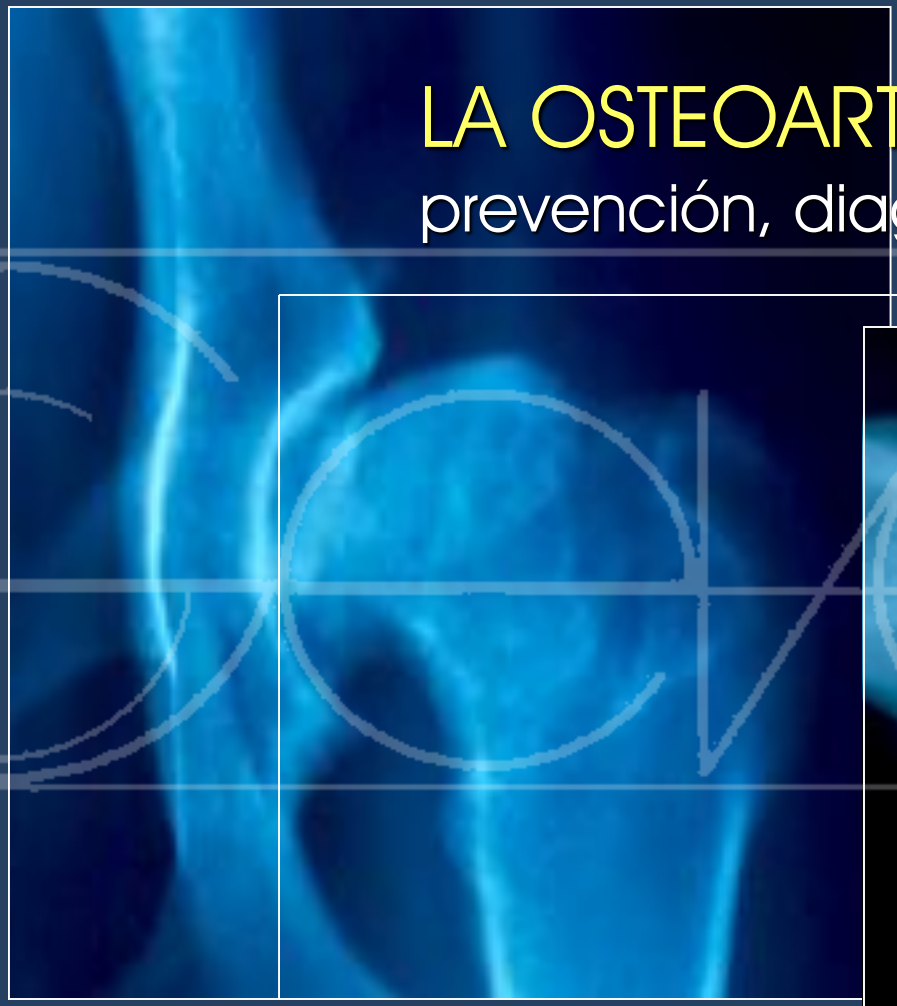
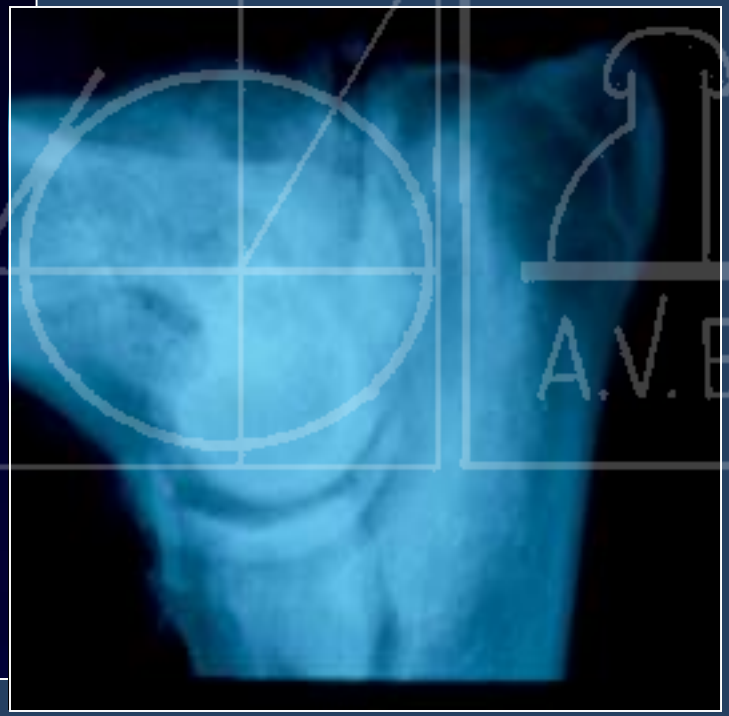
Grupo Veterinario KITICAN



LA OSTEOARTRITIS EN EL PERRO

prevención, diagnóstico y tratamiento



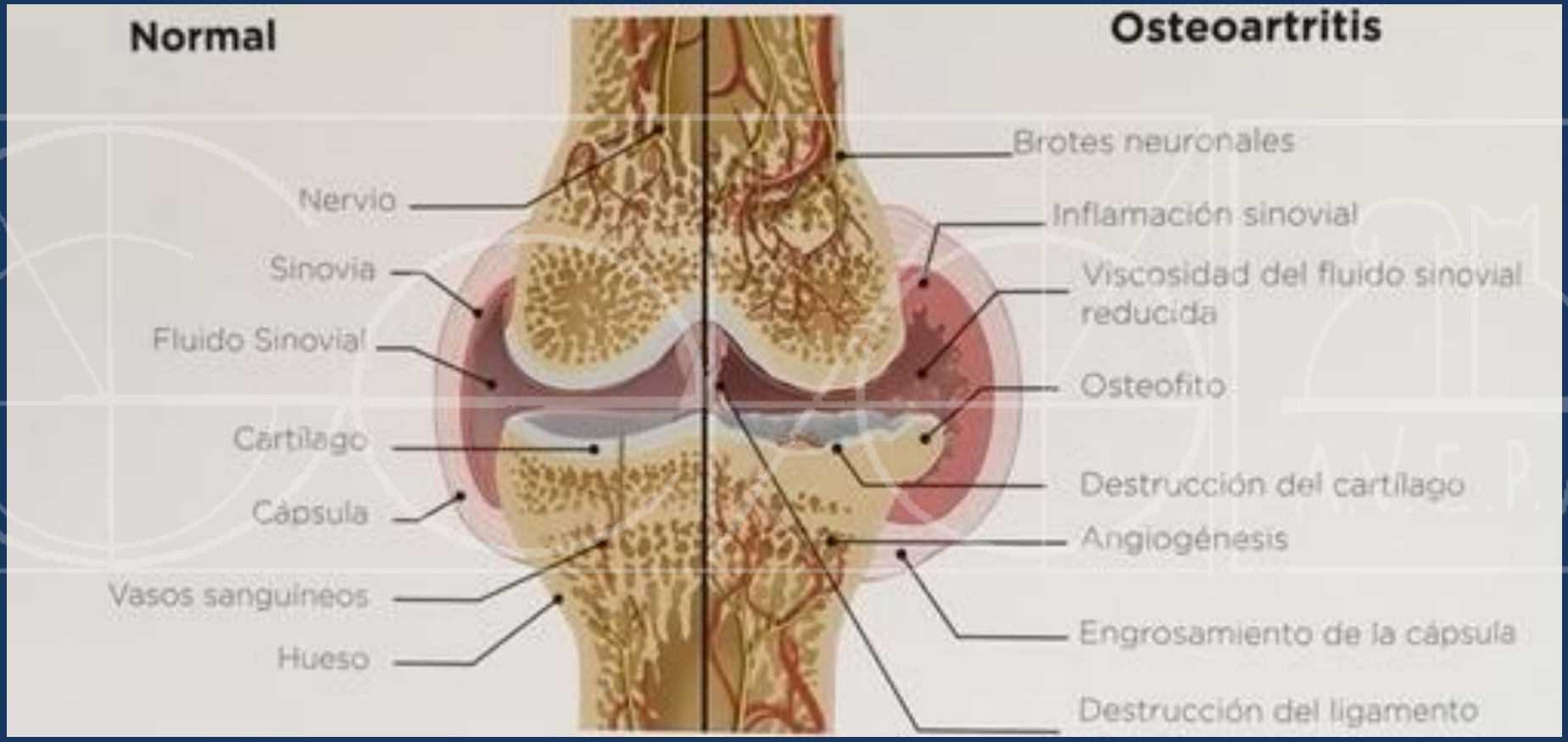
CENTRO MEDICO VETERINARIO *Delicias*

Tomás Fernández González DVM Ph D

Mar Lopez Faisano DVM

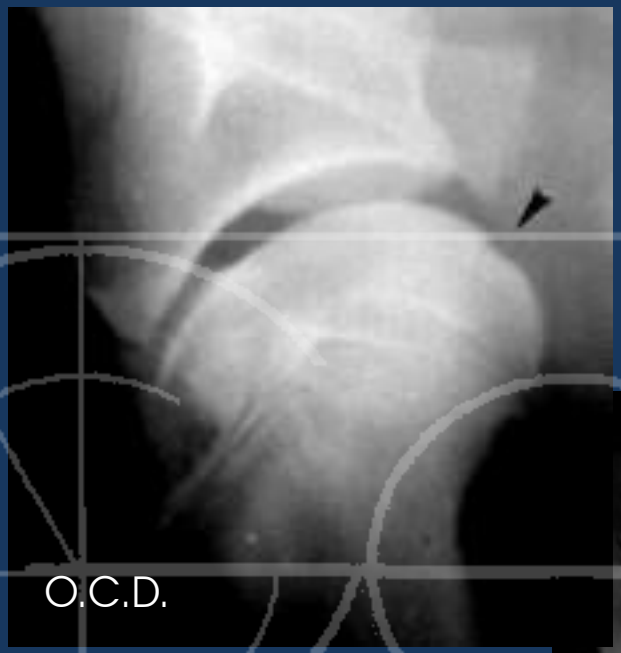
¿Que es la Osteoartritis?

