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RADIOLOGIC AND PATHOLOGIC ANALYSIS OF SOLITARY BONE LESIONS

Part II: Periosteal Reactions

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The periosteum is traditionally defined anatomically as an envelope, composed of an outer "fibrous layer" and an 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 growth and development of children or during reaction to injury, the periosteum may be thick and the two layers quite distinct.

Assigning names to the two layers of active periosteum is a convention that obscures the likelihood that the plane between them is a zone of transition, and that the fibrous layer can be replenished from surrounding parosteal 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 mitosis 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 achieved by direct modulation of parosteal soft tissue elements into osteogenic cells without a traditional dual-layered interface (or periosteum) (see Figs. 5B, 8D, 11B, 18D, and 22I).

The relative position of the periosteum shifts when bone is added to the diaphysis, increasing the shaft diameter (Fig. 1, top; arrow points to the interface between periosteum and bone). It also shifts when bone is deleted from the bone surface, as occurs in

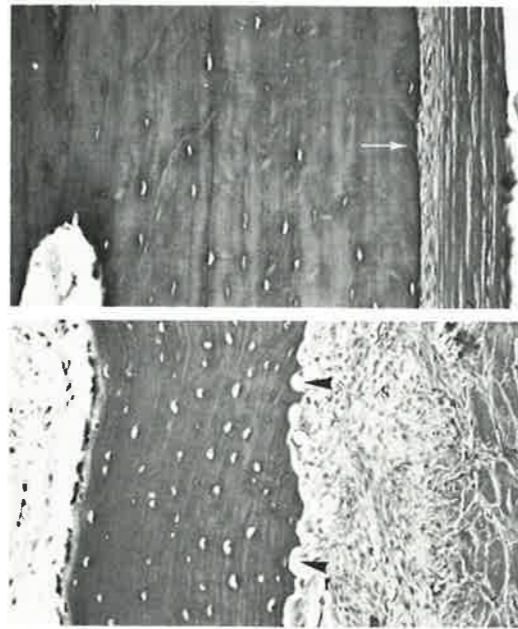


Figure 1. Normal periosteum. Tibia, five year old girl. Top. A photomicrograph (magnification $\times 157$) shows periosteal bone apposition to the midshaft cortex (arrow). Bottom. Subperiosteal bone resorption is evident in the metaphyseal "cut-back" area (arrows). (AFIP Neg. No. 81-14640.)

the tapered "cut-back zone" of the metaphysis during bone formation and modeling (Fig. 1, bottom; arrows point to the hollowed out areas where bone is being deleted at the interface between bone and periosteum). Such bone addition or deletion can be reawakened or exaggerated in disease. Positive periosteal reactions add bone to the surface and create the variety of reactive patterns schematically displayed in Figure 2. Negative periosteal reactions (subperiosteal resorption) remove surface bone as, for example, in hyperparathyroidism.

The slowest appositional (bone addition) activities of the periosteum are not thought of as "reactions." Such activities include the circumferential enlargement of bone shafts during normal growth (Fig. 3), and the increase in bone diameter with osteoporosis¹⁴ and Paget's disease. The expression "periosteal reactions," as conventionally used, implies a more vigorous participation of juxtacortical soft tissue in disease processes. 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 the new bone. For example, fibrosarcomas commonly erode through the cortex to form subperiosteal masses, but in doing so, stimulate little new bone forma-

tion. Since physical elevation of the periosteum from the bone is frequently present, but is not a consistent prerequisite for a "reaction," other factors must be operative. These include mechanical adaptation or compensation for weakness secondary to underlying osteolysis, attempts at tumor containment (teleologically speaking), altered circulation (passive hyperemia),^{10,17} and, perhaps, bone-inductive products emanating from tumors.^{2,18}

The configurations of periosteal reactions (see Fig. 2) 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 growth and development.

For a periosteal reaction to become visible on a radiograph, it must mineralize. This requires a period of 10 days to three weeks following the initial stimulus, varying somewhat with the age of the patient. Periosteal reactions become visible in radiographs sooner in younger patients.

Periosteal reactions may be classified basically as continuous and interrupted,^{5,8} but some are complex. The various subclasses, as outlined in Figure 2, are further detailed below.

PERIOSTEAL REACTIONS

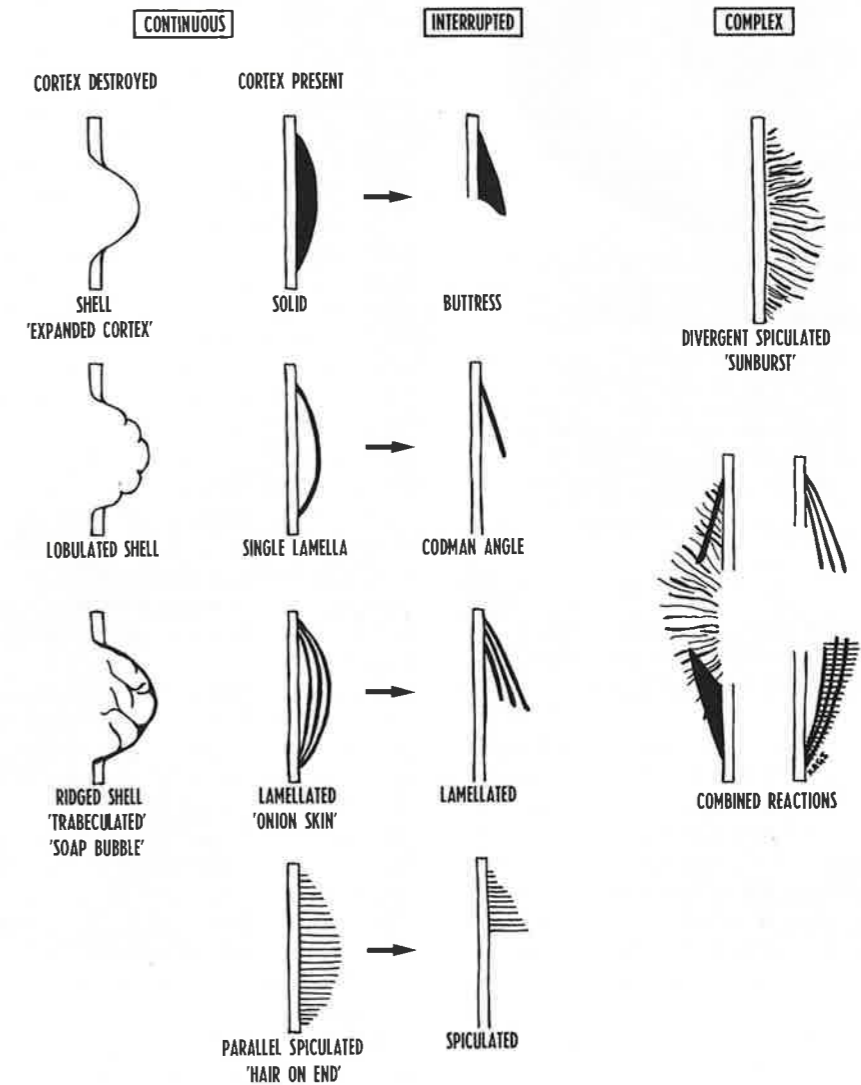


Figure 2. Schematic diagram of periosteal reactions. The arrows indicate that the continuous reactions may be interrupted.

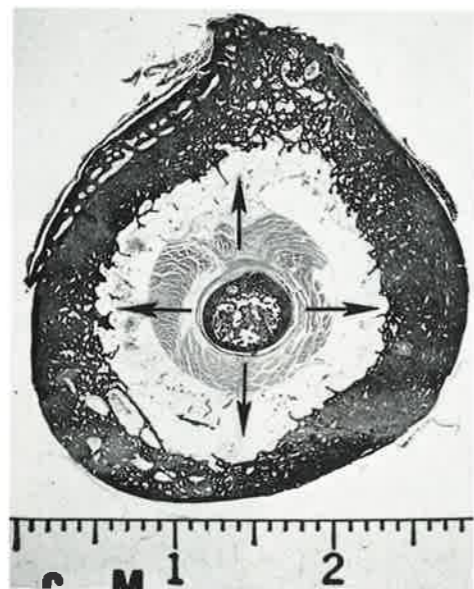


Figure 3. Normal cortical growth. A histologic section through the mid-shaft of the femur and thigh muscles of a six-month fetus is superimposed upon the medullary cavity of an adult femur (magnification $\times 3$). The increase in diameter (arrows) involves simultaneous endosteal osteolysis and cortical surface apposition. Therefore, none of the original fetal femur substance will persist in the adult. (AFIP Neg. Nos. 81-14801; 81-14802.)

CONTINUOUS PERIOSTEAL REACTIONS WITH DESTRUCTION OF CORTEX (SHELLS)

Any widening of bone contour represents periosteal activity. Use of the term "cortical expansion" for this change obscures 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 periosteal surface. Endosteal cortical resorption is effected by a front of osteoclasts stimulated either by pressure from impinging growth or by the presence of active hyperemia. 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 will result. Eventually, none of the original lamellar cortex remains. Only a shell of new periosteal bone, whether smooth, lobulated, or ridged, separates the tumor from extrasosseous soft tissue; the shell

(thick or thin) then serves as a substitute for the original cortex.

Smooth Shell. Shells with smooth outer contours (Fig. 4) signify uniform, expansile pressure; are usually, but not necessarily, eccentric; and favor benign lesions, such as giant cell tumor, enchondroma, lipoma, chondroblastoma, chondromyxoid fibroma, and fibrous dysplasia. Predominantly cystic lesions (for example, bone cysts) differ from focally cystic lesions in their tendency to be central rather than eccentric. 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."³ 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.

Lobulated Shell. A lobulated shell (Fig. 5) 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 tumor)¹⁵ in which the shaft is "saucerized" by surface osteolysis. The facility with which the lobulated shell evolves into the ridged shell suggests that it usually signifies a transitory, active phase of enlargement.

Ridged Shell. A ridged shell (Fig. 6) 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," "septate," or "soap 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 (Fig. 6, C and D). In a single projec-

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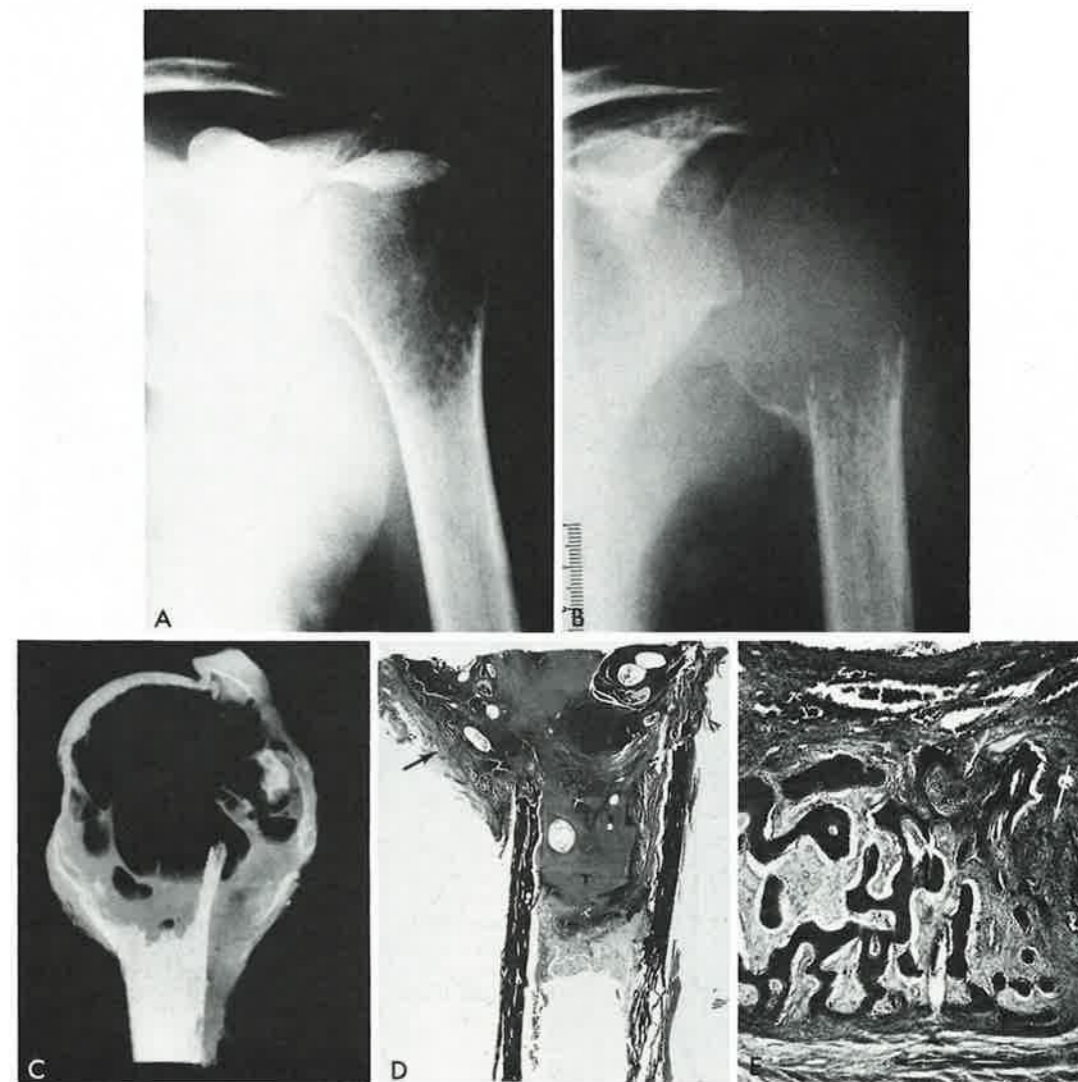


Figure 4. Shell periosteal reaction. Giant cell tumor of the humerus in a 17 year old woman. A, A clinical radiograph demonstrates a lytic lesion of the proximal humerus with thinning of the lateral cortex. B, A follow-up film obtained one and one half months after A documents progressive tumor enlargement and bone destruction, especially of the original cortex. As cortex was removed, a shell of periosteal new bone was added. C, A specimen radiograph (magnification $\times 1$) of the resected proximal humerus illustrates a widened bone outline secondary to the reactive bony shell which substitutes for the deleted cortex. D, A histologic section (magnification $\times 2.2$) demonstrates the junction between the remaining original cortex and the reactive bony shell (arrow); this area is further delineated in E. The end of the shaft is filled with giant tumor (T). E, Histologic examination (magnification $\times 63$) reveals that the shell is actually an interconnected trabecular system of periosteal new bone. Cystic tumor is present along the inner surface (top) and focally grows into the shell (arrows). (AFIP Neg. Nos. 79-2822-1, 2; 81-14658; 81-14800; 81-14568.)

