 18 hours

Test organism	No of paired samples	Filtration	Mean bacterial count		t	P
			Raw	Pasteurised		
E coli Oxford	12	No	$7·5 \times 10^4$	55×10^4	4·455	<0·001
Staph aureus Toxigenic	6	No	4×10^4	$1·8 \times 10^7$	>10	<0·001
Staph aureus	6	Yes	11×10^4	23×10^7	>10	<0·001

Comment

Pasteurisation reduces the concentration of IgA, virtually destroys IgM and lactoferrin,[4] and inactivates complement in human milk. Our findings show both the reduced antimicrobial properties of pasteurised milk and the inhibition of bacterial growth by untreated milk. Inhibition of coagulase-positive *Staphylococci* is greater than of *E coli*, which may be due to a specific heat-labile antistaphylococcal factor in raw milk.[5] Variable numbers of benign contaminants, predominantly coagulase-negative *staphylococci*, were found in raw milk samples and these may have competitively inhibited the growth of the experimental inoculum. Tests with bacteria-free filtered raw milk show that any such effect is negligible. Filtered raw milk inhibited bacterial growth to the same degree as unfiltered milk despite the inevitable removal of macrophages and neutrophils. This underlines the importance of non-cellular antibacterial constituents in raw milk. We conclude that unheated, compared with pasteurised, breast milk has bacterial growth inhibitory properties and that these may protect the neonatal gut against harmful bacterial colonisation. The need for careful and hygienic methods of milk collection in conjunction with bacteriological monitoring is clear.

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Serum 24,25-dihydroxyvitamin D and 25-hydroxyvitamin D concentrations in femoral neck fracture

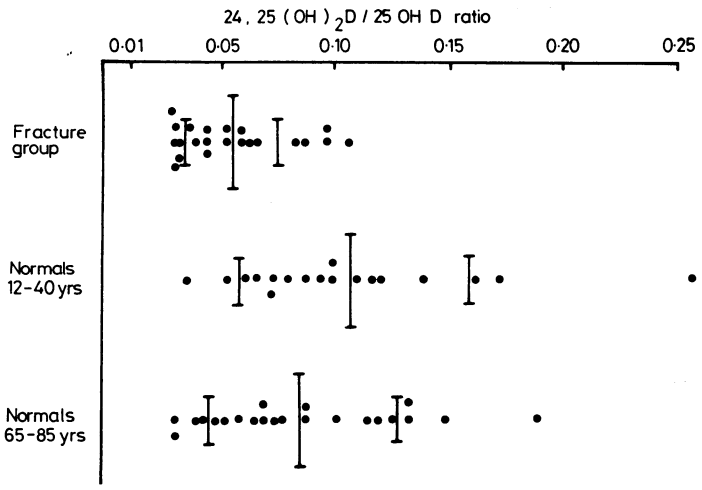
Low serum 25-hydroxyvitamin D (25-OHD) concentration and impaired conversion of 25-OHD to 1,25-dihydroxyvitamin D (1,25 $(OH)_2D$) have been suggested as important factors in the pathogenesis of osteomalacia and fracture of the proximal femur in elderly people.[1 2] Recent studies have shown the importance of another metabolite, 24,25-dihydroxyvitamin D (24,25$(OH)_2D$), in normal bone mineralisation and structure.[3] We have therefore investigated whether conversion of 25-OHD to 24,25$(OH)_2D$ is impaired in patients with fracture of the proximal femur.

Patients, methods, and results

Serum concentrations of radioassayable 25-OHD and 24,25$(OH)_2D$ were measured according to Weisman *et al*[4] in 22 patients (mean (±SD) age 74·7±8·4 years) admitted to Ichilov-Hospital, Tel-Aviv, with fracture of the proximal femur. Control sera were obtained from 18 young (mean age

32·4±10·2 years) and 22 elderly (mean age 76·2±9·4 years) people. Neither patients nor controls had any malabsorption, hepatic, or renal disease and were not taking barbiturates or anticonvulsants. All sera were collected from December 1977 to March 1978. Serum creatinine concentrations were <150 μmol/l (<1·5 mg/100 ml) in all subjects except one patient in whom it was <210 μmol/l (<2·1 mg/100 ml). One patient had hypocalcaemia and another had hypophosphataemia.

The mean (±SD) serum concentrations of 24,25(OH)$_2$D and 25-OHD were significantly (P<0·01) lower in patients with femoral fracture (1·37±0·52 nmol/l (0·55±0·21 ng/ml) and 27·0±9·2 nmol/l (10·8±3·7 ng/ml) respectively) and in elderly controls (1·87±1·22 nmol/l (0·75±0·49 ng/ml) and 24·5±14·0 nmol/l (9·8±5·6 ng/ml) respectively) than in young controls (5·82±2·75 nmol/l (2·33±1·10 ng/ml) and 58·0±21·2 nmol/l (23·2±8·5 ng/ml) respectively). No significant differences in mean serum 25-OHD and 24,25(OH)$_2$D were found between the fracture group and elderly controls.



