AN INTEGRATIVE APPROACH TO ARTHRITIS

OSTEOARTHRITIS (DJD) is a degenerative disorder of moveable joints, especially the weight-bearing joints, characterised by destruction of the articular cartilage and by formation of new bone at the articular margins. This is a narrow definition describing only the physical changes. In my view, it is a complex clinical entity requiring a comprehensive approach. It may affect all or part of the locomotory system and may be discrete or generalised, affecting one or more joints. It may be the result of anatomical, biochemical, metabolic, or physiological factors, or a combination of some or all of these factors. It is a slowly progressive disease amenable to preventive and therapeutic intervention.

The LOCOMOTORY SYSTEM (musculoskeletal system) is composed of bone, ligament, tendon, capsule, cartilage, synovia, and muscle. The performance of this system is dependent on the proper anatomy and functioning of all of these structures. Stress on one or more of these structures will impact on the rest, leading to the development of disease. In addition to joint disease there may also be bursitis, tendinitis, spondylitis, muscle wasting and weakness, and stretching and thickening of joint capsule. In my opinion, ankylosing spondylitis plays a significant part in the clinical syndrome seen in old dogs. The locomotory system is highly integrated with the cardiovascular, respiratory, neurologic, haemolymphatic, endocrine, and digestive systems. Primary disorders of any of these interrelated systems may produce symptoms directly related to the musculoskeletal system.

The SYNOVIAL JOINT is composed of two opposing articular surfaces lined with cartilage, which is attached to the underlying trabecular bone, and held in correct alignment by ligaments and capsule. The joint capsule envelops the joint and produces and contains the joint fluid, which lubricates the joint, and provides nutrition to the cartilage.

CARTILAGE is an avascular connective tissue composed of two elements – chondrocytes (5%) and extracellular matrix (95%). The chondrocytes produce and maintain the matrix and in turn the matrix provides support and nutrition for the chondrocytes. The matrix is composed of proteoglycan aggregates woven with collagen to form an elastic and compressible structure. The proteoglycans are composed of glycosaminoglycans (GAGs) attached to a core protein, forming a bottlebrush structure. The GAGs are mostly chondroitin-4-sulphate, and lesser amounts of chondroitin-6-sulphate and keratan sulphate. The chondroitins are long chain mucopolysaccharides built from repeating disaccharide units of glucuronic acid and galactosamine sulphate. The precusor for these disaccharides is glucosamine, an amino-monosaccharide, formed from glucose and an amino acid.

The PATHOGENESIS of DJD appears to be associated with a multitude of factors including abnormal loading forces on the joint, trauma, instability, degradation and loss of the cartilage matrix, nutritional imbalances, aging, and predisposing genetic and developmental (dysplastic) factors.

At the anatomical level there is initially a softening (chondromalacia) of cartilage, a loss of elasticity, and roughening of the surface. Later on there is loss of cartilage with fibrillation, cracking, and erosion. The underlying subchondral bone becomes sclerotic from mechanical pressure or the reaction to leakage of synovial fluid. The joint capsule thickens and the synovial surface develops a granular surface resulting in changes to the synovial fluid. Early on there is osteophyte development at the articular margins. The degree of synovial inflammation is variable. This may help explain the poor correlation between anatomical changes and the degree of immobility observed clinically.

At the biochemical level there is a normal turnover of cartilage with a fine balance between the degradation and production of cartilage matrix. If this balance is upset there is an overall loss of

cartilage. It appears that chondrocytes release a cascade of catabolic cytokines, leukotrienes, and prostaglandin derivatives which trigger an inflammatory response. In addition, these cytokins induce the release of lytic enzymes, including metalloproteinases, which degrade collagen and proteoglycans. Simultaneously, normal matrix synthesis is inhibited. These events result in a reduced amount of GAGs and collagen, a loss of binding between them, and as a result there is an increase in the amount of water in the matrix. These biochemical changes decrease the tensile strength and resilience of the matrix, and prevent it from functioning normally in transmitting forces, supporting chondrocytes, and protecting the subchondral bone. As a result there is further damage to chondrocytes and a vicious circle ensues. The disease progresses inexorably with the sloughing of cartilage, proliferation and micro-fractures of subchondral bone, formation of bone cysts and osteophytes, fibrosis of the capsule, joint effusion (loss of viscosity of the fluid and nutrient carrying capacity), and the production of joint mice. The clinical result is pain, deformity, swelling and reduced mobility.

At the metabolic level there are many nutritional factors which affect the biochemical processes described above. Since articular cartilage is avascular, it depends upon synovial fluid as a source of nutrition, and for the disposal of metabolic wastes. Nutritional excesses or deficiencies will affect the normal development and maintenance of the locomotor system. The provision of nutrients to the joint may be influenced by other organ function:

Liver – carbohydrate metabolism and detoxification

Circulation – perfusion of nutrients to the synovia and subchondral bone

Pancreas – digestion and glucose metabolism

Gut – absorption of nutrients (lectins)

The production of free oxygen radicals(FOS) causes tissue damage, reduces synovial fluid viscosity, and promotes inflammation. There is damage to proteins, lipids, GAGs, and proteoglycans. The provision of adequate anti-oxidants will minimise this damage.

