Obtaining radiographs for interpretation of the musculoskeletal system in the small animal patient is difficult due to necessity for appropriate radiographic positioning and technique. Radiographic interpretation is confounded by the numerous soft tissues and bone structures involved. Rapid assessment of the bone structure is routinely performed using radiographs; however, often the subtlety of disease can be confusing. The purpose of this lecture is to cover the identification of common developmental and acquired musculoskeletal diseases of the small animal patient.

When evaluating the skeletal system in a patient, the first thing to determine is if the lesion is aggressive or non-aggressive. Non-aggressive lesions include both developmental and acquired etiologies. Examples of non-aggressive lesions include osteochondrosis/osteochondritis dessicans, panosteitis, bone cysts, fracture healing (callulus/malunion), ununited centers of ossification, medial coronoid disease, panostitis, osteomas, osteoarthritis or metabolic disorders. Aggressive lesions are due to neoplasia or osteomyelitis.

When determining whether a lesion is aggressive or non-aggressive, there are multiple radiographic signs to take into account: pattern of bone lysis, cortical lysis, periosteal reaction, zone of transition and rate of progression. Bone lysis has three different appearances: geographic (focal), moth-eaten and permeative. Geographic lysis is easily defined and well-margined. A classic example of geographic lysis is a bone cyst. Geographic lysis is typically associated with non-aggressive lesions. Moth-eaten and permeative lysis is typically associated with aggressive disease. Moth-eaten lysis is seen as numerous small pinpoint lucent areas of lucency. Permeative lysis consists of larger regions of lysis but unlike geographic lysis, permeative lysis usually is irregularly marginated and/or has indistinct margins. It is important to note that detecting lysis can be difficult on radiographs, as it requires approximately 50% of the bone per unit area to be destroyed before the change is apparent. This makes subtle lysis hard to detect. The more lysis that is present, the easier it is to see on radiographs. Therefore, by the time lysis is seen on a radiograph, the lesion is quite severe.

Cortical lysis, as opposed to overall bone lysis, can be seen in aggressive bone lesions. If the cortex is thin, but no lysis is present, then it is more likely that the lesion is non-aggressive. If overall bone lysis and cortical lysis is seen, the lesion is likely aggressive.

Periosteal reaction can either be smooth (continuous) or interrupted (lamellar, palisading, irregular). Smooth periosteal reaction should be continuous from its proximal to distal aspect and can be easily traced from proximal to distal aspects without interruption. Smooth periosteal reactions are generally associated with non-aggressive lesions, specifically trauma. Interrupted periosteal reactions are due to an aggressive process.

Zone of transition refers to whether the lesion can be clearly demarcated from normal bone (short) or if the termination points are indistinct (long). Short zones of transition are often associated with non-aggressive lesions. Long zones of transition are more likely to be aggressive.

In addition, Rate of progression is probably the most overlooked method to assess an aggressive lesion. By the time a questionable aggressive lesion is seen on a radiograph, the lysis
is quite substantial. Therefore, the rate of progression in 2-4 weeks will also be dramatic. If a question exists between an aggressive and non-aggressive lesions, supportive medical management for 2-4 weeks then repeat radiographs to look for progression can aid in determining if the lesion is aggressive.

After evaluating for aggressive or non-aggressive change. The next step in interpretation is based on lesion location and number of bone involved. If the lesion is generalized, in that it affects all bones equally, then the primary differential diagnosis is a metabolic or nutritional abnormality. If only one bone is involved, this is a focal or monostotic lesion, primary osseous neoplasia often fits into this category. If multiple bones in the same region (locally extensive), different bones that are not in close proximity or multiple areas in the same bone are involved, this generally indicates a hematogenous spread or systemic disease such as bacterial osteomyelitis, metastatic neoplasia, panosteitis or hypertrophic osteodystrophy. Developmental lesions such as osteochondrosis, elbow and hip dysplasia, often affect the contralateral limb and thus are polyostotic.