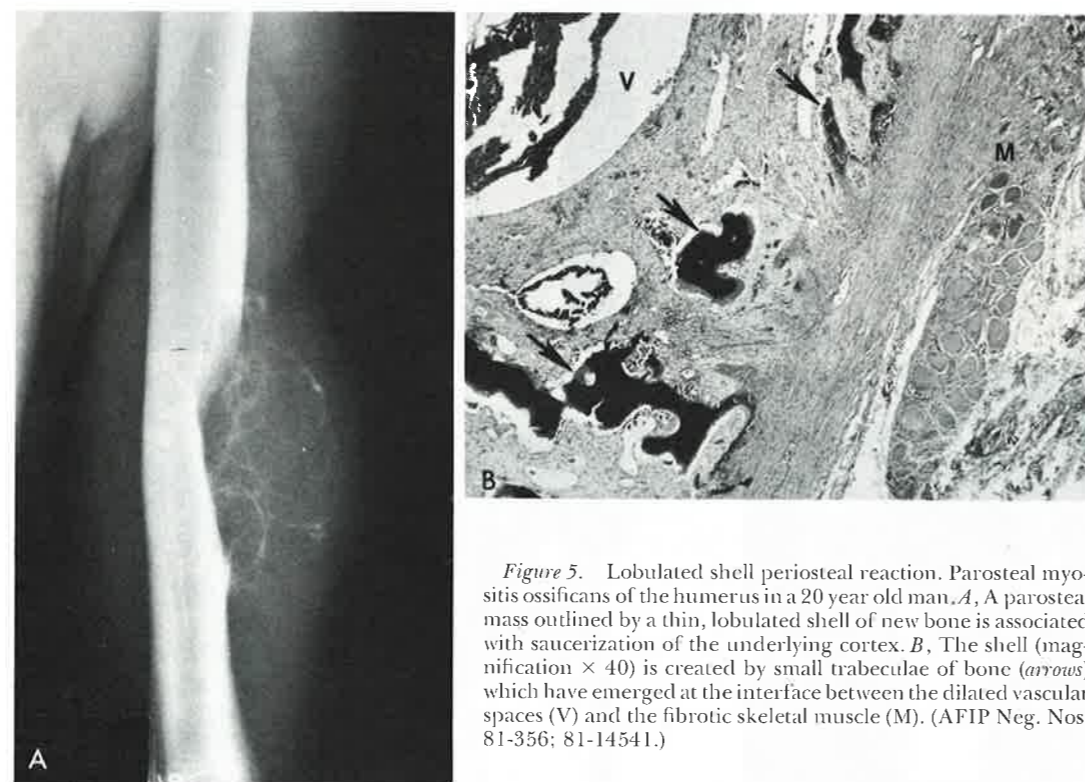


Figure 5. Lobulated shell periosteal reaction. Parosteal myositis ossificans of the humerus in a 20 year old man. *A*, A parosteal mass outlined by a thin, lobulated shell of new bone is associated with saucerization of the underlying cortex. *B*, The shell (magnification $\times 40$) is created by small trabeculae of bone (arrows) which have emerged at the interface between the dilated vascular spaces (V) and the fibrotic skeletal muscle (M). (AFIP Neg. Nos. 81-356; 81-14541.)



Figure 6. Ridged shell periosteal reaction. Giant cell tumor of the radius in a 38 year old man. *A*, An anteroposterior view of the distal radius shows a lytic defect with ridges associated with cortical destruction and a substituted shell. *B*, A specimen radiograph clearly demonstrates these ridges. *C*, The corresponding histologic macrosection (magnification $\times 2$) confirms that the tumor fills the entire radiolucent defect and has no internal bone trabeculae. An arrow marks a small ridge of bone on the inner surface of the shell. *D*, Magnification ($\times 15$) of the ridge (arrows) marked by the arrow in *C* shows osteoclastic resorption on the inner aspect of the shell, while apposition of new bone proceeds on the outer surface. (T, tumor; M, skeletal muscle.)

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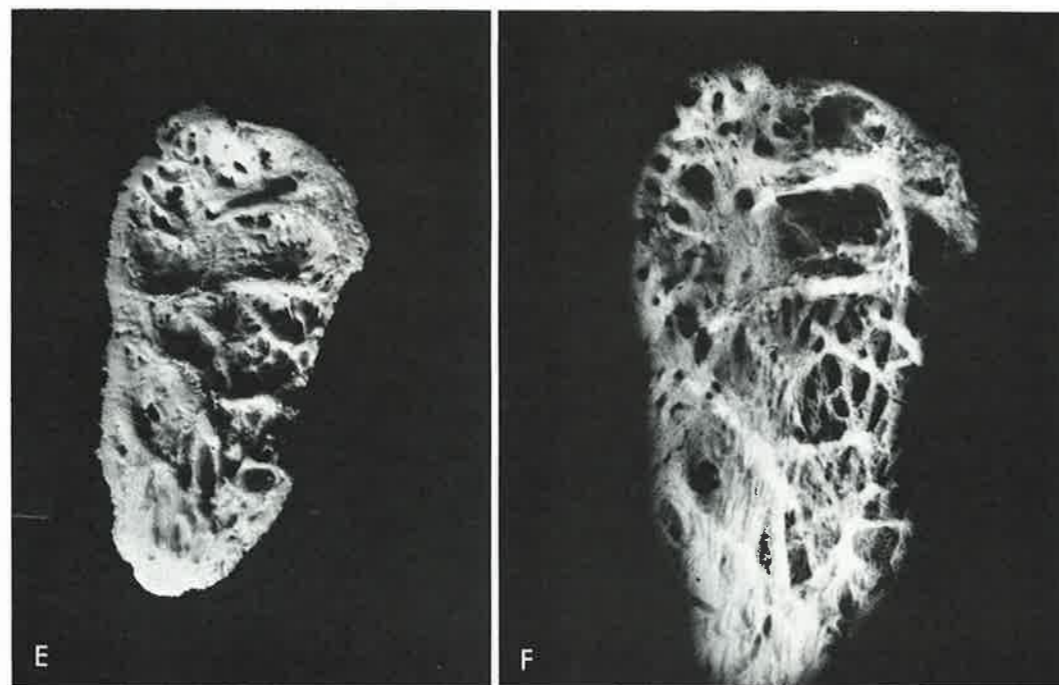


Figure 6 Continued. The macerated specimen (E, magnification $\times 2.3$) and its radiograph (F, magnification $\times 1.8$) effectively reveal the ridging of the inner aspect of the shell and confirm that the trabeculated or "soap-bubble" pattern seen radiographically is caused by the observation of such ridges en face. (AFIP Neg. Nos. 78-6679; 81-276; 81-14599; 81-14561; 81-14546; 81-14520.)

tion, the ridges may easily be misinterpreted as trabeculations or cords of bone within the lesion (Fig. 6B). The ridged shell is most often produced in fibroxanthomas (also called nonossifying fibromas) (Fig. 7), long-standing giant cell tumors, 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 ones seen with lesions such as giant cell tumors and angiomas.

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, beneath continuous periosteal reactions of the solid, single lamellar, lamellated, and parallel spiculated (osteophytic) types (see Fig. 2). Thus, these reactions are

additions to, rather than substitutes for, the original cortex. However, the persistence of the original cortex does not preclude some degree of penetration from the marrow space into the periosteal reaction and, in fact, such penetration is frequently encountered with the lamellated (Fig. 12E) and parallel spiculated (Fig. 13F) forms.

Solid Periosteal Reaction. A solid, continuous periosteal reaction (Fig. 8) represents multiple successive layers of new bone applied to the cortex about a chronic, indolent lesion in the marrow space, cortex, or adjacent soft tissue. This pattern, also referred to as the "dense-elliptical reaction"²⁵ and "cortical thickening" or "hyperostosis," connotes a slow rate of progression. The formative interface with surrounding soft tissue is not particularly cellular, and contributions from contiguous, involuting skeletal muscles may be seen (Fig. 8D).

A "solid" reaction may be created in a number of ways. Most simply, there may be the slow surface addition of layer after layer of compact lamellar bone which becomes incorporated or fused (Fig. 8). Alternatively, the soft tissue spaces between layers of a lamellated reaction may be reduced by con-

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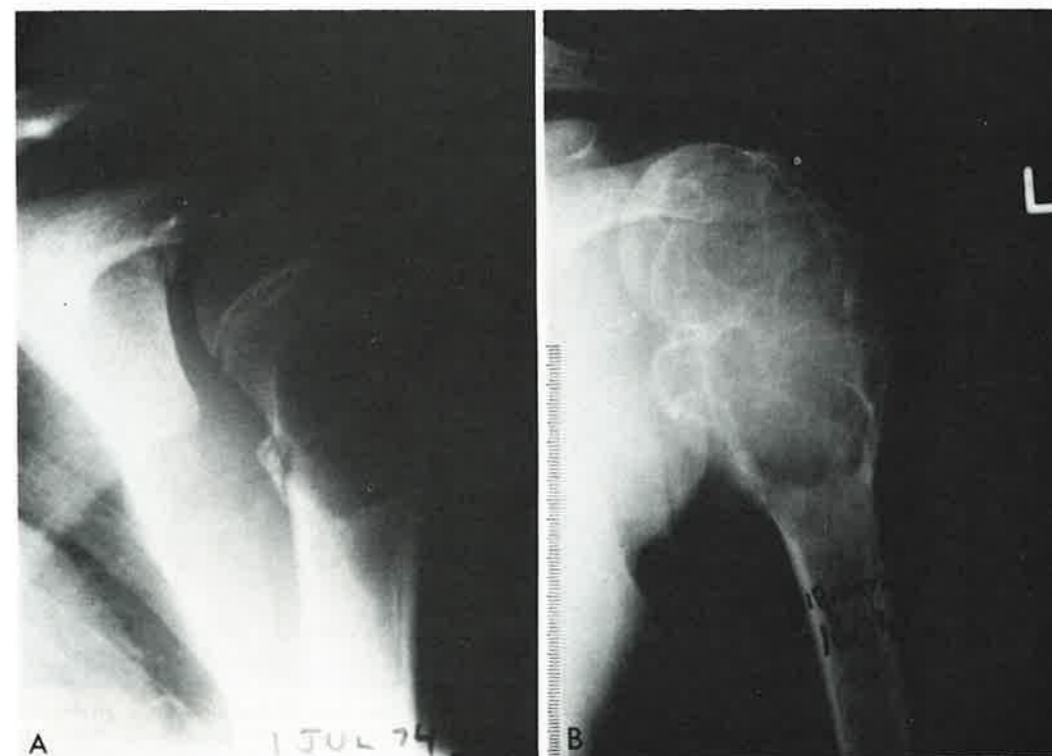


Figure 7. Ridged shell periosteal reaction, Cystic fibroxanthoma of the humerus in a 15 year old girl. A and B, Progressive enlargement of this active lesion over nearly a three-year interval has completely deleted the original cortex. A ridged shell of new bone has been substituted. (AFIP Neg. Nos. 77-6498-1,2.)

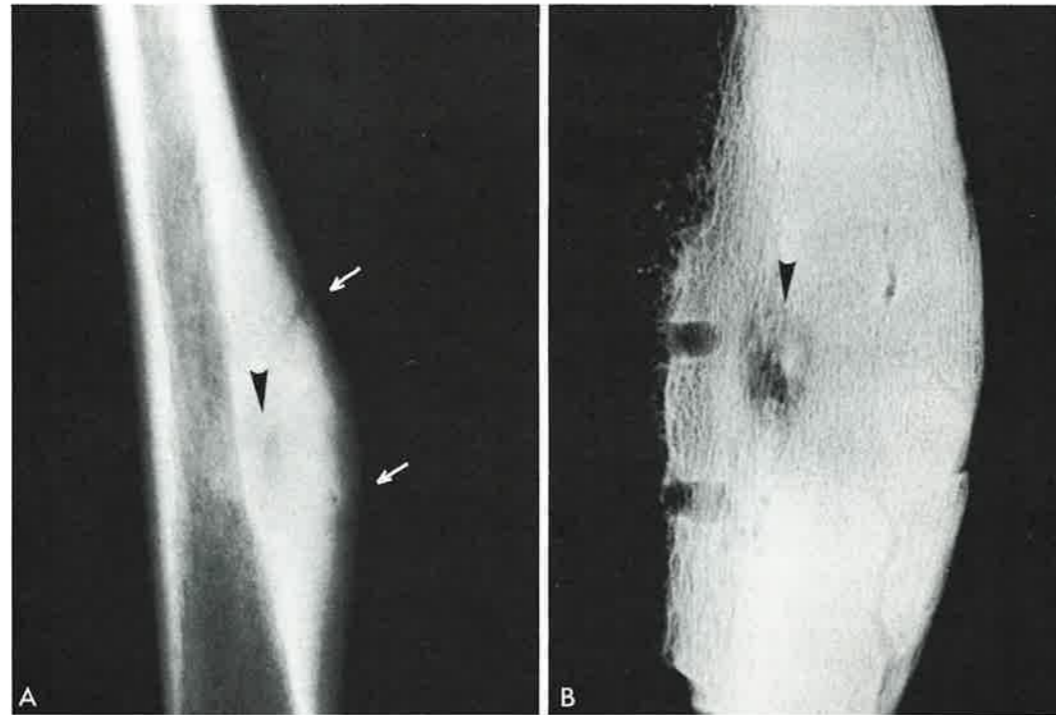


Figure 8. Solid continuous periosteal reaction. Osteoid osteoma of the femur in an 18 year old man. *A*, In the clinical radiograph, a small, lucent nidus is observed in a position near the original cortical surface (*arrow head*). A very thick, solid-appearing addition of new bone overlies and surrounds the nidus (*white arrows*). *B*, A specimen radiograph of a 4-mm thick slab of the resection specimen shows the nidus (*arrow*). The overlying and surrounding reactive new bone has a closely spaced, lamellated appearance which indicates that this spindle of new bone has been formed by apposition and incorporation of layer after layer of periosteal bone. The serpiginous lucent canals that move toward the nidus represent prominent vascular channels that supply the markedly hyperemic nidus. They can also be seen by careful inspection of the radiograph (*A*).