Degeneración gradual y dolorosa del cartílago de las articulaciones (según RAE)


Normal
Osteoartritis
Nervio
Sinovia
Fluido Sinovial
Cartílago
Cápsula
Vasos sanguíneos
Hueso
Brotes neuronales
Inflamación sinovial
Viscosidad del fluido sinovial reducida
Osteofito
Destrucción del cartílago
Angiogénesis
Engrosamiento de la cápsula
Destrucción del ligamento

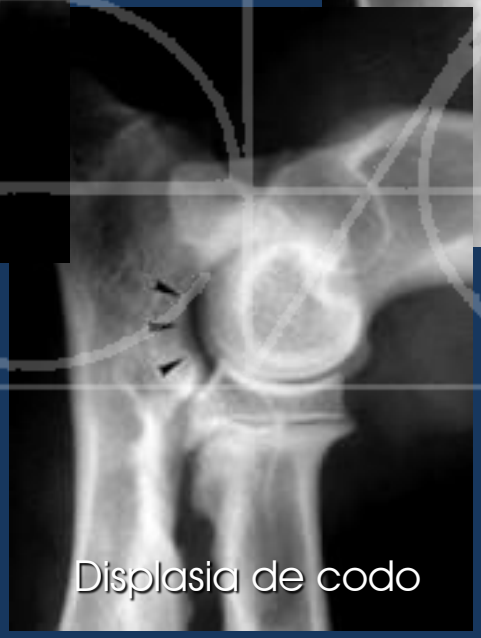
Zoetis

¿Qué causas generan osteoartritis?

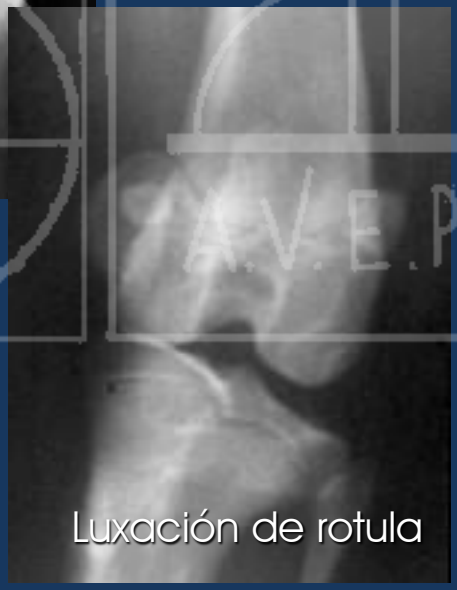


O.C.D.

