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A commonly made statement is that avian bone tends to be different to mammalian bone. This is relatively meaningless, given the huge diversity of birds and mammals. There are similarities in bone growth, microstructure and healing but there are some differences.

In the UK I treat mainly parrots and raptors and it is unusual for me to see anything else. These lectures will cover these birds with only occasional reference to other genera. Any comparison with mammals will be with dogs, cats and rabbits. The birds that I see are all able to fly. The raptors tend to fly to capture their prey, much of which is live animals. Many birds of prey are migratory, some between the northern and southern hemispheres. Although parrots are mostly vegetarian they often have to fly significant distances to find their food. Many parrots make local migrations, e.g. from lowlands to highlands depending on the season.

PNEUMATISATION

Birds that fly tend to have lighter skeletons than birds that do not. Flightless birds tend to be heavier and have thicker bone than birds that fly. The Frigate Birds (*Fregeta* spp.) are masters of flying and their bones weigh less than their feathers! This reduction in weight is achieved by pneumatisation of the bones. In most birds the vertebrae, pelvic, sternal and costal bones are invaded by diverticula of the air sacs which replace the bone marrow. The pneumatisation of the limb bones varies greatly between species. Whilst it is easy to show that a bone is aerated it is not so easy to prove that it is not. Accounts of which bones are pneumatised should always be read with this in mind. Although in some soaring birds nearly all the bones are pneumatised, there seems to be no correlation between the presence of air in the bone and the bird's flying habits. Many diving birds, such as penguins, diving ducks (Anatidae), cormorants and others have very poor or absent pneumatisation. However, gannets (Sulidae) have diverticula in the fascial planes beneath the skin and between the skeletal muscles as well as extensive pneumatisation of the bones. Most ratite birds are moderately pneumatised whereas the kiwi is not. Pneumatisation tends to be extensive in large powerful fliers such as eagles, swans and albatross but in the small birds noted for their flying ability, such as hummingbirds and swallows, there is minimal pneumatisation. It has been observed that pneumatised bones have a greater girth than other bones.

The cervical air sacs aerate the cervical and thoracic vertebrae and the vertebral ribs, the clavicular air sac the sternum, sternal ribs, pectoral girdle and humerus (and presumably the rest of the wing), and the abdominal air sac the synsacrum, pelvis and femur. The air sacs enter the bone through either a large opening, the pneumatic fossa or smaller holes, the pneumatic pores. The aerated bones are often more light lucent. Whilst some parts of the air sacs are lined with ciliated epithelium, the lining in the bone is a simple squamous epithelium.

BONE STRUCTURE

Much of the work on the bones of birds has been carried out on chickens but similar results have been obtained with other species on the rare occasions when they have been examined. A comprehensive overview of bone biology can be found in Whitehead (1992).

Bone has two functions. Firstly it acts as an interface between the muscles and as a structural support for the body. In young birds the bone has to be able to grow with the body and although growing bone is weaker than mature bone, it must provide appropriate strength whilst it does so. Secondly it serves as a calcium and phosphorus reservoir, which is particularly important for hard egg-shell production. Almost 99% of the body's total calcium and approximately 80% of its phosphorus is present in the skeleton; there is also some sodium and magnesium. These two roles can be contradictory. Bone grows and is maintained by a complex interaction between systemic hormones and local growth factors and between the cell types involved in bone metabolism – osteoblasts (+ osteocytes) and osteoclasts.

Bone is made of a hard mineral compound enmeshed within organic fibres. Hydroxyapatite represents the mineral phase of both types of avian bone. Collagen is the principal organic component. The mineral resists compression and the fibres withstand tension and torsion. The cortical bone has up to 70% of inorganic salts arranged in a hydroxyapatite lattice structure. In birds bone has to be laid down in a form that can be easily removed and replaced depending on the body's calcium requirements. The mineral has to be insoluble but when phosphorus or calcium is required for a metabolic reason it has to be readily broken down by a cellular control. Bone is also unique in its ability to remodel according to stress and strain. So although the general shape of a bone is predicted and controlled genetically, the internal organisation can vary widely due to adjacent environmental influences. It can vary the thickness of the cortex, the amount of trabecular or cancellous bone and the amount of membranous bone of the skull. Collagen is a major component of bone - it has high tensile strength: a collagen fibre of 1 mm diameter can support 40 kg. Once the collagen is impregnated with hydroxyapatite it provides high compressive stiffness. The collagen in bone is laid down by osteoblasts as parallel bundles of fibrils. The osteoblasts are able to sense the orientation of these fibrils and tend to lay them down with a cross-ply effect. The most important non-collagen protein in bone is osteocalcin. It is concerned with maturation of bone rather than bone growth. It is a good marker for bone metabolism in health and disease.

