

Radiographic Differential Diagnosis of Skeletal Diseases of Young Dogs¹

WAYNE H. RISER, D.V.M., M.S.²

INTRODUCTION AND LITERATURE REVIEW

When a young dog develops bowed legs, enlarged joints, and becomes lame, a diagnosis of rickets is commonly made. The diagnosis is often erroneous; true canine rickets is a rare disease in North America (11, 16) and a number of other skeletal disorders may cause the same general superficial clinical signs. Each disease has a different radiological and histological appearance because in each instance the skeleton responds differently. Unless the development and architecture of growing bone is understood in each disease, they will often be confused with one another. The purpose of this report is to review the changes which occur in normally growing long bones, in rachitic bones, and in some other bone disorders commonly encountered in young dogs. The bone diseases of young dogs and cats may result in radiographic densities ranging from osteoporosis to osteosclerosis (7, 8, 14).

GENERAL CONSIDERATIONS

Bone, even in the adult, is not a static tissue; it is under the influence of hormones, minerals, vitamins, and its mineral content is in constant exchange with the

extracellular fluid. Calcium, phosphorous, parathyroid hormone, and vitamin D are the main substances involved in bone metabolism. Imbalance of these substances produces a variety of diseases and the effects of their activity may be revealed in radiographs. Recognition of the bone response to imbalances as recorded in the radiograph is the key to the diagnosis.

NORMAL ENCHONDRAL BONE FORMATION

The main parts of the growing bone consist of the epiphysis, growth plate (epiphyseal line), metaphysis, cut back zone, and diaphysis (Fig. 1). Radiographically the normal epiphyses of the radius and ulna are well defined areas of bone that extend from the carpal articulation to the growth plate (epiphyseal line), and are crisscrossed by fine supporting trabeculae. Proximal to the epiphysis there is a radiolucent line, the growth plate or epiphyseal line. In the normal growing dog this area, composed of hypertrophied cartilage cells, is seldom more than a millimeter in width, even in the giant breeds. Proximal to the growth plate is the metaphysis, a radiopaque zone composed of calcified developing primary bone trabeculae. In the growing dog this area is radiopaque and extremely dense. Grossly the leg is "knobby" over the metaphyseal area, especially in the larger breeds. These metaphyseal prominences are normal for the young growing dog and are most obvious in the distal radius and ulna where growth is most rapid. The diameter of the bone decreases at the cut back or remodeling zone when resorption of primary and

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²Research Assistant Professor of Pathology, Department of Veterinary Biology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Penna.

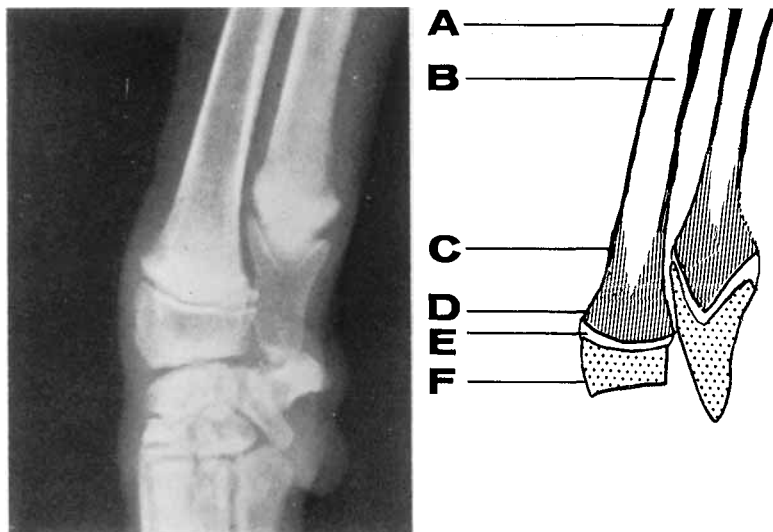


Fig. 1. Dalmatian, four months old, lateral radiograph of distal radius and ulna showing normal bone density and growth areas. (A) bone cortex, (B) diaphysis, (C) cut back zone, (D) metaphysis, (E) growth plate, and (F) epiphysis.

secondary trabeculae occurs in the diaphysis.

Histologically, at the proximal border of the radiolucent growth plate, the matrix left between the disintegrating cartilage cells becomes the calcified cartilage matrix which is first converted into primary, then secondary and tertiary trabeculae, and finally, at the diaphysis, is replaced by typical medullary and compact cortical bone (Fig. 2). This is enchondral bone formation.

In normal osteogenesis, enchondral bone formation is characterized by regular rows of cartilage cells lined up parallel to the long axis of the bone. In a relatively short distance the cartilage cells change from the resting stage to the hypertrophied stage and then disintegrate, leaving tongues of matrix to be calcified and covered with osteoid (unmineralized bone) and mineralized bone as these become the primary bone trabeculae. Histologically the growth plate is a relatively straight thin line of hypertrophied cartilage cells. In normal development, the margins of the bone in the region of the growth plate and metaphysis may bulge prominently. Radiographically, the primary trabeculae (the area just proximal to the growth plate) is the area of greatest radiopacity.

As the animal matures, bone growth slows when the rate of conversion of cartilage

to bone decreases. The bulging of the bone at the growth plate and metaphysis becomes less apparent and is not identifiable either radiographically or by palpation in the adult dog. In the growing dog the increased metaphyseal width is necessary to provide enough tissue to support the weight of the dog without injuring the newly formed, soft bone.

