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CASE REPORT

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The Effect of Severe Bedsores on Bone and Its Forensic Implications

Bedsores, also known as decubitus ulcers and pressure sores, are continual problems for the debilitated and disabled but are often not regarded with the seriousness they deserve. Pressure sores arise when localized areas of the body are subjected to prolonged pressure that occludes vascular flow. When blood flow is obstructed for a sufficiently long period, the mechanism of reactive hyperemia cannot compensate for inadequate cell nutrition and soft tissue necrosis begins. Pressure sores develop under bones that are close to the skin, where soft tissues are compressed by concentration of body weight. Particularly vulnerable areas are those over the sacrum, heel, greater trochanter, elbow, lateral and medial malleoli, scapula, and occiput in the bedfast patient and over the ischial tuberosities in the chairfast.

Although traditionally regarded as pathologies of the soft tissues, severe pressure sores may also affect the bone. It is known that septic ulcers can lead to osteomyelitis [*1,2*]. But the focus of this paper is on the extensive periosteal erosion, not directly attributable to infection, which may also result. Observation of two cases from the cadaveral collection of an anatomy laboratory will illustrate this little-known phenomenon.

The first case involves an 84-year-old female with several pressure sores, the most severe lying over the left greater trochanter (Fig. 1). The tissue visible at the center of the ulcer is the cancellous bone of the lateral surface of the trochanter. In Fig. 2 the soft tissue has been retracted to show the extent of bone involvement. In the affected area the periosteum and underlying compact bone have been completely eroded, exposing the spicules of cancellous bone. There is no evidence of reactive periosteal bone formation surrounding the lesion.

In the second case both femurs of a 70-year-old male are affected. The lateral surface of the right trochanter displays exposed cancellous bone, but the left femur shows even more extensive osseous involvement (Fig. 3). Periosteal erosion covers the entire lateral aspect of the greater trochanter and extends onto the anterolateral and posterolateral surfaces. On the posterolateral surface the lesion extends onto the diaphysis to a distance of 5 mm below the level of the lesser trochanter. As in the first case, there is no evidence of reactive bone or sinus formation.

Since the osseous lesions are solely destructive in character and do not display the usual hallmarks of osteomyelitis or periostitis, it is likely that they result from pressure atrophy. When bone is subjected to continuous pressure, there is active resorption by the osteoclasts

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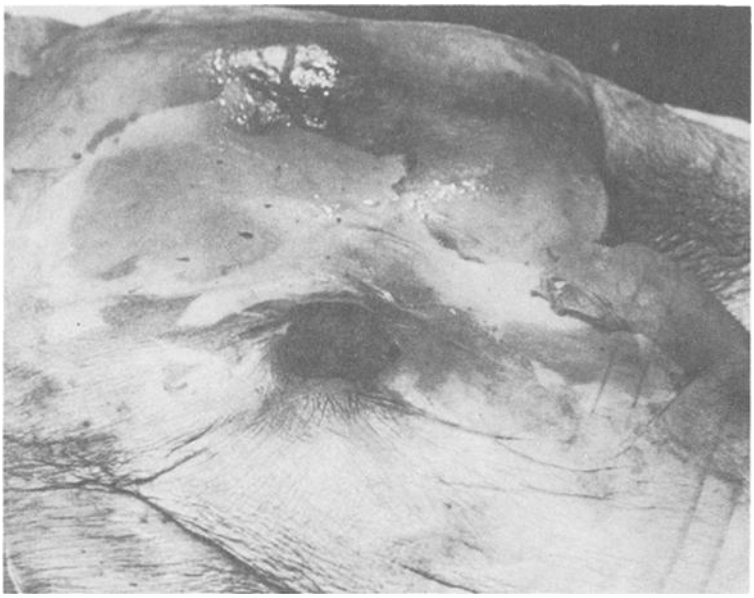