Compact (cortical or lamellar) bone has concentric layers of matrix surrounding longitudinal vessels – the Haversian system. It is found primarily in the shafts of the long bones of the appendicular skeleton and is stronger and more rigid than cancellous or trabecular bone. Lamellar is a term used when the collagen fibres are laid down in an orderly manner.

Cancellous (trabecular or spongy) bone is a soft bone consisting of thin intersecting lamellae internal to cortical bone. It is found throughout the avian skeleton. In the young growing bird it is filled with red marrow, but later this is replaced by fatty marrow or air filled cavities.

Intramembraneous bone is formed within a connective tissue membrane, under the periosteum. It is found primarily in the axial skeleton and consists of a framework of bony struts that is weaker than cortical bone but provides a metabolically active light-weight support. This bone is sandwiched between two plates of cortical bone and forms the flat bones – skull, scapula, vertebrae and so on.

Medullary bone is labile bone that normally occurs only in female birds in their reproductive phase.

It can also be formed by male birds with hormonal abnormalities. It resembles cancellous or embryonic bone and consists of interconnected spicules that grow out from the endosteal surface of the long bones. Medullary bone has no Haversian system and contains less collagen than normal bone. Phases of formation and destruction alternate during the laying cycle. The formation of medullary bone is controlled by oestrogens and androgens. Medullary bone is laid down in the medullary cavity of bones that contain red bone marrow. Occasionally it can be found at the endosteal part of pneumatized bones, i.e. where there is a blood supply. It is obvious in the long bones. In poultry it develops about 10 to 14 days before the start of laying. In poultry medullary bone does not fill the entire cavity. In pigeons, raptors and parrots it fills the whole cavity in the long bones that are not pneumatized. Medullary bone is laid down in response to androgen and oestrogen.

Some aspects of medullary bone are unique. The trabeculae are arranged randomly. The collagen filaments are arranged in a more haphazard manner. The hydroxyapatite is bound less closely to the collagen. The bone is also hypermineralised compared with cortical bone. Medullary bone osteoclasts are synchronised with egg shell production.

In poultry during the immediate pre-laying period the retention of calcium and phosphorus from the digestive tract is greatly increased. Excretion is probably decreased. The skeletal weight increases by about 20%. This represents about 4 to 5 g of calcium, mainly as medullary bone.

In spite of this, in poultry eating a diet containing adequate calcium and phosphorus, the calcium for the shell is produced mostly from intestinal absorption. Poultry are usually in negative calcium balance for the first 6 weeks of laying. Interestingly poultry that come into lay precociously lose all the mineral they have stored within the first 6 weeks. If the birds are prevented from laying by manipulating the photoperiod until they are more mature, they lose little of their skeletal calcium.

Much of the bone is laid down in successive layers to form dense compact bone that is covered on its outer surface by cellular periosteum. In the long bones there is an inner endosteal layer that contains many osteoblasts. There is a medullary cavity that is filled with air, fat, or marrow. The bone itself contains osteocytes that can be turned into osteoblasts. The osteocytes lie in the lacunae and communicate with each other by a series of canaliculi. Although they have lost their synthesising ability they seem to have a role in the way the bone responds to mechanical loading. The last members of the osteoblasts are the bone lining cells, which are found on the periosteal surfaces and on the surface of trabecular bone.

Bone formation is the responsibility of osteoblasts.

EMBRYOLOGY

In the domestic fowl, on day 4 of incubation the humeral girdle and forelimb starts to form. After this stage, it has four processes, one of which grows out to become the wing and three grow in to form the scapula, clavicle and coracoid. The clavicles are the first to ossify (6-7 days) and the right and left clavicles fuse at 12-13 days.

