Correspondence

Growth factors in arthrosis

Sir:

The observation by Benske et al. in this issue of *Acta Orthopaedica Scandinavia* offers a basis for speculation on the cause of changes in the subchondral bone in arthrosis. The authors of this interesting paper suggest that increased stress is the cause of the changes in the amount and architecture of bone. The question is whether such great changes can be explained only by increased stress. I would therefore like to suggest an additional mechanism: abnormal proliferation as a response to tissue injury, i.e., cartilage degeneration. Molecular interactions between disintegrating cartilage and blood elements in the vessels growing into the cartilage result in the release of various growth factors. The subchondral bone tissue becomes the target of these factors in the same way as the subendothelial myofibrotic cells react to the atherosclerotic endothelial lesion by factors released from platelets and monocytes (Ross 1986). In this disease, platelet-derived growth factor (PDGF) stimulates proliferation particularly at sites exposed to the greatest stress of the blood stream.

There is also reason to widen this concept to human arthrosis. The mice described by Benske et al. (1988) were still growing. In fact, even the human arthrotic cartilage is growing:"a reopened growth plate function" as Howell (1979) put it. The prerequisite for an injurious interaction between a sick cartilage and blood elements apparently exists; and, in addition, the abnormal cartilage is dependent on the exclusion of blood for its integrity. Weight bearing increases the release of growth factors and other molecular factors, for example, angiogenetic and pain factors. Growth factors are in the later course of the disease thought to be produced by the macrophages accumulating in the marrow cavities, but the rich presence of growth factors in the bone tissue itself, above all, transforming growth factor (TGFß), must not be forgotten (Canalis et al. 1988).

On the basis of this hypothesis, the whole gamut of joint changes secondary to the biochemical failure of the cartilage can be explained: the bone hypertrophy, deformation, local bone necroses due to failure of intratrabecular capillaries, increased vascularisation, stasis, cysts, breakdown of the joint surface, and fibrosis of the synovial membrane.

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