THE PHYSIOLOGY OF SUPPORTING TISSUE

By M. J. Dallemagne
Institute of Experimental Therapeutics
University of Liége, Belgium

Since Murray's review appeared (1), two important contributions have been added to the literature concerning supporting tissue: Weinmann & Sicher (2) dealt with various problems of physiology and pathology, while Lacroix (3) especially studied bone induction and growth. Leicester's monograph (4) is devoted to some aspects of calcification. Maynard & Smith (5) reported on mineral metabolism.

Structure of Bone

Microscopic structure.—By comparing the development of long bones in 350 different vertebrates ranging from the amphibia to man (6), it was shown that, during the prenatal stages, the structural features are common to all species (7). Differences which become manifest after birth depend upon the rate of bone development, which is a specific or racial character (8).

Structural changes occurring during growth, as well as the conditions responsible for these evolutional features (9), were studied in dogs (10). Functional factors evoke progressive changes in the ossified tendons of birds (11). Studies were devoted to the ossification of the distal epiphysis of the rat's humerus (12), of the mandibular joint (13), and of the third metacarpal (14). The vascular channels of bone (15) and the disposition of the fibrils (16) have been demonstrated.

Arteriographic and histological studies have shown that circulation in bone follows particular rules (17).

Submicroscopic structure.—The birefringence of total bone is due to a combination of the optical characters of the organic and mineral parts, in accordance with pure physical laws (18). These results have been questioned (19, 20, 22). The curve of structural double refraction of bone has been determined from the mineral fraction (18, 19). A mold of the submicroscopic spaces occupied by the organic constituents has been made by means of "plexiglas" (21). The refractive index of total bone depends upon the respec-

tive proportions of organic and mineral substances (23); it varies with age and species (24).

Growth of Bone

Embryonal grafts into the chorio-allantois survive unless they have been boiled (25). Their vessels anastomose with the pre-existing circulatory system and the explants develop in close relation to the vascular neoformation (26). Osteoclasts resulting from the fusion of several monocytes are released from the grafts (27).

Studies have been devoted to the displacement of the tendons (28) and of the nourishing artery during development of the bone (29). Reports have been made on the influence of x-rays (30) and pH on bone growth (31).

The evocation of bone formation.—The inductor of ossification (osteogenin) belongs neither to the lipids, nor to the steroids (32). The mechanism of its action is only the repetition of reactions occurring during the fetal stage (33). Osteogenin plays a part in periosteal osteogenesis (34). It forces hyaline cartilage transplanted into growth cartilage to function like the latter (35), and it has a role in the formation of the perichondreal ring (36). Osteogenin also determines the evolution of grafts transplanted under the renal capsule or under the skin (37). It is present in the shaft of growing bone (38) and in the bone marrow (39); the latter, when transplanted, gives rise to the formation of bone tissue (40). Nevertheless, the theory of the bone "organizer" has been criticized (25).

Chemical and enzymatic problems of ossification.—The latest concepts concerning the mechanism of ossification have been reviewed by Roche (41) and Moog (42). The chemical condition of calcium and phosphorus in the blood has lost some of its importance as far as ossification is concerned since it is known that the phosphorylases (43, 44, 45) can increase the local concentration of phosphoric esters from glycogen accumulated in the tissue undergoing ossification (41). By the action of phosphatase, the role of which has been reviewed (46, 47), phosphate ions are released from the phosphoric esters and temporarily fixed on the "pre-osseous" substance. This explains the variations of the ratio of calcium to phosphorus in growing bone without calling upon the theory of primary calcification, which has been criticized (48). Furthermore, *in vitro* studies of the reactions between calcium and phosphate

ions have shown that bicalcium phosphate cannot be formed in bone (49, 50). The phosphate ions then leave the pre-osseous substance as calcium ions are brought in by the blood, and tricalcium phosphate now precipitates in the organic matrix formed by polymerization of ossein (51).

The intervention of carbonic anhydrase in ossification is doubtful. Results indicating that sulfamides inhibit calcification (52) have not received support (53).

The influence of phosphates on the solubility of calcium carbonate, and vice versa, has been investigated (54). In order to clarify the process of precipitation of tricalcium phosphate, the titration curve of phosphoric acid with calcium hydroxide has been studied again in detail, as has also the nature of the precipitates appearing immediately and subsequently (55).

A physicochemical study of calcium and magnesium compounds in biological fluids has been published (56), also a report on the functional role of the decalcifying action exerted by phosphate (57). The presence of organic anions in the bone makes it interesting to determine the dissociation constants of their calcium salts (58).

Many investigators have been concerned with the histochemical detection of the phosphatases (59, 60, 61). The normal distribution of phosphatase has been demonstrated by histochemical methods (62). It has been found in the endosteum and periosteum, in the nuclei of the fibroblasts, in the collagen fibers, osteocytes, and haversian canals, but not in the organic matrix of the bone (63). During bone development, phosphatase becomes extracellular only at the sites of calcification (64, 65). Extraction methods for phosphatase have been reviewed (66). In tissue culture experiments, it was found that the addition of phosphatase extracted by Robison's method slows the development of chick embryos, by its protein impurities (67). Besides its relation to the phosphate ions involved in the process of ossification (68, 69), phosphatase influences the evolution of the tissues which are being ossified; it changes the distribution of nucleic acids in the periosteal fibroblasts evolving toward the osteoblastic stage (70) and its activity is related to the transformation of the hypertrophied cells in the growth cartilage (71, 72). Its influence on the maturation of the organic matrix has been said to be more important than its role in the precipitation of calcium salts (73). Phosphatase also

plays a part in tooth formation; it influences the histological differentiation of every organ in which it is present (74, 75) and contributes to the production of the shell in marine molluscs (76). Contrary to some previous results, it does not induce ectopic ossification when injected into muscular tissue together with calcium glycerophosphate (77). Its activity is inhibited *in vitro* by high concentrations of sulfamides (78). The role of phosphatase during the repair of bone defects has been studied again; the enzyme is elaborated by migrating cells gathering very early at the site of lesion and later by osteogenic cells, when the osteogenic fibres are being formed and joined together into trabeculae. The intensity of these phenomena is decreased by lack of vitamin C (79). Bone phosphatase is inactivated by α-amino acids (80).