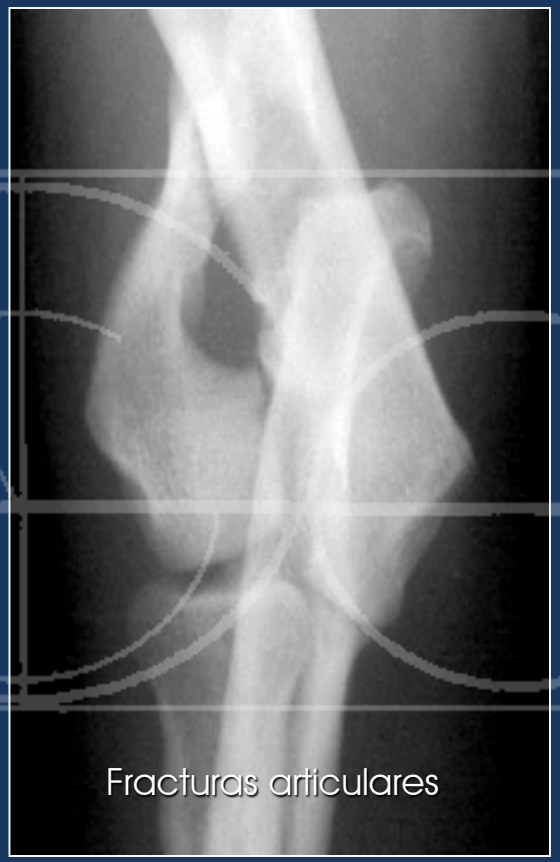
Displasia de cadera



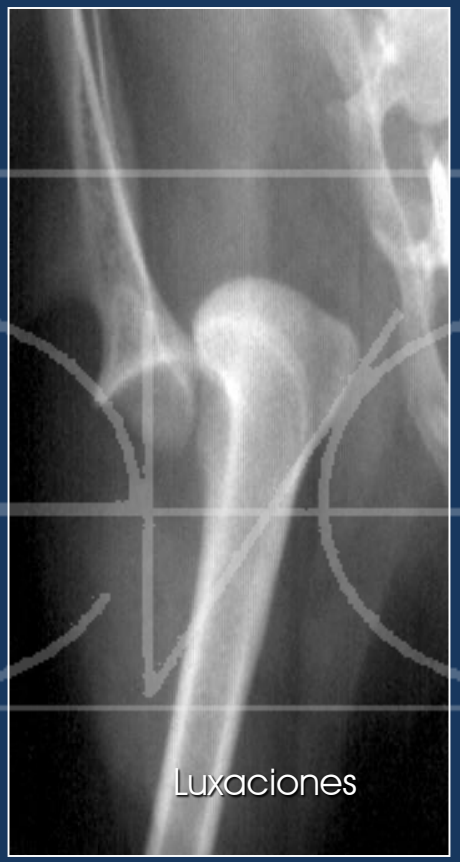
Displasia de codo



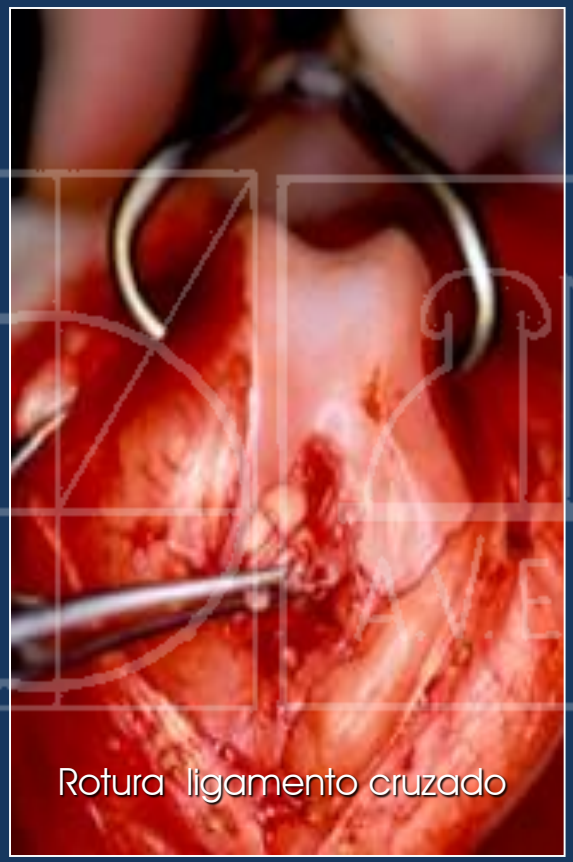
Luxación de rotula



Fracturas articulares

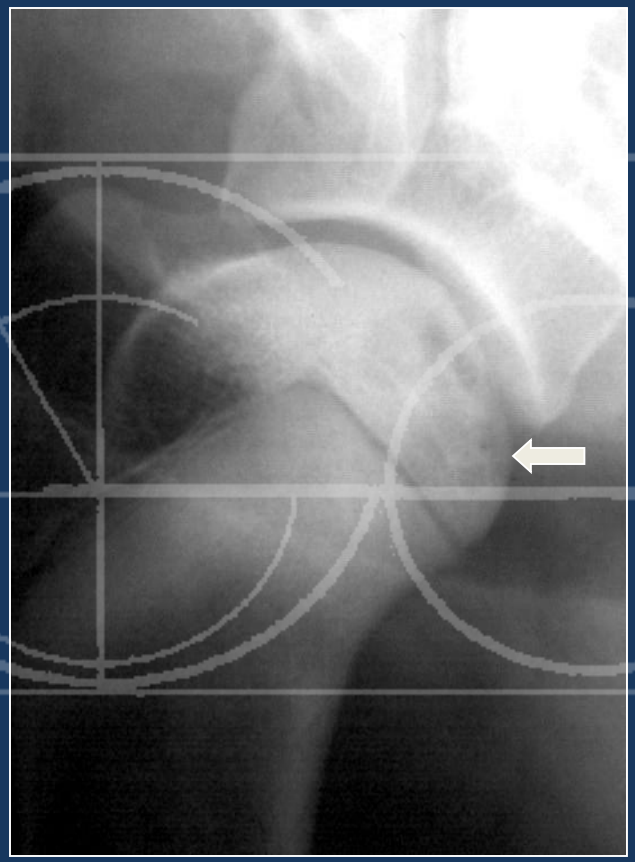


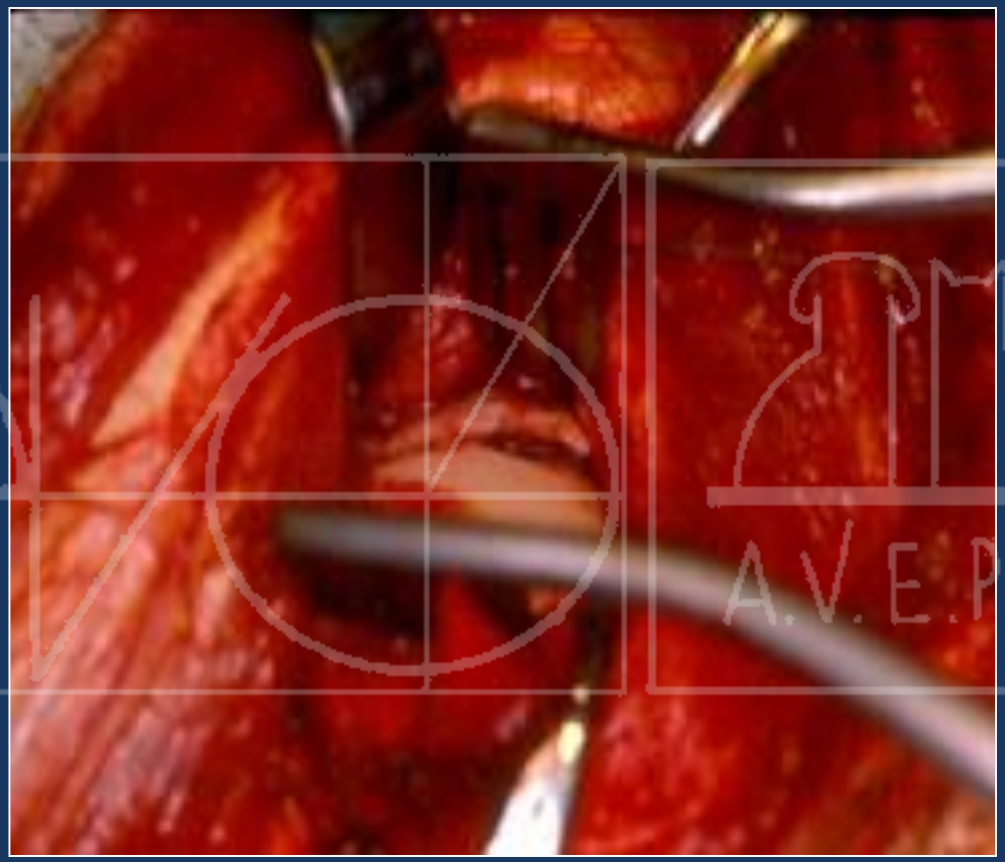
Luxaciones

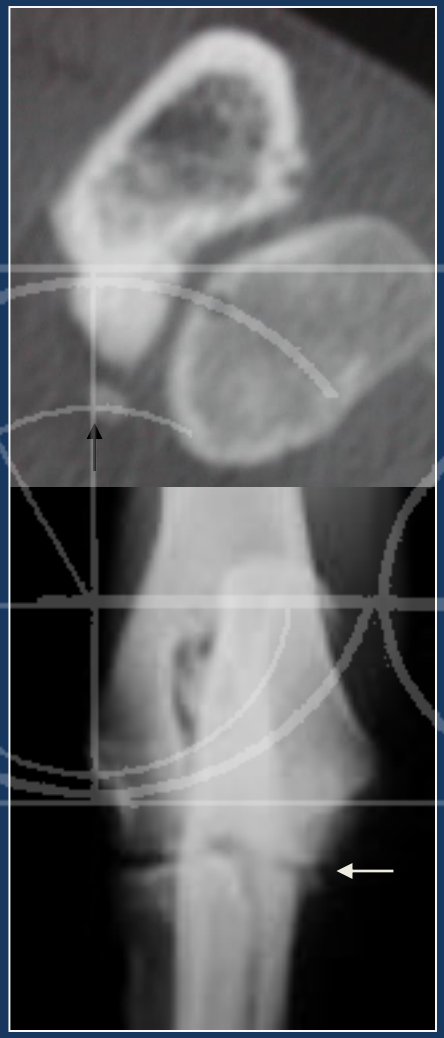


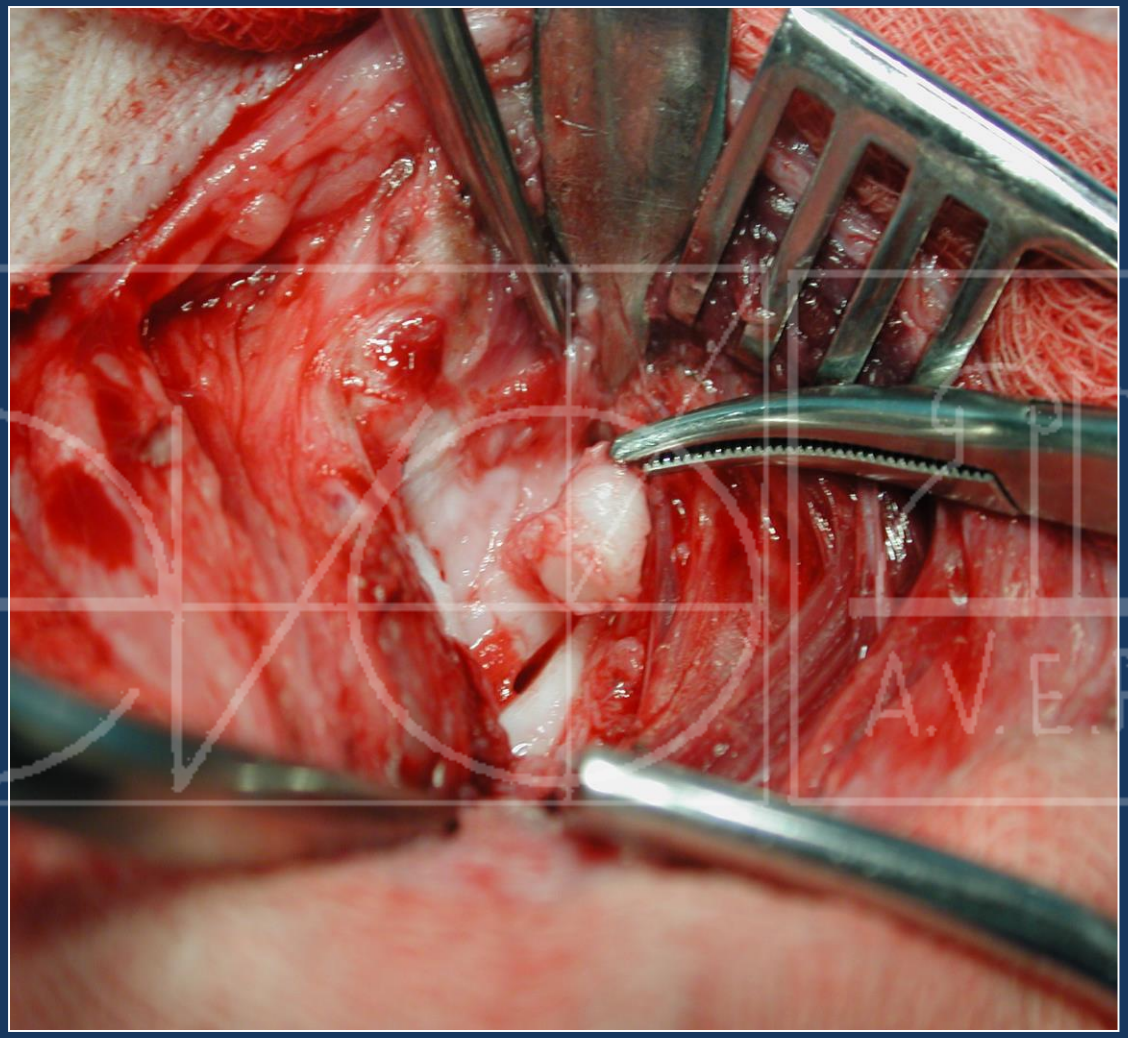
Rotura ligamento cruzado

¿Se puede prevenir la Osteoartritis?









elbow joint. There were precursors to this more refined procedure in the early 2000s, evidenced by the humeral wedge and humeral slide osteotomies.[5,6] Further research was done looking at medium-term and long-term outcomes, gaining this procedure a spot in the armament against MCD.[7]

