RADIOLOGICAL ASSESSMENT OF PERIOSTEAL REACTION

THESIS

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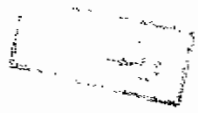

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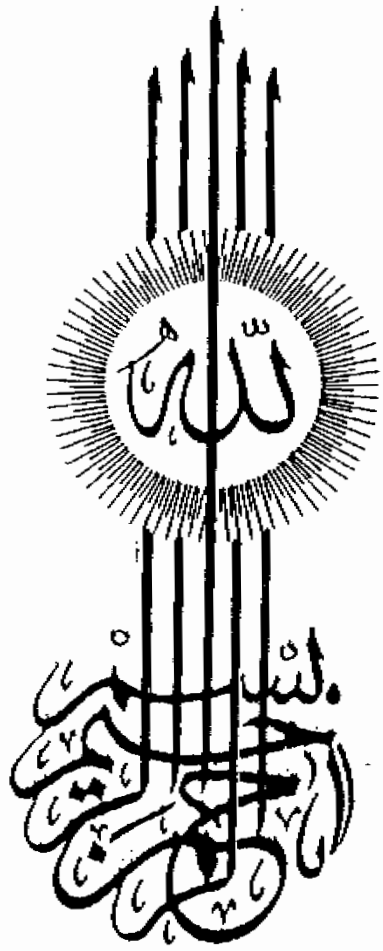