Birds are pentadactyl. The 6 day embryo has a spade-like wing. Two of the five digits disappear. There are also 7 carpal bones but with loss and fusion they end up as two free carpal bones. The skeletal development in the altricial bird is more rapid than the precocial bird. In transitional types (birds that have both altricial and precocial traits) the rate depends on the amount of yolk and the duration of embryogenesis.

The hind limb develops in a similar manner: there are more bones in the embryo than the adult. The fifth digit and its metatarsal disappear. Metatarsals fuse, three tarsal bones 'disappear', the pelvic girdle also forms from three bones. The ischiadic bone is the largest.

Long bones go through 3 developmental stages: membranous or precartilaginous, cartilaginous, and ossification. When the bones first form as a cartilage rod, ossification takes place at a central point in the diaphysis and gradually spreads towards the ends of the growing bone which always remain cartilaginous and ensure longitudinal growth of the bone. Transverse growth of the bone occurs from the periosteal side and there is simultaneous enlargement of the marrow cavity. The first calcium deposition in the chick is in the humerus on the eighth day of incubation; radius and ulna on 12 days of incubation.

Membranous bones develop independently of the cartilaginous skeleton and never have direct contact with cartilage. These bones develop in the connective tissue. In the embryo, a bony membrane plate is formed by osteoblasts. Bone spicules arise off it in between the connective tissue cells. The outer layer of this becomes periosteum that lays down layers of bone.

BONE GROWTH

Width is increased by deposition of new bone at the periosteum and resorption of old bone at the endosteum.

The length of the bone is by endochondral ossification. Bone growth occurs from an epiphyseal plate at each end of the bone in a similar position to that of the mammalian growth plate. The growing bird has cartilaginous epiphyses unlike mammals that have bony epiphyses.

During longitudinal bone growth, chondrocytes within the epiphyseal plate undergo a series of well defined stages characterised by changes in proliferation rate, shape and size, and synthesis and deposition of extracellular matrix components. Although the avian and mammalian growth plates show distinct structural differences, the cytological sequences are the same, which suggests a similar physiological control mechanism.

In birds there are far more chondrocytes: poultry have approximately double those of rats. Vascularisation is also different. In the rat the growth plate is avascular, the capillary bed terminating as loops overlying the chondrocyte columns. In the bird the epiphyseal blood vessels of the growth plate descend to the transitional zone; the metaphyseal vessels ascend to be very close to the epiphyseal vessels.

Regulation has been shown to be controlled by growth hormone, fibroblast growth factor, and metabolites of vitamin D.

The cartilage grows rapidly and is then eroded and replaced by long trabeculae of bone, which are frequently aligned along lines of stress to form spongy bone. Once the growth has finished the growth plates and the epiphyses calcify. The spongy bone is remodelled to form dense compact in concentric layers. The medullary cavity is formed and in many bones becomes pneumatised.

Huge amounts of work have been carried out on bone development in domestic poultry (Whitehead, 1992) and in embryos of other species (Starck, 1989; Starck and Ricklefs, 1998; Maxwell and Harrison, 2008). Very little has been published in non-domestic species. Most of the descriptions of bone

development in the embryo are based around whole alcohol-fixed specimens that have been cleared with potassium hydroxide (KOH) and stained with Alizarin Red and Alcian Blue to show bone and cartilage respectively. The bird is immersed in increasing strengths of glycerol that strengthens the specimen (softened by KOH). This technique is used routinely by toxicology laboratories looking for foetal deformities. It produces beautiful specimens of embryos, growing and even adult birds but obviously has its limits; one of which is size of the specimen. I have found it difficult to prepare limbs on adult birds bigger than a Kestrel (*Falco tinnunculus*).