The goal of the SHO is to shift the weight-bearing axis of the forelimb (digits to shoulder) as it crosses the elbow joint (in particular, the humeroulnar contact) laterally. This decreases the weight-bearing load within the medial compartment of the elbow joint, because this is where the majority of pathology resides (Fig. 3). The exact cause of MCD largely is unknown; however, it is evidenced as advancing cartilage wear and

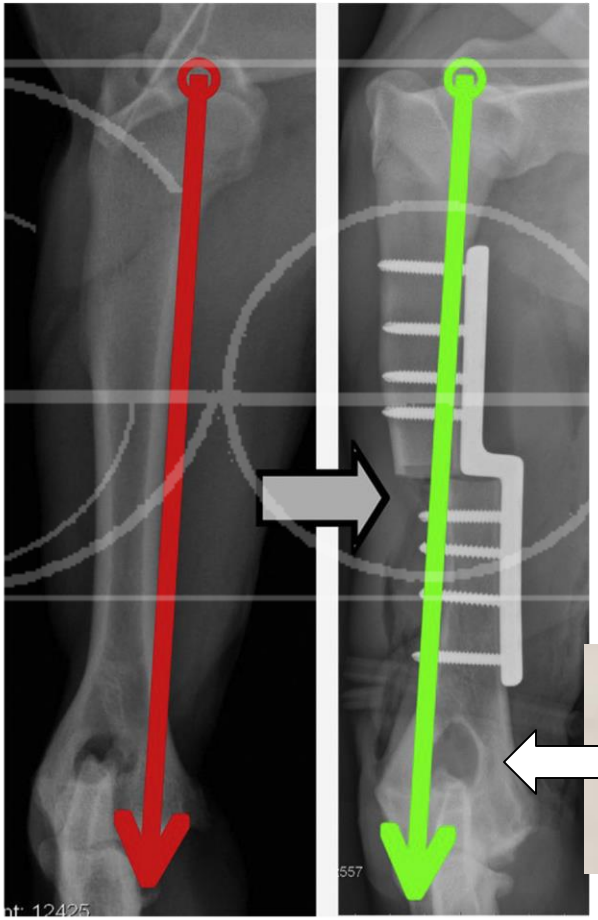


Fig. 3. Craniocaudal radiographs depicting the shift in weight-bearing access from medial (*red arrow* in the left image) to lateral (*green arrow* in the right image) following SHO.

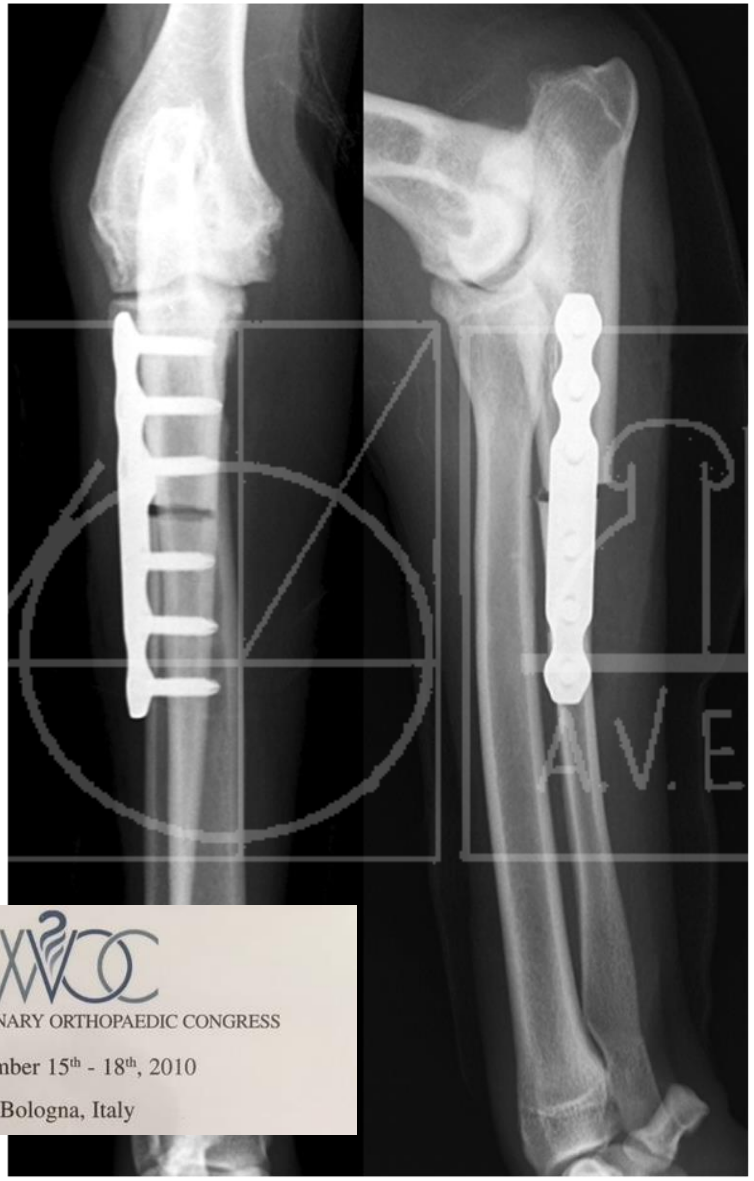


Fig. 14. Postoperative orthogonal radiographs depicting proper osteotomy and implant placement.

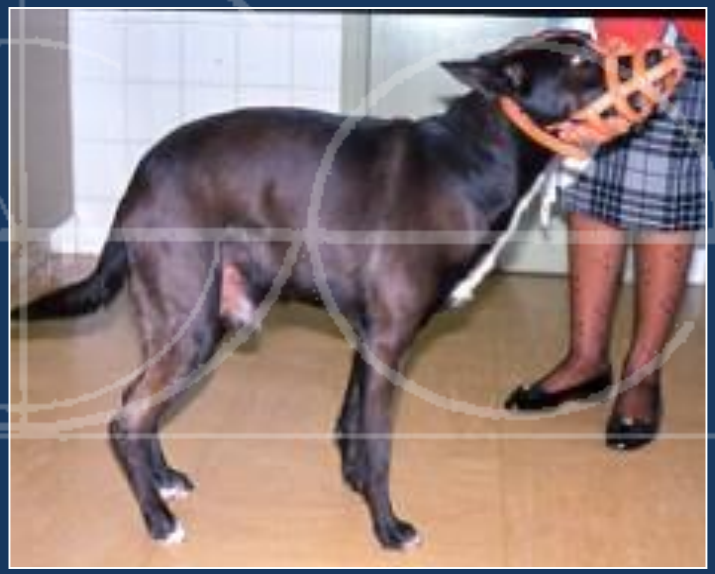
¿Como se diagnostica la Osteoartritis?

El diagnóstico de la osteoartritis se basará en:

1. Los signos clínicos

2. Las imágenes radiológicas

¡DOLOR!



1. Reacciones posturales
2. Temperamento
3. Vocalización
4. Locomoción
5. Otros

El dolor es el elemento central de la OA

Escalas de valoración

1. Breve cuestionario de dolor canino (Brown et al. 2007)
2. Indice de dolor crónico Helsinki (Hielm-Bjorkman et al. 2009)
3. Indice ortopédico canino (Brown 2013)
4. Cuestionario de Liverpool sobre OA (Walton et al. 2013)
5. Calidad de vida asociada a la salud (Reid et al. 2013)
6. Medidas específicas proporcionadas por propietario (Lascelles)
7. Herramientas de clasificación del grado de OA (Cachon et al. 2018)

El diagnóstico de la osteoartritis se basará en:

1. Los signos clínicos

2. Las imágenes radiológicas y TC

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DOI: 10.1111/vru.12718

ORIGINAL INVESTIGATION

WILEY

Stifle joint osteoarthritis at the time of diagnosis of cranial cruciate ligament injury is higher in Boxers and in dogs weighing more than 35 kilograms

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Abstract

Osteoarthritis is a ubiquitous disease in dogs. The purpose of this retrospective study was to characterize the severity and distribution of osteoarthritis (OA) within the joint and to identify differences among dog breeds in the severity of OA in the cranial cruciate ligament (CCL)-deficient stifle joint. Radiographs of 240 stifles from 51 Boxers, 66 German Shepherds, 100 Labrador Retrievers, and 23 Siberian Huskies with confirmed CCL rupture were included. Radiographs of the stifle joint were evaluated and OA severity was graded at 33 sites within and around the joint, and patella alta was graded as present or absent for a potential total stifle OA score of 100. Osteophyte size was correlated to OA severity score. Total OA scores were calculated and compared within and between breeds globally as well as at each joint site. Dogs weighing >35 kg had a higher total OA score than those weighing <35 kg. Osteoarthritis scores were highest at the apical patella, proximolateral tibia, and sesamoid bones, corresponding to the proximal, lateral, and caudal aspects of the joint, respectively. No statistically significant differences were found among the mean OA scores of various stifle joint regions. Boxer dogs had a higher total OA score than other breeds. We concluded that dogs have a consistent distribution pattern of OA within the stifle joint after CCL injury. Radiographic OA is more severe in the proximal, lateral, and caudal aspects of the joint. Boxers had more severe OA than the other breeds evaluated in the study.

