1 The role of nutrition in the pathogenesis of orthopedic problems

At the time of weaning, the human being takes over the responsibility for pet nutrition. A couple of weeks later, the puppy is handed over to the new owners, which may be less experienced than the breeder. This period is also the time of intensive turnover and growth. It is therefore obvious, that feeding imbalances in quantity and quality would have major impact on skeletal health in growing dogs.

Table 1: Daily weight gain in dogs of different body end weight in their first 12 months of age. The most intensive time is between 2 and 4 months of age.

The most deleterious effect comes from overweight by too high caloric intake. Increased muscular forces exert mechanical stress to the skeletal and joint tissues. A mismatch between body and bone growth is detected in ad libitum feeding of growing dogs. At last, Insulin-like growth factor-I and thyroid hormones are released. Both reflect a process, in which increased cartilage layers are broken down and resorbed. It can be concluded, that at least a part of the pathogenesis of elbow dysplasia, hip dysplasia, osteochondrosis and some angular deformity can be explained by overweight at a time between 3 and 8 months of age.

Calcium over supplementation has been a problem in the past. Breeders have recommended an addition of extra portions of “powder” to the normal diet in order to strengthen the skeleton. Furthermore, some puppy diets were not well balanced. These shortcomings have been corrected. Diets for fast growing large and giant breeds are supplemented by 0.8 to 1.0% of calcium in the dry substance. Calcium should be calculated in relation to the energy density of diet in order to avoid over supplementation. Calcium uptake in the intestines is well regulated from the 4 month of age by vitamin D. High intake at an earlier age would potentially lead to increased turnover of cartilage in the physeal region, leading to thickened hyaline cartilage.
layers. As it is nourished by synovial fluid, deeper layers may become necrotic, which is known as osteochondrosis. High calcium intake furthermore has experimentally led to Wobbler’s syndrome and angular deformity.

High phosphorus and low calcium diets, mainly as result of home made diets with meat and slaughtered offal, are rare in dogs, but can be seen occasionally in cats. They lead to secondary nutritional hyperparathyroidism, which is manifested with pathologic bone fractures.

The protein amount of the diet however does not seem to have an influence onto skeletal development. A dry amount of about 25 % is well tolerated. Low protein intake may disturb the development of the immune system.

2 Normal development and activity

As a rule of thumb, the puppy weight of large and giant breeds should not exceed one third of the adult weight of the sire or dam at the age of 4 months, 60 % after 6 months and 80 % after 12 months. Pet owners must be told, that the body size is genetically determined and will be reached the slower the healthier. Growth curves in middle sized dogs and toy breed dogs are different and end at the age of 12 months. Growth control is performed by weekly determination of body weight. The food amount is adjusted to be within the reference. Commercial diet is mandatory in the period of growth, whereas adult dog feeding allows the addition of home made ingredients in small amounts. Puppies should be fed three times a day until the age of 6 months because of the limited size of the stomach.

Table 2: Body weight development (in percentage of end weight) of different sized dogs. Note, that giant breed dogs should only have 80 % of total weight after 12 months
Activity levels of growing dogs are recommended as follows:

Dogs until 4 months of age:
- short walks of maximal 15 minutes length (1 minute walk length per week of age) at the leash
- playing with dogs of similar size, maximum 15 minutes
- no stairs downwards, but stairs upwards with leash

Dogs from 4 to 8 months of age:
- walks to 30 minutes length, several times a day
- if the dog gets tired (walks behind the owner), the walk should be stopped
- playing with similar sized dogs, intervention if the play is too intensive
- stairs on the leash
- start with swimming activity

Dogs after 8 months of age
- slow modulation of the activity level to normal references for the particular breed
- jogging and biking only with adult dogs
- sport activity (agility !) only with adult dogs

3 Common orthopedic problems in growing dogs

3.1 Elbow dysplasia

The etiopathogenesis of elbow dysplasia (ED) is controversial. Some confusion certainly exists, because the four diseases fragmented medial coronoid process (FMCP), osteochondrosis of the medial humeral condyle (OC), ununited anconeal process (UAP) and incongruity of the elbow joint (INC) have been summarized under the term ED. A genetic etiology has been established with a heritability index that varies from 0.27 to 0.77. The reminder is due to diet and management (see above).

The first philosophies on pathogenesis were deducted from the osteochondrosis complex. Later, asynchronous growth of radius and ulna was identified as the reason for pathologic load in the elbow joint. Current concepts suggest a trochlaer notch dysplasia resulting in incongruity characterized by an ulnear notch slightly elliptical in shape with an arc of curvature too small to fully encompass the humeral trochlea. At least the FMCP and the UAP can be explained. Osteochondrosis is either the result of malnutrition, malconformation (outward rotation of the elbow joint) or maluse of the dog at an early age.

Diagnosis of ED is not straight forward, because radiographic interpretation offers many difficulties and is not conclusive in all cases. Arthrosocopy may be a better tool. Current therapeutical options include removal or reattachment of the anconeal process (UAP), curettage of the osteochondritic lesion (OCD), coronoidectomy, sliding osteotomy of the humerus, corrective osteotomies of the ulna and bicipital tendon release (FMCP). These items wil be covered later.
3.2 Hip dysplasia

Hip dysplasia (HD) has undergone strict breeding control mechanisms in the last decades. Nevertheless, HD is still diagnosed often, which may be attributed to fast growing puppies or the tendency for breedings dogs very large.