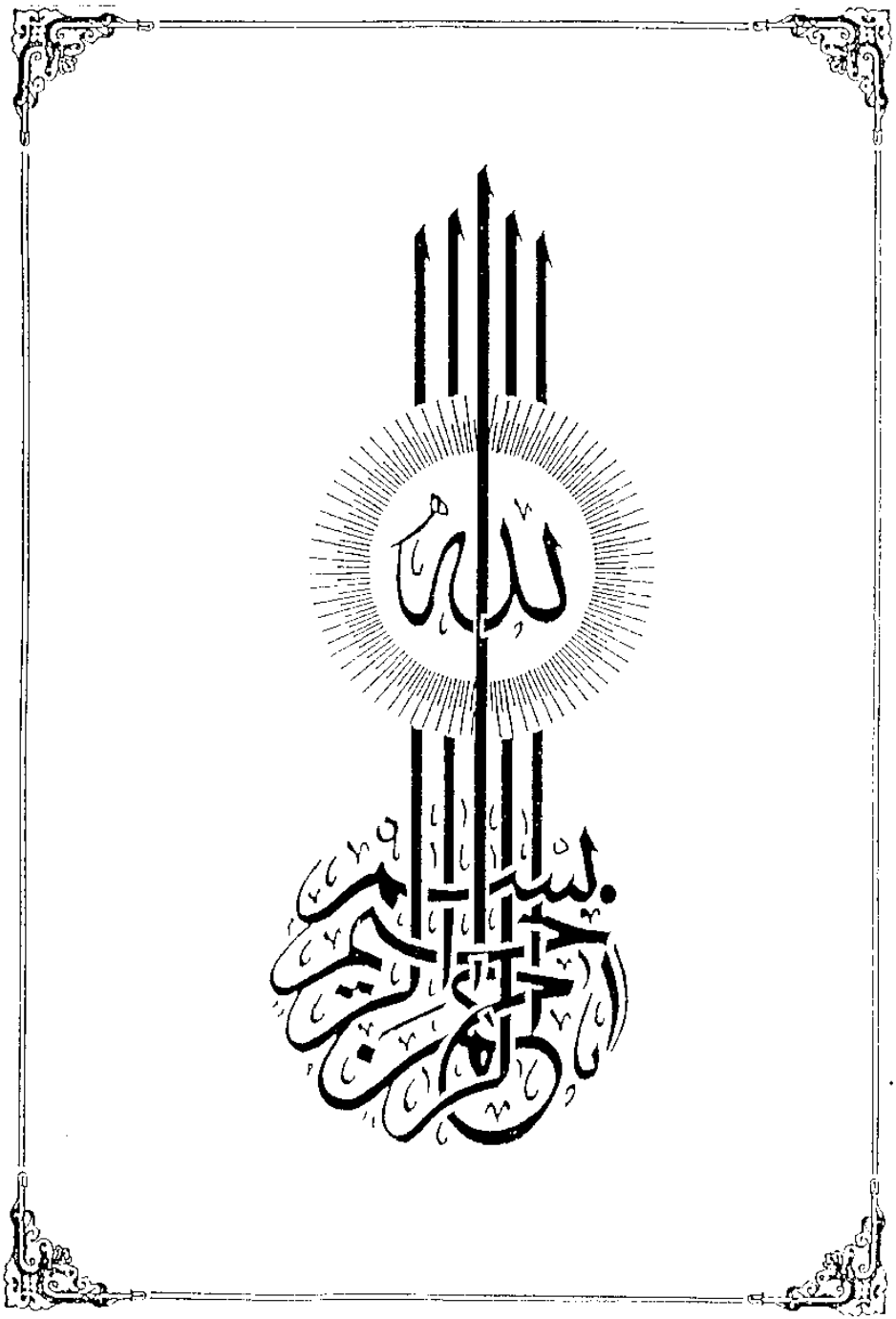

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The activated periosteum has a deceptive anatomic constancy amidst change. The change involves the production of matrix and, in the process, proliferation and expenditure of cells.

The configuration of a periosteal reaction is an index of the nature and intensity of the inciting process.

Periosteal reaction is one of the most common traumatic bone lesion, and occurs in many inflammatory and neoplastic conditions. It is seen in its most exuberant forms in long bones.

The aim of this study is to present different causes of periosteal reaction, their radiological picture and differential diagnosis.

PATHOGENESIS

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The periosteum is traditionally defined anatomically as an envelope, composed of an outer "fibrous layer", and inner cellular "Cambium layer", that demarcates the bone from surrounding soft tissue. The quiescent periosteum of an adult bone is minimally cellular and mainly fibrous, but during the normal gross and developement of children or during reaction to injury, the periosteum may be thick and the two layers quite distinct.

There is a plane between the two layers of the active periosteum called the zone of transion, and the fibrous layer of the periosteum can be replenished from surrounding periosteal soft tissues, such as fascia, fat, and muscle. The earliest phase in this process is the progressive modulation of "fibroblasts" of the fibrous layer into "preosteoblasts" through nuclear enlargement and acquisition of cytoplasm. Subsequent mytosis combines with further cell enlargement to create the cambium layer within which osteoid-secreting cells emerge.

Often the creation of new bone about the cortex appears to be ashieved by direct modulation of parosteal soft tissue elements into osteogenic cells without a traditional dual-

layered interphase (or periosteum). (Brailsford, J.E., 1953)

The relative position of the periosteum shifts when bone is added to diaphysis, increasing the shaft diameter. It also shifts when bone is deleted from the bone surface, as occurs in the tapered "cut-backzone" of the metaphysis during bone formation and modeling. Such bone addition or deletion can be reawakened or exagerated in disease. Positive periosteal reactions add bone to the surface and create a variety of different reactive patterns, negative periosteal reactions (subperiosteal resorption) remove bone from the surface as, for example, in hyperparathyrodism.

The slowest appositional activities (bone addition) of the periosteum are not thought of as "reactions". Such activities include the circumferencial enlargement of bone shaft during normal gross, and the increase in bone diameter with osteoprosis and Paget's disease.

The expression "periosteal reaction", as conventionally used, implies a more vigrous participation of juxta cortical soft tissue in disease process.

In general, the amount of periosteal new bone production (in a "reaction") relates not only to the degree of periosteal elevation but also to the level of activity of the process stimulating new bone. For example, fibrosarcoma commonly erode through the cortex to form subperiosteal

masses, but in doing so, stimulate little new bone formation. (Brunschwig, A., 1935).