A very interesting study was carried out by Carrier and Leon (1990) where they looked at skeletal growth and related it to function in the Californian gull. Gull chicks are precocial and able to run around almost as soon as they hatch. However they cannot fly until they are nearly fully grown. Carrier and Leon (1990) caught and killed 32 birds of known ages (2 days to adult) and examined their bones. Their findings and conclusions were:

- Growing bones are weak but must function adequately;
- Soft growing bone is much thicker than mature bone;
- Post-hatching growth sees strength and stiffness of skeleton increase 6-10 x;
- Gulls walk soon after hatching but only fly when fully grown. This difference is reflected in the skeletal development; and
- The growing leg bones are thick (and therefore strong) for most of the young bird's development whereas the wing bones are comparatively underdeveloped until the very last stages of the birds growth when they develop very quickly.

There are various reasons for looking at ossification sequences (see Maxwell et al., 2010).

GROSS ANATOMY

As Cuvier noted in the 18th century much of an animal's anatomy predicts the way that it lives, eats, breeds and so on.

Embryonic birds start life with pentadactyl limbs. The leg ends up with a single metatarsus and four digits; the wing has a single metacarpus and three digits. There has been huge debate about which bones are lost in this process.

The sternum supports the thoracic girdle, which consists of the scapula, clavicle, and coracoid bones. The coracoid acts as a strut, holding the shoulder a constant distance from the sternum. The scapulae lie adjacent to the ribs and the left and right clavicles are fused into a structure known as the furcula (it is absent in some parrots e.g. lovebirds and rosellas). The furcula acts as a spring and stores energy during the downbeat as it is compressed. These three bones are joined by ligaments at their proximal ends to articulate with the head of the humerus; their jointed articular surfaces form the triosseal foramen through which the tendon of the supracoracoideus muscle passes.

The wing bones are the humerus, radius and ulna, and the manus – a semi-fused three fingered hand. There are separate radial and ulnar carpal bones and the distal carpals and metacarpals are fused into a carpometacarpus. The alula digit (thumb) has a good range of movement; the major and minor digits form an integrated unit. The wing is very mobile when flexed but when extended it tends to move at the shoulder joint and otherwise resists dorsal and ventral forces. The shafts of the flight feathers are closely attached to the dorsal aspect of the ulna and manus; the primary feathers are more firmly attached than the secondary feathers.

The wing has its main muscle mass on the sternum. The pectoral muscle contracts to cause the down stroke of the wing and forms 15 to 20% of the total body weight of the bird. Dorsal (or deep) to the pectoral muscle is the supracoracoideus muscle. Its tendon of insertion runs through the triosseal foramen and inserts on the dorsocranial edge of the humerus. During normal flapping flight the supracoracoideus muscle rotates the humerus causing the wing to raise its leading edge. This allows the bird to maintain its position in the air between down strokes. During slow flight and take off, the shoulder muscles elevate the wing. A triangular portion of skin, the propatagium, is present from the cranial aspect of the shoulder to the carpus and caudally to the elbow. The leading edge of the propatagium is supported internally by an elastic tendon that is joined by various other muscles and tendons, all of which maintain the aerofoil shape of the wing. The elbow joint has a wide range of movements when flexed and the radius and ulna pronate and supinate.

The leg has a femur, a tibiotarsus (the tibia fused to the proximal row of tarsal bones), and a short fibula that does not extend below the fibula crest. The tarsometatarsus is also a short bone formed by the distal row of tarsal bones combined with the fused second, third and fourth metatarsal bones. The first metatarsal bone is separate. The parrot's foot is zygodactyl: digits 2 and 3 face cranially, digits 1 and 4 caudally. In falcons and hawks it is anisodactyl: digit I caudal and II, III, and IV cranial. Owls, ospreys and turacos are semizygodactyl in that they can place digit IV cranial or caudal. The pelvis is formed by fusion of the ilium, ischium, and pubis. The acetabulum is formed from a bony rim and a fibrous cup. Caudodorsal to the acetabulum is the antitrochanter, which articulates with the trochanter of the femur and prevents abduction of the limb when in a normal standing position. Because of their foot shape and short tarsometatarsus, parrots are very good at climbing and manipulating their food but when walking on a flat surface they have a typical waddling/rolling gait especially those, like macaws, that walk on their caudal tarsometatarsus as well as their foot. Like the wing, the main muscle mass of the leg is close to the body, so many muscles have long tendons of insertion. When the toes are flexed and gripping there is a locking mechanism between the flexor tendons and their sheath that maintains grip with a minimum of muscle activity. In parrots the long digital extensor muscle moves all four digits. In falcons the soft tissue in the distal limb are modified to take the strains of hunting. There are a number of ossifications, e.g. in the flexor tendons, that distribute these extreme forces. These are discussed later.