KEYWORDS
cranial cruciate ligament, dog, osteoarthritis, osteophytes

1 | INTRODUCTION

Cranial cruciate ligament (CCL) rupture is one of the most common causes of pelvic limb lameness in dogs.[1] In large breed dogs, CCL problems are suspected to result from a multifactorial degenerative process that leads to rupture of the ligament.[2,3] Cranial cruciate ligament problems lead to osteoarthritis (OA)[4] that can start before CCL rupture and reportedly progresses when the CCL rupture is managed conservatively or when it is managed by use of surgery.[5–8] The severity of OA at the time of presentation influences the prognosis after CCL injury and may influence management decisions.[5]

Radiography is the standard diagnostic modality for assessment of stifle OA in dogs.[4,9,10] Radiography is widely available and allows a clear visualization of bone changes resulting from OA. Stifle radiographs obtained at the time of presentation after CCL injury are commonly used to rule out problems other than CCL rupture, to assess presurgical OA, and to serve as a baseline for serial radiography monitoring postsurgical healing and long-term OA progression. Several radiographic grading schemes have been described to quantify stifle OA.[4,9,11,12] A 32-point scale based on the presence and severity of radiographic changes such as osteophytes, enthesophytes, joint effusion, capsular thickening, and subchondral cysts has been reported.[9,10] In humans with CCL rupture, OA is most severe in the patellofemoral joint space.[13] It is unclear whether stifle OA in dogs follows a similar pattern.

Genetic predisposition to CCL disease has been reported.[14,15] The prevalence, age at diagnosis, and clinical progression of CCL disease vary among dog breeds.[16–19] Several theories for the pathophysiology of CCL disease have been proposed, some relate to stifle joint biology[2,20] and others to pelvic limb conformation.[21] Several

Abbreviations: CCL, cranial cruciate ligament; OA, osteoarthritis; TPLO, tibial plateau leveling osteotomy; TTA, tibial tuberosity advancement

Presented at the 2017 ACVS Surgery Summit, Indianapolis, IN.

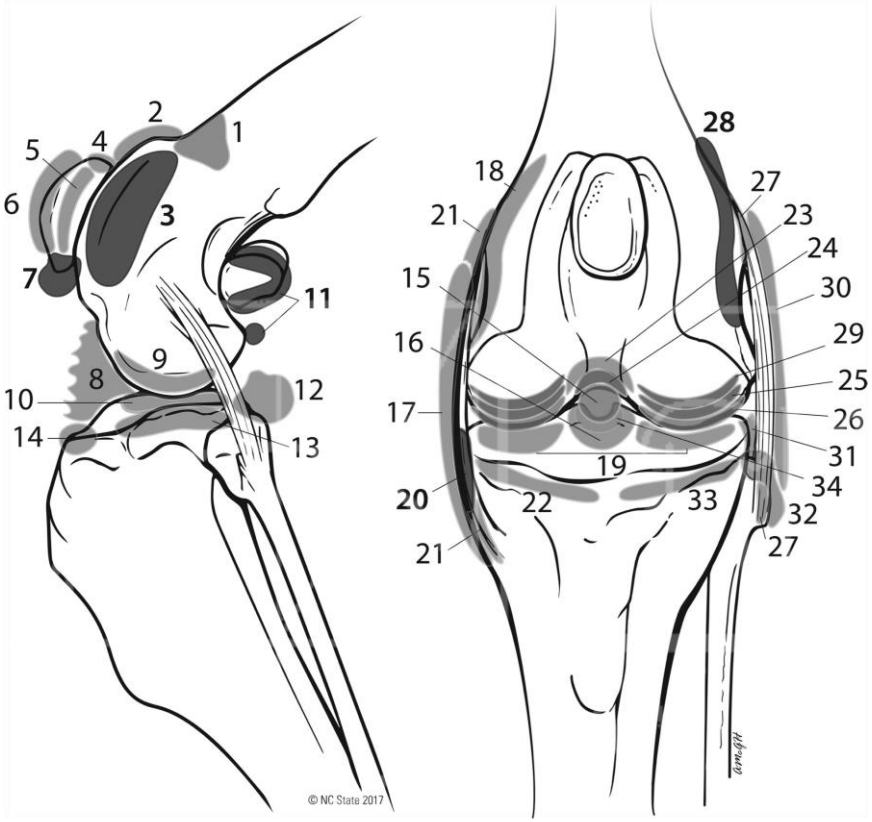
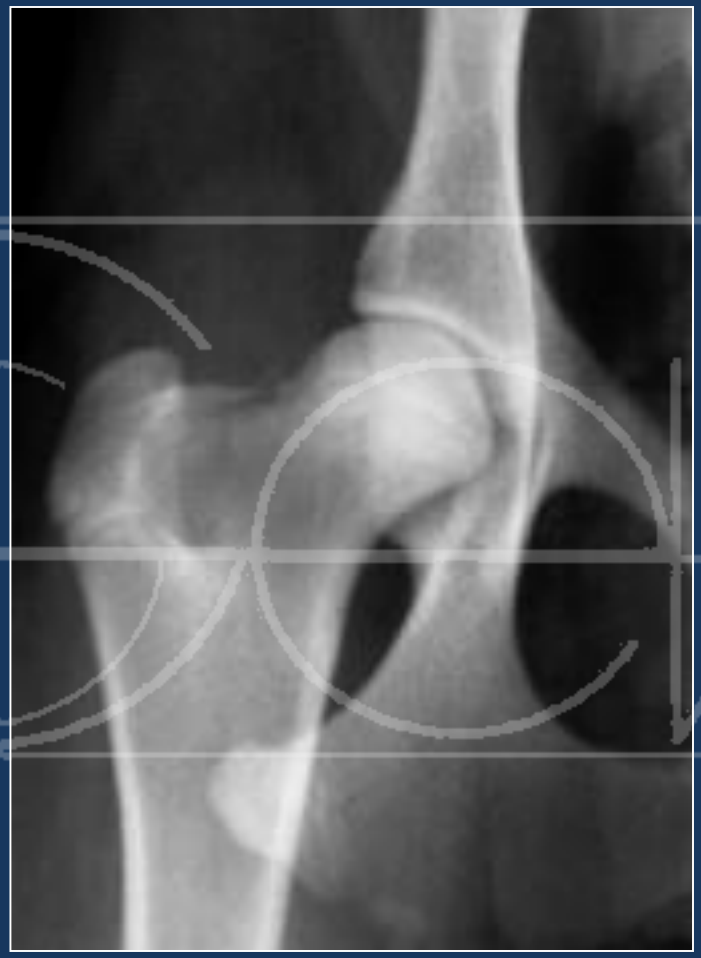
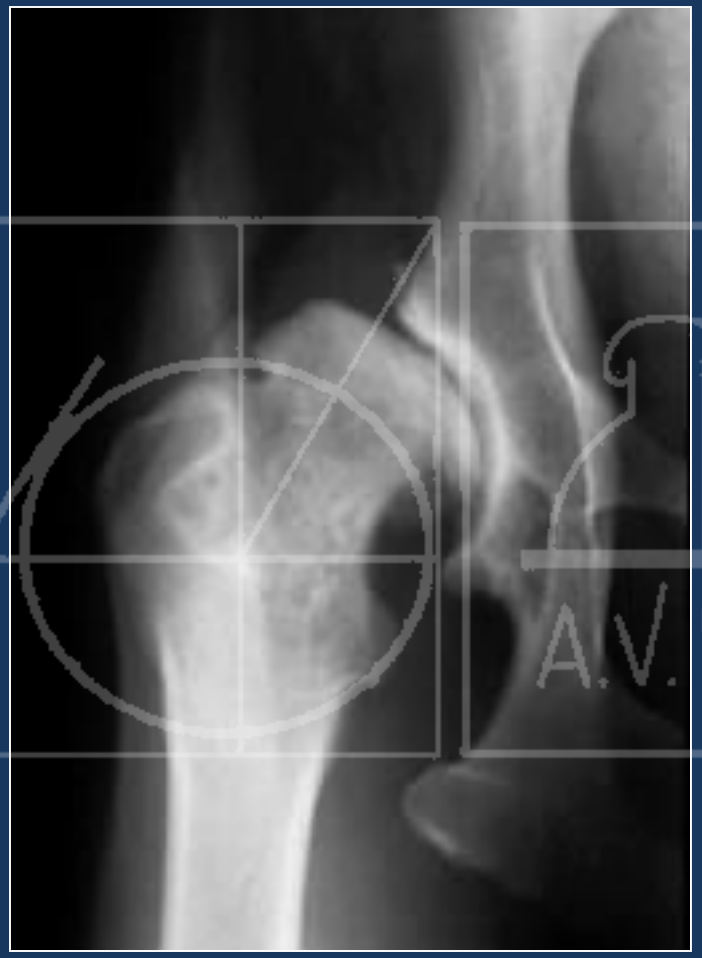
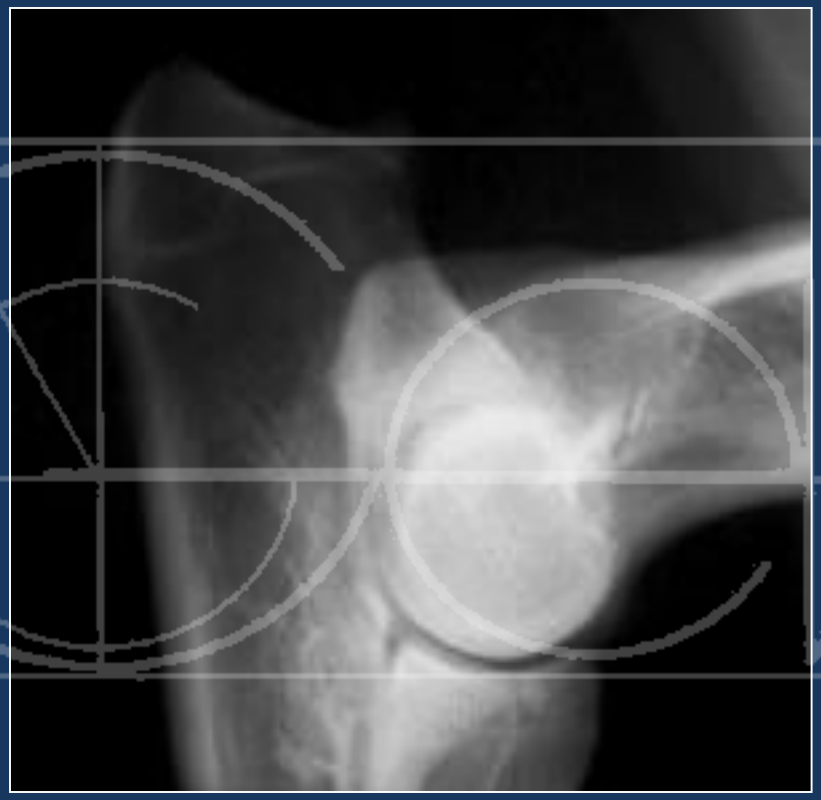
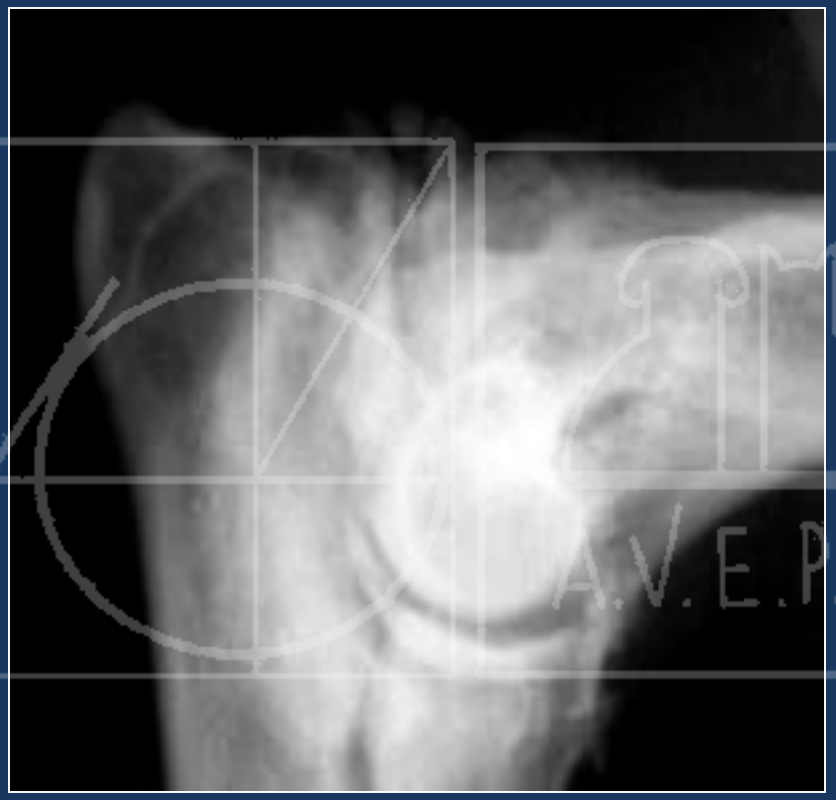