Chemical Nature of Bone

Bone salts.—Dallemagne *et al.* (84, 85) have brought new evidence in favor of their opinion concerning the chemical nature of bone salts. According to these authors, bone salts isolated after hydrolysis of the organic matrix contain α-tricalcium phosphate and various carbonates not in chemical combination with one another. Phosphate and carbonates combine to form carbonato-apatite at temperature of 900°C. and in other well-determined conditions. The isomorphism of α-tricalcium phosphate and apatite has again been insisted upon (81, 82, 83).
the study of refractive indices in bone submitted to various treatments agree with those given by the examination of x-ray diffraction spectra (84, 85).

Whereas a method for preparation of pure α-tricalcium phosphate has been described in detail (86), its existence has been questioned; the reaction between lime and phosphoric acid would always result in an alkaline salt, hydroxylapatite (87). In fact, depending upon the conditions of the experiment and the reagents used, one obtains α-tricalcium phosphate either pure or more or less contaminated with absorbed lime (88). These results which establish beyond question the existence of pure α-tricalcium phosphate (89) have been recently re-emphasized (90). Most of them have been confirmed (91). These views have been objected to on the basis that synthesis of carbonato-apatite is not possible because the size of the crystalline lattice of apatite is incompatible with the inclusion of carbonate groups; however, it should be

borne in mind that the structure of the apatites is not exactly hexagonal as was thought before. The different apatites can be differentiated from each other by minute characters of their x-ray diffraction spectrum (92).

Organic constituents of bone.—The organic component of bone produces a deformation of the mineral crystallites (93). The structure of the organic matrix in mammals and fishes has been discussed (94).

Besides proteins, some other organic molecules, principally citric acid (95, 96), are also present in bone. Solubility measurements have been said to show that about 10 per cent of total calcium and phosphorus is fixed by the proteinic constituents (97). A proteolytic enzyme is present in the metaphyses of young animals (98). It originates in the marrow and plays a part in the formation of the preosseous substance (99). Glycerol extracts of these bones exhibit a peptidase activity (100).

Bone Metabolism Studied by Means of Radioisotopes

The use of isotopically marked elements in the study of bone metabolism has been mentioned in many general reviews (101, 102, 103). Exchanges of mineral phosphates between blood and bone have been studied *in vitro* (104) and *in vivo* (105) on bones from normal or denervated limbs. Phosphorylcholine does not give more phosphorus to the bone than do mineral phosphates, and the replacement of phosphorus seems to be due essentially to ionic exchanges (106).

Replacement of bone salts requires at least 230 days (107), and the metabolism of phosphorus is more active than that of calcium (108). Radiostrontium follows the same physiological rules as calcium, which is not true for plutonium, yttrium, cerium (109), or uranium; the uptake of the latter element by bone tissue is not parallel to the calcification process (110). Phosphorus fed to chickens appears in the egg only one month later; it is stored in muscle and bone (111). On the contrary, intra-esophageally administrated radiocalcium can be detected in the egg laid 15 min. later (112). The uptake of radiocalcium by the skeleton of the rat has been studied (113). Tyrosine, marked with one atom of radiocarbon, does not accumulate in bone (114). A new method for tissue microanalysis has also been used on bone; it is based on induced radioactivity (115).

Radiocarbon parenterally administered as carbonate is found in the mineral fraction of bone but also, to a smaller extent, in the organic substance (116). After inhalation of labeled carbon dioxide carbon is also fixed by bone (117).

The hypothesis that the isotopic composition of potassium would be different in a mineral or biological medium has not been confirmed (118).

Influence of Dietary Factors on Bone

Reducing the caloric value of the diet given to rats does not produce any bone disease, but retards the process of ossification (119).

Proteins.—Protein insufficiency in the diet also slows bone lengthening, but the ash content remains unchanged (120).

Lipids.—Coconut oil has a beneficial influence on rachitic bone by improving absorption and retention of phosphorus (121).

Calcium and phosphorus.—Excess or deficiency of dietary calcium produces a loss of calcium and phosphorus from the entire organism (122) and reduces the ash content of bone (123). In growing animals receiving a diet made rachitogenic by excess of calcium, it has been confirmed that starvation soon causes the appearance of a positive line test (124). Severe calcium and phosphorus deficiency in the diet, as well as extensive restriction of a normal diet, produces a decrease of ash, especially in the bones of limbs paralyzed by nervous section (125).

A diet rich in phosphorus and poor in calcium lowers the Ca/P ratio in the bones of young rats; this has been interpreted as evidence in favor of the presence of dicalcium phosphate in the mineral constituents (126). Calcium carbonate added to bread can correct an otherwise calcium-deficient diet (127). Changes in nerve and muscle irritability have been studied by chronaximetric measurements in animals receiving an acidotic rachitogenic diet (128). Such a diet is apt to inhibit vitamin D synthesis produced by ultraviolet rays (129).

Fluorine.—Whereas there have been many reports concerning the protective influence of fluorine on tooth enamel, only a few deal with its action on bone. The enzymatic and protoplasmic action of fluorine promotes rickets-like changes in the bones of puppies, but its effect on the bones of older dogs is different from rickets (130). Besides, sodium fluoride does not affect the calcemia

or phosphoremia of normal rats unless the diet is not properly balanced (131). In a review dealing with the biological importance of phosphomonoesterase (42), it has been recalled that fluorine reduces the accumulation in bone of the glycogen related to the production of phosphoric esters.

Vitamin A.—The action of vitamin A on bone is exerted through the osteoblasts (132); its deficiency produces an accelerated remodeling of bone and a laying down of defective tissue (133). The activity of the osteoclasts is also affected. The disturbances are reversible if the vitamin A deficiency is corrected (134). Hypervitaminosis A has some action on bone growth (135). A study of its clinical features (136) has been published.

Vitamin B.—Deficiency in riboflavin leads to slowing of chondrogenesis and endochondreal ossification in rats (137). Those phenomena, which have been shown in mice to be reversible (138), are also produced by deficiency in total vitamin B complex (139), pantothenic acid (140), and pyridoxine; in this last case, the disturbances are accentuated by excess dietary proteins (141).

Vitamin C.—Growth of the odontoblasts in the rat's incisors is directly related to the vitamin C content of the diet (142). It has been emphasized that vitamin C is necessary for tooth growth (143). Its importance in fracture repair and ossification has been reviewed (144). With partial vitamin C deficiency, compact bone becomes porotic, but this is often accompanied by hyperostosis: the callus of fractured bone fails to become compact and retains trabecular structure (145).