ABNORMAL ENCHONDRAL BONE GROWTH

There are a number of mineral deficiencies, congenital defects, and growth deformities of small animals which are frequently observed. Most of these have been erroneously called rickets (11, 15, 16). Rickets and three other diseases which are sometimes confused with rickets (nutritional secondary hyperparathyroidism, hypertrophic osteodystrophy, and osteodystrophy II) will be discussed and illustrated.

RICKETS

Rickets has been recognized since the advent of recorded history. Well defined references to rickets in man and animals date back to 1650 (3, 9, 10) when it became a prevalent and crippling disease

ENCHONDRAL BONE FORMATION OF THE RADIUS

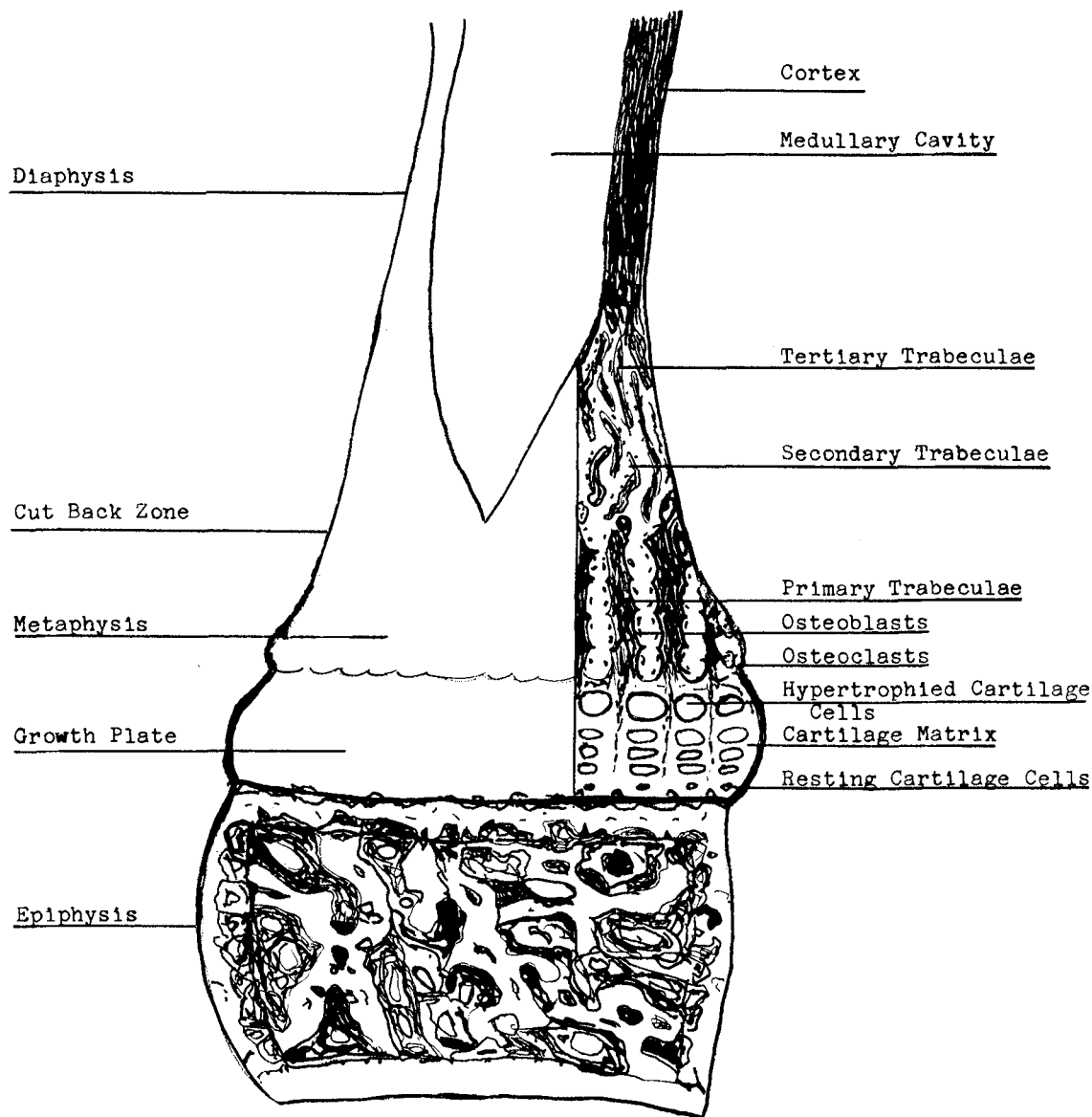


Fig. 2. Sketch of normal enchondral bone development of the distal radius. The areas listed to the left are those visible radiographically (see Fig. 1). The areas listed on the right are those seen histologically.

in Great Britain, Scotland, Europe, and America. In Great Britain in the seventeenth century, when tenement living became common, the incidence of rickets

increased and the condition became known as "the English Disease." The treatment at that time, empirical in nature, was sunshine and fish oil. Seventeenth cen-

tury physicians considered this "old wives" remedy to be ridiculous, as the effects of sunshine were held to be dangerous. From the medical viewpoint, smelly fish oil was classified with disapproved potions.

Rickets was not only a disease of man; young animals were affected as well. So serious and widespread was this ailment by the beginning of the twentieth century, that organized groups strongly urged the British government to support research on its causation.

In 1915, with the aid of state grants, scientists from Great Britain began a study of rickets. The dog was chosen as the chief experimental animal because the disease at that time was prevalent in dogs and closely resembled rickets in man. By 1918 the results of these studies began to show that minerals, vitamin D, and sunshine were effective antirachitic agents (3, 9).