The author’s experience with HD shows, that holding a dog lean in the first 12 months together with restricted motion can cover vast parts of a genetical predisposition. Strong musculature, as seen in English bulldogs, compensate even for severely osteoarthritic hips.

Early interventions with triple pelvic osteotomies are successful, when performed under 10 months of age, with no signs of osteoarthritis and an acceptable acetabular coverage of the femoral head. Juvenile pubic symphysiodesis, carried out between 12 and 20 weeks of age, is a risky procedure, because HD can never be diagnosed that early, the benefits of the surgery are reduced with late timing and no proof of the correction is visible on radiographs made for breeding permission. Total hip replacement with non cemented systems are recommended from 10 months of age with good results.

Conservative management and postoperative plans include weight control, locomotion control, physiotherapy, new generation analgetics and chondroprotectives.
3.3 Patellar luxation

Measurements on the skeleton of small breed dogs have not identified risk factors for patellar luxation (PL). The only predictor was body weight: the smaller the dog, the more risk for PL. Diagnosing PL is still a clinical procedure based on the US standard with four degrees. Switzerland and other central Europe countries have tightened the diagnostic standard through examinations in different physiological positions and notifying the worst result.

PL is seen from the age of 4 months. The gait is very typical. Medial luxations are mostly seen in small dogs, whereas lateral luxations are far less common and more seen in large dogs. Chihuahuas may have lateral and medial luxations on the same stifles. Appenzeller dogs are prone to medial luxations.

Surgical therapy is successful, if the alignment can be corrected rigidly. The classical surgery includes a femoral sulcoplasty and a transposition of the tibial tuberosity. Soft tissue reconstructions alone (muscle releases) are indicated only in dogs less than 3 months. Any other non osseous reconstruction will lead to relapse because of the strong forces exerted by the quadriceps mechanism. A novel technique is the replacement of the femoral sulcus with a titanium patellar groove. In this procedure, the tibial attachment remains untouched.

Prognosis after PL surgery is generally excellent. Osteoarthritis hardly progresses. Implants of the tension band wire must be removed especially in small dogs.
Fig. 5: Grading patellar luxation by assessing the spontaneous patellar position and the movement, by which a luxated patella is brought back into the femoral sulcus.

3.4 Osteochondrosis

Osteochondrosis is a generalized problem. The medial condyle of the distal humerus (see chapter on ED) and the caudocentral part of the proximal humerus are predisposed areas, followed by the lateral condyle of the distal femur, the talus and the lumbosacral joint. Pathophysiological explanations are based onto malnutrition, mainly with excessive calcium, and too early and too high loads on the joints. Border collies are diagnosed more often with osteochondrosis of the shoulder joint.

Standard therapy of osteochondrosis is removal of the cartilage flap together with curettage of the hyaline cartilage in order to allow the subchondral bone to replace and grow into the defect. Prognosis is quite well in the shoulder and elbow joint, whereas osteochondrosis of the tarsal joint often leads to severe osteoarthritis.

Fig. 6 Histologic section through a hyaline cartilage demonstrating the creation of an osteochondritic flap

Fig. 7: Caudocentral lesion in the proximal humerus, a typical location for osteochondrosis
3.5 Panosteitis

Etiology of panosteitis (or enostosis) remains undetected. Large breeds, rapid growth and extra calcium alimentation may play a role. Through an unknown mechanism, endosteal pressure is increased, which leads to pain and lameness. Fat cells die and are replaced by a thin grid of lamellar bone, which is visible a couple of weeks after the onset of lameness.

Most dogs are between 5 and 9 months of age. Some giant breed dogs have been diagnosed with panosteitis at the age of 3 years. The affected limb changes in periods of 6 weeks. The disease is self limiting. Pain medications and diet restriction are recommended in cases with severe lameness.

Panosteitis is a major differential diagnosis in ED and should be ruled out with radiographs.

3.6 Hypertrophic osteodystrophy

Hypertrophic osteodystrophy (HOD) is a disease of the metaphyseal bone. Some researchers believe, it may be part of the osteochondrosis complex, whereas others claim to have found infectious agents such as E. coli or RNA of distemper viruses. Independent of the etiology, HOD normally takes a serious course with painful swelling of the distal radius und ulna, fever and anorexia. Fatal outcome is reported and experienced by the author.

Initial palliative and supportive measures are taken to save life (infusions, analgetics, antibiotics, forced feeding). A possible destruction of the distal ulnar and radial growth plate is monitored over weeks with radiographs. Because of the lack of a proximal ulnar growth plate between radiocarpal and elbow joint, a bowing, outward rotation and valgus deviation of the radius can be noticed (radius curvus syndrome). Early ulnar osteotomies relieve tension and allow self alignment of the radius and the elbow joint. Severe angular deformities need radial und ulnar corrective osteotomies and fixation with plates or external skeletal fixators.
3.7 Other diseases of growing dogs

Other diseases of growing dogs, which are not discussed here, are: Calvé Legg Perthes disease, retined cartilage cones and islands, Wobbler’s syndrome, secondary nutritive hyperparathyroidism, hypervitaminosis A.

Selected references