Vitamin D.—The fundamental process of ossification in rachitic rats is only slightly abnormal, the reduced strength of rachitic bone being due solely to insufficient mineralization (146). Rickets lesions are often accompanied by osteopetrosis (147). Histological and histochemical methods have been applied to the detection of the early signs of avitaminosis D and the quantitative determination of bone phosphatase (148, 149). Rickets also produces some changes in the ionic product of serum calcium phosphates (150).

The mode of action and the therapeutic indications of vitamin D have been reviewed (151, 152). It has been said to act essentially by preventing the solution of bone calcium (153). The influence of vitamin D on calcium balance is very different in normal individuals and in subjects suffering from rickets or other bone disease (154, 155, 156).

Studies were devoted to the action of vitamin D on different types of rickets: rickets due to an unbalanced Ca/P ratio in the diet (123), to liver disturbances (157), and to excess mineral sulfates in drinking water (158). Rickets produced by a diet with a high Ca/P ratio is cured by a normal diet, but the curative action is different from that exhibited by vitamin D (159) or citric acid (160). In South Africa, summer sunshine provides full protection against rickets; its action has been estimated equivalent to 2,500 I.U. a day (161).

The widely accepted opinion that vitamin D is stored in the liver has been criticized (162).

A detailed critical study of the chicken assay for vitamin D has been made (163 to 169); the line test and the chemical methods have been compared (170). A rachitogenic index representing the rachitogenic value of the diet has been defined (171).

As it has been emphasized again in many publications, hypervitaminosis D produces metastatic calcifications especially in the lungs, the myocardium, the kidneys, and the gastric mucosa. Abnormal calcification can occur either after one high dose of vitamin D or after repeated and prolonged administration (172 to 178). Hypervitaminosis D also causes atrophy of the parathyroids (175). Hypercalcemia cannot be considered as a toxicity test for vitamin D (179).

Influence of Hormonal Factors on Bone

The influence of hormones on osteogenesis in man has been detailed in Albright's extensive report (180).

Adrenals.—It has been confirmed that adrenocorticotropic hormone retards chondro- and osteogenesis (181).

Hypophysis.—The role of the pituitary growth hormone has been recently reviewed (182). Hypophysectomy is followed by acceleration of ossification and senescence of the mandibular joint (183), but stops chondrogenesis and osteogenesis in the third metacarpal (184). The hormone restores growth processes in the senescent mandibular joint (185) and in the epiphyseal cartilage plate of the third metacarpal of hypophysectomized rats (186). In normal rats, it prolongs the period of growth especially at the mandibular condyle (187), to a smaller extent at the tibia and at the costo-chondral junction (188), but not at all at [redacted] third metacarpal (189).

Thyroid.—Thyroidectomy transitorily retards the ossification of the mandibular joint (190), the tibia, the metacarpals, and the caudal vertebrae (191). In hypophysectomized rats, thyroxine failed to reactivate growth at the mandibular joint and at the third metacarpal; moreover, it inhibited the response to the growth hormone (185, 186). Thyroxine enhances the formation of the organic bone matrix (192).

Parathyroids.—From some studies devoted to the relations between parathyroids and mineral metabolism, it has been concluded that parathormone influences through the kindey the metabolism of phosphorus (193) and, secondarily, of calcium and strontium (194). The hypercalcemia due to injections of parathyroid extracts is said to be directly related to their diuretic effects (195). Increased diuresis is accompanied by an increased renal blood flow; the responsible pressor substance might not be the hormone which mobilizes calcium (196). Urinary variations of phosphate concentration seem to be parallel to those of glucose (197). Parathyroid extract has been said to have a direct action on bone (198). In young dogs, prolonged administration modifies the composition of bones more than dietary mineral variations are able to (199). The relations between hyperparathyroidism and decalcification of the skeleton have been reviewed (200). Studies were devoted to the pathogenesis of the clinical manifestations of hyperparathyroidism (201). Nephrectomy or ligation of the ureters is soon followed by hypertrophy and hyperplasia of the parathyroids; the histological features of the glandular cells in such cases have been described and discussed (202). Parathyroid hypertrophy can be produced by renal lesions (203), liver disturbances (204), and low Ca/P ratio in the diet (205). It has been shown in dogs that renal lesions can be followed by osteofibrosis (206). The relations between parathyroids and the skeleton in kidney disease have been dealt with in Gilmour's recent monograph (207). Parathyroid activity partially depends upon the anterior pituitary body (208).

It seemed that the existence of a functional equilibrium between parathyroid secretion and vitamin D has been clearly established by Studitskii (209), who has shown in chick embryos that anterior pituitary grafts into the chorio-allantois are frequently followed by chondro-dystrophy; the mechanism of this effect would be a stimulation of the parathyroids. However, these conclusions have been criticized by Landauer (210); in the chick, the

causes for the abnormal bone evolution exist long before the embryonal development of the endocrines.

Sexual hormones.—Injection of estradiol benzoate to chick embryos does not induce hypercalcification of the femur (211) as it does in adults. Endosteal osteogenesis requires constant administration of estrogen. In parathyroidectomized ducks receiving folliculin, medullar ossein trabeculae are formed normally, but their calcification is prevented (212). The action of folliculin is local as it is present after injection into a long bone (213). In ducks receiving a calcium-deficient diet together with injections of folliculin, ossein trabeculae develop, but there is only a partial calcification at the expense of the mineral reserves in bone (214). These purely organic trabeculae do not attract osteoclasts (215). Blood depletion is followed by medullary hyperplasia and increases bone response to estrogen (216). The osteoblasts have an important role in endosteal osteogenesis: they derive from the medullar reticulum and their activity is maintained by folliculin. When folliculin adminstration is discontinued, the osteoblasts become osteoclasts and destroy the trabeculae (217).

Estrogen can cause hypomineralization, which is a consequence of abnormal formation of the proteinic matrix by the osteoblasts (218). The Ca/P ratio in recently built trabeculae is higher than in normal bone. These trabeculae have been said to contain apatite and calcium carbonate in an irregular crystalline mixture; the same condition would occur in the femoral cortex under the influence of folliculin (219). However, the principal constituent of new bone is tricalcium phosphate, as can be shown by chemical analysis, determination of refractive indices, and study of x-ray diffraction spectra (220).