Prophylactic measures brought about radical changes in the diets of both animals and children as manufactured foods were fortified with vitamin D and minerals. Today, years later, because of the fortification of foods, rickets has vanished from most parts of the world.

PATHOGENESIS

Rickets, caused by a deficiency of vitamin D, minerals, and/or sunshine, is primarily a disease affecting the process of normal bone ossification and occurs in most species of young animals. In the dog, the disease usually appears at six to fourteen weeks of age. The chief clinical signs of the disease are delayed growth, apathy, muscular weakness, and bending and distortion of the bones under the weight of walking and the stress of muscular contraction. Gross nodular enlargements are visible at the ends of the long bones. These nodular enlargements arise from the growth plates (epiphyseal lines). They are especially noticeable proximal to the carpal joints and at the costochondral junctions.

In dogs, the normal serum calcium level is approximately 10 mg. per cent; the normal serum phosphorus level is approximately 4.2 mg. per cent. When multiplied, the product of these two figures is approximately 40. If the serum calcium-phosphorus product drops to 30 or under, the development of true rickets in the young, or osteomalacia in the adult dog may be expected (11).

The radiographic features of rickets are best observed in radiographs of the epiphyses, growth plates (epiphyseal lines), metaphyses, and diaphyses of the radius and ulna. Knowledge of the normal radiographic appearance of the young dog's skeleton is essential for purposes of comparison with diseased bone.

BONE CHANGES IN RICKETS

Radiographs of the rachitic dog (Figs. 3 and 4) are characterized by:

1. Widened radiolucent growth plate (epiphyseal line).
2. Failure of adequate mineralization of all bones.
3. Bowing of the long bones and extreme enlargement (bulging) of the metaphyses adjacent to the growth plates, especially the distal radius, ulna, and costochondral junctions.
4. Widened, arched, irregular epiphyseal and metaphyseal borders at the growth plate.
5. Poor mineralization or lack of density in the metaphyses from failure of the metaphyseal osteoid to calcify.

Generally poor mineralization of the bones, thin cortices, bowed legs, arched and irregularly widened radiolucent growth plates, and poor mineralization of the metaphyses were observed in radiographs obtained from Scotland of two dogs with rickets (Figs. 3 and 4).

Histologically, a section of rib from one of these two dogs showed excessive bulging at the growth plate and the adjacent metaphyseal area. Clinically this is known as

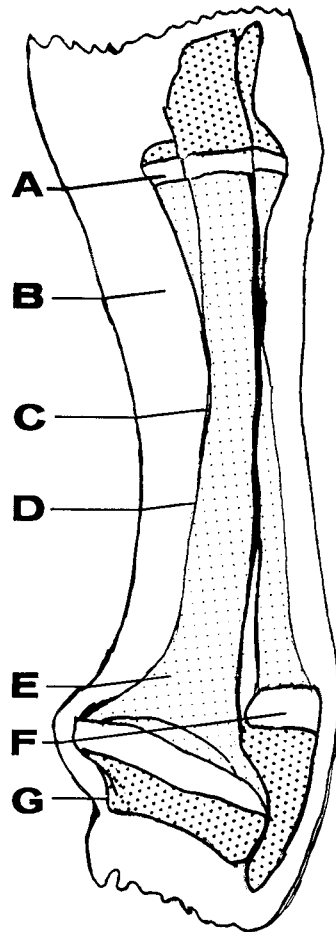


Fig. 3. Advanced rickets in a three-month-old dog. The bowed radius and ulna are poorly mineralized, there is a wide irregular growth plate (epiphyseal line), and the metaphysis is poorly mineralized. (A) growth plate, (B) soft tissue, (C) bowed radius and ulna, (D) cortex, (E) metaphysis, (F) growth plates, and (G) epiphysis.

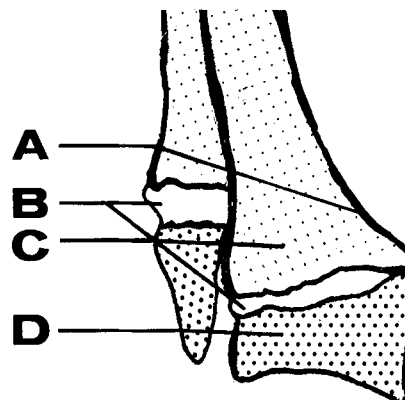


Fig. 4. Rickets in a four-month-old dog. The foreleg is bowed, the cortex and metaphysis are poorly mineralized, and the growth plate is widened and irregular. (A) cut back zone, (B) growth plates, (C) metaphysis, and (D) epiphysis.