Since physical elevation of the periosteum from the bone is frequently present, but is not a consistant prerequisite for a "reaction", other factors must be operative.

These factors include mechanical adaptation or compensation for weakness secondary to underlying osteolysis, attempts at tumour containment, altered circulation (passive hyperaemia), and perhaps, bone-inductive products emaneting from tumours. (Urist, M.R., 1979).

The configurations of periosteal reactions relate to their manner and time course of production. They are a biologic measure of the intensity, aggressiveness, and duration of an inciting process.

Their production involves the reawakening and accentuation of mechanisms that normally modify the surface of bone in gross and developement. (Hedhammer, A., 1974).

For a periosteal reaction to become visible in a radiograph it must mineralise, this requires a period of 10 days to 3 weeks following the initial stimulus, varying somewhat to the age of the patient. Periosteal reactions become visible in radiographs sooner in younger patients. (Johnson, L.C., 1964).

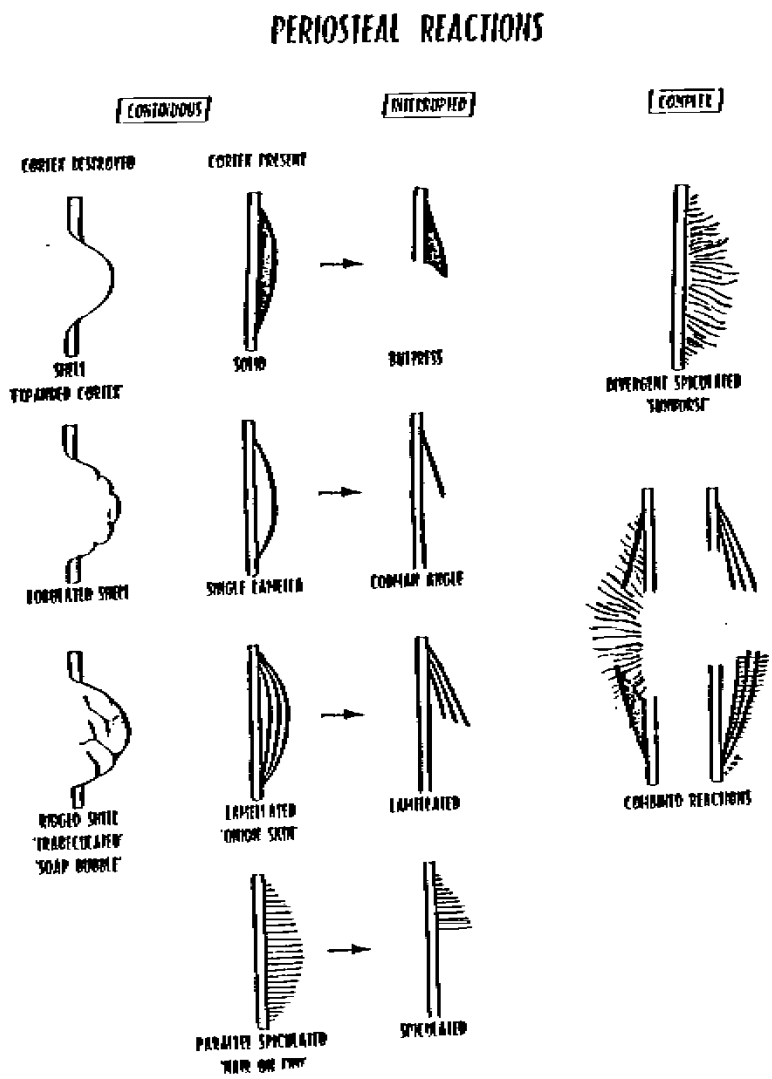

Fig. 1
Schematic diagram of periosteal reactions.
The arrows indicate that
the continuous reaction may be interrupted,
(from "Radiologic Clinics of North America" Dec. 1981.)

