Canine Osteoarthritis

Following the talks given in March and April on osteoarthritis in dogs here is a summary of the main points discussed.

Osteoarthritis (OA)

1. OA affects synovial joints. These are where two (or more) bones meet and articulate with each other. The main anatomical features of synovial joints are:
   i. Bone
   ii. Cartilage – to line the bone ends; articular cartilage is very smooth and has a firm but spongy texture to resist damage when weight-bearing
   iii. Synovial fluid – the liquid within joints that bathes and nourishes the cartilage
   iv. Synovial membrane – encloses the joint and produces the synovial fluid
   v. Joint capsule – a strong fibrous capsule with a mixture of collagen (strength) and elastin (flexible) fibres to hold the joint together
   vi. Ligaments – attach bone to bone to ensure that movement of the joint is maintained within an appropriate range
   vii. Blood supply – to supply nutrition and remove waste products
   viii. Nerves – for sensation

2. In dogs OA usually develops following injury to a joint. This injury may be the result of trauma, but most frequently is caused by a joint that has grown and developed inappropriately, so that instead of an even distribution of weight over the joint surfaces, forces acting through the joint are focused onto small discrete areas. This leads to cartilage damage which initiates the vicious cycle of OA.

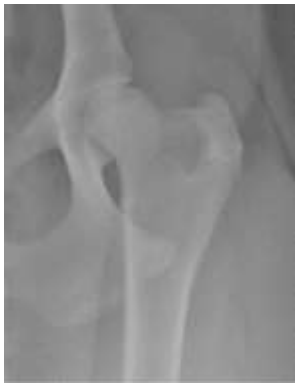
Figure 1 

Figure 2 

Hip dysplasia: Figure 1 shows a hip joint with the leg outstretched – it appears that there is good conformation of the joint with the curve of the ball apparently matching the curve of the socket. However, as can be seen from figure 2, when the leg is moved into a weight-bearing position the ball slips out of the socket so that the dog's weight is resting on the rim of the joint, rather than being distributed evenly across it.

3. This is further exacerbated by obesity and inappropriate exercise:
   i. Obesity – the forces acting through a joint are considerably more than the dog's bodyweight (think about the force that is passed through a stiletto heel!), so a small amount of extra weight can have a very detrimental effect on the joint
   ii. Exercise – young pups have very "elastic" joints, so boisterous exercise can severely affect misshapen joints as the bone ends pull apart and then collide within the range of elasticity

4. The first stage of OA is damage to the cartilage. There are two inherent problems:
   i. Cartilage itself is devoid of sensation, so the body is not aware of the damage until it is severe enough to lead to inflammatory changes in the wider tissues of the joint
   ii. Cartilage has a limited capacity to repair itself and any repair is slow. With all but minor damage the repair is through scar-tissue cartilage rather than specialised articular cartilage

   The result is that very often by the time OA is recognised as being a clinical problem, the cartilage is damaged beyond repair.

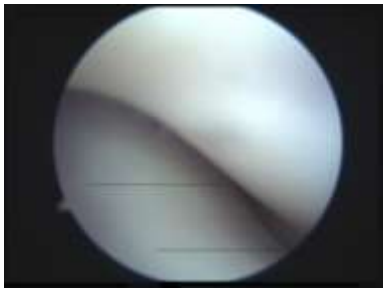
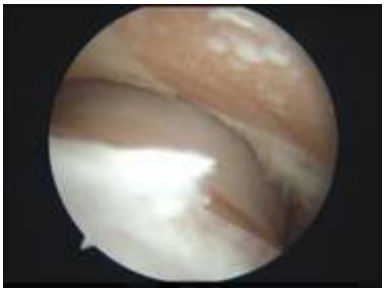
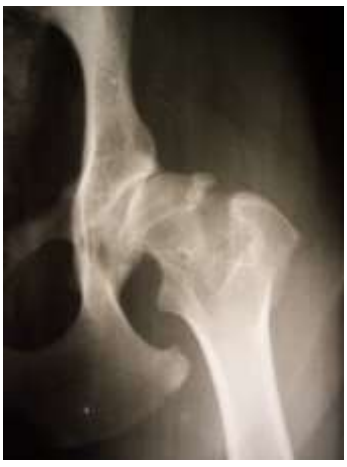
Figure 3  Figure 4 

Figure 3 shows normal cartilage within an elbow joint; figure 4 shows damaged cartilage (and pink exposed bone) in the elbow of a 6 month old Labrador

5. Cartilage damage sets up a vicious cycle of inflammatory changes within the joint, which affect all the tissues described above:
   i. Bone – bone beneath the joint surfaces becomes thickened (sclerosis) and new bone is formed at the perimeter of the joint within the capsule (periosteal new bone)
   ii. Synovial fluid – becomes a soup of inflammatory chemicals which are toxic to cartilage and stimulate nerve endings around the joint
   iii. Synovial membrane – becomes inflamed and thickened
   iv. Joint capsule – becomes thick and leathery, which reduces the degree to which a joint can bend comfortably
   v. Blood supply – becomes congested, leading to stasis
   vi. Nerves – nerve endings that previously conveyed messages such as pressure now send pain impulses to the brain

6. With time the joint becomes thickened and deformed as can be seen in the case of this hip joint



7. Pain – instead of the sudden sharp pain that we are all familiar with, the dog enters a chronic pain state in which the brain and spinal cord have been over-stimulated and sensitised by pain impulses. A small stimulation leads to an excessive response

8. Chronic pain is easily overlooked as it is often manifested as subtle changes, for example dogs seeming slow or miserable

9. Muscles begin to waste away due to reduced capacity for exercise

Treatment

There are three vital components to treatment and there is little benefit in looking for other modalities until these three are in place:

1. **Pain Control**
   The main challenge is to recognise that dogs with OA are in pain, because it is easy to dismiss these signs as "getting old and stiff". Pain should be controlled by using medication to act directly on the inflamed joint and possibly centrally acting drugs to block the pain within the nervous system

2. **Weight Control**
   Losing excessive weight is important to try to reduce ongoing damage in the joint and to help ease the burden for surrounding muscles. This is often a lifestyle change for many families

3. **Exercise Control**
   Exercise is good for joints as it promotes circulation and helps to maintain muscle condition. However, the type and amount of exercise should be tailored to the current clinical stage of the disease

With these three things in place the next stages for treatment are:

4. **Regular reassessment**
   OA is not a static condition and it is important to have regular veterinary assessments of the whole patient

5. **Maintaining Good Muscle Condition**
   This is best achieved through the use of appropriate exercise, physiotherapy and hydrotherapy

The least important consideration, but the one that is often first to come to mind is:

6. **Joint Supplements**
   There is no evidence to show beneficial effects for their use (with the possible exception of very high dose omega 3 fatty acids) and they should never be used in place of the proven treatments listed above.

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