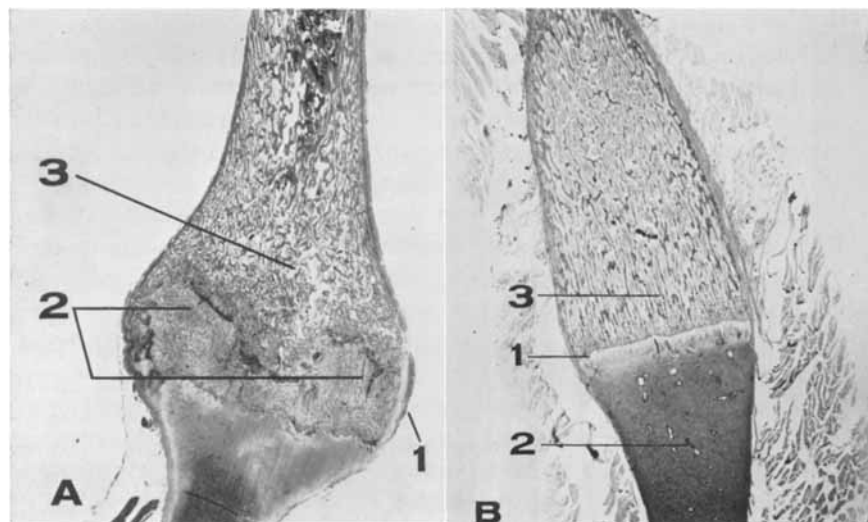


Fig. 5. Figure 5A is a histological cross section of a rachitic rib and the costochondral junction showing the "rosary" like bulge at the metaphysis. (1) The irregular line of the growth plate, (2) the persistent hypertrophic cartilage, and (3) the osteoid and retained cartilage cells in the metaphysis. Figure 5B is a normal rib of a growing dog showing (1) normal growth plate, (2) resting cartilage, and (3) metaphysis.

"rosary bulging." Tufts or nests of persistent cartilage cells can be seen histologically in the metaphyseal-diaphyseal area (Fig. 5).

In rickets the primary trabeculae are scanty, short, thick, distorted, and surrounded by wide bands of pink-staining osteoid (Fig. 5). The unmineralized osteoid and the poorly ossified bony trabeculae have a radiographic density of soft tissue. Radiolucent widened cartilage bands of the growth plates are characteristic of well established canine rickets (Figs. 3 and 4). Grossly the metaphyseal areas are greatly enlarged (rosary bulging). Tongues of hypertrophied cartilage cells in irregular disorderly rows distend the metaphyseal area; uncalcified bone, known as osteoid, is seen adjacent to the hypertrophied cartilage. This tissue at the metaphysis accounts for the bulging observed grossly.

DISCUSSION

There has been confusion about the diagnosis of rickets in animals (11, 16). Diseases in animals now diagnosed as rickets often have little resemblance radiographically or histologically to rickets in man (2). In America, one investigator attempted to

demonstrate rickets in farm animals for more than twenty years, but failed (11). The same investigator observed, "... so-called rickets in dogs and cats comprises a miscellaneous group of calcium and phosphorus deficiencies, hereditary bone and cartilage diseases, and skeletal deformities Rickets is frequently diagnosed in the dog, but the lesions of rickets are not present at necropsy examination" (16).

The author of this paper has attempted to produce rickets in young dogs on two occasions, one with Beagles and the other with Greyhounds (15). Both attempts failed. The dogs in both instances were well nourished and forty-five days old when the experiments were begun. They were kept in darkened rooms and fed a rachitogenic diet³ for one hundred days before being killed. During this one hundred twenty day period, the dogs were radiographed and weighed weekly. The dogs did not eat the diet well. They became underweight and grew very little during the experiments. At necropsy, the bones (including the growth plates) varied very little radiographically and histologically from normal. At no time during the course of the experiments was evidence of rickets observed radiographically.

³ A mixture of oatmeal, white flour, and ground corn meal. Distilled drinking water was provided

True rickets seldom occurs in either young dogs or calves anymore in England around Oxford and Cambridge where the pioneer research on this disease was done forty years ago. Today it is hard to imagine that rickets in 1900 was prevalent in this same area of Great Britain and in Europe. The two dogs with rickets mentioned previously came from the hills of Scotland where there is little sunshine or available fortified food (Figs. 3 and 4). Some of the hills of this northern country are isolated and dark in climate. Food is all home raised and not supplemented with minerals and vitamins. Both the histological sections and radiographs of the growth plates of the bones were characteristic of rickets in these two dogs.

Today in Great Britain and Europe, as in America, the people depend on at least some commercially prepared foods. These are fortified with minerals and vitamin D. In these areas rickets has almost disappeared. Because of this, it is theorized that to produce rickets, the experiment must start with a mother that is fed a rachitogenic diet in the almost complete absence of sunshine. It is believed that healthy pups by birth have passed the period of development when classical rickets may occur (11). In young animals born from a mother with rickets, the young can be expected to develop clinical and radiographic rickets by ten weeks of age if the deficiencies are maintained.

In Sweden, where there is little sun during the winter, rickets occurs in calves and young dogs if the deficiency in minerals and vitamin D is not corrected (12).

NUTRITIONAL SECONDARY HYPERPARATHYROIDISM (OSTEODYSTROPHIA FIBROSA, JUVENILE OSTEOPOROSIS)

Once called osteogenesis imperfecta, a congenital disease of man, this disease of the young dog and cat is often mistaken for rickets because of the bowing and folding fractures of the long bones. The dis-

ease is a nutritional one that develops when the calcium intake is low and the phosphorus intake is high. This disease occurs frequently in young cats and dogs when they are fed exclusively on diets of horsemeat, beef heart, beef and pork kidney, or liver. These meats are low in calcium and high in phosphorus (ratio 1:20).

For example, if a cat was given an exclusive diet of beef heart at the rate of 150 gm. per day, this would not provide the animal with over 15 mg. of calcium per day. The daily requirement of calcium for a normal young cat is 70 to 90 mg. per day. Beginning with normal young cats, pathological fractures have been produced in ninety days by the use of a diet consisting exclusively of horsemeat and distilled water. Pathological fractures were observed within thirty-five days after well-nourished thirty-day-old German Shepherd dogs and Greyhound dogs were fed a diet consisting exclusively of horsemeat and distilled water (13).

