Adaptation of bone to mechanical loads

Sir—It is proposed that bone adapts to mechanical loads through control of bone strength and mass by bone strain.[1] By contrast, little is known about bone quality (bone material properties), although bone strength is measured by bone mass and other factors such as bone quality. On the basis of this theory, bone quality could explain some discrepant results in bone.

Bone mass in the radius is low, whereas that in the lumbar is high in children with X-linked hypophosphataemic rickets (XLH) characterised by hypomineralised bone (poor bone quality),[2] but the mechanism remains unclear. In a girl aged 7 years with XLH, we noted that a rope-skipping vertical jumping exercise, 50 jumps once every 2–3 days for 6 months, strikingly increased the bone mass on the lumbar bones by 17·0% but by only 1·5% on the radius. Serum intact osteocalcin concentration increased by 34·5%, and urinary deoxypyridinoline concentration decreased by 29·6% (unpublished data). Serum concentrations of calcium, inorganic phosphorus, and alkaline phosphatase did not change.

Poor bone quality increases bone strain from mechanical loads, and impairment of bone quality could be compensated by raising bone mass in weight-bearing bone, as in XLH patients—ie, the increased total bone volume with low mineral content per unit in histology and the increased bone area with low bone mineral density in CT measurement. The compensation mechanism could explain the difference in lumbar and radius bone mass in patients with XLH, because lumbar bone is weight-bearing.

This concept could explain the controversial effects of warfarin, a vitamin K antagonist, on fracture risk. Vitamin K seems to lower fracture risk through improvement of bone quality resulting from an increase of osteocalcin carboxylation.[3] By contrast, the increased fracture risk due to chronic use of warfarin seems to be limited to the rib and vertebra, and the risk for the hip does not change.[4] Warfarin decreases osteocalcin carboxylation and could, therefore, induce impairment of bone quality, but the bone strength at the hip might not be weakened by the compensation mechanism because degree of bone strain from mechanical loads in daily life is higher at the hip than at the rib or vertebra.

Patients with type 2 diabetes have a normal or high bone mass and simultaneously have an increased fracture risk. This apparent paradox could be explained partly by poor bone quality. The high bone mass may be associated with the compensation mechanism between bone mass and quality. Furthermore, the faster bone loss in the hip of these patients[5] may be related to their lowered physical activity, which leads to a decrease of bone strain level, because a gain of bone mass induced by the compensation mechanism is lost because of decreased physical activity.

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1 Schneider P, Reiners C. Bone density in cosmonauts. *Lancet* 2000; **356:** 1851–52.
2 Oliveri MB, Cassinelli H, Bergadá C, Mautalen CA. Bone mineral density of the spine and radius shaft in children with X-linked hypophosphatemic rickets (XLH). *Bone Miner* 1991; **12:** 91–100.
3 Sugiyama T, Kawai S. Carboxylation of osteocalcin may be related to bone quality: a possible mechanism of bone fracture prevention by vitamin K. *J Bone Miner Metab* 2001; **19:** 146–49.
4 Booth SL, Mayer J. Warfarin use and fracture risk. *Nutr Rev* 2000; **58:** 20–22.
5 Nelson DA, Jacober SJ. Why do older women with diabetes have an increased fracture risk? *J Clin Endocrinol Metab* 2001; **86:** 29–31.

Position of cardiac monitor and defibrillator

Sir—It is a pity that a journal held in such high esteem by many in the medical profession should resort to using a stock and frankly incorrect photograph (Dec 8, Talking Points).

The defibrillator is in use, presumably for a cardiac arrest, since the patient is intubated. The UK Resuscitation Council teaches that the cardiac monitor and defibrillator should be on the left of the patient, and not have the wires draped across their chest. In addition, the oxygen should be disconnected when defibrillation is about to occur.

I would hope that your editorial staff are not so far removed from the shop floor that they no longer realise the importance of proper form, especially during such an acute incident as a cardiac arrest.

I am a UK Resuscitation Council advanced life-support course instructor.

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DEPARTMENT OF ERROR

Predictors of mortality in acute myocardial infarction—In this Commentary by C Varma and S J D Brecker (Nov 3, 2001, p 1473) some of the details given in the figure were wrong. The correct figure is printed below.