Anatomic location is also a key into the differential diagnoses. If the lesion is epiphyseal or physeal in origin, then it is likely secondary to infection, trauma or potentially a nutritional abnormality. These lesions are generally in juvenile dogs and cats. If the lesion is in the metaphyseal region, then a primary bone tumor or hematogenous infection is most likely due to the proximity of the nutrient foramen. If the lesion is diaphyseal, then the lesion is likely metastatic neoplasia, a soft tissue mass with secondary bone involvement or a focal infection related to a penetrating trauma.

With interpretation paradigms in mind, it is important to be familiar with the radiographic findings seen in common musculoskeletal diseases. For the purpose of this discussion, the lesions covered are categorized as developmental/juvenile lesions and acquired osseous lesions.

The most commonly seen developmental/juvenile diseases include osteochondrosis/osteochondritis desicans, elbow dysplasia, hip dysplasia, panosteitis and hypertrophic osteodystrophy. Osteochondrosis (OC) occurs secondary to failure of endosteal ossification. If a cartilage fragment or flap is present, the lesion is then termed osteochondritis dessicans (OCD). Endochondral ossification is not complete until 5-6 months in the dog, therefore this diagnosis should only be made after this age. OC can occur in any bone that undergoes endosteal ossification, however commonly affected sites in the canine patient include: caudal humeral head, (medial) humeral condyle, (lateral) condyle of the femur and talar ridges (medial in most breeds, lateral in Rottweilers). Regardless of location OC lesions are seen radiographically as a flattening of the articular margin at the site of the lesion. Often these lesions are surrounded by an opaque rim (sclerosis) and secondary degenerative joint changes. For the talar OC, a flexed dorsoplantar projection of the tarsus and a flexed lateral can help to remove the superimposition of the distal tibia to identify the lesion.

Elbow dysplasia is a blanket term for multiple disease processes. Although there is some discrepancy in sources on exactly what lesions are classified as elbow dysplasia, for the purpose of this lecture, elbow dysplasia includes: medial coronoid disease of the ulna, ununitedanconeal process, humeral condyle OC and flexor enthesopathy (ununited medial humeral epicondyle). These disease processes are often bilateral and multiple lesions may be seen. Medial coronoid disease is often difficult to appreciate radiographically due to the superimposition of the radius. In many cases, medial coronoid disease is suspected when degenerative elbow disease is present and there is no evidence of other lesions associated with elbow dysplasia present. A blunted medial coronoid process can be sometimes seen radiographically, however the absence of
coronoid blunting does not exclude medial coronoid disease. Computed tomography (CT) is the gold standard for medial coronoid disease. It is not uncommon on CT to only see a small fissure or change in density (lucent or sclerotic) of the medial coronoid process. These subtle changes are not visible radiographically. The anconeal process should fuse to the ulna by 5 months. An ununited anconeal process should be diagnosed if the process is not fused to the ulna after 5 months of age. Flexor enthesopathy is oftencategorized with elbow dysplasia. Radiographically, this is apparent as mineral fragment(s) continuous with or immediately adjacent to the origin of the flexor tendon (medial epicondyle of the humerus). The fragments can become quite large and often are curvilinear in shape. Soft tissue swelling may also be present. Recent literature supports that flexor enthesopathy can exist as a sole etiology or concomitantly with other causes of elbow dysplasia.

Hip dysplasia is an orthopedic disorder that causes widespread confusion among breeders mainly due to the difficulty of predicting the likelihood of young animals developing osteoarthritis later in life due to hip laxity. Radiographically laxity can be assessed in juveniele dogs by distraction radiography, most commonly PennHip is used. With PennHip, greater than 50% is considered a pass and hips with a distraction index of < 0.3 are considered unlikely to develop osteoarthritis later in life. Additional radiographic findings in a juvenile with hip dysplasia includes coxofemoral joint widening or subluxation. As the animal ages, degenerative changes and alterations to the coxofemoral joint, such as thickening of the femoral head and neck, shallow acetabular cup and/or subluxation/luxation of the coxofemoral joint are often used to make the diagnosis of hip dysplasia.