A low dietary intake of calcium eventually produces a low serum calcium. This stimulates the parathyroid glands to secrete parathormone which causes skeletal resorption of calcium in an attempt to correct the hypocalcemia. Such a drain demineralizes the bones; the thin cortices and trabeculae, deficient in calcium, bow and fracture as the result of compression and stress. The osteoporosis of the cortices, the bowing of the long bones, and the presence of folding fractures are often mistaken for rickets.

Radiographs of nutritional secondary hyperparathyroidism (Fig. 6) are characterized by:

1. Inadequate mineralization of all bones (osteoporosis).
2. Normal growth plates but poorly mineralized metaphyses.
3. Folding fractures at the points of greatest compression and stress.
4. Folding fractures present in long bones and in thoracic and lumbar vertebrae.

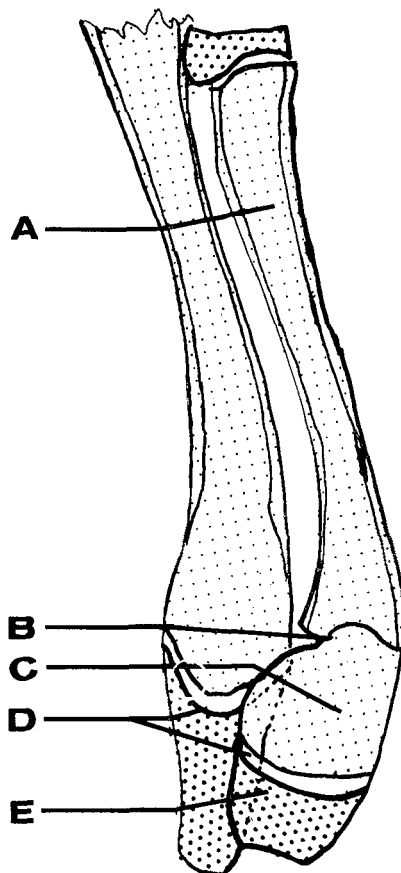


Fig. 6. A German Shepherd dog, four months old. In a lateral radiograph of the foreleg there is (E) a normal epiphysis and (D) growth plates. The (C) metaphyseal area of the radius is demineralized and a fracture is present in the proximal metaphyseal area (B). The cortices of the diaphyses (A) are very thin and osteoporotic. The (C) metaphysis of the ulna is osteoporotic.

DISCUSSION

When normal mineral metabolism is threatened by inadequate dietary mineral intake, the mineral reserve in the bones is utilized to provide normal blood levels of these elements.

Radiographically, growth in length of the long bones continues. The epiphysis is of normal size but the cortex and trabeculae are very thin. The radiolucent growth plate is of normal width. Cartilage proliferation and turnover rates are normal. In the metaphysis the radiodensity is decreased because of the decrease in calcium available to mineralize the cartilage matrix of the primary and secondary trabeculae. The diaphyseal cortex is also osteoporotic, as the available bone calcium is being mobilized to keep blood calcium at a normal level to support metabolic processes where calcium is needed to support life.

The new bone being produced under this mineral-restricted regime is so deficient in calcium that the areas in the metaphysis and diaphysis that normally show a detailed radiopaque image are reduced to densities sometimes as radiolucent as soft tissue. The osteoporotic bone thus produced is not strong enough to withstand the compression and stress forces of the body. Bowing and folding fractures of the long bones and vertebrae result.

Other comparable bone changes occur in the dog from excessive excretion of calcium in the urine because of renal disease (1). One, usually seen in older dogs, is called renal osteodystrophy and is most obvious in the bones of the head, particularly the mandibles (rubber jaw). An osteodystrophy, characterized radiographically by generalized osteoporosis, has been reported as the result of hypercalcuria associated with renal cortical hypoplasia (4, 5). An-

other generalized osteodystrophy occurs in monkeys in which the diet has been restricted largely to fruits and nuts (6). These foods are low in calcium.

HYPERTROPHIC OSTEODYSTROPHY

Hypertrophic osteodystrophy in young dogs of the giant type is a disease in which excessive quantities of radiopaque min-

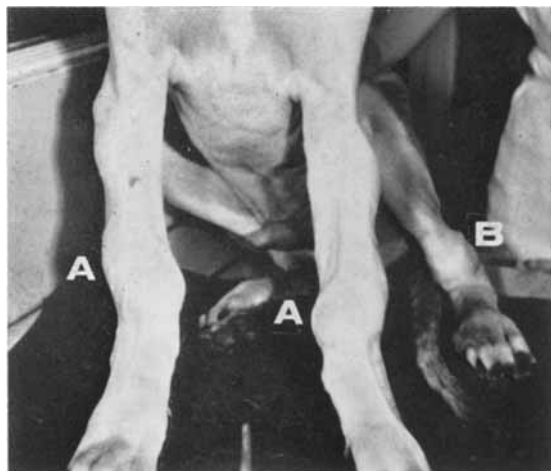


Fig. 7. A Great Dane dog, six months old. There is extensive enlargement of the metaphyseal areas of the distal ends of the radius and ulna (A) and the (B) tibiae.