FIG. 1—*Case 1: Pressure sore over left greater trochanter.*

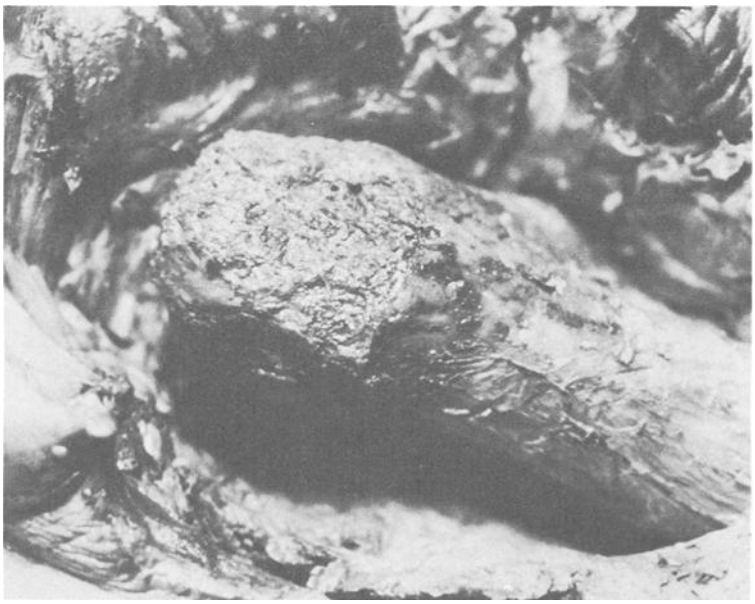

FIG. 2—*Case 1: Trochanteric pressure sore with soft tissue retracted.*

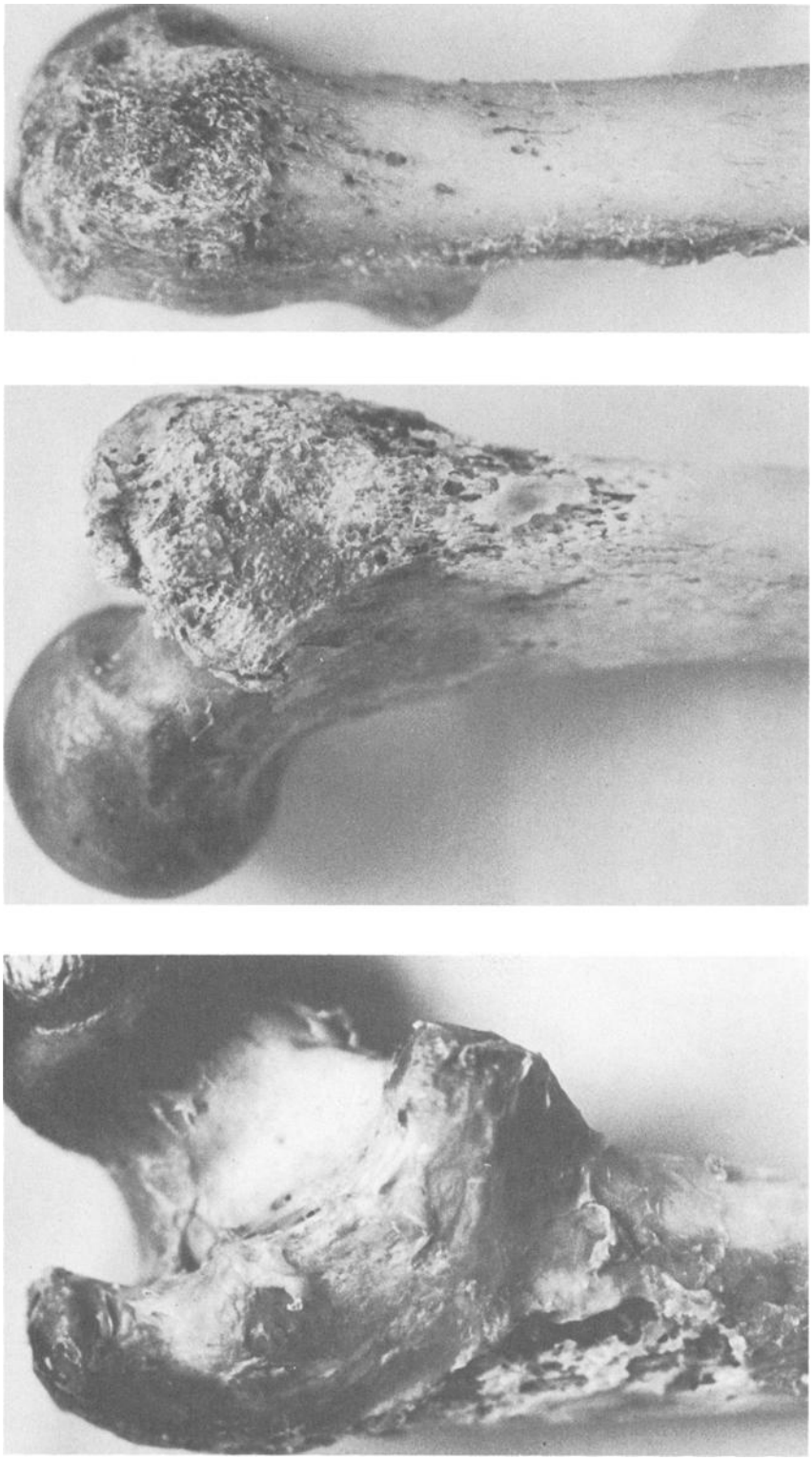

FIG. 3—*Case 2:* (left) *Lateral view of right proximal femur;* (center) *anterolateral view of left proximal femur; and* (right) *posterior view of left proximal femur.*

[3]. In decubitus ulcers pressure damage usually begins and is more severe on or near the body surface, but in some cases, such as over the trochanters or heels where body prominences are especially close to the surface, pressure is concentrated and necrosis may involve deep tissues from the beginning [4].

The forensic implications of the effect of pressure sores on bone are two-fold. For the forensic anthropologist recognition of osseous lesions resulting from pressure sores may aid the skeletal identification of recently bedfast or chairfast individuals. Although the effect of severe bedsores on bone mimics the effect of postmortem erosion, the two possible causes can be distinguished by the extent and distribution of the lesions within the skeleton. Unlike weathering, erosion from bedsores will selectively affect those localized areas where the bone is relatively superficial and bears a high proportion of sitting or reclining body weight.

