could be replicated in Britain for about £50m.¹ The current British system compensates about three people out of every 100 000 of the population with an award of £15 000, whereas the Swedish system compensates 10 times as many people with much greater efficiency and an average award of about £3200.

Now that lawyers and economists are supporting no fault

compensation it is surely time for the government to think again—particularly when it ultimately meets most of the costs.

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Catching up the orthopods

Mechanical forces matter in tissues other than bone

For almost a century it has been recognised that bone responds to applied mechanical forces with resorption and new bone formation (Wolff's law).¹ This functional adaptation fashions bone to suit its mechanical environment, endowing optimum strength and stiffness² while keeping its weight and hence metabolic cost to a minimum. Our understanding of this mechanism is incomplete, but its therapeutic implications have been used in treating fractures and in operations to replace joints.^{3,4} Increasing the mechanical loading on bone increases its formation locally. Conversely, reducing the loading as with immobilisation results in disuse osteoporosis.⁵ More recently the fact that exercise reduces generalised osteoporosis has also been recognised.⁶

Not surprisingly similar effects have been observed in other components of the musculoskeletal system. In articular cartilage moderate physical activity changes the mechanical properties of the tissue.^{7,8} The tissue biochemistry of the tendons changes rapidly in response to different loads—again implying changes in mechanical behaviour.⁹ And we now know that this dynamic relation is also important in other connective tissues.

In hypertension arterial smooth muscle cells produce a factor that stimulates fibroblasts to synthesise elastin, which in turn modifies the extensibility of the vessel walls.¹⁰ When the smooth muscle cells are stretched they synthesise collagen and glycosaminoglycans, which also alter the mechanical behaviour of the tissue.¹¹ Endothelial cells respond to shear forces by releasing vasoactive substances that manipulate the contractility of the smooth muscle of the vessel wall.¹² In the cervix there are gross alterations in tissue microstructure before and during labour.^{13,14} This is physiological soft tissue remodelling on an enormous scale and is at least partly a response to mechanical stimuli.

Responses to mechanical forces have been reported in other sites including the skin¹⁵ and lung,¹⁶ with postulated mechanosensors to account for the changes in blood vessels.¹² An alternative, or parallel, mechanism may include the transduction of mechanical stimuli into electrical potentials that are then decoded by the cells, a possible explanation for the sensitivity of fibroblasts to applied electrical fields.^{17,18}

The importance of mechanical effects in the pathophysiology of many disorders may have been overlooked because we tend to relate all diseases to specific cellular abnormalities. But we need also to consider the interaction between the cells and the tissue matrix on which they depend for their

mechanical support. This has required an increasing awareness of biomechanics. The principles embodied in Wolff's law are no longer confined to orthopaedic conditions. Orthopods are, however, 100 years ahead.

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Correction

Silent myocardial ischaemia

In the editorial on silent myocardial ischaemia by Dr Stephen Campbell (24 September, p 751) the first sentence of the second paragraph should have read: "Silent ischaemia is defined as 'objective evidence for myocardial ischaemia in the absence of angina or equivalent symptoms,' and its prevalence is unknown," although over a quarter of all myocardial infarctions are unrecognised and a half of those that are unrecognised cause no symptoms at all."