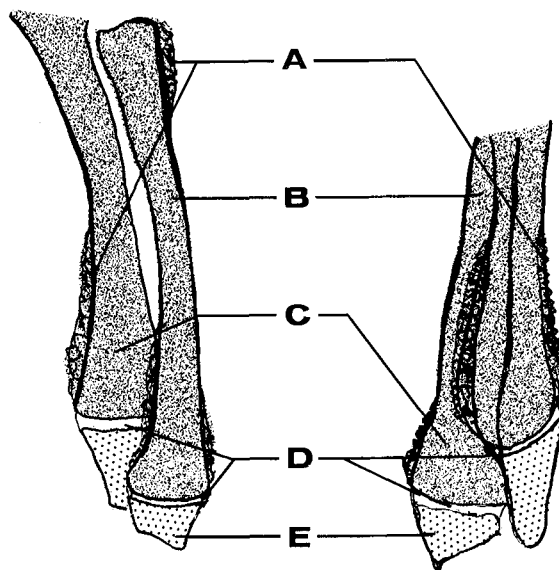


Fig. 8. Hypertrophic osteodystrophy of the radius and ulna. (A) extra-ossification along the metaphyseal areas of the proximal and distal ends of the radius and the distal end of the ulna, (B) diaphyses, (C) increased radio-density of the metaphyses, (D) normal growth plates, and (E) normal epiphyses.

erals are deposited in the metaphyses where enchondral osteogenesis is most active (distal radius and ulna, and distal tibia). In instances of extreme osteogenic activity, ossification occurs in the soft tissue outside the periosteum adjacent to the metaphysis. This excessive osteogenesis causes grossly visible hypersensitive enlargements in the metaphyseal area (Fig. 7). If severe, the disease affects the dog systemically. The metaphyseal swellings are warm, sometimes even hot, and the dog appears depressed and is reluctant to stand on his legs or to move. The dog becomes anorectic and may have a temperature of 104 to 106° F.

Radiographs of hypertrophic osteodystrophy (Fig. 8) are characterized by:

1. Increased radiopacity at the metaphyses.
2. Excessive enlargement of the metaphyses.
3. Beaded radiopaque deposition of bone surrounding the metaphyses in the soft tissue outside the periosteum.
4. Normal appearing epiphyses and growth plates.

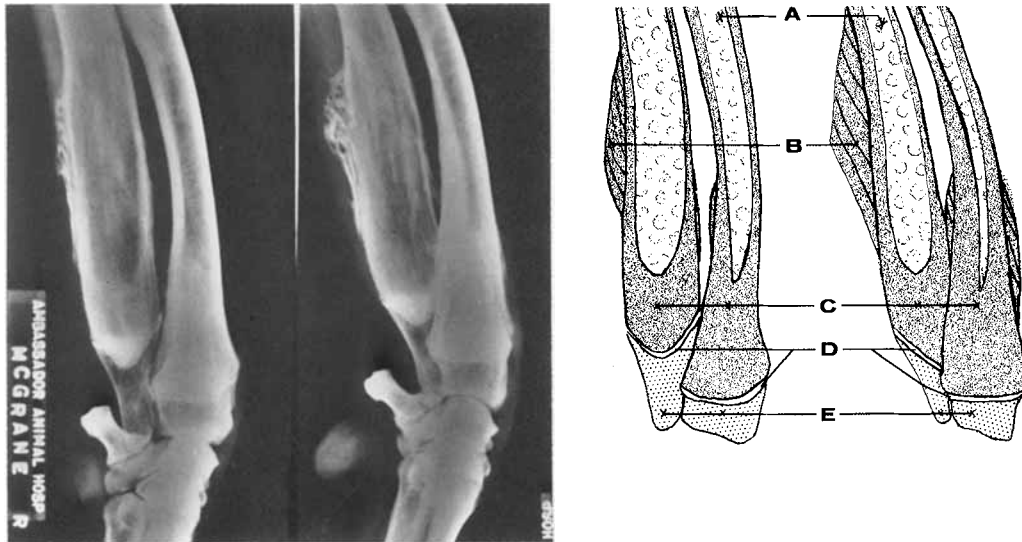


Fig. 9. Hypertrophic osteodystrophy in the healing phase. (A) diaphyses, (B) areas of extra-ossification being absorbed, (C) extremely radiopaque newly formed metaphyseal bone returning to normal in appearance with no newly formed extra-periosteal ossification, (D) normal growth plates, and (E) normal epiphyses.

Radiographically, the skeleton as a whole appears normal except for the increased radiopacity of the metaphyses, and the accompanying beaded radiopaque deposition of bone surrounding the metaphyses outside the periosteum from the growth plates proximally toward the diaphyses. In the metaphyseal areas where bone growth is not normally rapid, e.g., proximal radius and tibia, the metaphyses are extremely radiopaque but calcification may not occur in tissues outside the periosteum.

In dogs in which the hypertrophic osteogenesis subsides, the appearance of the metaphyses returns to normal density and contour (Fig. 9). When the rate of osteogenesis returns to normal, no additional radiopaque material is deposited outside the periosteum. The extra-periosteal mineralization previously deposited is absorbed slowly. The normal tubular diaphyseal contour returns. Normal locomotion is resumed, the fever subsides, and the appetite returns to normal.

The etiology of hypertrophic osteodystrophy is unknown. In the author's experience, one predisposing factor seems to be the administration of excessive amounts

of vitamin D, minerals, and other food and vitamin supplements. It appears reasonable to hypothesize that forced supplemental feedings may stimulate hypertrophic osteogenesis.