Part of the phosphorus present in endosteal bone is provided by the skeleton, as has been shown in pigeons receiving a normal amount of dietary phosphorus supplemented by injected radiophosphorus (221). The same procedure has shown that the exchanges of phosphorus are significantly accelerated by folliculin; this happens in all bones, especially those undergoing endosteal osteogenesis (222). Only the bones of this latter group, however show accelerated calcium exchanges under the influence of estrogen, as has been demonstrated by a parallel study using radiocalcium. In other bones, for instance the humerus, bone calcium metabolism is not different from the controls receiving no folliculin.

Furthermore, folliculin has been said to influence the intestinal resorption of calcium (223). Estrogen has no influence on the bones of puppies (224); its action on bone absorption and relaxation of pelvis has been studied in guinea pigs (225).

In pigeons androgen is necessary to the action of folliculin on bone; in sparrows, only age and seasonal factors seem to have a role (226). Dietary restrictions associated with administration of sesame oil induce bone atrophy in limbs either normal or paralyzed by nervous section; this effect is minimized by estrogen (125). While the male sexual hormone is rachitogenic, folliculin has the opposite effect; alone, or associated with vitamin D, it is apt to antagonize hyperparathyroidism (227). Castration retards growth by inhibiting the hypertrophic development of cartilaginous cells (228); this effect is antagonized by thyroxine which stimulates this hypertrophy (229). In ducks, thyroidectomy slows the formation of the proteinic trabeculae; and even though there is a slower rise in calcemia under the influence of folliculin (230), calcification of endosteal bone is more active (231). These results emphasize the general stimulating action of thyroxine on tissue metabolism as well as its property of increasing calcium excretion. While thyroxine apparently does not inhibit estrogen action on endosteal osteogenesis, it reduces the rise in calcemia following administration of folliculin (232).

In growing mice, the effects of estrogen given together with thyroxine are altered: each hormone predominates during the stage where its action would have been most intense if it had been injected alone (233). In impuberal rats, natural as well as synthetic estrogens produce a narrowing of the epiphyseal lines followed by hyperossification. These changes do not occur in adult rats (234). The estrogen-induced formation of endosteal bone in rats requires the presence of the pituitary body, but not of the parathyroids. The amount of bony trabeculae is increased because estrogen makes bone resorption deficient (235).

In growing guinea pigs suffering from avitaminosis C, estrogen administration is unable to induce the evolution of the proteinic matrix into bone; however, the appearance of scurvy lesions is prevented in these animals as well as in adults (236). Estrogen does not alter the metabolism of ascorbic acid (237).

The physiology of antlers is an interesting problem related to the sexual glands; it has been mentioned in many biological

reviews (238, 239). Thorough studies have been devoted to the seasonal changes affecting antlers (240, 241, 242).

LITERATURE CITED

1. MURRAY, P. D. F., *Ann. Rev. Physiol.*, **9**, 103 (1947)
2. WEINMANN, J. P., AND SICHER, H., *Bone and Bones—Fundamentals of Bone Biology*, 464 pp. (Kimpton, London, 1947)
3. LACROIX, P., *L'Organisation des os*, 230 pp. (Desoer, Liége, 1949)
4. LEICESTER, H. M., *Biochemistry of the Teeth*, 306 pp. (Mosby, St. Louis, 1949)
5. MAYNARD, L. A., AND SMITH, S. E., *Ann. Rev. Biochem.*, **16**, 273 (1947)
6. AMPRINO, R., AND GODINA, G., *Commentationes Pontificia Acad. Sci.*, **11**, 329 (1947)
7. AMPRINO, R., AND GODINA, G., *Atti accad. nazl. Lincei, Classe sci. fis. mat. e nat.*, **1**, 648 (1946)
8. AMPRINO, R., *Arch. biol. (Liége)*, **58**, 315 (1947)
9. AMPRINO, R., *Arch. sci. biol. (Italy)*, **31**, 208 (1946)
10. GODINA, G., *Arch. ital. anat. embriol.*, **52**, 161 (1947)
11. AMPRINO, R., *Acta Anat.*, **5**, 291 (1948)
12. BECKS, H., ASLING, C. W., SIMPSON, M. E., EVANS, A. M., AND LI, C. H., *Am. J. Anat.*, **82**, 203 (1948)
13. COLLINS, D. A., BECKS, H., SIMPSON, M. E., AND EVANS, H. M., *Am. J. Orthodontics Oral Surg.*, **32**, 431 (1946)
14. BECKS, H., ASLING, C. W., COLLINS, D. A., SIMPSON, M. E., AND EVANS, H. M., *Anat. Rec.*, **100**, 577 (1948)
15. RUTH, E. B., *Anat. Rec.*, **98**, 59 (1947)
16. RUTH, E. B., *Am. J. Anat.*, **80**, 35 (1947)
17. LAMAS, A., AMADO, D., AND CELESTINO DACOSTA, J., *Presse méd.*, **54**, 862 (1946)
18. DALLEMAGNE, M. J., AND MELON, J., *J. Wash. Acad. Sci.*, **36**, 181 (1946)
19. ASCENZI, A., *Atti accad. nazl. Lincei, Classe sci. fis. mat. e nat.*, **5**, 171 (1948)
20. ASCENZI, A., *Atti accad. nazl. Lincei, Classe sci. fis. mat. e nat.*, **5**, 100 (1948)
21. BAUD, C. A., AND DALLEMAGNE, M. J., *Science*, **110**, 90 (1949)
22. ASCENZI, A., *Nature*, **163**, 604 (1949)
23. DALLEMAGNE, M. J., AND MELON, J., *Compt. rend. soc. biol.*, **141**, 539 (1947)
24. ASCENZI, A., *Atti accad. nazl. Lincei, Classe sci. fis. mat. e nat.*, **4**, 777 (1948)
25. HANCOX, N. M., *J. Physiol. (London)*, **106**, 279 (1947)
26. HANCOX, N. M., *J. Physiol. (London)*, **107**, 513 (1948)
27. HANCOX, N. M., *J. Physiol. (London)*, **105**, 66 (1946)
28. LACROIX, P., *Arch. biol. (Liége)*, **59**, 391 (1948)
29. LACROIX, P., *Arch. biol. (Liége)*, **59**, 379 (1948)
30. VINCENT, J., *Bull. histol. appl. à physiol. et path. et tech. microscop.*, **25**, 180 (1948)
31. PAFF, G. H., *Proc. Soc. Exptl. Biol. Med.*, **68**, 288 (1948)
32. WILLSTAEDT, H., LEVANDER, G., AND HULT, L. (Personal communication, 1949)
33. LEVANDER, G., *Acta Path. Microbiol. Scand.*, **26**, 113 (1949)
34. LACROIX, P., *Arch. biol. (Liége)*, **57**, 99 (1946)
35. LACROIX, P., *Bull. acad. roy. méd. Belg.*, **10**, 517 (1945)