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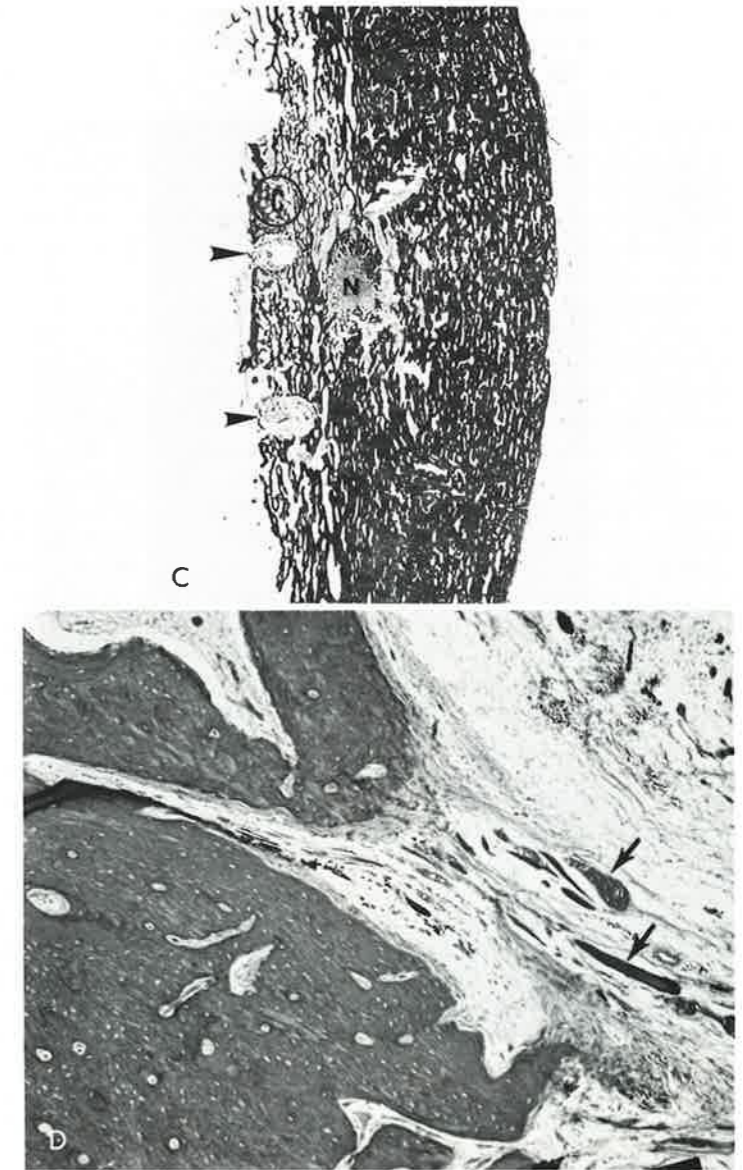


Figure 8 Continued. *C*, The original cortex (C) underlying the nidus (N) is more porous than the thick periosteal addition (magnification $\times 2.4$). The round cortical defects (*arrow heads*) are surgical drill holes and are also seen in the specimen radiograph (*B*). *D*, A selected photomicrograph (magnification $\times 63$) shows that, at its formative surface, the new bone emerges, in part, from edematous skeletal muscle interstitium. Residual skeletal muscle fibers are marked by arrows. (AFIP Neg. Nos. 80-11508; 81-257; 81-14591; 81-14550.)

tinued apposition to the lamellae. Still another mode of formation is analogous to that of early fetal cortical development in which an initial spicular ("streamer") woven bone network, produced by the periosteum, is subsequently filled in with an "inlay" of lamellar bone (Fig. 10). Lesions that often are accompanied by the solid reaction pattern include osteoid osteomas, large enchondromas (especially of the intertrochanteric zone of the femur [Fig. 9]), and eosinophilic granulomas.

Undulating Periosteal Reaction. The undulating periosteal reaction (Fig. 10) is a variant of solid reaction that can be seen with low grade osteomyelitis, long-standing varicosities, pulmonary osteoarthropathy, chronic lymphedema, periostitis, chronic osteitis, and rarely, with neoplasms.

Single Lamellar Reaction. The single lamella is a periosteal reaction consisting of one sheet of new bone (Fig. 11, *arrow*). It begins as a faint, radiodense line that is 1 to 2 mm from the cortical surface, and that may or may not join the cortex at its proximal and distal extremes. In three dimensions, such a reaction is a folded disc if partly circumferential, and a cylinder if completely circumferential. It consists of a woven bone network (Fig. 11B). As the "hallmark of a benign process,"¹⁵ especially when it is coarse or thick, it is often seen with acute osteomyelitis (Fig. 11), eosinophilic granuloma, and occasionally, with neoplasms. In time, succeeding lamellae may be added or the space between the shaft and reactive layer may fill in to produce a solid-appearing periosteal reaction.

Lamellated Reaction. The lamellated ("onion-skin") continuous reaction (Fig. 12) is created by concentric planes of ossification beyond the cortex. The inconstant finding of tumor between the lamellae, and the occurrence of this type of lamellated reaction in several non-neoplastic diseases (such as osteomyelitis, stress fracture, and hypertrophic pulmonary osteoarthropathy) negate the usual explanation that it results from cyclical variation in subperiosteal tumor growth with repeated phases of rapid periosteal elevation. The radiographically lucent zones between the new bone lamellae are initially occupied by prominent, dilated vessels in loose connective tissue (Fig. 12D). Only later are the spaces colonized by tumor, at first only focally (Fig. 12E).

In non-neoplastic conditions, the continuous lamellated periosteal reaction follows excessive ("explosive") cortical tunneling caused

by active hyperemia. When associated with tumor, this configuration may also relate to regional vascular adjustments. An alternative explanation is the presence of sequential osseous modulation within several adjacent parosteal fascial planes and collagenous septa normally found beside some major long bones. It may also occur in new fibrous planes created by involution of soft tissue.

The initial bone of a reactive lamella is a fine network of woven bone appearing in fibrous tissue (Fig. 12D). This becomes thickened by surface apposition of lamellar bone into a plate fenestrated by numerous vessels which cross at right angles (Fig. 12E). Prominent, dilated vessels in loose areolar tissue lie between the layers of bone. In long-standing reactions of this type, longitudinally oriented cement lines and surface osteoblastic activity testify to sequences of remodeling which, over time, make the layers progressively thicker. On each lamellation, osteoblastic activity⁷ tends to predominate on the surface facing soft tissue, and osteoclastic activity¹³ predominates on the side facing the cortex. Thus, through dynamic refinement, the layers tend to become radiographically more distinct with the passage of time. If the intervening soft tissue zones are nearly extinguished, the reaction may become "solid" or fused (see Figs. 8 and 15).

Lamellated, continuous periosteal reactions are seen with cellular bone sarcomas, especially Ewing's sarcoma and osteosarcoma (Fig. 12). Occasionally, eosinophilic granuloma in the very young patient and osteomyelitis (acute suppurative, and luetic) may present this pattern. It can be seen as a transient feature of normal growth.⁶

Parallel, Spiculated Reaction. The parallel, spiculated, continuous ("hair-on-end") reaction (Fig. 13) implies a more rapidly unfolding process than does a solid or lamellar reaction. The texture of individual spicules ranges from a uniform, fine, velvet-like texture to lengthy, linear shadows. The spicules show a gradual reduction in height in each direction along the shaft from the midzone of the reaction. When such a reaction is sectioned parallel to the bone surface, its true configuration is seen to be an interconnected compartmental system that resembles a honeycomb (Fig. 13, D and F). The spicular shadows are cast by compartment walls that happen to lie parallel to the radiographic beam. The honeycomb morphology is traceable to the regular spac-

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Figure 9. Solid continuous periosteal reaction. Active enchondroma involving the femur in a 49 year old man. A solid periosteal reaction has widened the bone's outline and thickened the cortex. The central, flocculent mineralization is calcified cartilage. Endosteal resorption is extensive and correlates with the increased cellular activity seen on histologic examination. (AFIP Neg. No. 72-18224-3.)

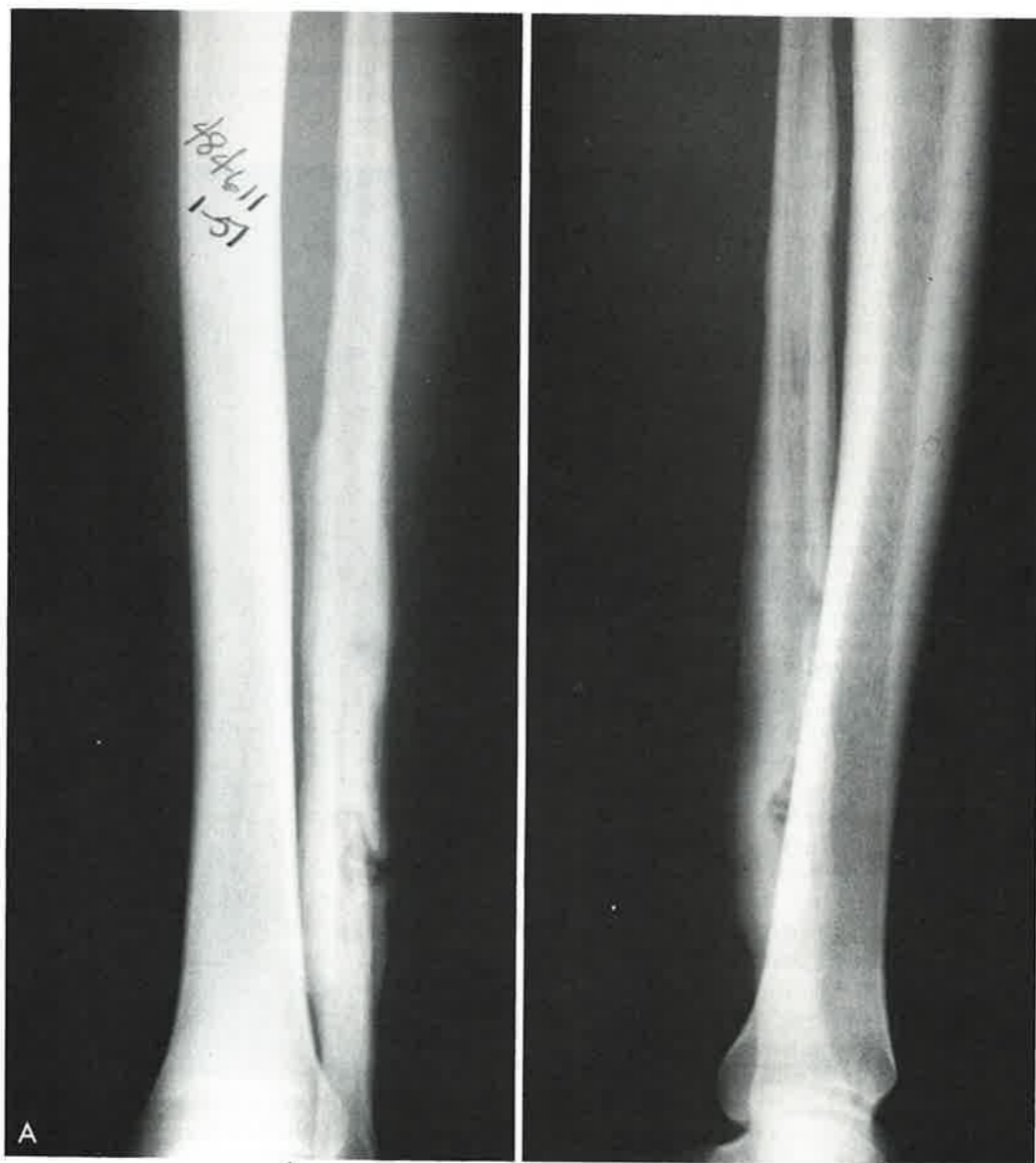


Figure 10. Solid undulating periosteal reaction. Chronic osteomyelitis of the fibula. A, Clinical radiographs reveal the varying thickness of the periosteal reaction pattern and endosteal sclerosis.

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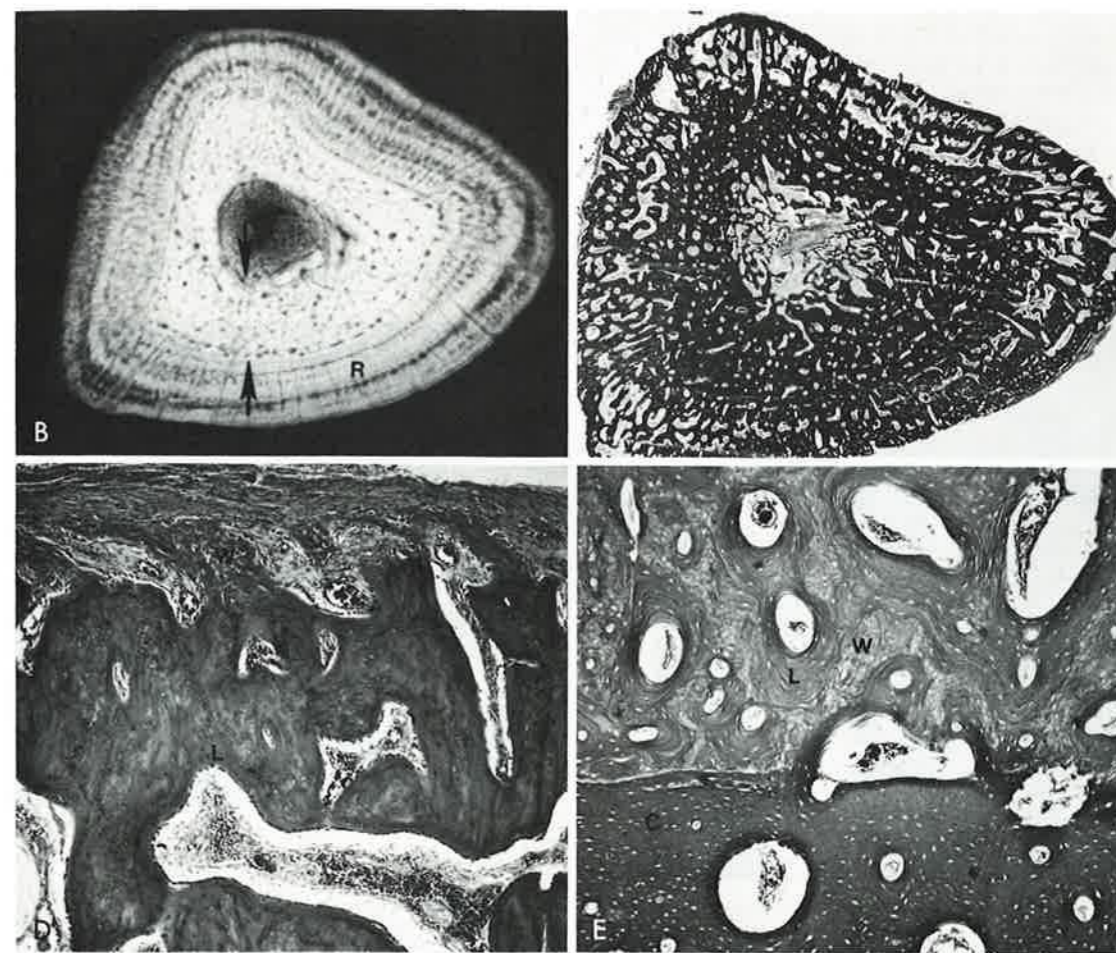


Figure 10 Continued. A specimen radiograph of a fibular cross-section B and its corresponding whole-mount histologic section (C, magnification $\times 4.6$) reveal a circumferentially layered addition of new bone (R) which now approximates the thickness of the original fibular cortex (area between the arrows). The fibular cortex is seen to be more compact and dense, and some medullary new bone (endosteal sclerosis) is seen near the very center. D, A photomicrograph (magnification $\times 63$) of the outer limit of the periosteal reaction shows streamers of new woven bone (W) extending into the parosteal soft tissue. Deeper within the periosteal reaction (L), rows of osteoblasts deposit lamellar bone on the earlier formed woven bone, a process which is analogous to early fetal cortical growth. E, Another photomicrograph (magnification $\times 63$) indicates that an "inlay" ossification process is filling in the spaces in the depth of the reaction where it meets the original cortex (C). (Reticulin stain). (W, woven streamer bone; L, inlay bone) (AFIP Neg. Nos. 15-5-1; 81-14573; 81-14580; 81-14638; 81-14508.)