CHONDROPROTECTIVE AGENTS have the following attributes:

- Support chondrocyte synthesis of collagen and proteoglycans in matrix
- Support production of hyaluronans in joint fluid
- Inhibit cartilage degradation enzymes
- Prevent fibrin formation in synovial fluid and plaque formation in subchondral blood vessels.

Compounds that exhibit some of these characteristics are the endogenous components of articular cartilage – hyaluronic acid, glucosamine, and chondroitin sulphate.

EXAMINATION AND DIAGNOSIS are best helped by observing the animal walking, trotting, and running, and provides information on gait and visible lameness. Turning in decreasing circles and climbing steps may accentuate abnormalities. Simply watching the animal sit and stand can provide useful information. Stiffness upon rising appears to be due to changes in elasticity of peri-articular structures.

A good history will reveal how the lameness is affected by exercise, rest, the conditions where it is better or worse, time of day, changes in weather, the activities the patient is unwilling to do (climbing stairs, jumping into the car), and whether it is acute or chronic in onset.

Careful manipulation may elicit areas of tenderness within muscle, ligaments, bones, tendons and joints. Rotating joints through their full range of movements helps determine joint function and the presence of pain. Loss of joint range of motion may be due to joint surface incongruity, muscle spasm and contracture, capsular contraction, or mechanical block from osteophytes or joint mice. The presence of bilateral or multi-joint disease is suggestive of a more systemic problem.

Palpation will reveal joint changes (effusion, capsule thickening), conformation, crepitus, muscle wasting, instability (luxation, ligament rupture), and deformity.

A neurological examination may be warranted. Further diagnostic tests may include radiology, joint fluid cytology, and blood analysis. There is a poor correlation between the degree of

lameness and radiographic changes.The older, large breed dogs, are those most commonly affected.

THERAPEUTIC OPTIONS

The main objectives of therapy are relief of pain, restoration of joint function, correction of any underlying structural problem, prevention of avoidable disability or progression of the disease, and attention to any systemic disease. From the discussion on pathogenesis the focus on treatment is the restoration of balance. Prevention may prove to be the best management tool we have.

The use of several of the options below provides the basis for an integrated approach.

* provision of essential nutrients – diet – balance (Ca and protein)
supplements (chondroprotectives)
phytonutrients(herbs) – lectins
anti-oxidants (free radical scavengers)
* lifestyle management – weight control exercise – swimming
rest support – strapping/immobilisation
heat or cold compresses
*manipulative therapy – physiotherapy osteopathy – craniosacral manipulation
chiropractic
*drugs – anti-inflammatory – steroidal/ non-steroidal (destroy cartilage, loss of proteoglycans, inhibit chondroitins, gut erosion)
inhibitors of MMPs
analgesics
slow acting drugs (SAARDS) – injectable/oral
polysulphated GAGs
sodium hyaluronate
* holistic – acupuncture
homoeopathy
organ support – liver detoxification/bowel cleansing/insulin balance
*surgery – joint irrigation/ ligament repair/ correction of deformity/ debridement of osteophytes and removal of joint mice/prostheses/ muscle release (pectinectomy)/ arthrodesis/ arthroplasty/ wedge osteotomy/ amputation/ neurectomy

PERSONAL APPROACH

- natural diet – mimic the wild diet – fresh/raw/balance/prevention
  white meats-minced bone/cartilage
  homoeopathy/acupuncture
  supplements – supply nutrients, anti-oxidants, and anti-inflammatory agents
  EFAs and herbs
  judicious use of drugs
  surgery
  treatment of concurrent disease – eg diabetes, Addisons, SLE, hypercalcaemia, hypothyroidism.

JOINT FLEX (Great & Small)

A nutraceutical source of type II collagen, hydroxyappatite, fatty acids, botanicals, amino acids, GAGs (chondroitins), glucosamine, trace elements, minerals, and vitamins. These nutrients are sourced from whole animal extracts which have been carefully processed to minimise nutrient damage. This combination provides a broad range of nutrients with inherent natural balance.

Active ingredients in 3.6gm:

Glucosamine sulphate 400mg	Vitamin C 200mg
Shark cartilage 450mg	Boron (from sodium tetraborate) 500mcg
Bovine cartilage 450mg	Magnesium (from aspartate) 25mg
Green lipped muscle 500mg	Manganese (from chelate) 2mg
Baical skullcap 200mg	Selenium(from selenomethionine) 75mcg

Willow bark 100mg	Zinc(from chelate) 5mg
Natural vitamin E 20mg	Methyl sulphonyl methane(MSM) 250mg
Niacinamide(B3) 5mg	Kelp 200mg
Copper (from gluconate) 500mcg	Yeast 480mg

This provides approximately 150mg (minimum) of GAGs, mostly from shark cartilage 7-12% and bovine tracheal cartilage 20-28%. Contrary to popular opinion green lipped muscle only contributes 2-3%. The variation in GAGs depends on the age and species used, and varies slightly from one batch to the next.

There appears to be a link between angiogenesis and the progression of OA. The anti-angiogenetic effect of shark cartilage may prevent the vascularisation and degradation of cartilage and block the progression of disease.

CONCLUSION

This approach addresses the whole locomotor system and considers the impact of systemic disease. Treatment is primarily focused on prevention – mostly through sound nutrition. Where there is significant pathology, the aim is to supply additional nutrients including chondroprotective, anti-inflammatory, and anti-oxidant agents.

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