36. LACROIX, P., *J. Bone Joint Surg.*, **29,** 292 (1947)
37. LACROIX, P., *Arch. biol. (Liége)*, **60,** 1 (1949)
38. TEUCQ, E., *Arch. biol. (Liége)*, **59,** 1 (1948)
39. LACROIX, P., *Arch. biol. (Liége)*, **60,** 14 (1949)
40. PFEIFFER, C. A., *Anat. Rec.*, **102,** 225 (1948)
41. ROCHE, J., *Experientia*, **2,** 325 (1946)
42. MOOG, F., *Biol. Revs. Cambridge Phil. Soc.*, **21,** 41 (1946)
43. GLOCK, G. E., *J. Physiol. (London)*, **98,** 1 (1940)
44. GUTMAN, A. B., AND GUTMAN, E. B., *Proc. Soc. Exptl. Biol. Med.*, **48,** 687 (1931)
45. GUTMAN, A. B., WARRICK, F. B., AND GUTMAN, E. B., *Science*, **95,** 461 (1942)
46. ROCHE, J., in *Actualités Biochimiques*, 10 (Desoer, Liége, 1947)
47. LORCH, I. J., *J. Bone Joint Surg.* [B]**31,** 94 (1949)
48. DALLEMAGNE, M. J., *Nature*, **161,** 115 (1948)
49. MELON, J., AND DALLEMAGNE, M. J., *Bull. soc. chim. Belges.*, **55,** 38 (1946)
50. MELON, J., AND DALLEMAGNE, M. J., *Bull. soc. chim. Belges.*, **56,** 180 (1947)
51. DALLEMAGNE, M. J., *Acta physiother. Rheumatol. Belgica*, **3,** 77 (1947)
52. BENESCH, R., CHANCE, M. R. A., AND GLYNN, L. E., *Nature*, **155,** 203 (1945)
53. MILLER, Z. B., WALDMAN, J., AND MCLEAN, F.C., *Nature*, **161,** 272 (1948)
54. GREENWALD, I., *J. Biol. Chem.*, **161,** 697 (1945)
55. DALLEMAGNE, M. J., AND MELON, J., *Bull. soc. chim. biol.*, **28,** 566 (1946)
56. MONNIER, A. M., BONNET, V., AND BRARD, R., *Arch. intern. physiol.*, **54,** 188 (1946)
57. MONNIER, A. M., AND BONNET, V., *Arch. sci. physiol.*, **1,** 91 (1947)
58. JOSEPH, N. R., *J. Biol. Chem.*, **164,** 29 (1946)
59. CAPPELLIN, M., *Mon. zool. ital.*, **56,** 256 (1948)
60. LORCH, I. J., *Nature*, **158,** 269 (1946)
61. BARGER, J. D., *Arch. Path.*, **43,** 620 (1947)
62. DANIELLI, J. F., *J. Exptl. Biol.*, **22,** 110 (1946)
63. LORCH, I. J., *Quart. J. Microscop Sci.*, **88,** 367 (1947)
64. LORCH, I. J., *Quart. J. Microscop. Sci.* (In press)
65. GREEP, R. O., FISCHER, C. J., AND MORSE, A., *J. Am. Dental Assoc.*, **36,** 427 (1948)
66. NGUYEN-VAN THOAI, ROCHE J., AND ROGER, M., *Biochem. et Biophys. Acta*, **1,** 61 (1947)
67. CAPPELLIN, M., *Sperimentale*, **99,** 133 (1948)
68. WISLOCKI, G. B., AND DEMPSEY, E. W., *Am. J. Anat.*, **76,** 277 (1945)
69 ROCHE, J., in *Biochemische Aktualiteiten* (Vlaamse Chemische Vereniging, Antwerpen, 1947)
70. CAPPELLIN, M., *Boll. soc. ital. biol. sper.*, **24,** 1228 (1948)
71. CAPPELLIN, M., *Atti soc. med. chir. Padova*, **25,** 86 (1947)
72. CAPPELLIN, M., *Rass. Biol. Um.*, **3,** 35 (1948)
73. MCKELVIE, A. M., AND MANN, F. C., *Proc. Staff Meetings Mayo Clinic*, **23,** 449 (1948)
74. JOHNSON, P. L., BUTCHER, E. O., AND BEVELANDER, G., *Anat. Record*, **93,** 355 (1945)
75. BEVELANDER, G., AND JOHNSON, P. L., *J. Cellular Comp. Physiol.*, **28,** 129 (1946)