MUSEUM SPECIMENS

Many museums hold avian specimens. Some have more extensive collections than others. The Natural History Museum in the UK has a huge collection because during Victorian times there were many wealthy people who indulged themselves by sending out collectors across the world. Their collections were usually donated or sold to a museum. Nowadays new specimens are difficult to obtain as collectors do not shoot birds, as they did.

Museum specimens are prepared bones, skins, and whole bird fixed in spirit (alcohol). They also have eggs and nests but tend not to have growing birds or embryos. In the UK anyone can have supervised access to the collection at Tring, provided they can prove that they have some scientific purpose. Like most institutions, they want a publication to help justify their existence.

Radiology is used in all branches of clinical veterinary science. Most of the techniques used on dogs and cats are applicable to birds. Whole body radiographs should be used as part of the routine examination of most ill birds.

Any small animal radiography set will be able to produce good avian radiographs. It is unusual to need more than 60 kV. A large mA is useful as it will allow fast exposure times, but perfectly adequate radiographs can be produced with 20 mA and exposure times of up to 0.2 seconds. Exposure times of greater than 0.2 seconds will produce blurred images on the radiograph. Because of this, image-intensifying screens are necessary. Fine definition screens that are used for the extremities in man and are very useful for cats and small dogs as well as birds. Fine definition screens require more mAS than fast screens, but a lot less mAS than non-screen film. Fast screens and a grid can be necessary for large birds such as eagles and vultures as they are more than 10 cms thick. The choice of film and processing chemicals is decided by the type of screen to be used in the cassette. Mammography film used with appropriate screens and cassettes will give extremely good detail, however processing temperature is critical, mAS requirements are increased, and the screens are easily marked with scratches or dust. Digital processing is said to be even better but my personal experience is limited.

Positioning the patient

It is important to consider what information one wants from the radiograph. A bird that only needs to be screened for the presence of metal in its gizzard requires a different technique to the bird that is lame. For accurate diagnosis it is usually important to have two correctly positioned views, the second view taken at 90° from the first. To allow perfect positioning and to keep within the law in the UK some form of restraint is necessary that allows the radiographer to stand away from the patient whilst the X-rays are emitted. It is possible to restrain conscious parrots using various positioning devices and these allow whole body radiography if used sensibly. Most parrots are best restrained using a general anaesthetic (GA) which allows perfect positioning using sand-bags and ties. Raptors legs etc need proper positioning so again GA and ties, plus zinc oxide tape to stick the toes down.

Normal Bones and Soft Tissue

It is really important to know what is normal for the species. The shape of the bones can vary widely, even between closely related species. Without an appreciation of the normal it is very difficult to spot subtle differences in shape and even pathology. There are several books that portray normal radiographs, see end of section.

Pneumatisation varies between genera, e.g. the femur is pneumatised in raptors and not in parrots. All bones contain marrow but this is replaced by air sac as part of maturation. There is no guide to relate pneumatisation to age in parrots or raptors. The humerus of dusky parrots appeared to be pneumatised at 45 days old, i.e. before emerging from the nest chamber (personal observation, 2006). There has been no other confirmation of this – either dissection or histology.

In many birds there are intratendinous ossifications. Of the birds I see, parrots and hawks have none, falcons have some, and owls have lots. They are found in various families of birds from woodcreepers

to grouse. Intratendinous ossifications are considered a way of strengthening a tendon similar to a sesamoid bone such as the patella. They have been used for taxonomic relationships e.g. woodcreepers (Bledsoe et al., 1993; Harcourt-Brown, 2001). There is a good review by Vanden Berge and Storer (1995).

Most of the information is about intratendinous ossifications in the leg but these are found in the wing as well as the leg. This has confused me. In owls there are large numbers of tendons in the leg and wing. It makes sense that for wrestling prey the legs have to be strong but owls do not seem to be really strong fliers so maybe the intratendinous ossifications are in the wing because of gene expression; that is, if you have them in the leg they have to be in the wing.