OSTEODYSTROPHY II

A variant of hypertrophic osteodystrophy, or perhaps a third disease, is seen in Fig. 10. This disease was observed in a five-month-old Irish Setter. The metaphyses are enlarged, and proximal to the carpal joints an area of increased density is seen. Immediately proximal to the growth plates in the region of the primary trabeculae disintegration of the tissues has taken place in the radiopaque area. The radiopaque continuity is broken, giving the tissue a disorganized, irregular, granular appearance. The areas proximal to this appear normal, indicating that when this portion of the bone was developing no disease was present. The cortices in the diaphyseal area also appear normal. The dog recovered with rest and an adequate diet.

Radiographs of osteodystrophy II (Fig. 10) are characterized by:

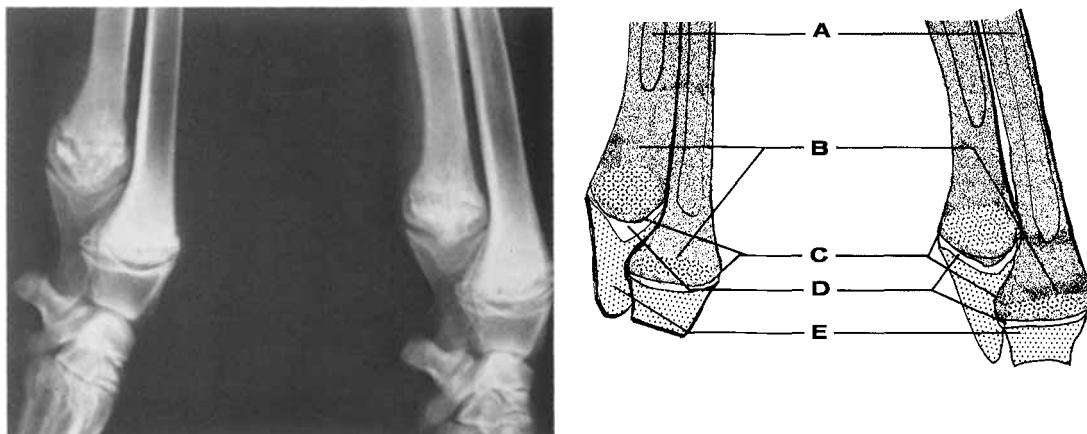


Fig. 10. Osteodystrophy II in an Irish Setter, five months old. (A) diaphyses, (B) metaphyses, (C) disorganized and irregular granular appearance of the metaphyses just proximal to the growth plates, (D) normal growth plates, and (E) normal epiphyses.

1. Increased enlargement and radiopacity of the metaphyses.
2. Smooth homogenous bone opacity is broken and becomes disorganized, irregular, and granular in the metaphyses immediately proximal to the growth plate.
3. The epiphyses, the growth plate, and the cortices are normal in appearance.
4. There is no evidence of extraperiosteal ossification.

This is a disease characterized by hyperossification of the metaphysis, normal turnover of cartilage at the growth plate, but granular disintegration of the metaphysis just proximal to the growth plate.

SUMMARY AND CONCLUSIONS

The differentiating characteristics of rickets and three other diseases often mistaken for rickets (nutritional secondary hyperparathyroidism, hypertrophic osteodystrophy, and osteodystrophy II) have been discussed and compared to normal enchondral ossification.

By understanding normal enchondral bone formation, the characteristics of each of these diseases and how they differ from each other can be more fully comprehended.

Rickets

Because of a lack of vitamin D and minerals the cartilage cells of the growth plate pile up, do not complete their cycle, and disintegrate. The absence of rows or tongues of cartilage matrix provides no tissue templates on which bone can form. Even if they were present, bone could not be formed on them as there is no conversion of osteoid to bone without vitamin D. Osteomalacia occurs as a result of poor quality bone formation. Under present conditions with food fortified with vitamin D, rickets is a rare disease and it is seen only under very unusual circumstances of mineral, vitamin, and sunshine deprivation.

Nutritional Secondary Hyperparathyroidism (*Osteodystrophia Fibrosa, or Juvenile Osteoporosis*)

The essential element lacking in the bones is calcium. Osteogenesis is normal, but the bones do not become adequately mineralized because of the lack of minerals. Fibrous tissue fills the areas where supporting bone and normal bone marrow cells should be. The disease occurs in young animals where the diet is solely or largely muscle meat and visceral organs.

Hypertrophic Osteodystrophy

This is a disease in which there is excessive bone formation in the metaphyses and extra-metaphyseal periosteal calcification. Excessive amounts of bone are laid down in the metaphyses of all long bones. If the osteogenesis is too great, there is bone formation in the soft tissue outside the periosteum surrounding the metaphyses. The cause of this disease is not known, but it seems reasonable that bone metabolism is hyper-stimulated.

Osteodystrophy II

This may or may not be a separate disease and may be a variant of hypertrophic

osteodystrophy. There is hyperosteogenesis with degeneration, breaking up, or failure of the supporting tissue immediately proximal to the growth plate. The exact microscopic features of this disease are not yet known. This seems to be too rapid growth for the minerals being laid down.

University of Pennsylvania
School of Veterinary Medicine
39th and Pine Streets
Philadelphia, Penna. 19104

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ZUSAMMENFASSUNG

Die Unterscheidungsmerkmale der Rachitis und drei weiterer Krankheiten, die fälschlicherweise oft für Rachitis gehalten werden (sekundärer Ernährungs-Hyperparathyreoidismus, hypertrophische Osteodystrophie und Osteodystroph II), wurden besprochen und mit normaler enchondraler Knochenbildung verglichen. Die Kenntnis der normalen enchondralen Knochenbildung verglichen. Die Kenntnis der normalen enchondralen Knochen-

RÉSUMÉ

Les caractéristiques qui permettent de différencier entre le rachitisme et trois autres maladies souvent prises à tort pour du rachitisme (Hyperparathyroïdisme, nutritionnel secondaire, Osteodystrophie hypertrophique et osteodystrophie II) ont été discutées et comparées à celles de l'ossification enchondrale normale.