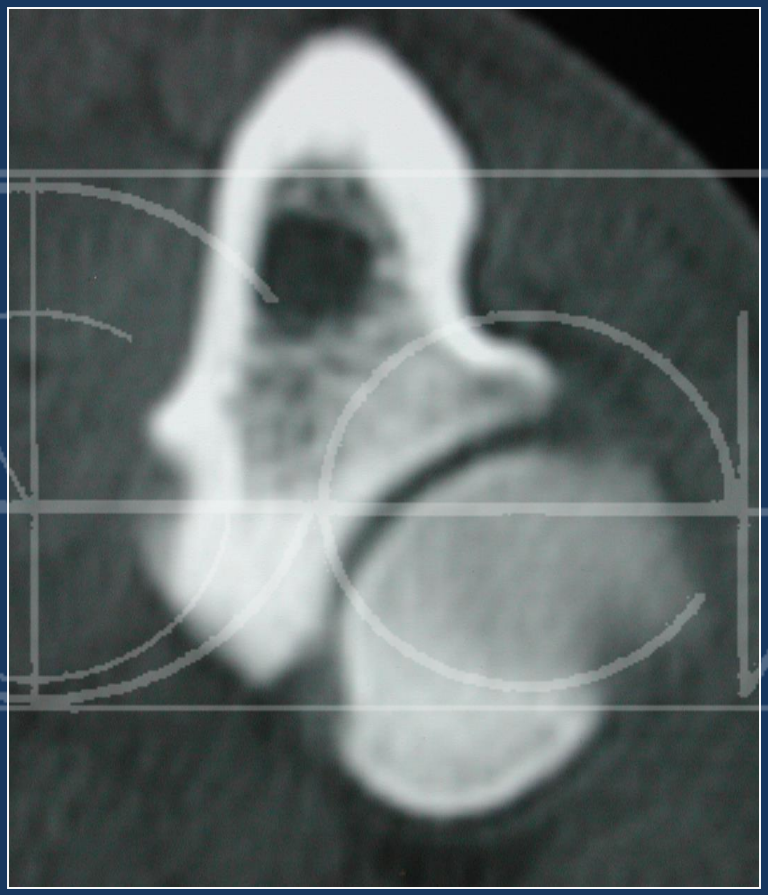
FIGURE 1 Osteoarthritis was graded as absent, mild (osteophytes or enthesophytes measuring <2 mm), moderate (≥2 mm and ≤4 mm), or severe (>4 mm) in 33 regions of the canine stifle joint, including femoral supratrochlear lysis (landmark 1), osteophytes along the femoral trochlear groove (landmark 3), patellar basilar osteophytes (landmark 4), patellar subchondral cystic lucencies (landmark 5), cranial apical patellar enthesopathy (landmark 6), patellar apical osteophytes (landmark 7), stifle joint effusion or capsular thickening (landmark 8), femoral condylar subchondral cystic lucencies (landmark 9), tibial condylar remodeling (landmark 10), osteophytes of the fabellae and popliteal sesamoids (landmark 11), osteophytes of the caudoproximal tibia (landmark 12), proximal tibial epiphyseal subchondral cystic lucencies (landmark 13), osteophytes of the cranioproximal tibia (landmark 14), intercondylar avulsion fracture fragments (landmark 15), osteophytes of the central tibial plateau (landmark 16), medial soft tissue thickening (landmark 17), osteophytes of the medial femoral epicondyle (landmark 18), meniscal mineralization (landmark 19), osteophytes of the proximomedial tibia (landmark 20), medial collateral ligament enthesopathy (landmark 21), proximomedial tibial subchondral sclerosis (landmark 22), osteophytes of the femoral intercondylar region (landmark 23), intercondyloid fossa subchondral cystic lucencies (landmark 24), femoral condylar subchondral cystic lucencies (landmark 25), femoral subchondral sclerosis (landmark 26), lateral collateral ligament enthesopathy (landmark 27), osteophytes of the lateral femoral epicondyle (landmark 28), distal femoral condylar remodeling (landmark 29), lateral soft tissue thickening (landmark 30), osteophytes of the proximolateral tibia (landmark 31), fibular osteoarthritis (landmark 32), proximolateral tibial subchondral sclerosis (landmark 33), and intra-articular mineralized osseous fragments (landmark 34). Patella alta (landmark 2) was graded as absent or present. For all breeds combined, OA scores at landmarks 3, 7, 11, 20, and 28 correlated most highly with the overall OA score (Table 2) and are highlighted

