

Thermal Monitoring of Paget's Disease of Bone

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In 1876, Sir James Paget, in London, described osteitis deformans, a progressive disease of the skeleton that affects the shape and size of bone. The main feature of the disease (called Paget's disease) is an increase in osteoclastic bone resorption. In a proportion of cases the disease can progress rapidly, with persistent bone pain.¹ Deformity of weight-bearing bones, and an enlarged skull, if the skull is affected, leads to many complications. The disease has also been called "chronic inflammation of bone," and increased skin temperature over the active lesion is a common finding. In 1953 Edholm et al. showed that increased peripheral blood flow frequently occurred and that increased periosteal vascularity was found during the active phase of the disease. The vascular bed, acting as an arteriovenous shunt, can be so increased that high-output cardiac failure may occur.²

The tibia and skull are two skeletal sites affected by Paget's disease. Lumbosacral spinal and pelvic involvement are particularly common, but the femur, sternum, and humerus are also affected. Many of these sites are not near the skin surface and are less likely to affect skin temperature. The forehead and tibiae, however, are so close to the skin that changes in bone temperature have a marked effect on the temperature of overlying tissues.

Infrared thermography is a convenient and noninvasive technique that can add valuable information in the assessment of the disease when the tibia or skull are affected. Radiological changes usually occur too slowly to be useful for monitoring the effects of treatment. Biochemical markers, such as alkaline phosphatase and urinary hydroxyproline levels, are widely used but they cannot indicate specific sites of changes of disease progression. To accurately monitor the effects of therapeutic agents on the disease, objective and site-specific techniques that are sensitive to both short- and long-term action are required. As temperature changes appear to be associated with Paget's disease, thermal imaging should be the ideal means of objective measurement. Infrared thermography is most applicable to monitoring the disease in skeletal sites lying near the skin. We have also investigated the use of a microwave

radiometer at 3 GHz to detect active Paget's disease at some of the other, more deep-seated osteoid lesions.

Experimental Studies on the Tibia

There are many unanswered questions about the pathology of Paget's disease, particularly the large changes in temperature that can be measured over the tibia. This weight-bearing long bone is ideally situated for heat-transfer experiments on living bone. By a combination of experimental measurements, with calculations based on a single model, we have investigated the thermal processes occurring in normal and Pagetic bone. The tibia and dermis were described by 17 differential equations, producing a close analogy between heat flow and electric current, and between temperature and voltage (Figure 1). Thermal conductivity was expressed as thermal resistance R , and heat transfer from skin to ambient temperature by R'' . Heat from arterial blood is delivered to tissue at a rate proportional to $T_c - R$, i.e., core temperature and local tissue temperature, flowing in the thermal resistances R'' . Metabolic heat production is represented by Input, I , and thermal capacitance of sections of tissue by C . The parameters of the model were calculated from the basic tissue properties, that is, thermal conductivity, density, specific heat, blood perfusion rate, and metabolic heat. However, there are few data for blood perfusion or metabolic heat in these circumstances. Geometric measurements were obtained from a CT scan of a normal leg.

Based on the above model, a graph was constructed to show the calculated temperature distributions for normal and active Pagetic tibia (Figure 2). The skin temperature values were brought into agreement with those measured by infrared thermography by adjusting the values for blood perfusion in the cortical bone. The graphs labeled B were obtained by reducing metabolic heating to zero in the cortical bone, skin temperature then being controlled more by perfusion. The apparent insensitivity to metabolic heat is remarkable because the value taken for the Paget model was estimated as three times the rate for liver, i.e., 10^4 Wm^{-3} , representing a very active tissue. The blood flow rates that gave the correct skin temperatures seemed to be of the right order of magnitude.

To test the thermal capacitance and thermal resistance in vivo, the dermis was cooled by a metal block at 18C

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