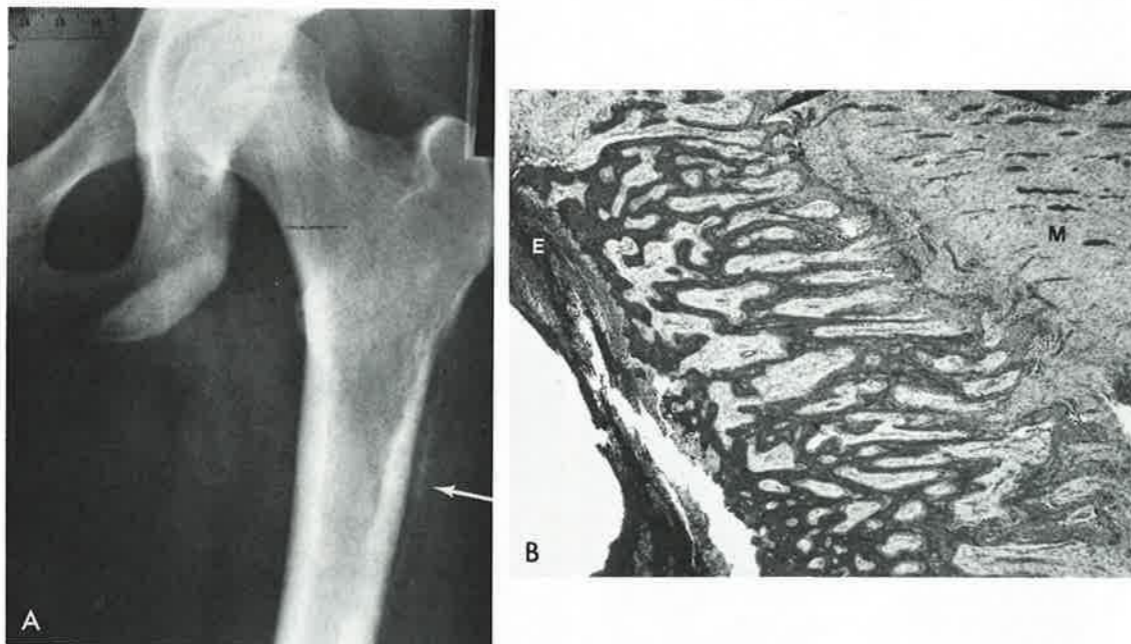


Figure 11. Single lamella periosteal reaction. Acute osteomyelitis of the femur in a 13 year old girl. *A*. A clinical radiograph shows a single continuous lamella of reactive new bone (arrow) just beyond the outer cortical limit where cortical osteolysis is evident. *B*. A photomicrograph (magnification $\times 40$) of the single lamella reveals that it consists of streamers of an interconnected osseous network which appears to be formed by osteoblasts modulating directly from the fibrotic interstitium of adjacent atrophic skeletal muscle (M). Purulent exudate (E) of the osteomyelitis is present beneath the reactive layer. (AFIP Neg. Nos. 79-1983; 81-14548.)

ing of radially oriented, enlarged periosteal vessels that extend from periosteum to cortex and the tendency of osteoblasts to become active in the region midway between adjacent vessels. The loose areolar tissue in individual compartments may, in time, be replaced, for instance, by tumor in Ewing's sarcoma (Fig. 13E) or red marrow in the calvarial reaction of thalassemia. This pattern is not usually seen with benign tumors. Certain inflammatory states, such as syphilis, myositis, and Caffey's disease, can stimulate spicular growth,¹ as well as lamellated reactions.

INTERRUPTED PERIOSTEAL REACTIONS

The route by which a bone tumor traverses the cortex and attains a subperiosteal position may or may not be apparent radiographically. Percolation through enlarged Volkmann's canals and haversian spaces often leaves the cortex radiographically intact (Fig. 18, *A* and *C*). The other extreme is the complete cortical destruction that is seen radiographically with direct extension of neoplasm into the periosteal reaction (Fig. 20A). Two mechanisms result in less prominent periosteal bone when tumor colonizes the soft tissue space within a periosteal reaction. The first, occupation by tumor, denies space for elaboration of new bone, and the second, pressure by the tumor (especially as sarcomatous matrix synthesis occurs), stimulates osteoclasts to remove the reactive bone (Figs. 18 and 19). Through these mechanisms, an interrupted periosteal reaction is formed.

"When periosteal accretions are broken down in the middle so that they form a cuff on either side of the primary focus (i.e., interrupted periosteal reactions), the appearances are more likely to be those of sarcoma."¹ The key word is "likely." Other clues, such as evidence of mineralizing tumor matrix in the area of tumor breakout or lytic margins, or both, should be integrated with the periosteal reaction data before a radiographic diagnosis is established.

Buttress. A solid-appearing wedge of reactive bone, or a buttress, tends to form at the lateral extraosseous margins of slowly enlarging bone lesions (Fig. 14). The cortex beneath the buttress is frequently intact, whereas the cortex just beyond it is usually deleted, with or without creation of a shell. Such a re-

action may, on histologic examination, bear evidence that it was originally lamellated and filled in, through osteoblastic apposition, at the expense of the intervening fibrovascular tissue (Fig. 15). Buttresses may be seen at the lateral margin of shell-type periosteal reactions (Fig. 14) or they may be created by central destruction of an originally continuous solid reaction (Fig. 15). The latter process should prompt consideration of a malignant change in a long-standing, antecedent, benign lesion, such as an enchondroma or a cyst⁹ (Fig. 15). With periosteal or outer cortical lesions, the cortex beneath the tumor may remain intact while a buttress forms at the edge.

Codman Angle. Following its initial description by Ribbert in 1914, Codman⁴ drew further attention to "the little cuff of reactive bone trumpet shape which surrounds the upper limit of [a] tumor" and "appears in the x-ray as a triangular space on each side of the shaft." Although a "sure indication of subperiosteal extracortical involvement, this reactive triangle in itself is not diagnostic of sarcoma" since it "sometimes occurs as a defense against inflammation." Brailsford reports similar periosteal cuffs in acute osteomyelitis aborted by drug treatment,¹ and Edeiken reports their occurrence with subperiosteal hematoma.⁵ However, most of the time, this finding is the harbinger of an aggressive, malignant lesion which has broken out into soft tissue.

The term Codman "angle" is preferred to "triangle" in the usual example since the side facing the tumor is lucent (Fig. 16). This focus of reactive periosteal bone occupies the space between the shoulder of the neoplastic mass and the cortical line, and may be unilateral, thus appearing only in certain projections. The outermost plane of reactive bone is usually the thickest (Figs. 16A and 21B).

Codman angles are usually tumor-free, but tumor cells may infiltrate, individually or en masse, into the angle from its open end (Fig. 16D) or through the cortex and within vascular channels from below. Although labile and changing (Fig. 19, *A* and *C*), Codman angles are not necessarily completely resorbed after being overrun by tumor. Figure 17 presents successive illustrations of three somewhat spiculated Codman angles on the posterior surface of a femur which were buried by sarcoma.

Interrupted Lamellated Reaction. The abrupt histologic truncation of the inter-

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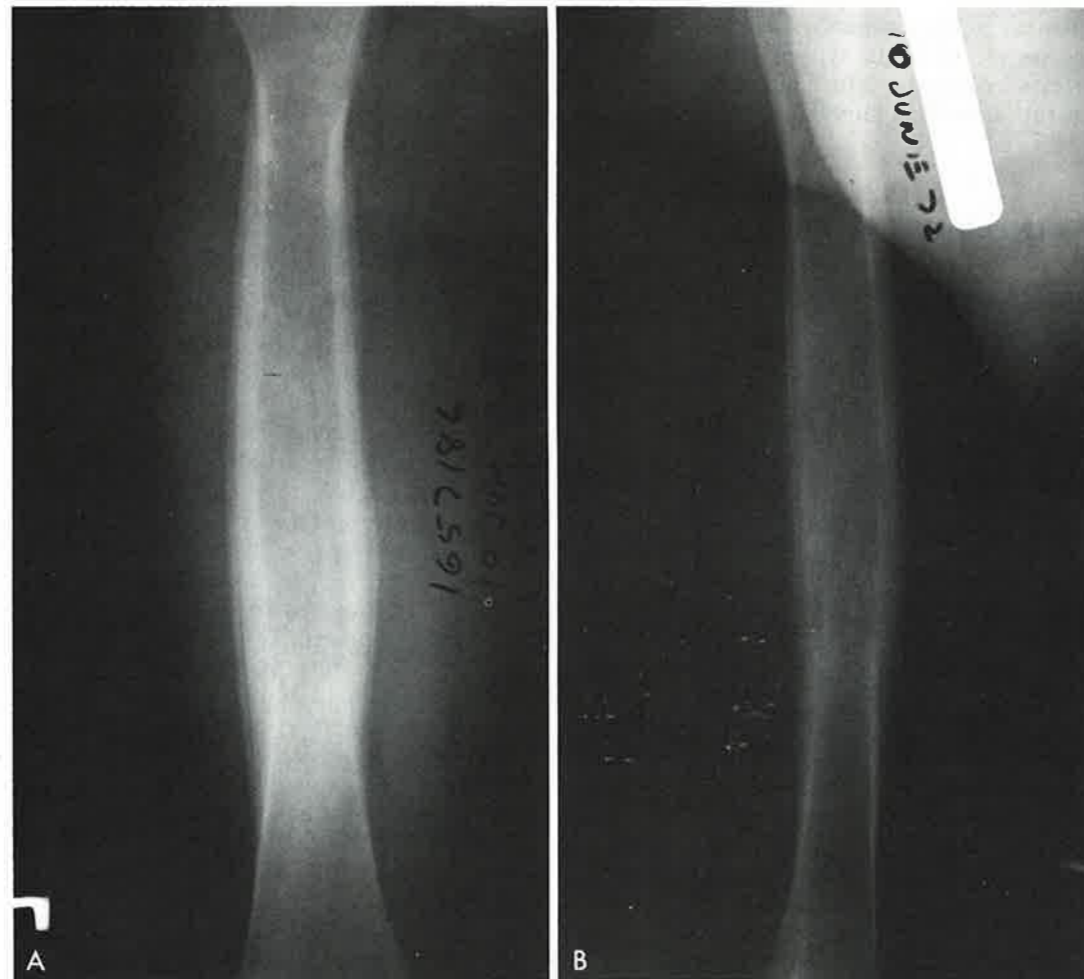


Figure 12. Lamellated ("onion-skin") periosteal reaction. Osteosarcoma of the femur in a six year old boy. *A* and *B*, Anteroposterior and lateral radiographs show multiple continuous lamellae widening the femoral contour. The underlying cortex is partially destroyed. No matrix density pattern is seen because tumor osteoid was scant and poorly mineralized. *C*, Individual lamellae are best seen near the arrows in this longitudinal macroscopic view (magnification $\times 3$) of the proximal shaft specimen.

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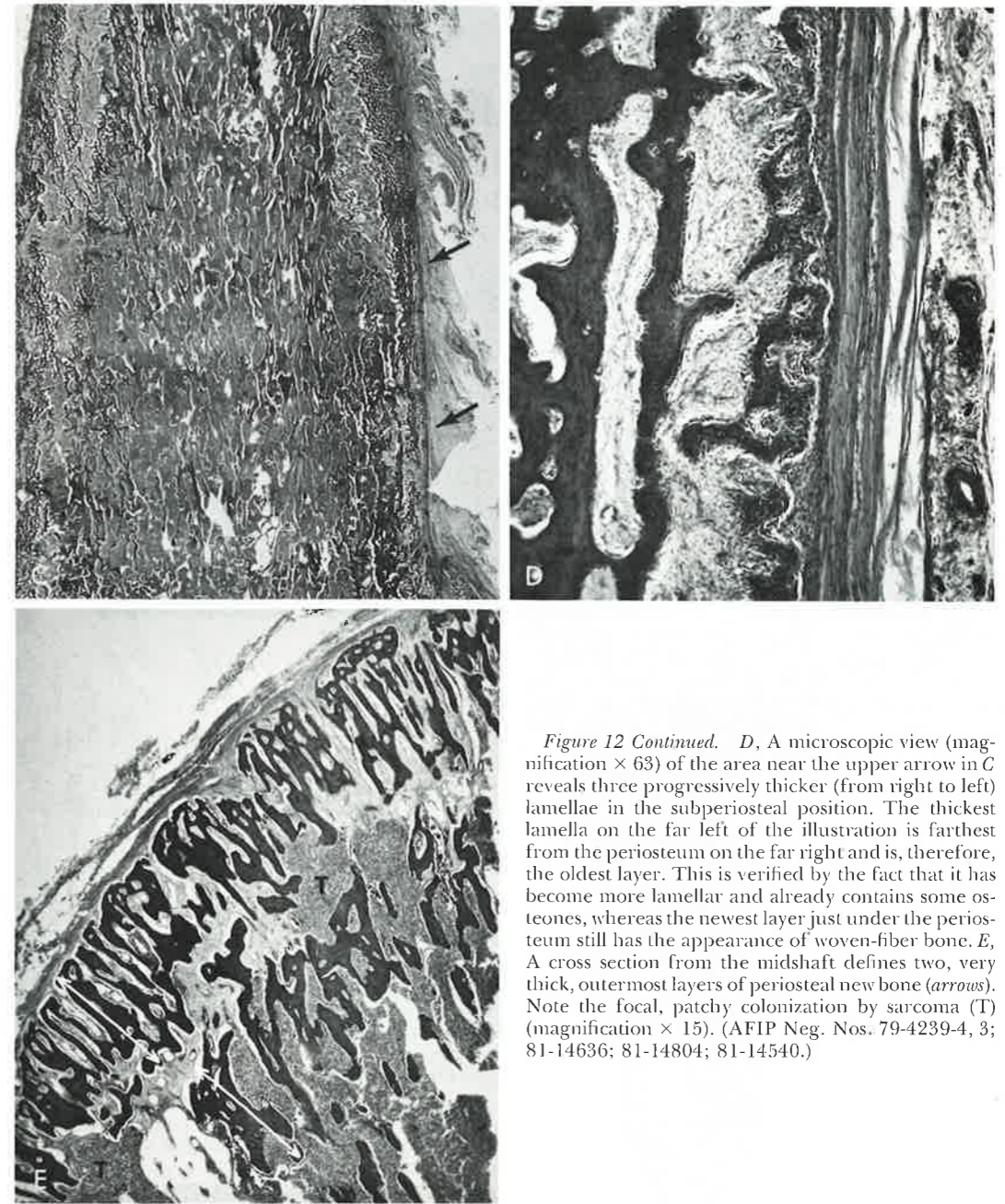


Figure 12 Continued. *D*, A microscopic view (magnification $\times 63$) of the area near the upper arrow in *C* reveals three progressively thicker (from right to left) lamellae in the subperiosteal position. The thickest lamella on the far left of the illustration is farthest from the periosteum on the far right and is, therefore, the oldest layer. This is verified by the fact that it has become more lamellar and already contains some osteons, whereas the newest layer just under the periosteum still has the appearance of woven-fiber bone. *E*, A cross section from the midshaft defines two, very thick, outermost layers of periosteal new bone (arrows). Note the focal, patchy colonization by sarcoma (T) (magnification $\times 15$). (AFIP Neg. Nos. 79-4239-4, 3; 81-14636; 81-14804; 81-14540.)