76. BEVELANDER, G., AND BENZER, P., *Biol. Bull.*, **94,** 176 (1948)
77. SLESSOR, A., AND WYBURN, G. M., *Lancet.*, **I,** 212 (1948)
78. SILVER, P. H., AND GOLDING, J. S. R., *Lancet*, **I,** 528 (1945)
79. BOURNE, G. H., *J. Anat.*, **82,** 81 (1948)
80. BODANSKY, O., *Federation Proc.*, **8,** 185 (1949)
81. LOMBARD, P., VERAIN, ROYER, AND COUILLAUD, *Mem. acad. chir.*, **71,** 180 (1945)
82. BEVERS, C. A., *Brit. Sci. News*, **1,** 4 (1947)
83. BEVERS, C. A., AND MCINTYRE, D. B., *Mineralog. Mag.*, **27,** 254 (1946)
84. BRASSEUR, H., DALLEMAGNE, M. J., AND MELON, J., *Nature*, **157,** 453 (1946)
85. DALLEMAGNE, M. J., AND BRASSEUR, H., *Experientia*, **3,** 469 (1947)
86. MACINTYRE, W. H., PALMER, G., AND MARCHALL, H. L., *Ind. Eng. Chem.*, **37,** 164 (1945)
87. BRANDENBERGER, E., AND SCHINZ, H. R., *Experientia*, **4,** 59 (1948)
88. BRASSEUR, H., DALLEMAGNE, M. J., AND MELON, J., *Experientia*, **3,** 421 (1948)
89. DALLEMAGNE, M. J., BRASSEUR, H., AND MELON, J., *Bull. soc. chim. France*, **D.,** 138 (1949)
90. DALLEMAGNE, M. J., BRASSEUR, H., AND MELON, J., *Bull. soc. chim. biol.*, **31,** 425 (1949)
91. CARTIER, P., *Bull. Soc. chim biol.*, **30,** 65 (1948)
92. BRASSEUR, H., AND DALLEMAGNE, M. J., *Bull. soc. chim. France*, D, 135 (1949)
93. DAWSON, I. M., *Nature*, **157,** 660 (1946)
94. HENNY, G. C., AND SPIEGEL-ADOLPH, M., *Am. J. Physiol.*, **144,** 632 (1945)
95. THUNBERG, T., *Acta Physiol. Scand.*, **14,** 245 (1947)
96. THUNBERG, T., *Acta Physiol. Scand.*, **15,** 38 (1948)
97. CARTIER, P., *Bull. soc. chim. biol.*, **30,** 73 (1948)
98. POLONOWSKI, M., AND CARTIER, P., *Bull. soc. chim. biol.*, **28,** 247 (1946)
99. CARTIER, P., *Bull. soc. chim. biol.*, **28,** 273 (1946)
100. CARTIER, P., *Bull. soc. chim. biol.*, **28,** 258 (1946)
101. SACKS, J., *Chemical Rev.*, **42,** 411 (1948)
102. DOUGHERTY, E. C., AND LAWRENCE, J. H., *Calif. Med.*, **69,** 1 (1948)
103. HEVESY, G., *Radioactive isotopes*, 556 pp. (Interscience, New York 1948)
104. FALKENHEIM, M., NEUMAN, W. F., AND HODGE, H. C., *J. Biol. Chem.*, **169,** 713 (1947)
105. NEUMAN, W. F., AND RILEY, R. F., *J. Biol. Chem.* **168,** 585 (1947)
106. RILEY, R. F., MCCLEARY, B., AND JOHNSON, R. E., *Am. J. Physiol.*, **143,** 677 (1945)
107. ARMSTRONG, W. D., *Federation Proc.*, **6,** 235 (1947)
108. ARMSTRONG, W. D., AND BARNUM, C. P., *J. Biol. Chem.*, **172,** 199 (1948)
109. COPP, D. H., AXELROD, D. J., AND HAMILTON, J. G., *Am. J. Roentgen. Radium Therapy*, **58,** 10 (1947)
110. NEUMAN, W. F., NEUMAN, M. W., AND MULRYAN, B. J., *J. Biol. Chem.*, **175,** 705 (1948)
111. O'NEIL, J. B., JOWSEY, J. R., LEE, C. C., READE, M. A., AND SPINKS, J. W. T. *Science*, **107,** 295 (1948)

112. COMAR, C. L., AND DRIGGERS, J. C., *Science*, **109**, 282 (1949)
113. HARRISON, H. E., AND HARRISON, H. C., *Federation Proc.*, **8**, 68 (1949)
114. REED, J. C., AND JONES, H. B., *J. Biol. Chem.*, **174**, 427 (1948)
115. TOBIAS, C. A., AND DUNN, R. W., *Science*, **109**, 109 (1049)
116. ARMSTRONG, W. D., SCHUBERT, J., AND LINDENBAUM, A., *Proc. Soc. Exptl. Biol. Med.*, **68**, 233 (1948)
117. DELLUVA, A. N., AND WILSON, D. W., *J. Biol. Chem.*, **166**, 739 (1946)
118. MULLENS, L. J., AND ZERAHN, K., *J. Biol. Chem.*, **174**, 107 (1948)
119. SAXTON, J. A,. AND SILBERBERG, N., *Am. J. Anat.*, **81**, 445 (1947)
120. ESTREMERA, H. R., AND ARMSTRONG, W. D., *J. Nutrition*, **35**, 611 (1948)
121. DUTTA, N. C., *Ann. Biochem. Exptl. Med. (India)*, **8**, 69 (1948)
122. HOLTZ, F., POPPER, H., AND SILBERMANN, L., *Biochem. Z.*, **318**, 149 (1947)
123. MIGICOVSKY, B. B., AND EMSLIE, A. R. G., *Arch. Biochem.*, **13**, 175 (1947)
124. IRVING, J. T., *J. Physiol. (London)*, **105**, 16 (1946)
125. ARMSTRONG, W. D., *J. Nutrition*, **35**, 597 (1948)
126. HIRSCHMAN, A., SOBEL, A. E., KRAMER, B., AND FANKUCHEN, I., *J. Biol. Chem.*, **171**, 285 (1947)
127. CROSNIER, R., GIRARD, P., RENAULT, J., AND GROUSSAULT, S. *J. Physiol. et path. gén.*, **39**, 331 (1947)
128. LECOQ, R., CHAUCHARD, P., AND MAZOUÉ, H., *Thérapie*, **2**, 272 (1947)
129. LECOQ, R., *Thérapie*, **2**, 73 (1947)
130. BAUER, W. H., *Am. J. Orthodontics Oral Surg.*, **31**, 700 (1945)
131. IRVING, J. I., AND NIENABER, M. W. P., *J. Dental Research*, **25**, 327 (1946)
132. IRVING, J. I., *Nature*, **162**, 377 (1948)
133. WOLBACH, S. B., *Proc. Inst. Med. Chicago*, **16**, 188 (1946)
134. MELLANBY, E., *J. Physiol. (London)*, **105**, 382 (1947)
135. VAN METRE, T. E., Jr., *Bull. Johns Hopkins.*, **81**, 305 (1947)
136. TOOMEY, J. A., AND MORISSETTE, R. A., *Am. J. Diseases Children*, **73**, 473 (1947)
137. NELSON, M. M., SULON, E., BECKS, H., AND EVANS, H. M., *Proc. Soc. Exptl. Biol. Med.*, **66**, 631 (1947)
138. LEVY, B. M., AND SILBERBERG, R., *Proc. Soc. Exptl. Biol. Med.*, **63**, 355 (1946)
139. SILBERBERG, M., LEVY, B. M., AND YOUNGER, F., *Proc. Soc. Exptl. Biol. Med.*, **67**, 185 (1948)
140. LEVY, B. M., AND SILBERBERG, R., *Proc. Soc. Exptl. Biol. Med.*, **63**, 380 (1946)
141. SILBERBERG, R., AND LEVY, B. M., *Proc. Soc. Exptl. Biol. Med.*, **67**, 259 (1948)
142. CRAMPTON, E. W., *J. Nutrition* **33**. 491 (1947)
143. GIROUD, A., AND PARANT, M., *Rev. stomatol.*, **48**, 44 (1947)
144. BOURNE, G. H., *Proc. Nutrition Soc. (Engl. and Scot.)*, **4**, 204 (1946)
145. MURRAY, P. D. F., AND KODICEK, E., *Proc. Nutrition Soc. (Engl. and Scot.)*, **4**, 200 (1946)
146. BELL, G. H., CHAMBERS, J. W., AND DAWSON, I. M., *J. Physiol. (London)*, **106**, 286 (1947)
147. PINCUS, J. B., GITTLEMAN, I. F., AND KRAMER, B., *Am. J. Diseases Children*, **73**, 458 (1947)
148. LECOQ, R., AND CHAMPEAU, M. F., *Compt. rend. soc. biol.*, **141**, 995 (1947)