PATHOLOGICAL CLASSIFICATION OF PERIOSTEAL REACTIONS

Periosteal reactions may be classified basically as continuous and interrupted, but some are complex (Fig. 1), with various subclasses as delaited below :

I. Continuous periosteal reactions with destruction of cortex (shells)
II. Continuous periosteal reactions with cortical persistance.
III. Interrupted periosteal reactions
IV. Complex periosteal patterns.

I. Continuous periosteal reactions with desturction of cortex shells :

Any widening of bone contour represents periosteal activity. The use of the term "cortical expansion" for this change obscurs the true mechanism, since bone is solid and therefore cannot "expand" like a balloon.

Widening of the cortical outline while maintaining cortical thickness signifies a relatively slow process which has evoked removal of bone from the inner (endosteal surface) of the cortex at a rate balanced by apposition of new bone on the outer priosteal surface.

Endosteal cortical resorption is affected by the osteoclasts which are stimulated either by pressure from impringing gross or by the presence of active hyperaemia. At the same time, the periosteum adds new bone to the outer surface of the cortex.

If endosteal resorption slowly or transiently exceeds periosteal apposition, a thinner bony shell wil result. Eventually, none of the original lamellar cortex remains. Only a shell of new periosteal bone, whether smooth, lobulated, or ridged seperates the tumour from extraoseous soft tissue; the shell (thick or thin) then serves as substitute to the original cortex. (Brailsford, J.E., 1948).

Types of Shells:

(1) Shells with smooth contour signify a uniform external pressure. They are usually, but not necessary excentric; and favor benign lesions such as giant cell tumour, enchondroma, chondroblastoma, lipoma, chondromyxoid fibroma, and fibrous displasia.

Predominantly benign lesions (for example, bone cysts) differ from focally cystic lesions in their tendency to be central rather than excentric. They create a circumferential shell, leaving no residual of original cortex around the perimeter. This is the morphologic consequence of equality

of hydrostatic force in all directions. Shells, then are created by synchronous endosteal resorption and periosteal apposition, not "bone expansion". By this same mechanism, the transverse endosteal (medullary) diameter eventually comes to exceed the original total diameter of the normal bone. "The shell is new-bone-not the old expanded shaft". The longer the lesion has been present and the slower it progresses, the thicker the periosteal new cortex. The most rapid rate of progression is denoted by complete cortical lysis and no visible periosteal reaction, indicating that the process is advancing so fast that time does not permit production and mineralization of a new outer bone layer. (Codman, E.A., 1925).

(2) Lobulated Shell :

A lobulated shell results when a lesion has focal variation in growth rate. The more rapidly enlarging areas correlate with the bulges (lobules), and may be either solid or cystic. An example is the pattern in parosteal myositis ossificans (subperiosteal giant cell tumour) in which the shaft is "saucerized" by surface osteolysis. The facility with which the lobulated shell involves into the ridged shell suggests that it usually signifies a transitory, active phase of enlargement. (Thompson, P.C., 1954).

(3) Ridged Shell :

A ridged shell is formed when a proliferative lesion of intermediate speed slows down. This type of shell tends to be thicker and better defined than a lobulated shell.The ridged shell is also known as a trabeculated "seperate" or "soop bubble" reaction. The most active lobules become outlined by ridges on the inner surface of the periosteal shell. The ridges are areas where bone removal lags behind adjacent, faster growing areas, and in fact, new bone production may be seen along such ridges.

The ridged shell is most often produced in fibroxanthomas (also monossifying fibroms), long standing giant tumours, enchondroma, and even in slowly growing malignant processes such as chondrosarcoma, fibrosarcoma, plasmacytoma, and metastatic carcinoma of renal or thyroid origin.

The ridges of certain lesions (for example, desmoplastic fibromas and enchondromas), tend to be thicker and coarser than are the more delicate one seen with lesions such as giant cell tumours and angiomas. (Thompson, P.C., 1954)

II. Continuous periosteal reactions with cortical persistence.

In contrast to its destruction in shell reactions, the original cortex tends to persist, in full or in part,