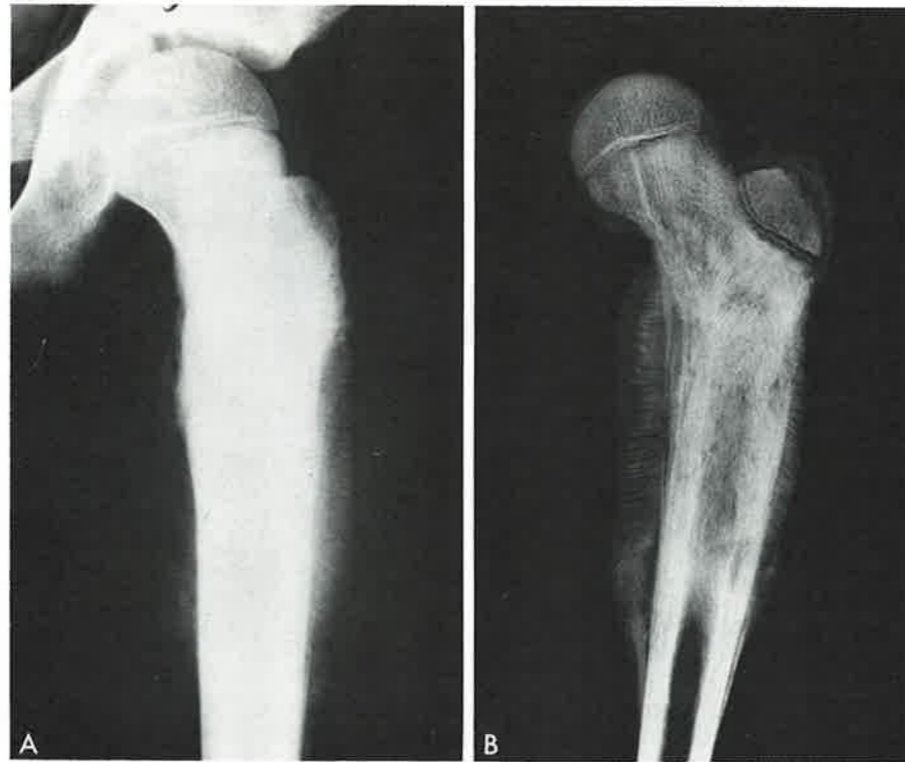


Figure 13. Parallel spiculated ("hair-on-end") periosteal reaction. Ewing's sarcoma of the femur in a 10 year old boy. *A*, The clinical radiograph features a spiculated reaction which is most prominent along the lateral femur. *B*, The specimen radiograph of a 4-mm thick longitudinal slab records the thin linear appearance of the periosteal spiculation and its decreasing amplitude proximally and distally. The underlying cortex is the site of permeative osteolysis and the cancellous bone is partially resorbed. *C*, Correlation of this whole-mount histologic section (magnification $\times 1.1$) with the specimen radiograph (*B*) confirms that the medullary extent of the tumor is from the proximal growth plate to the distal end of the specimen.

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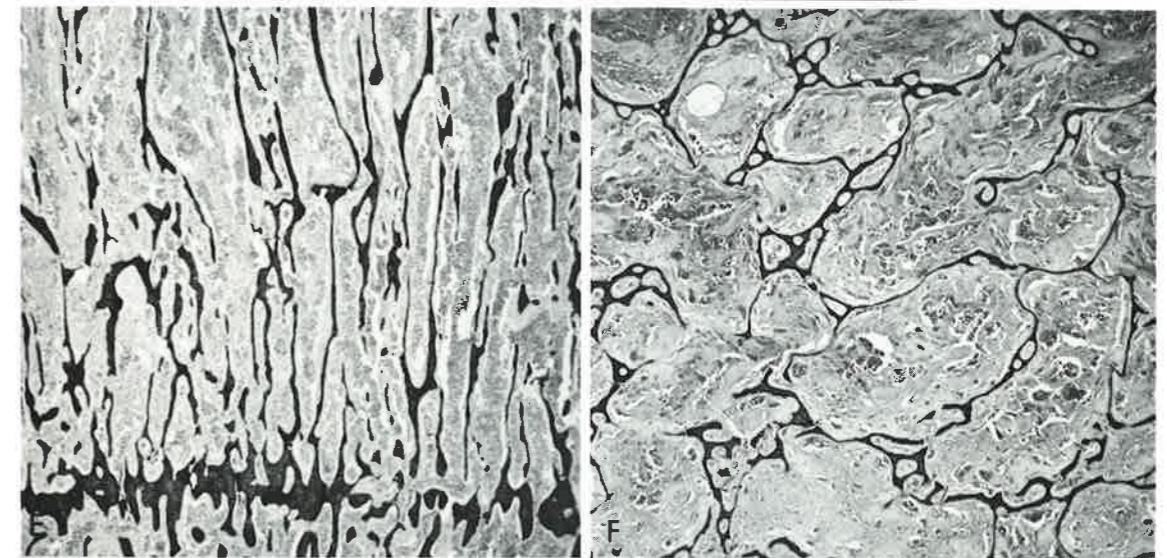
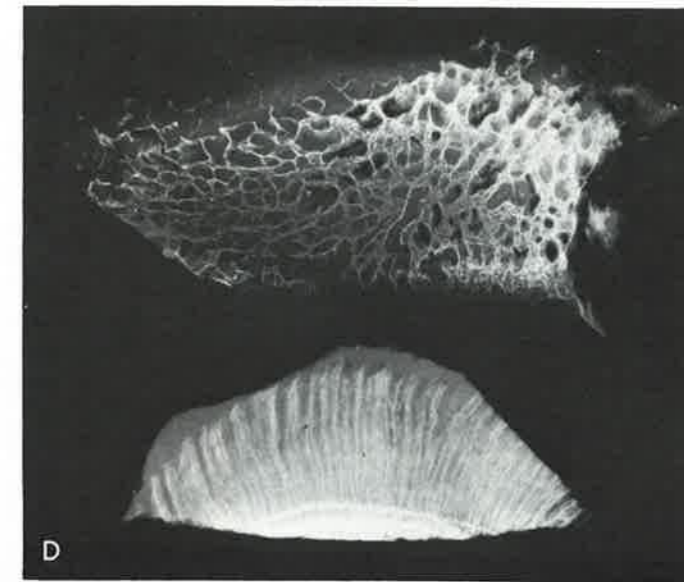


Figure 13 Continued. *D*, The specimen radiograph at the bottom represents an outer-quadrant cross section (magnification $\times 2.5$) and shows the thin linear configuration and circumferential distribution of the periosteal spiculation. The specimen radiograph at the top is a longitudinal, 2 mm-thick, slab section (magnification $\times 2.5$) of the periosteal reaction, which reveals its honeycomb configuration. The apparent spiculation on the radiographs is caused by those honeycomb chamber walls which happen to be oriented parallel to the beam. *E* and *F*, Histologic sections (magnification $\times 15$) of the same specimens shown in *D* confirm that the plane of view determines whether linear spicules or an interconnected osseous network honeycomb (*F*) are seen. Tumor cells occupy most of the intervening soft tissue spaces. (AFIP Neg. Nos. 81-14665; 566653-2; 81-14664; 81-14496; 81-14501; 81-14499.)

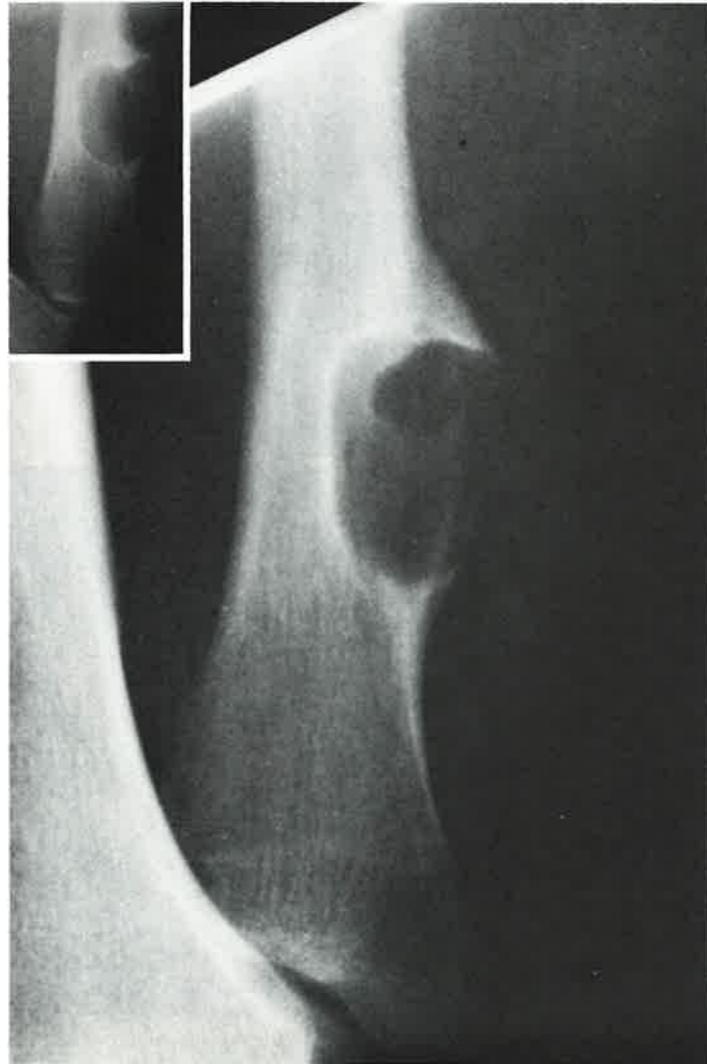


Figure 14. Buttress interrupted periosteal reaction. Chondromyxoid fibroma of the fibula in a 20 year old man. Anteroposterior (*inset*) and lateral views demonstrate an eccentric lucent lesion in the metadiaphysis, with a sclerotic margin and an interrupted shell. A buttress periosteal reaction smooths the contour where the shell and original cortex meet. (AFIP Neg. No. 75-13296.)

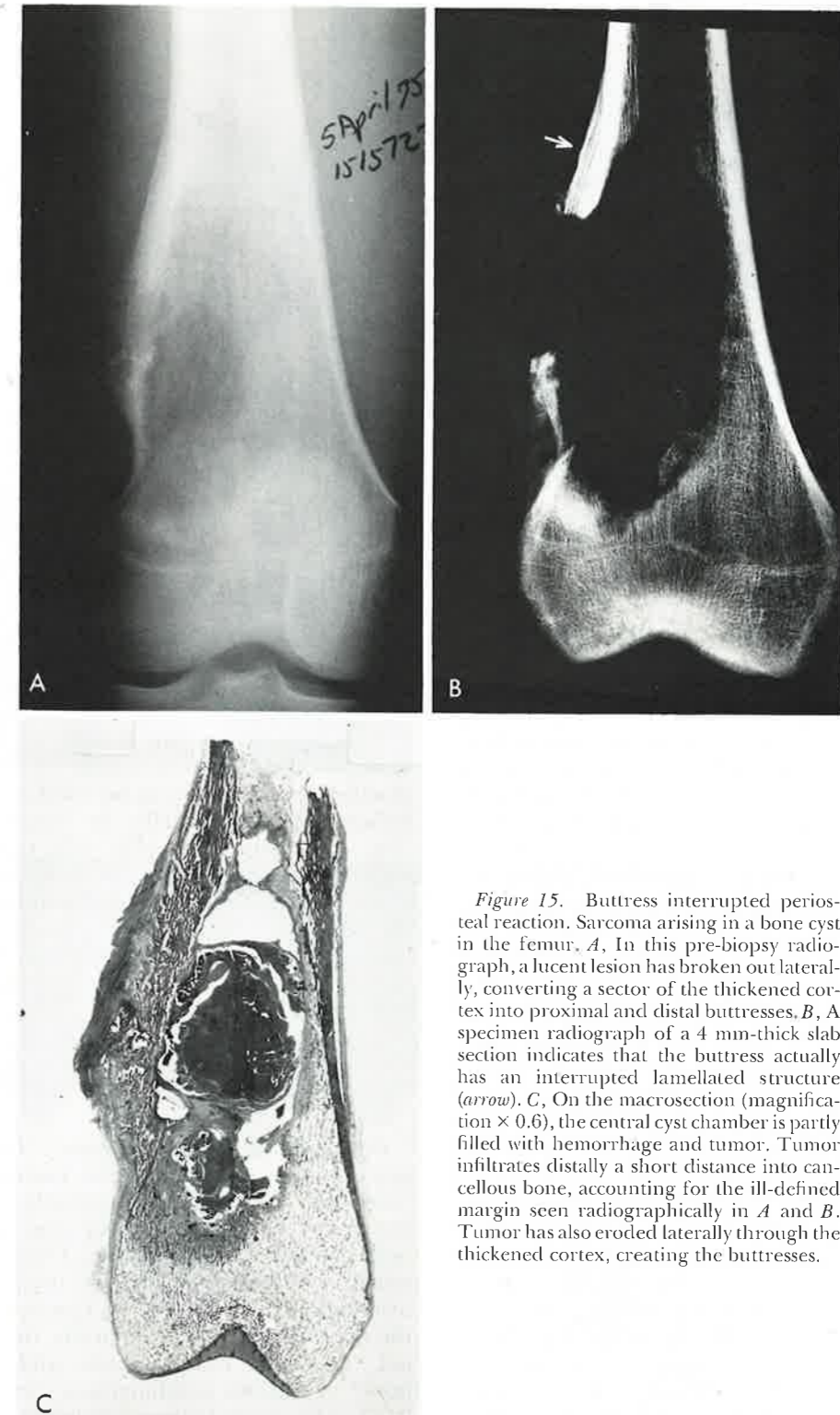


Figure 15. Buttress interrupted periosteal reaction. Sarcoma arising in a bone cyst in the femur. *A*, In this pre-biopsy radiograph, a lucent lesion has broken out laterally, converting a sector of the thickened cortex into proximal and distal buttresses. *B*, A specimen radiograph of a 4 mm-thick slab section indicates that the buttress actually has an interrupted lamellated structure (*arrow*). *C*, On the macrosection (magnification $\times 0.6$), the central cyst chamber is partly filled with hemorrhage and tumor. Tumor infiltrates distally a short distance into cancellous bone, accounting for the ill-defined margin seen radiographically in *A* and *B*. Tumor has also eroded laterally through the thickened cortex, creating the buttresses.