Serum 24,25(OH)$_2$D: 25-OHD ratios in patients with proximal femoral fracture compared with elderly and young controls (solid lines = mean ± SD).

Serum 24,25(OH)$_2$D was undetectable (<1·0 nmol/l (<0·39 ng/ml)) in 19 (43·1%) out of 44 patients and elderly controls. The mean serum 24,25(OH)$_2$D: 25-OHD ratio was significantly lower in the fracture group than in either young (P<0·01) or elderly (P<0·05) controls (see figure).

Comment

This study shows that serum concentrations of 24,25(OH)$_2$D are undetectable or very low in patients with fracture of the proximal femur. Furthermore, the decreased serum 24,25(OH)$_2$D: 25-OHD ratio in these patients indicates that serum 24,25(OH)$_2$D concentrations are relatively low for the respective serum 25-OHD values. Nutritional osteomalacia will first develop at serum 25-OHD concentrations lower than 12·5 nmol/l (5·0 ng/ml). In our study, however, serum 24,25(OH)$_2$D concentrations were undetectable or extremely low in 7 out of 14 (50%) patients with femoral neck fracture in whom the serum 25-OHD concentrations were above 25·0 nmol/l (10·0 ng/ml). Therefore the steep rise in femoral neck fracture with advancing age cannot be explained by low serum 25-OHD concentrations. It probably results from a decline in renal ability to convert 25-OHD to its active metabolites 24,25(OH)$_2$D and, probably, 1,25(OH)$_2$D. The basic and best known function of 1,25(OH)$_2$D is to stimulate intestinal calcium transport. Edelstein *et al*[3] have shown the importance of 24,25(OH)$_2$D in normal bone mineralisation and structure, and Kanis *et al*[5] showed that calcium retention improved in anephric patients treated with 24,25(OH)$_2$D. Thus impaired conversion of 25-OHD to its active dihydroxy-metabolites could be responsible for the progressive decline in calcium absorption[1] and change in normal bone mineralisation in elderly people. The low vitamin D concentrations in our elderly controls suggests that elderly people are susceptible to osteomalacia and fracture of the proximal femur even in sunny climates. A tendency to be house-bound together with inadequate dietary intake are probably the major contributing factors. We also investigated a younger group (mean age 62·1±10·2 years) of 17 patients with vertebral osteoporosis. Their mean concentrations of serum 25-OHD (82·8±35·6 nmol/l (20·7±8·9 ng/ml)), 24,25(OH)$_2$D (6·8±3·36 nmol/l (1·70±0·84 ng/ml)), and 24,25(OH)$_2$D: 25-OHD ratio (0·086±0·04) were similar to those in the young controls (unpublished). This indicates that elderly patients with fracture of the proximal femur probably differ from patients with vertebral osteoporosis in their vitamin D state and metabolism.

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Communication with Asian diabetics

To achieve optimum control the diabetic needs education about diet, about the use of insulin, and on how to avoid problems. Asians pose special problems because of differences in diet, language, and customs. Several Asian diabetics attend Dudley Road Hospital, and efforts to improve communication have included the distribution of literature and erection of signs in Asian. We conducted a small survey to assess the impact of these measures in our diabetic clinic.

Patients, methods, and results

Two Asian-speaking medical students assessed the ability to read and write English and native language among Asian patients and their relatives. A standard questionnaire was used. We thought that some patients might falsely claim to be literate and so we asked whether multilingual signs prominently displayed in the hospital had been seen. These should be immediately obvious to literate Asians.

Seventy-seven diabetics had Asian origins and native language (see table). Most (59) were Punjabi-speaking Sikhs from India who had lived in Britain for an average of 15 years (range 10 months to 23 years, based on information from 45 patients). There were 43 men and 34 women. The patients' ages ranged from 17 to 75 years, with most (63) aged 30 to 60. Fifty-eight of the patients were not dependent on insulin.

Forty patients were illiterate in both their native language and English and 26 claimed literacy in their native language alone. Sixty-two households had a person literate in English who could communicate verbally, if sometimes imperfectly, in Asian without necessarily being able to read or write it. None of the women could read or write English. Only six patients had noticed the Asian signs despite 26 claiming literacy in their native language. Four patients thought them useful but only one was illiterate in English. Sixty-eight patients had received a diet sheet—53 in English, four in their native language, and 11 in both English and the native language. Thirty-eight of the 53 receiving

Nationality and language of 77 Asian diabetics

	Men	Women	Total
Nationality			
Indian	32	27	59
Pakistani	2	1	3
Bangladeshi	4	1	5
African Asians	5	5	10
Language			
Punjabi	33	29	62
Gujerati	3	3	6
Bengali	4	1	5
Urdu	3	1	4