Panosteitis is a self-limiting disorder that is hard to diagnose because of the subtlety of the radiographic changes. It ranges from increased opacity of the medullary cavity to a decreased opacity or smooth periosteal proliferation. The ulna, radius and distal humerus are the most commonly affected areas. The lesion is often polyosteotic and the contralateral limb should be obtained for comparison. Generally, lateral radiographs are all that is needed to make the diagnosis.

Hypertrophic osteodystrophy (HOD) affects the metaphyseal region of long bones, although the periosteal reaction appears aggressive, this lesion is purely proliferative and no lysis is present. HOD is only seen in young dogs and is typically polyostitic. The adjacent soft tissues are often swollen. The appearance of a double physeal line is often seen. Animals with this condition often show systemic signs of illness such as a fever and/or an inflammatory leukogram. This condition is self-limiting and typically resolves when growing ceases. In some cases, this disease is seen concurrently with cranio mandibular osteopathy (CMO). CMO is a recessive gene in west highland white terriers and tends to only affect the skull in this breed. CMO is characterized radiographically by irregular new bone formation in along the mandible, tympanic bullae and petrous temporal bone, it is often bilateral but can be assymmetric. CMO can be seen concurrently with HOD. Like HOD, CMO is self-limiting.

Other less common developmental diseases include multiple cartilaginous exostosis, retained cartilaginous core and calvarial hyperostosis of mastiffs. Multiple cartilaginous core is a benign proliferative disease and any bone that has enchondral ossification can be affected. The growth of these masses usually ceases when animal reaches skeletal maturity. On radiographs the osseous mass are often located along the ribs, long bones and vertebrae; the rib masses are amorphous in appearance, radiolucent and opaque with irregular contours. The long bone and vertebral body mass are usually more organized with radiolucent cartilage and trabecular bone. Retained cartilaginous core appears
radiographically as a cone shaped radiolucent area surrounded by a narrow zone of sclerosis. Retained cartilaginous cores can occur along any physis, however are most common in the distal ulnar physis. Their presence may result in an angular limb deformity due to retardation of ulnar growth. Calvarial hyperostosis is smooth thickening of the bones of the calvarial and is seen in young bullmastiffs. In one case report, concurrent HOD was reported.

Numerous additional congenital osteochondrodysplasias have been described for various breeds including Alaskan malamutes, Labrador retrievers, Scottish fold cats, Norwegian elkhounds and beagles.

Nutritional diseases such as nutritional hyperparathyroidism, rickets, and hypervitaminosis A, are not commonly seen since most small animal patients receive a balanced diet. Metabolic diseases such as renal secondary hyperparathyroidism are occasionally seen in patients and associated with severe renal disease. Hyperparathyroidism manifests as severe osteopenia; the bones are often only minimally more opaque than the surrounding soft tissues on radiographs. This is most apparent in the spine and along the mandibular and maxillary bones where the teeth remain the strongly mineral opaque against a faint mineral opaque bone. In severe cases, fibrous osteodystrophy of the mandible may be present with the classic “rubber jaw” finding on physical exam. Congenital hypoparathyroidism is rare but has been described in numerous breeds of dogs (i.e. Boxers, Scottish deerhounds, Giant schnauzers, Affenpinchers, Great Danes) and cats. Congenital hypothyroidism is characterized radiographically by epiphyseal dysplasia, delayed cuboidal bone ossification, short vertebral bodies, short and broad skull. These animals are often dwarf-like in appearance. Although rare, appearing somewhat similar to congenital hypoparathyroidism is mucopolysaccharidosis, a group of lysosomal storage diseases. Radiographically these cases are characterized by generalized epiphyseal dysplasia with the ossified regions of the epiphyses being smaller than normal and non-uniform with a granular appearance, cuboid and short vertebral bodies, short and flat maxilla, small or absent frontal sinuses, severe progressive degenerative joint disease +/- coxofemoral subluxation or luxation.