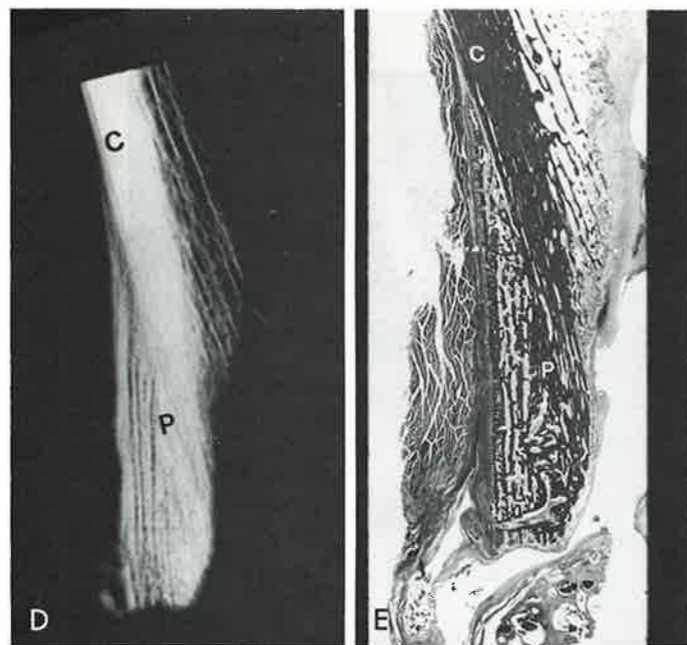


Figure 15 Continued. D and E, A specimen radiograph (magnification $\times 1.8$) and corresponding histologic section (magnification $\times 2.1$) show the truncated original cortex (C) and buttress interrupted periosteal addition (P). (AFIP Neg. Nos. 75-8624-1; 81-14519; 77-7771; 81-14593; 81-14505.)

rupted lamellated reaction (Fig. 18) and the presence of numerous Howship's lacunae where the layers terminate into tumor fit the concept that the lamellated reaction is continuous prior to tumorous invasion. Occasional cases have afforded sufficient serial radiographs to document central removal of previously continuous lamellae (Fig. 19). A benign condition with which this reactive pattern is associated is eosinophilic granuloma in young children, which is particularly prone to stimulate complete lysis of the cortex and to invade deeper layers of the reaction rapidly.

Interrupted Spiculated Reaction. Central invasion and destruction of a spiculated reaction is one means of creating an interrupted spiculated reaction, or spiculated wedge at the side of a tumor. More often it is a localized, reactive condensation at the lateral border of a juxtacortical neoplastic mass. Such a wedge appears to "fill in" the contour between the side of the mass and the shaft of the bone, just as molding does at the point where the floor meets the wall. This can be seen with solid and cystic benign and malignant tumors of medullary origin (Fig. 20) or with exclusively surface lesions, such as parosteal sarcomas. The common denominator is rapid development of an extraosseous mass.

Spiculated wedges usually have a homoge-

neous density on clinical radiographs and seldom look "spiculated." Furthermore, solid, lamellated, and spiculated reactions often cannot be distinguished in clinical radiographs because the fine details are lost as a result of density summation. A periosteal reaction that appears to be solid on clinical radiographs can actually be "nearly solid" (Fig. 8) or lamellated (Fig. 15), tightly spiculated, or any gradation between. A transition from lamellated to spiculated interrupted patterns (Fig. 22, B and C) occurs frequently and suggests an acceleration in the rate of formation from the surface of the bone outward.

COMPLEX PERIOSTEAL PATTERNS

Divergent Spiculated Patterns. The divergent spiculated pattern, also known as the "sunburst" (Fig. 21), is commonly a sign of malignant osteoid production and only partially consists of reactive bone. The individual streaks of density of variable thickness and orientation point toward an epicenter within the marrow space. Histologically, the individual "rays" are reactive bone and sarcoma bone² or various combinations thereof (Fig. 21E). The space between individual rays in osteosarcoma is occupied by cellular tumor and tumor products, usually chondroid and

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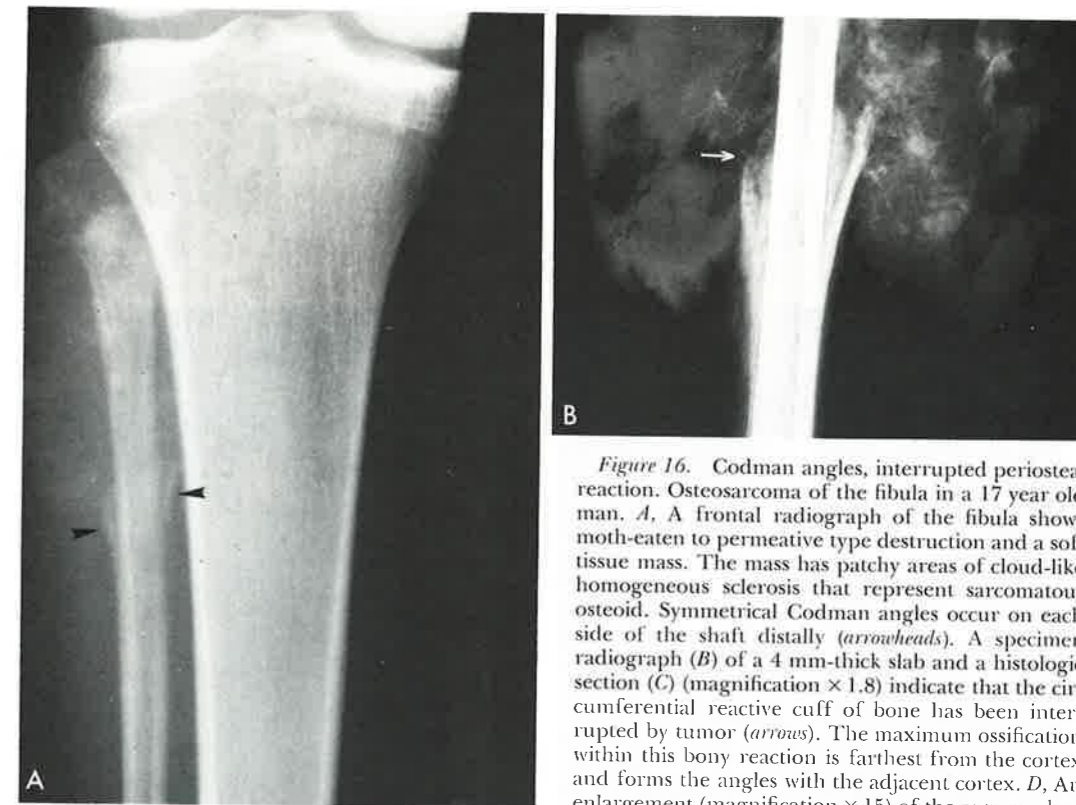
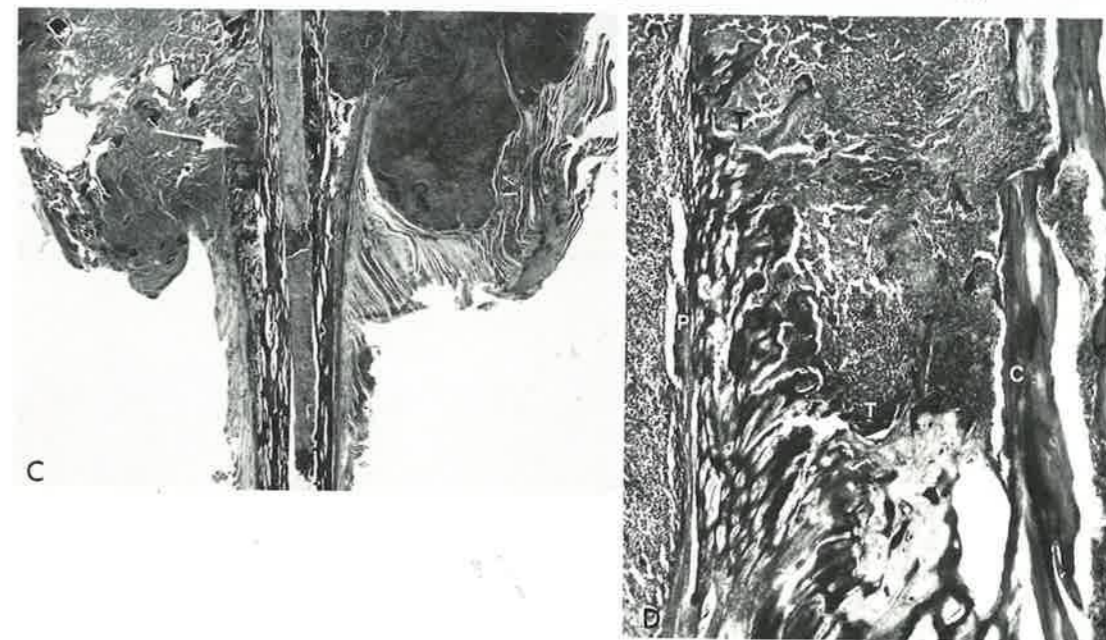


Figure 16. Codman angles, interrupted periosteal reaction. Osteosarcoma of the fibula in a 17 year old man. A, A frontal radiograph of the fibula shows moth-eaten to permeative type destruction and a soft tissue mass. The mass has patchy areas of cloud-like homogeneous sclerosis that represent sarcomatous osteoid. Symmetrical Codman angles occur on each side of the shaft distally (arrowheads). A specimen radiograph (B) of a 4 mm-thick slab and a histologic section (C) (magnification $\times 1.8$) indicate that the circumferential reactive cuff of bone has been interrupted by tumor (arrows). The maximum ossification within this bony reaction is farthest from the cortex and forms the angles with the adjacent cortex. D, An enlargement (magnification $\times 15$) of the area marked by the arrow in C reveals tumor (T) filling the open end of the angle. (C, cortex; P, periosteum.) (AFIP Neg. Nos. 79-6000; 81-14554; 81-14555; 81-14551.)



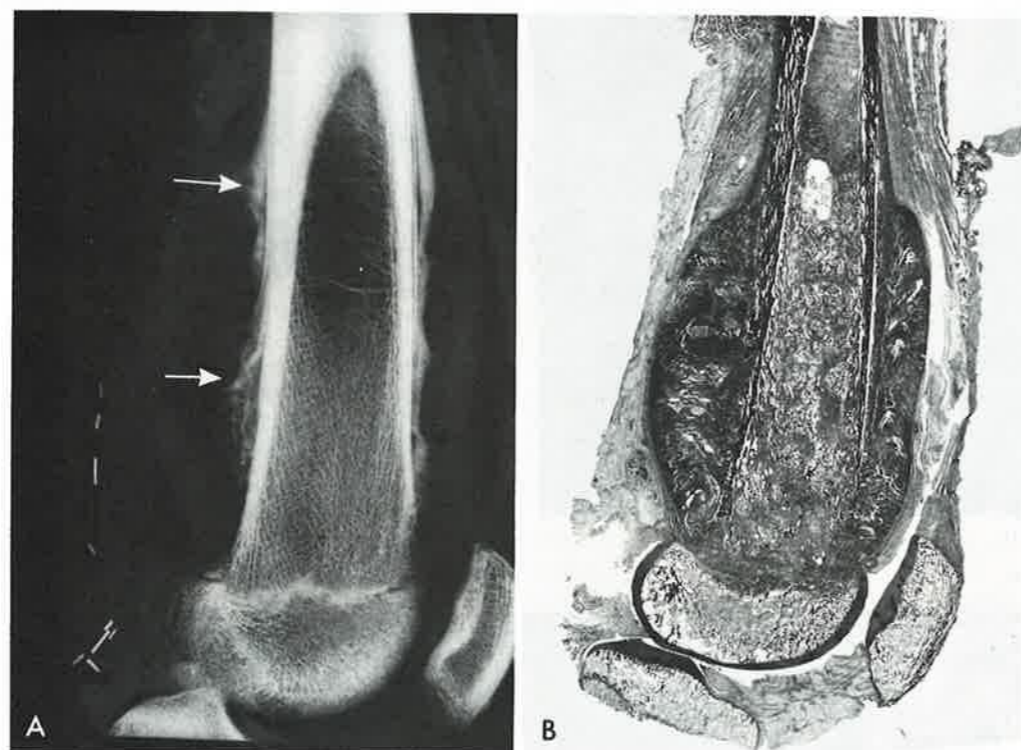


Figure 17. Codman angles, interrupted periosteal reaction (multiple). Chondrosarcoma of the femur in a 13 year old girl. *A*, In the specimen radiograph, a sequence of three overlapping, slightly spiculated Codman angles (arrows) on the posterior surface of the femur have been buried by progressive extraosseous growth of this originally intramedullary tumor. *B*, The slab specimen (magnification $\times 0.7$) demonstrates this very effectively. (AFIP Neg. Nos. 81-14656; 81-14577.)