149. CHAMPEAU, M. F., AND LECOQ, R., *Compt. rend. soc. biol.*, **141,** 993 (1947)
150. DIKSHIT, P. K., AND PATWARDHAN, V. N., *Indian J. Med. Research*, **34,** 263 (1946)
151. MACH, R. S., in *3e journée de thérapeutique clinique* (A. Skira, Genève, 1947)
152. VOLLMER, H., AND OSER, B. L., *J. Pediat.*, **30,** 446 (1947)
153. MIGICOVSKY, B. B., AND EMSLIE, A. R. G., *Arch. Biochem.*, **20,** 325 (1949)
154. MACH, R. S., FABRE, J., AND DELLA SANTA, R., *J. suisse med.*, **78,** 453 (1948)
155. HOUET, R., *Ann. Paediat.*, **167,** 127 (1946)
156. HOUET, R., *Ann. Paediat.*, **166,** 177 (1946)
157. HOUET, R., *Ann. Paediat.*, **167,** 113 (1946)
158. GLANZMANN, E., MEIER, K., AND UEHLINGER, E., *Z. Vitaminforsch.*, **17,** 130 (1946)
159. IRVING, J. I., *J. Physiol. (London)*, **104,** 253 (1946)
160. GLANZMANN, E., MEIER, K., AND WALTHARD, B., *Z. Vitaminforsch.*, **17,** 159 (1946)
161. IRVING, J. I., AND SCHWARTZ, H. M., *Clin. Proc. (Cape Town)*, **4,** 260 (1945)
162. HOUET, R., *Ann. Paediat.*, **166,** 169 (1946)
163. CAMPBELL, J. A., AND EMSLIE, A. R. G., *Poultry Sci.*, **26,** 255 (1947)
164. CAMPBELL, J. A., AND EMSLIE, A. R. G., *Poultry Sci.*, **24,** 296 (1945)
165. CAMPBELL, J. A., AND EMSLIE, A. R. G., *Poultry Sci.*, **24,** 201 (1945)
166. CAMPBELL, J. A., AND EMSLIE, A. R. G., *Poultry Sci.*, **26,** 568 (1947)
167. CAMPBELL, J. A., AND EMSLIE, A. R. G., *Poultry Sci.*, **26,** 573 (1947)
168. CAMPBELL, J. A., MIGICOVSKY, B. B., AND EMSLIE, A. R. G., *Poultry Sci.*, **24,** 3 (1945)
169. CAMPBELL, J. A., MIGICOVSKY, B. B., AND EMSLIE, A. R. G., *Poultry Sci.*, **24,** 72 (1945)
170. BILLS, C. E., *Biol. Symposia*, **12,** 409 (1947)
171. MIGICOVSKY, B. B., AND EMSLIE, A. R. G., *Arch. Biochem.*, **13,** 185 (1947)
172. MORGAN, A. F., AXELROD, H. E., AND GROODY, M., *Am. J. Physiol.*, **149,** 333 (1947)
173. HENDRICKS, J. B., MORGAN, A. F., AND FREYTAG, P. M., *Am. J. Physiol.*, **149,** 319 (1947)
174. MULLIGAN, R. M., *Am. J. Path.*, **22,** 1293 (1946)
175. MULLIGAN, R. M., AND STRICKER, F. L., *Am. J. Path.*, **24,** 451 (1948)
176. FROST, J. W., SUNDERMAN, F. W., AND LEOPOLD, I. H., *Am. J. Med. Sci.*, **214,** 639 (1947)
177. KAUFMAN, P., BECK, R. D., AND WISEMAN, R. D., *J. Am. Med. Assoc.*, **134,** 688 (1947)
178. MACH. R. S., AND DUCOMMUN, P., *J. suisse med.*, **78,** 732 (1948)
179. REED, C. I., *Federation Proc.*, **6,** 185 (1947)
180. ALBRIGHT, F., *Recent Progress Hormone Research*, **1,** 293 (1947)
181. BAKER, B. L., AND INGLE, D. J., *Endocrinology*, **43,** 422 (1948)
182. FØNSS-BECH, P., *Acta Pharmacol. Toxicol.*, **3,** Suppl. 3 (1947)
183. COLLINS, D. A., BECKS, H., SIMPSON, M. E., AND EVANS, H. M., *Am. J. Orthodontics Oral Surg.*, **32,** 443 (1946)
184. COLLINS, D. A., BECKS, H., ASLING, C. W., SIMPSON, M. E., AND EVANS, H. M., *Anat. Record*, **101,** 13 (1948)
185. COLLINS, D. A., BECKS, H., SIMPSON, M. E., AND EVANS, H. M., *Am. J. Orthodontics Oral Surg.* **32,** 447 (1946)