Growing Bones

Although bone growth occurs at a similar position to that of the mammalian growth plate, the growing bird has cartilaginous epiphyses. No growth plates are visible but the distal tibiotarsus and proximal tarsometatarsus look as if they have due to the presence of the tarsal bones. The same is seen in the carpometacarpus. Metatarsals II, III and IV and the two metacarpals can be seen as separate entities in the first third of growth.

The radiographic anatomy of growing parrots has been described using the Dusky Parrot *Pionus fuscus* as a model (Harcourt-Brown 2004). These birds weigh 220grms and the very young birds are difficult to image. They do have the advantage of laying 4 or 5 eggs and they do this every other day so a single clutch can potentially be examined radiographically every 10 days to give an almost entire sequence of bone growth.

On the day of hatching very little skeletal development was visible on radiographs: only some parts of the skull, ribs, femur, and tibiotarsus could be detected.

By day 16 most of the bones were visible, except the tarsal and carpal bones.

By day 31 the distal tibiotarsus, tarsometatarsus, and synsacrum were fully developed.

By day 39 the tibiotarsus and femur were fully formed, by day 43 the radius was fully formed, and by day 45 the ulnar carpal bone, humerus and pectoral girdle were fully formed.

The birds left the nest at 56 days by which time the bones were fully grown and the feathers nearly fully grown.

Examination of many joints, such as the elbow or knee showed no radiodensity; this was normal. The carpal and tarsal bones arose as separate ossification centres that eventually fused to an adjacent bone. Whilst growing, these zones of proliferation gave the appearance of a typical mammalian growth plate.

The diaphysis of each growing bone was considerably narrower than the growing ends (metaphyses), especially the tibiotarsus. This narrow portion of bone had an apparently well-developed cortex. During the mid part of the growth period it was very difficult to measure the length of the cortical bone in the tibiotarsus as the beginning and end of the cortex was indistinct.

The synsacrum and notarium grew differently from the long bones and could be seen to arise as a

series of vertebrae that enlarged and finally fused. Once the bones were fused the synsacrum seemed to cease growing.

In this study, the humerus appeared pneumatized when fully formed at 45 days, as its medullary cavity was less radiodense than that of the adjacent radius and ulna. Radiographic examination of other *Pionus* spp. was similar. Raptors have similar events in their bones but no-one has described the sequence of events in a similar manner to the Dusky Parrot.

Nutritional Osteodystrophy (Metabolic Bone Disease)

This is a common condition in captive animals. Many museums will not accept bones from zoo animals or long term captives as they find that the bones are abnormal. There is argument as to how much of this is behavioural (and in poultry, genetic) and how much is nutritional but there is no doubt that many captive animals are fed a calcium deficient diet as well as having little access to UVB radiation so they are deprived of vitamin D₃. The cause of juvenile osteodystrophy in non-domestic birds is thought to be a calcium and phosphorus imbalance in the diet, and/or a deficiency of vitamin D (Fowler, 1978). A lack of minerals and vitamin D (or a deficiency in ultraviolet light) are recognised as causes of metabolic bone disease in domestic poultry. However, many other ancillary factors, such as rate of growth, have been implicated and these have been reviewed in Whitehead (1992). It has been recognised in poultry that genetics plays a part (Kestin et al., 1999) as does the amount of exercise (Classen and Riddell, 1989). In these birds the resulting skeletal deformity is not an osteodystrophy as it may be caused in otherwise normal bones by physical means. Breeding abnormally huge birds causes abnormal strains on their legs. Poor substrate will also play a part.

Bony deformity that arises from dietary deficiency whilst the bones are forming will be present for the rest of the bird's life. In a study (Harcourt-Brown, 2003) where hand-reared grey parrots (*Psittacus erithacus*) were examined radiographically for signs of juvenile osteodystrophy it was found that the incidence of bony deformity varied between bones: furcula 0%, keel 25%, ribs 41%, humerus 8%, radius 33%, ulna 33%, pelvis/synsacrum 25%, femur 25%, and tibiotarsus 100%. All the grey parrots affected by osteodystrophy to any degree had deformity of the tibiotarsus whereas no bird had a deformed furcula. Although the cause was probably nutritional deficiency, the distribution of lesions could be modified by abnormal activity. Of these 28 grey parrots, none of whom were presented for lameness or related conditions, 43% had signs of bony deformity.