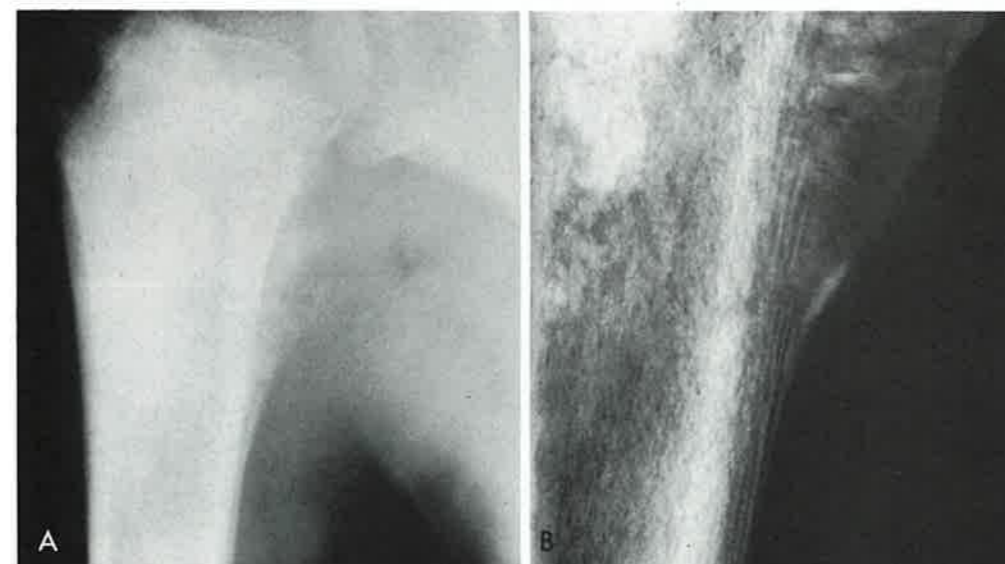


Figure 18. Lamellated interrupted periosteal reaction. Osteosarcoma of the humerus in a 19 year old man. *A*, In the radiograph, an angled periosteal reaction terminates at the medial distal margin of the tumorous soft tissue extension. Diffuse sclerosis with homogeneous density within and beside the bone is osteosarcomatous matrix. The lateral periosteal reaction is more uniform. *B*, The radiograph (magnification $\times 1.5$) of a slab specimen (magnification $\times 1.5$) indicates that the Codman angle at the medial humeral metaphysis consists of multiple lamellae that terminate in the sarcoma. Partially mineralized sarcomatous osteoid occupies the medullary space.

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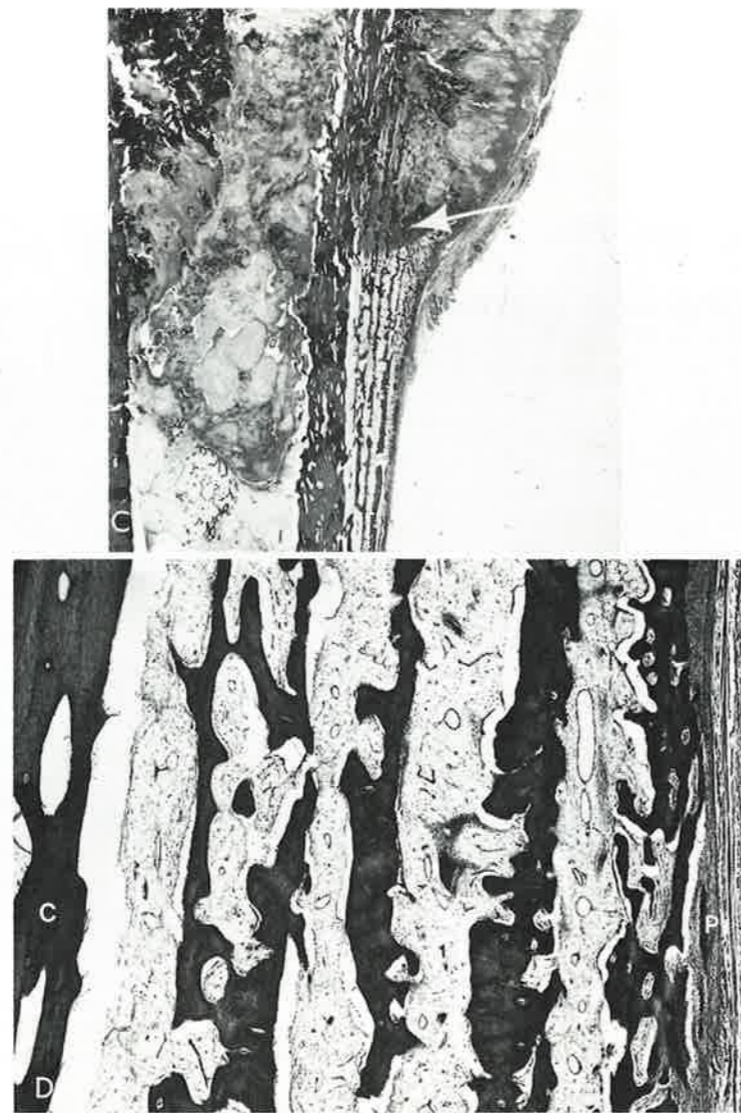


Figure 18 Continued. C, The macrosection (magnification $\times 2$) that corresponds to B confirms the multilayered appearance and the interruption of the reaction by sarcoma (arrow). D, A histologic view (magnification $\times 21$) of the lamellated reaction reveals loose, well-vascularized areolar tissue between adjacent lamellae. (C, original cortex; P, the periosteum.) Again, note the progressive maturation (from fiber bone to cortical [lamellar] bone) of the layers as they proceed from right (just under the periosteum) to left (toward the cortex). (AFIP Neg. Nos. 57-8981-2; 81-14598; 81-14597; 70-11524.)

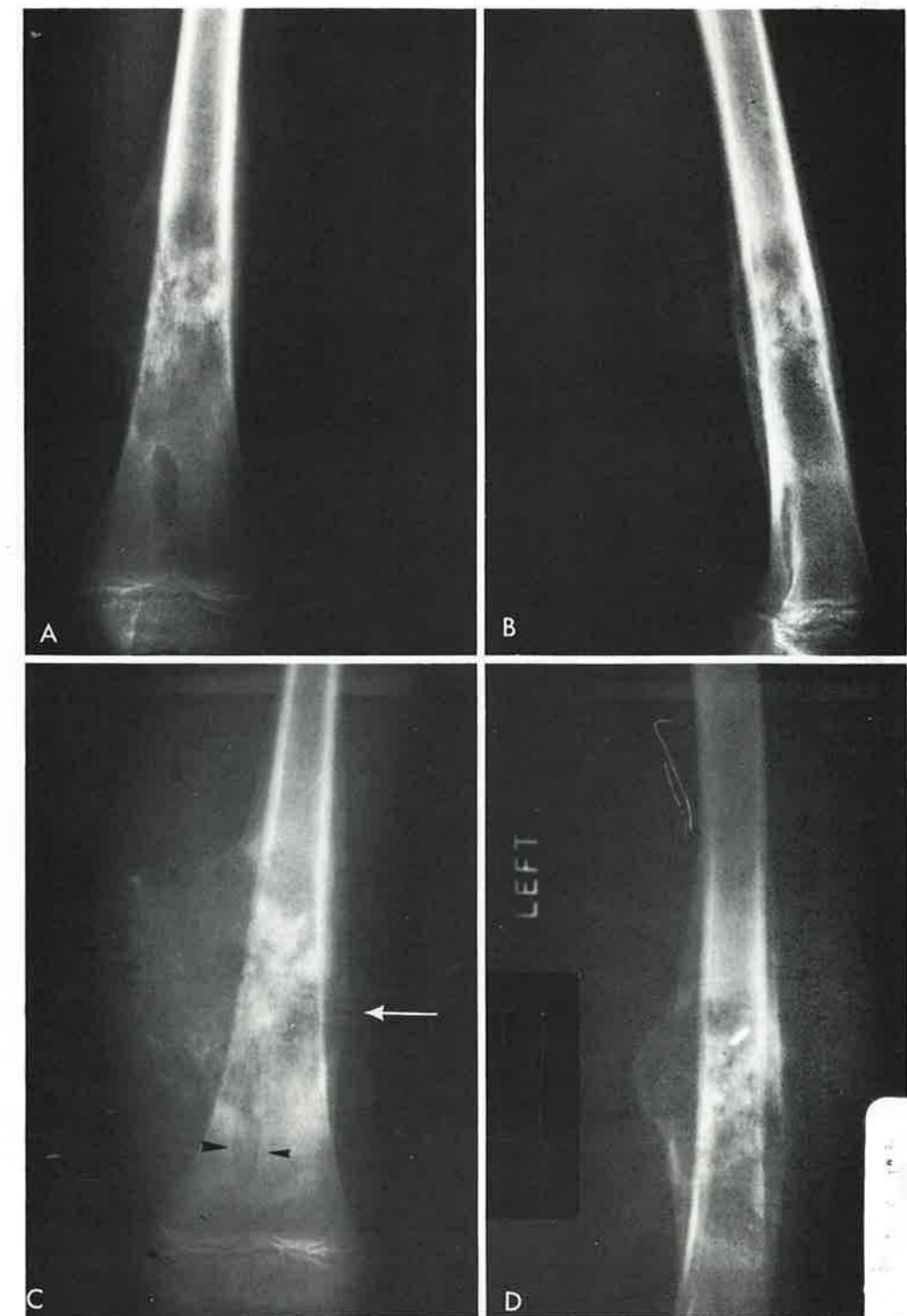


Figure 19. Conversion of a lamellated continuous reaction into a lamellated interrupted periosteal reaction. Osteosarcoma of femur in an eight year old boy. The interval of time between radiographs A and B versus C and D is 20 days. What was originally a continuous reaction on the posterior surface B is interrupted by the extraosseous extension of tumor (D). A comparison of A and C shows progression of the lamellated reaction along the lateral aspect of the femur, with early tumorous encroachment distally in C (arrow). The distal ovoid lucency (arrowheads) is probably an incidental benign lesion, such as a fibrous cortical defect. (AFIP Neg. No. 79-5920.)

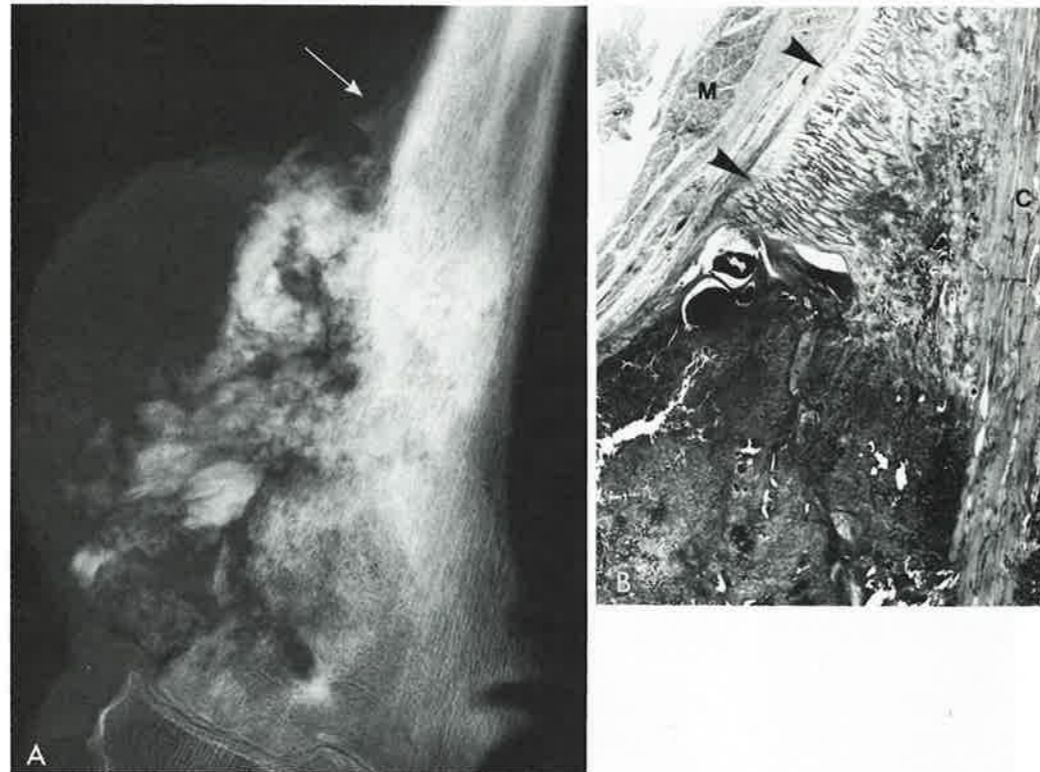


Figure 20. Spiculated interrupted periosteal reaction. Osteosarcoma of the femur. The small, wedge-shaped reaction (arrow) in the slab radiograph (A) correlates with a truncated, spiculated reaction (arrowheads) in the histologic view (B) (magnification $\times 3.5$). (C, original cortex; M, skeletal muscle.) (AFIP Neg. Nos. 59-6677; 70-11538.)

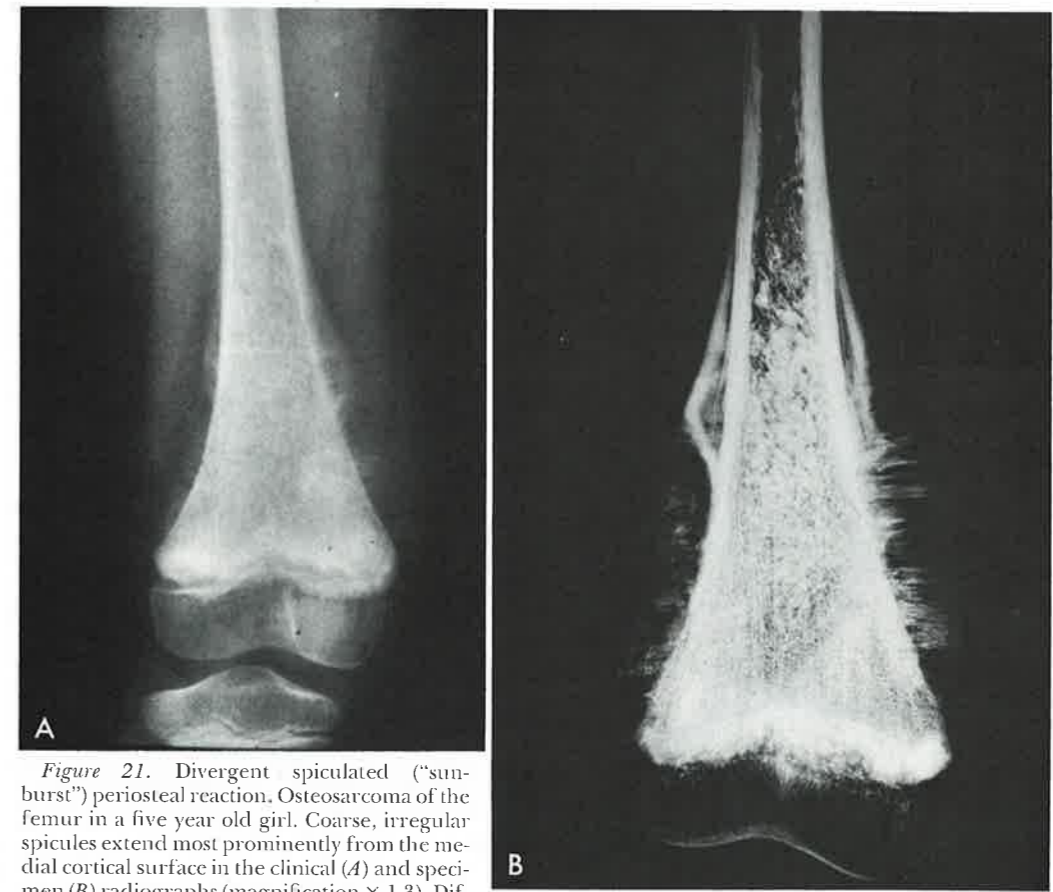


Figure 21. Divergent spiculated ("sunburst") periosteal reaction. Osteosarcoma of the femur in a five year old girl. Coarse, irregular spicules extend most prominently from the medial cortical surface in the clinical (A) and specimen (B) radiographs (magnification $\times 1.3$). Diffuse sclerosis in the marrow space represents mineralizing tumor osteoid.