Les caractéristiques de chacune de ces maladies, et la manière dont elles diffèrent les unes des autres

bildung erlaubt ein besseres Verstehen der Eigenschaften jeder dieser Krankheiten und wie sie sich voneinander unterscheiden.

Rachitis

Wegen eines Vitamin D und Mineralmangels häufen sich die Knorpelzellen in der Proliferationszone an, vervollständigen ihren Lebenslauf nicht, und zerfallen. Knochenbildung kann hier nicht stattfinden, da ein Knorpelgerüst nicht vorhanden ist. Selbst wenn ein Knorpelgerüst vorhanden wäre, könnte keine Knochenbildung stattfinden, da eine Umwandlung von Osteoid in Knochen ohne Vitamin D nicht möglich ist. Als Folge schlechter Knochenbildung entsteht eine Osteomalazie. In der heutigen Zeit, in der Lebensmittel Vitamin-D Zusatz erhalten, ist Rachitis eine seltene Krankheit. Rachitis ist nur unter ganz ungewöhnlichen Umständen wie Mineral-, Vitamin- und Sonnenscheinmangel anzutreffen.

Sekundärer Ernährungs-Hyperparathyreoidismus (Osteodystrophia Fibrosa Oder Juvenile Osteoporose)

Das mangelnde Element ist Kalk. Knochenbildung ist normal, aber die Knochen erhalten nicht genügend Minerale, da ein Mineralmangel besteht. Bindegewebe füllt die Gebiete an, in welchen Knochenbälkchen und normales Knochenmark sein sollten. Diese Krankheit tritt bei jungen Tieren auf, deren Ernährung nur oder grösstenteils aus Muskelfleisch und inneren Organen besteht.

Hypertrophische Osteodystrophie

Das Hauptmerkmal dieser Krankheit ist übermässige Knochenbildung in der Metaphysis. Ausserdem findet extra-metaphyseale periosteale Verkalkung statt. Übermässige Knochenmengen lagern sich in der Metaphysenzone aller langen Knochen ab. Wenn die Knochenbildung zu gross ist, kann es auch zur Knochenbildung in den Geweben ausserhalb des Periosteums in der Metaphysenumgebung kommen. Die Ursache dieser Krankheit ist unbekannt, aber es erscheint, dass Knochenbildung überstimuliert ist.

Osteodystrophie II

Dies ist möglicherweise eine gesonderte Krankheit und könnte eine Variante der hypertrophischen Osteodystrophie sein. Es erscheinen Hyperosteo-genese mit Degeneration, ein Zerfall oder ein Ausbleiben des Stützgewebes, das sich unmittelbar proximal an die Proliferationszone anschliesst. Die genaue Histopathologie dieser Krankheit ist noch unbekannt. Das Wachstum scheint hier viel zu schnell zu sein ohne dass genügend Mineralien abgelagert werden.

peuvent être mieux comprises grâce à la connaissance de la formation normale de l'os endochondral.

Rachitisme.—Dû au manque de vitamine D et de minéraux, les cellules cartilagineuses de la plaque de croissance s'accumulent, ne complètent pas leur cycle et se désintègrent. L'absence de rangées ou de langues de matrix cartilagineuses ne fournissent pas de plaques réceptrices sur lesquelles l'os peut se former. Même si elles étaient présentes, l'os ne pourrait pas se former dessus puisque sans vitamine D l'ostéode ne peut se transformer en os. L'ostéomalacie a pour cause la formation d'os de mauvaise qualité. Dans les conditions présentes, grâce à la nourriture enrichie de vitamine D, le rachitisme est une maladie rare, et on la rencontre seulement dans des circonstances très extraordinaires comportant la privation de minéraux, de vitamines et de soleil.

Hyperparathyroidisme Secondaire de Nutrition (osteodystrophie fibreuse ou osteoporose juvenile). Le calcium est l'élément essentiel manquant dans les os. L'ostéogénèse est normale mais les os ne se minéralisent pas suffisamment du au manque de minéraux. Du tissu fibreux remplit les espaces où le tissu osseux et les cellules normales de la moelle devraient être. La maladie se produit chez les animaux jeunes dont le régime alimentaire comporte uniquement ou principalement de la viande rouge ou des organes viscéraux.

Osteodystrophie Hypertrophique.—Cette maladie est caractérisée par une formation osseuse excessive de la metaphyse, et une calcification periostale, extra metaphysale. Des quantités excessives de tissu osseux sont déposées sur la metaphyse de tous les os longs. Si l'ostéogénèse est trop considérable, la formation de tissu osseux prend place dans les tissus mous qui entourent les metaphyses à l'extérieur du périoste. Les causes de cette maladie ne sont pas connues, mais raisonnablement il apparaît que le métabolisme osseux soit hyperstimulé.

Ostéodystrophie II.—Ou ne sait si cette maladie est distincte ou non de l'ostéodystrophie hypertrophique, elle en est peut-être une variante. On peut observer une hyperostéogénèse avec dégénération, cassure, ou défaut du tissu de support immédiatement adjacent à la plaque de croissance. Les caractéristiques microscopiques exactes de cette maladie ne sont pas connues pour l'instant. Il semble qu'il y ait une croissance trop rapide pour que les minéraux puissent être déposés.