186. BECKS, H., ASLING, C. W., COLLINS, D. A., SIMPSON, M. E., LI, C. H., AND EVANS, H. M., *Anat. Record*, **101,** 17 (1948)
187. BECKS, H., COLLINS, D. A., ASLING, C. W., SIMPSON, M. E., LI, C. H., AND EVANS, H. M. *Growth*, **12,** 55 (1948)
188. EVANS, H. M., BECKS, H., ASLING, C. W., SIMPSON, M. E., AND LI, C. H., *Growth*, **12,** 43 (1948)
189. ASLING, C. W., BECKS, H., SIMPSON, M. E., LI, C. H., AND EVANS, H. M., *Anat. Record*, **101,** 23 (1948)
190. BECKS, H., COLLINS, D. A., ASLING, C. W., SCOW, R. O., SIMPSON, M. E., AND EVANS, H. M., *Oral Surg. Med. Path.*, **1,** 315 (1948)
191. BECKS, H., SIMPSON, M. E., SCOW, R. O., ASLING, C. W., AND EVANS, H. M., *Anat. Record*, **100,** 561 (1948)
192. BENOIT, J., AND CLAVERT, J., *Arch. Anat. Micro. Morph. Exptl.*, **37,** 214 (1948)
193. TWEEDY, W. R., CHILCOTT, M. E., AND PATRAS, M. C., *J. Biol. Chem.*, **168,** 597 (1947)
194. TWEEDY, W. R., *J. Biol. Chem.*, **161,** 105 (1945)
195. MOEHLIG, R. C., AND ABBOTT, H. L., *J. Michigan State Med. Soc.*, **46,** 1397 (1947)
196. HANDLER, P., AND DE MARIA, W. J. A., *Federation Proc.*, **8,** 204 (1949)
197. CARGILL, W. H., AND WITHAM, A. C., *Federation Proc.*, **8,** 21 (1949)
198. MCCHESNEY, E. W., AND GIACOMINO, N. J., *J. Clin. Invest.*, **24,** 680 (1945)
199. BURNS, C. M., AND HENDERSON, N., *Biochem. J.*, **40,** 501 (1946)
200. MASMONTEIL, F., LEURET, J. AND CARTIER, P., *Rev. Orthop.*, **34,** 120 (1948)
201. STEPHENSON, H. V., AND MCNAMARA, W. J., *Am. J. Med. Sci.*, **215,** 381 (1948)
202. BAKER, B. L., *Anat. Record*, **93,** 125 (1945)
203. LIEGEOIS, F., AND DERIVAUX, J., *Compt. rend soc. biol.*, **140,** 1143 (1946)
204. MULLIGAN, R. M., *Arch. Path.*, **40,** 182 (1945)
205. LIEGEOIS, F., AND DERIVAUX, J., *Compt. rend. soc. biol.*, **143,** 128 (1949)
206. LIEGEOIS, F., AND DERIVAUX, J., *Rev. path. comp.*, **46,** 486 (1946)
207. GILMOUR, J. R., *The Parathyroid Glands and Skeleton in Renal Disease*, 157 pp. (Oxford Medical Publications, London, 1947)
208. BENOIT, J., AND CLAVERT, J., *Acta Anat.*, **4,** 49 –1947)
209. STUDITSKII, A. N., *Nature*, **157,** 427 (1946)
210. LANDAUER, W., *Nature*, **157,** 838 (1946)
211. GABRIO, B. W., AND SALOMON, K., *Endocrinology*, **42,** 73–76 (1948)
212. BENOIT, J., AND CLAVERT, J., *Compt. rend. ass. anat.*, **50,** 2734 (1947)
213. BENOIT, J., AND CLAVERT, J., *Compt. rend soc. biol.*, **139,** 728 (1945)
214. BENOIT, J., AND CLAVERT, J., *Compt. rend soc. biol.*, **139,** 737 (1945)
215. BENOIT, J., AND CLAVERT, J., *Compt. rend. soc. biol.*, **141,** 911 (1947)
216. SALOMON, K., GABRIO, B. W., REINHARD, E., AND SILBERBERG, R., *Arch. Path.*, **43,** 76 (1947)
217. CLAVERT, J., *Arch. Anat. Micro. Morph. Exptl.*, **37,** 41 (1948)
218. REIFENSTEIN, E. C., AND ALBRIGHT, F., *J. Clin. Invest.*, **26,** 24 (1947)
219. REED, C. I., REED, B. P., AND GARDNER, W. U., *Endocrinology*, **38,** 238 (1946)
220. DALLEMAGNE, M. J., AND MELON, J., *Compt. rend soc. biol.*, **141,** 539 (1947)
221. DALLEMAGNE, M. J., GOVAERTS, J., AND SUE, P. *Compt. rend. soc. biol.*, **141,** 541 (1947)

222. GOVAERTS, J., AND DALLEMAGNE, M. J., *Nature*, **161**, 977 (1948)
223. DALLEMAGNE, M. J., GOVAERTS, J., AND MELON, J., *Experientia*, **5**, 331 (1949)
224. BAUER, W. H., *J. Am. Coll. Dentists*, **12**, 192 (1945)
225. YOUNG, W. C., AND EMERY, F. E., *Federation Proc.*, **8**, 174 (1949)
226. PFEIFFER, C. A., *Anat. Record*, **94**, 362 (1946)
227. NORMAN, G. F., AND MITTLER, A., *Proc. Soc. Exp. Biol. Med.*, **67**, 104 (1948)
228. SILBERBERG, M., AND SILBERBERG, R., *Anat. Record*, **95**, 97 (1946)
229. SILBERBERG, M., AND SILBERBERG, R., *Anat. Record*, **98**, 181 (1947)
230. BENOIT, J., AND CLAVERT, J., *Compt. rend soc. biol.*, **141**, 1256 (1947)
231. BENOIT, J., AND CLAVERT, J., *Arch. Anat. Micro. Morph. Exptl.*, **37**, 214 (1948)
232. MC DONALD, M. R., RIDDLE, O., AND SMITH, G. C., *Endocrinology*, **37**, 23 (1945)
233. SILBERBERG, M., AND SILBERBERG, R., *Am. J. Path.* **22**, 1033 (1946)
234. PINDBORG, J. J., *Acta Path. Microbiol. Scand.*, **22**, 290 (1945)
235. BAKER, B. L., AND LEEK, J. H., *Am. J. Physiol.*, **147**, 522 (1946)
236. SILBERBERG, M., AND SILBERBERG, R., *Am. J. Path.*, **24**, 1019 (1948)
237. SILBERBERG, M., AND SILBERBERG, R., *Anat. Record*, **102**, 141–160 (1948)
238. THOMSON, J. A., *Science Old and New*, 192 pp. (Melrose, London, 1946)
239. BEACH, F. A., *Hormones and Behaviour*, 368 pp. (Hoeber, New York, 1948)
240. WISLOCKI, G. B., AND SINGER, M., *J. Comp. Neur.*, **85**, 1 (1946)
241. WISLOCKI, G. B., AUB, J. C., AND WALDO, C. M., *Endocrinology*, **40**, 202 (1947)
242. WISLOCKI, G. B., WEATHERFORD, H. I., AND SINGER, M., *Anat. Record*, **99** 265 (1947)