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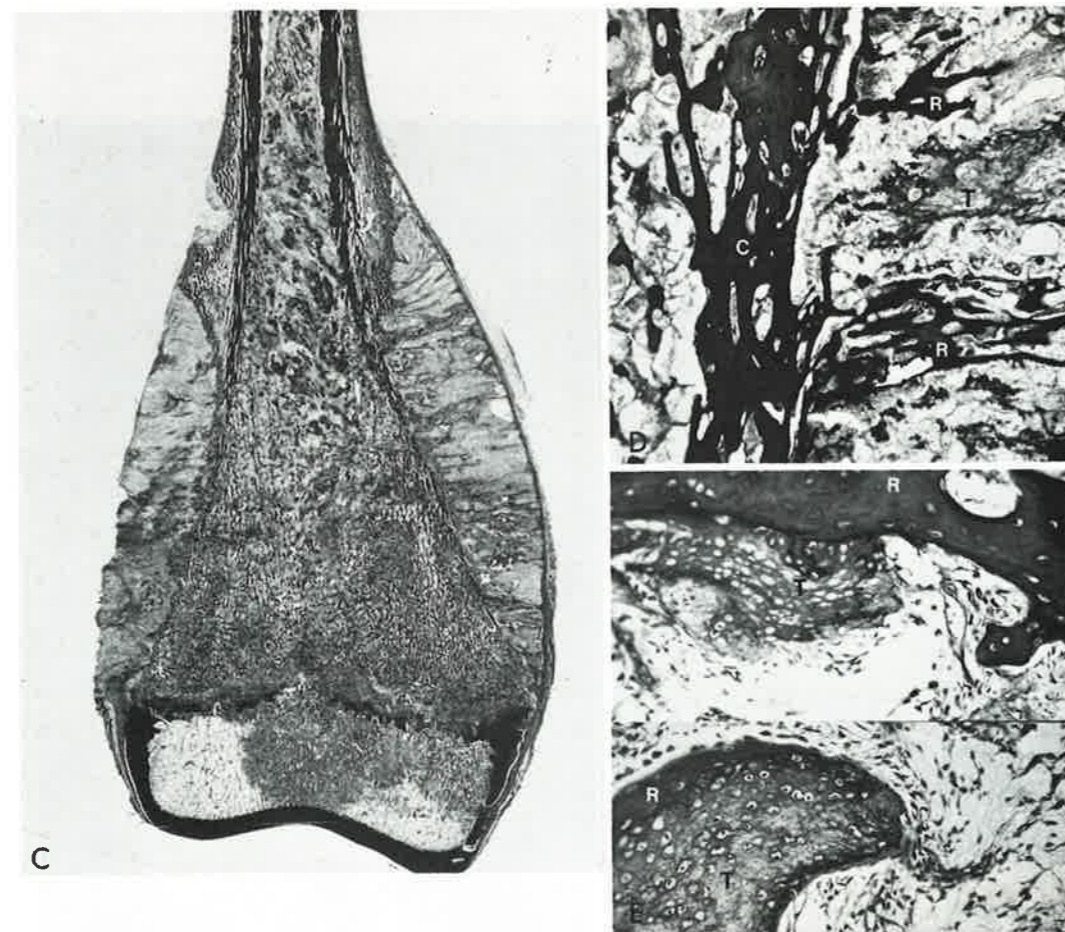


Figure 21 Continued. C, The corresponding slab section (magnification $\times 1.4$) confirms the irregular spacing and width of the spicules and shows their tendency to diverge. The tumor has filled the marrow space extensively and has crossed the fenestrated growth plate into the epiphysis. D, A photomicrograph (magnification $\times 15$) within the "sunburst" zone shows a combination of tumor (T) and reactive (R) bone to the right of the cortex (C). Both contribute to the radiographic impression of spiculation and "sunburst," as further documented in E. E, "Hybrid" spicules can consist of reactive bone (R) with superimposed tumor (T) matrix additions, as shown in the top field of this illustration (magnification $\times 157$), or they may be composed of tumor matrix spicules with superimposed reactive bone additions, as is shown in the bottom field (magnification $\times 157$). (AFIP Neg. Nos. 81-702; 81-14663; 81-14662; 81-14517; 81-14518.)

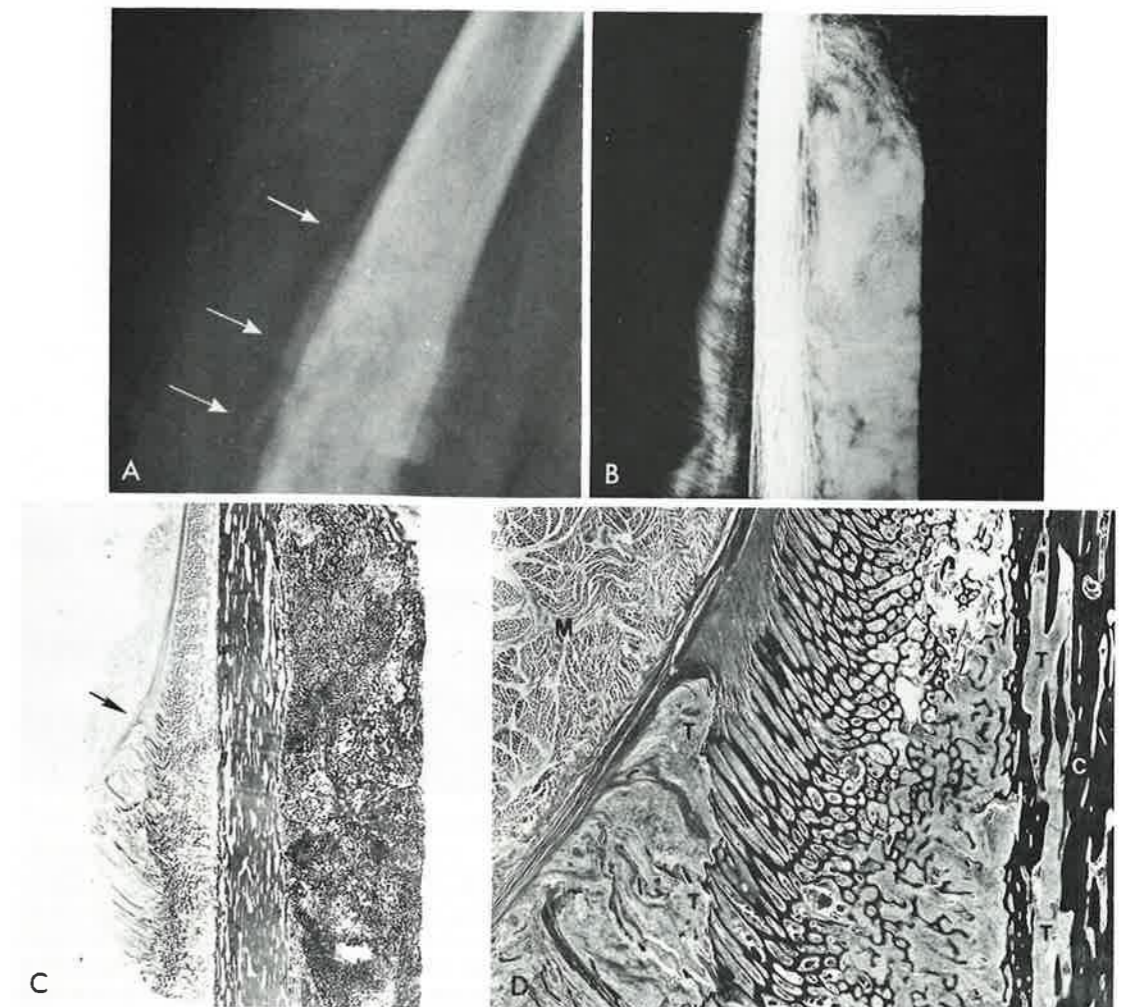
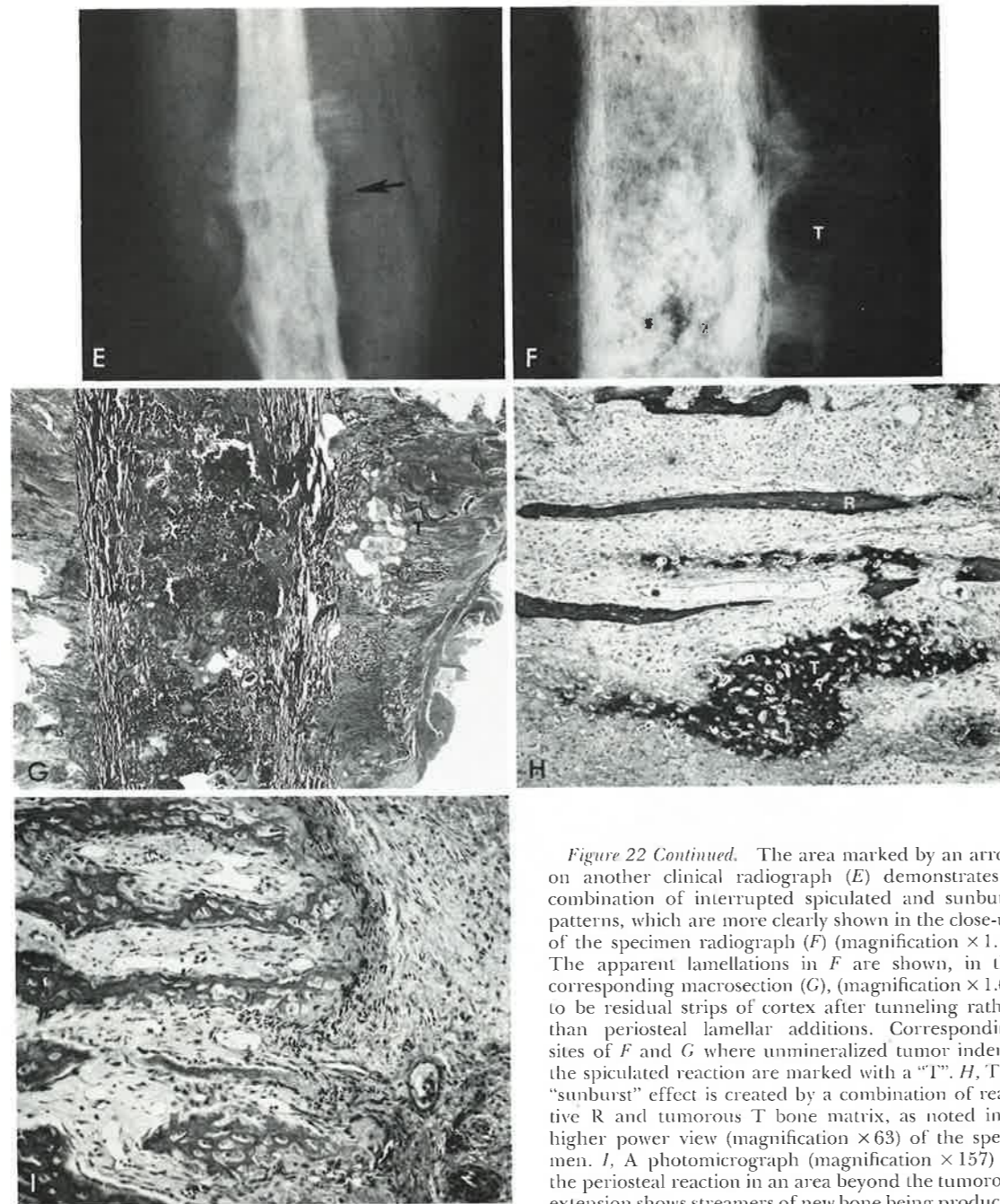


Figure 22. Combined periosteal reactions. Osteosarcoma of the femur in a 13 year old boy. The area marked by the arrows in the clinical radiograph (A) is noted in the specimen radiograph (B) (magnification $\times 1.8$) to consist of combined lamellated and spiculated reactions. C shows the line of impingement of subperiosteal tumor (T) against the outer spiculated reaction and the manner in which it denies space for continued spicular growth. The tumor has also colonized an area of accentuated tunneling through the outermost layer of cortex (C), thereby simulating a single reactive lamella. (M, muscle outside the periosteum.)

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by cells derived from activated periosteal soft tissue. (AFIP Neg. Nos. 79-5718; 81-14590; 81-14589; 81-14587; 79-5718; 81-14504; 81-14502; 81-14506; 81-14513.)

Figure 22 Continued. The area marked by an arrow on another clinical radiograph (E) demonstrates a combination of interrupted spiculated and sunburst patterns, which are more clearly shown in the close-up of the specimen radiograph (F) (magnification $\times 1.1$). The apparent lamellations in F are shown, in the corresponding macrosection (G), (magnification $\times 1.6$), to be residual strips of cortex after tunneling rather than periosteal lamellar additions. Corresponding sites of F and G where unmineralized tumor indents the spiculated reaction are marked with a "T". H, The "sunburst" effect is created by a combination of reactive R and tumorous T bone matrix, as noted in a higher power view (magnification $\times 63$) of the specimen. I, A photomicrograph (magnification $\times 157$) of the periosteal reaction in an area beyond the tumorous extension shows streamers of new bone being produced

myxoid. Reactive bone that is admixed with sarcoma will have the greatest radiographic density nearest the cortex since it is the oldest at that location. The advance of mineralized spiculation from the cortex into the juxtacortical mass seen on serial films should not be misinterpreted as production of a periosteal reaction in the depths of the tumor. The sunburst appearance may eventually disappear as the extracortical mass becomes more densely consolidated.¹¹ It is highly suggestive of, but not pathognomonic for, osteosarcoma since it can be seen with other tumors such as blastic metastasis and hemangioma.

Combined Reactions. Combined reactions encompass transitions between and combinations of the various patterns diagrammed in pure form in Figure 2. It is likely that an individual case for diagnosis will present one or more transitional or mixed patterns. A case example is presented in Figure 22.

Focal periosteal reactions superimposed on bone lesions that appear to be chronic suggest a transition to more aggressive behavior if a mechanical complication (such as a fracture) can be excluded. Similarly, the combination of slow and rapid periosteal reaction patterns in a single lesion suggests an accelerated growth rate such as occurs with malignant change of an antecedent benign lesion.

SUMMARY

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. When the demand for a reaction is excessive, nearby extraperiosteal soft tissue serves as a ready source for additional modulating cells, just as it does for fracture callus. The configuration of a periosteal reaction is an index of the nature and intensity of the inciting process.

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