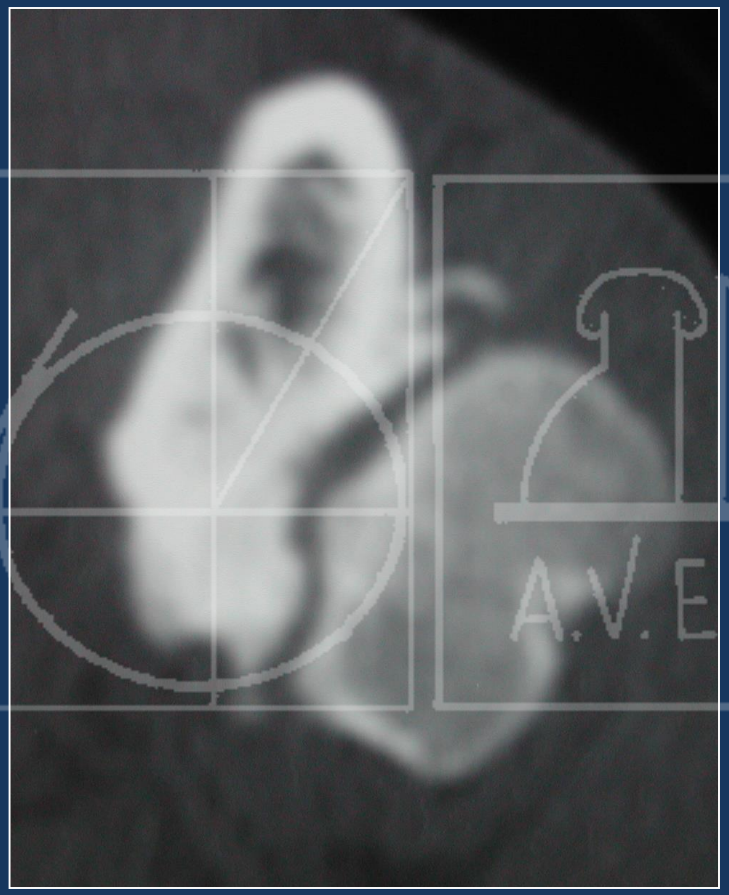












COMPUTED TOMOGRAPHIC IDENTIFICATION OF DYSPLASIA AND PROGRESSION OF OSTEOARTHRITIS IN DOG ELBOWS PREVIOUSLY ASSIGNED OFA GRADES 0 AND 1

CHELSEA M. KUNST, ANTHONY P. PEASE, NATHAN C. NELSON, GREG HABING, ELIZABETH A. BALLEGEER

Elbow dysplasia is a heritable disease that is a common cause of lameness and progressive elbow osteoarthritis in young large breed dogs. The Orthopedic Foundation for Animals (OFA) screens elbow radiographs, and assigns grades 0–3 based on presence and severity of bony proliferation on the anconeal process. Grade 1 is assigned when less than 3 mm is present and considered positive for dysplasia. We investigated the incidence of elbow dysplasia and progression of osteoarthritis in elbows with grades 0 and 1 in 46 elbows screened at least 1 year previously, using CT as a gold standard and with the addition of CT absorptiometry. The incidence of dysplasia based on CT was 62% in grade 0, and 75% in grade 1 elbows, all of which had medial coronoid disease. Progressive osteoarthritis at recheck was consistent with elbow dysplasia. The sensitivity and specificity of the OFA grade for elbow dysplasia compared to CT findings was 75% and 38%, respectively. Increased bone mineral density of the medial coronoid process as characterized by osteoabsorptiometry warrants further investigation with respect to elbow dysplasia. Proliferation on the anconeal process without CT evidence of dysplasia or osteoarthritis was present in 20% of the elbows, and is theorized to be an anatomic variant or enthesopathy of the olecranon ligament/synovium. Results of our study suggest that the "anconeal bump" used for elbow screening by the OFA is a relatively insensitive characteristic, and support the use of CT for identifying additional characteristics of elbow dysplasia. *© 2014 American College of Veterinary Radiology.*

Key words: canine orthopedic foundation for animals, computed tomography, elbow dysplasia, medial coronoid process, osteoabsorptiometry.

Introduction

ELBOW DYSPLASIA IS A common heritable disease in dogs. Elbow dysplasia involves one or more of four distinct pathologic processes: medial coronoid disease (which is the most common type of dysplasia),[1–5] and includes but is not limited to fragmented medial coronoid process), ununited anconeal process, osteochondrosis of the humeral condyle, and elbow incongruity.[6] Elbow dysplasia can cause debilitating lameness, and arthroscopic treatment does not palliate pain in all dogs.[7] Additionally, elbow dysplasia is characterized by progressive osteoarthritis,[2,3,8–11] which surgical treatment does not resolve.[7] Therefore, it is important to identify and remove affected dogs from the breeding pool to decrease prevalence of elbow dysplasia in the canine population.

The Orthopedic Foundation for Animals (OFA) in the United States maintains a screening process of pure bred dogs for elbow dysplasia. Dogs must be at least 2 years of age and be registered with a recognized organization, such as the American Kennel Club. The Foundation assesses a single mediolateral radiographic projection of the elbow during extreme flexion for the presence of bony proliferation on the anconeal process, as this is often the first place in the elbow that evidence of osteoarthritis occurs secondary to a dysplastic lesion, and diagnosis of dysplasia based on the presence of secondary signs has been well documented.[1,2,8,12–21] The OFA grades range from 0 to 3. A grade 0 is assigned when there is no evidence of proliferation on the anconeal process, and is considered negative for the presence of elbow dysplasia. Grades 1–3 are assigned when

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Funding sources: Michigan State University, College of Veterinary Medicine Endowed Research Funds Project Grant.

Previous abstract: Kunst CM, Habing G, Ballegeer EA. CT identification of dysplasia and progression of osteoarthritis in dog elbows previously assigned Orthopedic Foundation for Animals (OFA) grade 1. 2012 ACVR Annual Scientific Conference, Las Vegas, Nevada: Vet Rad Ultrasound, Vol. 53, No. 6, 681–682.

Previous presentation: 2012 ACVR Annual Scientific Conference, Las Vegas, Nevada.

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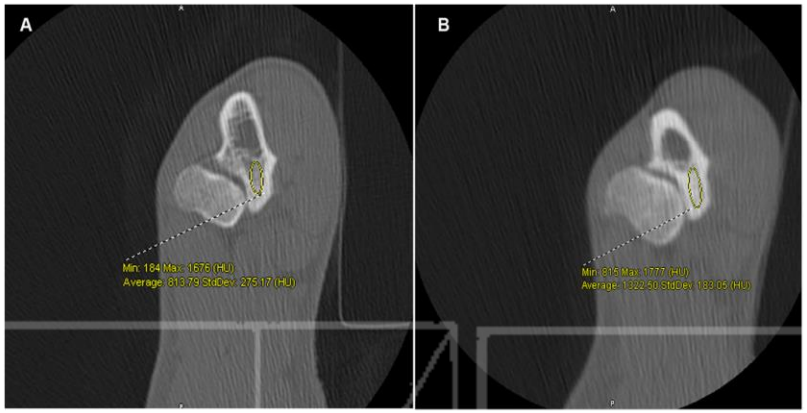

FIG. 2. Demonstration of the ellipsoid region of interest drawn on the transverse plane CT images at the slice location with the largest medial coronoid process height for the purpose of recording the regional Hounsfield units. (A) is an elbow with a presumed normal medial coronoid process, and (B) is an elbow with a presumed diseased medial coronoid process.

was present between reviewers, a consensus was reached after mutual examination of images; these were mostly limited to minimal differences in height measurements, and subjective assessments of sclerosis or medial coronoid process blunting, as seen on Time 0 radiographs. In addition to subjective assessment, a similar-sized ellipsoid region of interest was hand drawn on transverse plane images at the slice of largest medial coronoid process height to include the subchondral bone of the ulna in the medial coronoid process and ulnar notch (Fig. 2) to more quantitatively measure Hounsfield units. Care was taken to include only the perceived medullary bone, and not cortical bone, by following visible cortices adjacent to the nonhyperattenuating medulla, and excluding the bone following the same thickness along the entire cortex. Additionally, the region of interest edge was placed at the corticomedullary junction (when visible) and the caudal extent of the region of interest only to the level of the lateral coronoid process. Additional regions of interest (ROIs) were drawn on each bar of the previously described phantom, taking care not to include edges, or recognizable beam-hardening artifact.

Statistical Analysis

All statistics were performed by an epidemiologist (G.H.) using commercially available statistics software (SAS v. 9.2, Cary, NC). All p values for significance were set at 0.05. Descriptive statistics were calculated, including the proportion of elbows at each time point with each grade, osteoarthritis, and fragmented medial coronoid process. Additionally, linear correlation between height of proliferation on the anconeal process on radiographs obtained at Time 1 compared to the CT images was performed using the PROC CORR procedure. Attenuation correction for ulnar Hounsfield units on CTs was performed by translating Hounsfield units to bone mineral densities with phantom attenuation corrections, as a more accurate means of reporting bone density, as previously described.[30,35,36] Four elbows were excluded due to phantom absence from the scan. The remaining 42 BMD values were calculated for each elbow from attenuation measurements from phantom bars, linear regression slope and intercept values, and the equation: BMD = (region of interest value – intercept)/slope. To test differences in the mean BMD between dogs with and without medial coronoid process disease, a generalized linear model was constructed using the PROC GENMOD procedure. To account for the expected nonindependence of elbows within dogs, a repeated statement was included for dog. Bone mineral density was tested for correlation with age, separated into separate elbows to remove the correlation of elbows within dogs, by calculating the Pearson correlation coefficient using PROC CORR procedure. Exact confidence intervals for the proportions, including sensitivity and specificity, were calculated using the PROC FREQ procedure.