Diagnosis with radiography is based on longitudinal or rotational deformity in the long bones and general deformity in the other bones. It is important that there are normal skeletons in similar views to make the comparison. In young growing birds the first sign of nutritional osteodystrophy is bone deformity. Bones tend to bend and the first change observed are trabeculae in unusual places within the bone. Next the bones bend. In birds that have some calcium and bony strength the deformity seems to occur between the formed cortices and the growth plate. Some birds get a folding fracture that allows the periosteum to elevate like a bow string on a bow. It then lays down a new (secondary) cortex. Growing birds that have very little calcium in their diet and also probably had calcium/vitamin D deficient parents show loss of density and folding fractures along the entire length of the bone. Although breeders wish to blame genetics the cause is always dietary deficiency or occasionally excess. It may be genetic in poultry but with modern breeds it is very likely that the members of a flock are more like clones when compared the genetic variation between recently wild birds, even if they are siblings.

Osteoporosis

In adult birds advanced osteoporosis causes loss of bone density, gross irregularity of the cortices of long bones, loss of the central part of the cortex giving a double cortical line and multiple fractures. Using radiography, early or mild osteoporosis is difficult to diagnose with certainty. It is more reliable to use a dexta scanner, and there are models that are used in man for scanning small extremities, such as fingers and these can be adapted for use in birds. Fusi (1953) found that in man up to 60% of bone mineral detected on dexta scans could be lost from vertebrae before it could be detected on radiography. Finsen and Anda (1988) found that 30% bone density loss was required in their study of the peripheral skeleton. Experienced radiologists were able to detect the changes earlier and with greater certainty than less experienced colleagues. The detection of osteoporosis in dogs was similar in that significant loss was required for it to be obvious on a radiograph. It is likely that this is true for birds. Comparison with the surrounding soft tissue and great confidence in both the radiographic technique and the processing are required to make diagnosis of osteoporosis. It can be useful to have a normal bone that can be placed on each cassette next to the bird that is then used as a standard reference.

Birds that have obvious bone loss need emergency treatment - oral administration of high levels of calcium and an initial oral dose of vitamin D₃. After this, vitamin D should be supplied by using UV B light, preferably sunshine. It is possible to overdose vitamin D if given orally as it is absorbed from the gut on a diffusion gradient whereas in the skin, once the body is fully supplied the vitamin D; precursors are converted into an inert compound so toxicity does not occur. It can be dangerous to inject multi-vitamin preparations. However a general vitamin and mineral supplement is required to cover all the other deficiencies that will be accompanying this malnourished bird. Calcium in the drinking water is not satisfactory.

Infection

The appearance of osteomyelitis in birds differs to that in mammals because of the caseous nature of avian pus. Abscesses that form in the medullary cavity slowly enlarge. The bone is distorted around the abscess and may either form a bubble-like appearance or the cortex may disappear.

Some birds are presented with multiple osteomyelitis lesions. Sometimes they seem to have bilateral symmetry. Many of these cases have avian tuberculosis.

Septic arthritis is different to osteomyelitis as the joint fluid keeps the pus more liquid. The surfaces of the joint are eroded and the joint space expands. The edge of the lesion is not clear cut and has a more roughened outline.

A bony sequestrum is often surrounded by a lytic area (like a halo) where the bone has been removed by the infectious process. The sequestrum is often more radiodense. In some cases the cortical bone is 'pushed' into a bulge around the sequestrum by the necrotic purulent material – another indication of the effects of pressure on osteoblasts and osteoclasts.

Bone Tumours

These are uncommon and cause osteolysis, increased soft tissue density and often a radiating periostitis. These changes do not cross a joint.

Bone healing and Non-healing

In skeletally normal birds, fracture and subsequent callus formation is similar to mammals. Malunion of fractures is usually atrophic and shows as rounding of the ends of the fracture with no periosteal proliferation.

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