As mentioned above, acquired disease can be classified as aggressive or non-aggressive. Non-aggressive acquired lesions are typically associated with trauma. Aggressive lesions are either neoplastic or secondary to osteomyelitis. This distinction cannot be definitively determined without biopsy, however clinical judgement can often be implemented to prioritize these two differentials. For instance, a 2 year old hunting dog with an aggressive bone lesion in the proximal metaphysis of the humerus is more likely to have a fungal infection; however an 8 year old Rottweiler with the exact same radiographic findings is more likely to have osteosarcoma. Although osteosarcoma is the most common appendicular skeletal neoplasia other differentials for primary osseous neoplasia include chondrosarcoma or fibrosarcoma. Histiocytic sarcoma and hemangiosarcoma can also present as a solitary osseous neoplasm.

Fractures are acquired diseases that can fall into either aggressive or non-aggressive depending on their underlying etiology. Fractures are classified by their location, orientation, open vs. closed and displacement. Fracture orientation describes the actual fracture line +/- fragments and includes incomplete, transverse, oblique, spiral, segmental and comminuted. If the fracture itself or the surrounding soft tissues have gas within them, this fracture is considered open, if not it is closed. Fracture displacement is described in relationship to how the distal fracture is displaced. The greater the degree of displacement, the more blood supply compromise. Fracture healing following appropriate apposition of the fragments is patient
dependent with young healthy dogs healing quicker than older dogs with concurrent diseases. Stages of healing are roughly as follows at 5-10 days lose sharp margins with a slight increased fracture gap, 10-20 days there is minimal callus and decreased opacity of fragments, 30 days the fracture gap disappearing, more callus formed and at 3 months remodeled callus and re-establishment of the medullary cavity. Not all fractures heal as planned however and abnormalities do occur. If the fracture implant is infected, radiographic signs usually occur around 14 days and progress with time, these include periosteal proliferation, increased lucency around implants, soft tissue swelling +/- draining tract. If anatomic alignment is not achieved, a malunion may occur. In some cases, delayed or non-unions are also possible.

Acquired arthropathies are evaluated the similar to bone lesions, however the terminology is slightly different, erosive is aggressive and non-erosive is non-aggressive. When evaluating an arthropathy, the step based approach begins with whether the joint has characteristics of erosive or a non-erosive lesion. Followed by, is it a mono-arthrosis or poly-arthrosis? Then, what joint(s)? Remembering to evaluate the radiographic findings with the signalment and clinical picture. Characteristics of an erosive joint include subchondral bone lysis and proliferation, narrowing of the joint space, bone fragmentation and joint subluxation/luxation. In non-erosive arthropathies, the subchondral bone may be normal or have osteophytosis, well circumscribed geographic lucencies, sclerosis of the subchondral bone and increased synovial mass. Differentials for erosive arthropathies include septic or mycotic arthritis, neoplasia (synovial cell sarcoma, histocytic sarcoma, hemangiosarcoma), immune-mediated (rheumatoid), chronic hemarthrosis and osteochondrosis. Non-erosive differentials include degenerative joint disease and immune-mediated arthropathies (systemic lupus erythematosus, rickettsial arthropathies, idiopathic). Monoarthropathies are often degenerative, traumatic or a result of direct inoculation. Polyarthropathies are more likely to be hematogenous or immune mediated.

Radiographic findings of bone lesions can be confusing if one does not consider the vast number of differential diagnoses possible and then makes an educated decision to prioritize the lesion. At the end of this lecture the goal is to provide the practitioner with numerous examples and a better appreciation of how to evaluate a radiograph for musculoskeletal lesions of the appendicular and axial skeleton.