In addition, these lesions may potentially serve as evidence of marginal institutional care. Effective prevention of bedsores consists of keeping the patient dry, clean, and well-nourished and of turning the immobile patient frequently. Despite such straightforward prophylaxis, pressure sores continue to be a serious problem. Berecek [5] suggests that the following factors may be largely responsible: the health care staff does not realize the necessary frequency for turning the patient; the staff does not accept the necessity for frequent turning; the number of staff is inadequate to carry out the task properly.

Not only do bedsores slow the recovery of a patient, but in extreme cases they may be a major factor contributing to his death [6]. For instance, Galpin and his co-workers in a study of 21 patients with sepsis attributable solely to pressure sores reported that 10 patients died, 8 despite the administration of antibiotics [2]. Decubitus ulcers severe enough to erode the bone cannot but have a detrimental effect on the patient's health.

Because adequate nursing care can prevent bedsores in most cases, it is not unreasonable that charges of negligence could be leveled against health care institutions, which are being held increasingly accountable for standards of care [7]. In regard to hospital liability the landmark case of *Darling v. Charleston Community Hospital* is pertinent. The Illinois Supreme Court held that all hospitals must have a sufficient number of trained nurses and ancillary personnel for bedside care [8]. Considering recent trends in tort law, it is conceivable that such judgments could, in some cases, be extended to cover the most common of iatrogenic diseases, the bedsore.

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