Results

Twenty-three dogs met the inclusion criteria. They ranged in age from 3.2 to 8.7 years (mean 5.6 years old). Ten breeds were represented: six Golden Retrievers, four Bernese Mountain dogs, three each of English Springer

¿Como se trata la Osteoartritis? ¿Hay novedades?

El tratamiento de la osteoartritis seguirá siendo:

1. Conservador

2. Quirúrgico

Nonsurgical Management of Osteoarthritis in Dogs

Spencer A. Johnston, VMD[a,*], Ronald M. McLaughlin, DVM, DVSc[b], Steven C. Budsberg, DVM, MS[a]

KEYWORDS

• Osteoarthritis • Nonsteroidal anti-inflammatory drug
• Disease-modifying agent of osteoarthritis • Physiotherapy
• Medical management • Evidence-based

Occam's razor is a philosophical statement that is often used as a guideline in the practice of medicine. Originally referred to as the Law of Parsimony, it essentially states that the simplest explanation of a problem is frequently the best.[1] In medicine, it is often used to suggest that a patient's clinical signs can usually be explained by one disease process instead of a complex interaction of multiple disease processes. By extension, it is often suggested that the abnormality is treated in the least complex manner possible.

Although Occam's razor can be used to explain how a multitude of clinical signs can be attributed to a condition like osteoarthritis (OA), it does not necessarily extend to the treatment of this condition. OA, although superficially considered to be deterioration of the joint associated with pain and dysfunction, is actually quite a complex condition. When considering treatment of OA, a multitude of biochemical, physical, and pathologic alterations must be recognized.[2] Because our knowledge of OA and factors contributing to its development suggest that OA has existed for as long as the diarthrodial joint has existed, and because no known cure or even universally accepted treatment for OA exists, it is probably safe to assume that a single simple treatment does not exist. This does not seem to hinder the quest to find one, however. The search for the Holy Grail of OA treatment continues, and is likely to continue, well past the career longevity of the authors of this article.

Treatment for OA is effectively limited to the available products. The number of products proved to provide safe and effective treatment does not change rapidly. The approved pharmaceutic agents are the most extensively reviewed products. There is a constant search to find new and improved treatments, however, and

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doi:10.1016/j.cvsm.2008.08.001 vetsmall.theclinics.com
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nonpharmaceutic treatments are often suggested and embraced despite a lack of proved efficacy or safety. Journal articles, podium presentations at major and minor veterinary meetings, and popular press articles frequently address the treatment of OA. Seemingly, most of these presentations are based on the same data, or include that author's or speaker's opinion variably based on scientific data or anecdotal experience.

In practice, the decision of when and how to treat OA is often based on a combination of factors. These factors include the available data regarding efficacy but also incorporate the frequency of administration, product formulation, cost, promotions and advertisements by the manufacturer or distributor of the drug or supplement, personal experience, and success or failure of prior treatments used by the client and patient. Treatment is further influenced by the ability or willingness of the client to understand or implement weight control, exercise modification, and physical therapy as part of the management strategy. This article presents a review of the published material regarding various treatments for OA. When there are no data regarding a specific treatment or when a statement is the opinion of the authors, such a deficiency is identified.

TREATMENT OF OSTEOARTHRITIS

Treatment of OA has traditionally been directed toward palliation of the painful symptoms associated with the condition. It is generally recognized that a variable degree of pathologic change, including bone and soft tissue alterations, exists, and the degree of pathologic change and clinical signs associated with OA must be considered on a continuous scale. The severity of discomfort, often manifest as lameness, can be inconsistent with the degree of pathologic or radiographic change. Furthermore, the severity of the associated symptoms may be related to recent use, or stress, placed on the articular and periarticular tissues. The combination of these variables may lead to chronic pain, often characterized as a dull ache, or acute pain, more typically characterized as a sharp shooting pain. The wide range of factors affecting joint health and pain status makes it difficult to provide a specific recommendation for the treatment of OA that is applicable in all situations. Part of the challenge of OA treatment is that the goal is often variable between patients or within an individual patient. As a result, multimodal therapy is often necessary to address this complex problem.

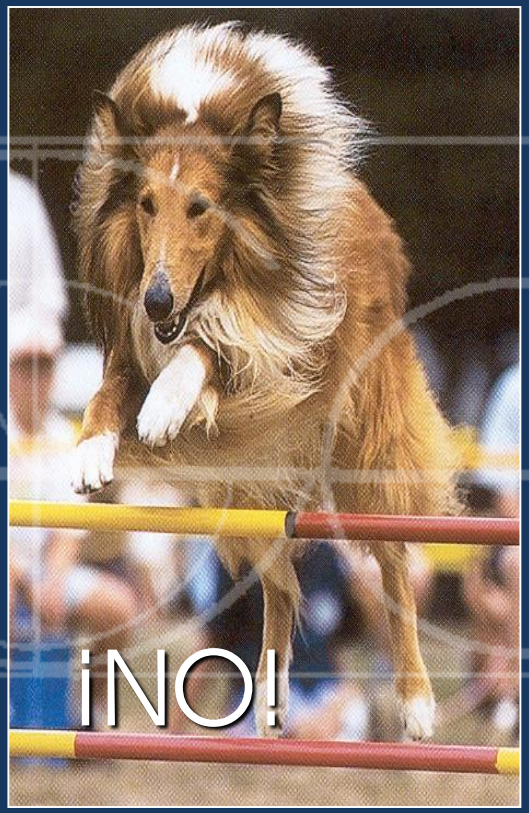
NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most frequently recommended treatment for OA. The popularity of this class of drugs is typically attributed to the effectiveness of NSAIDs for palliating the painful symptoms associated with OA and their relative ease of administration. An excellent thorough review of NSAIDs approved in the United States for use in small animals was recently published.[3] It is not the intention of this article to repeat such an extensive review but to focus on the use of these products for the treatment of OA.

Although acetaminophen, an analgesic, is often recommended for the treatment of OA in human beings because of a decreased side effect profile, NSAIDs remain popular despite well-known side effects that may occur with their use.[4–6] When used by people who have OA, NSAIDs are generally considered to provide a greater global relief score than does acetaminophen.[7] A greater global relief score is generally a result of treatment effect (including decreased pain, improved functioning, or both), decreased side effects, and patient (or client) expectation.[8] Although use of acetaminophen is

El tratamiento conservador de la osteoartritis se basará en:

1. Ejercicio controlado
2. Dieta
3. Medicación

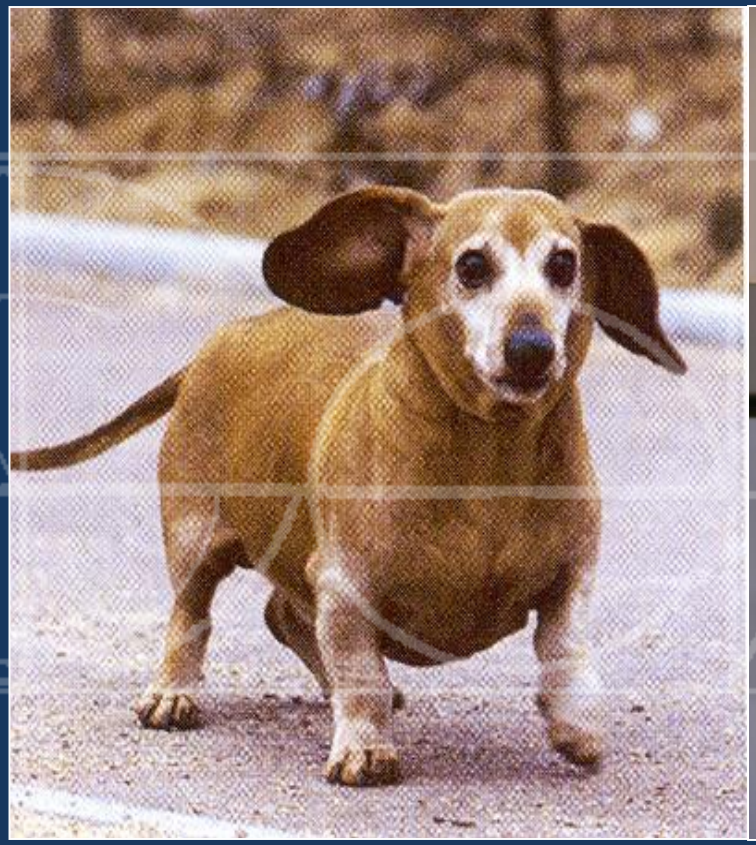

¡NO!


¡NO!

El tratamiento conservador de la osteoartritis se basará en:

1. Ejercicio controlado
2. Dieta
3. Medicación

Obesidad: Proteinas del tejido adiposo (Leptinas)



A review of osteoarthritis and obesity: current understanding of the relationship and benefit of obesity treatment and prevention in the dog

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Keywords
Obesity, osteoarthritis, canine, human

Summary
Obesity is an increasingly important health problem for both man and dog. Osteoarthritis (OA) is a significant cause of pain and disability in both species. A link between obesity and OA has been established in man, though the exact mechanism of the relationship remains to be fully elucidated – current research supports both biomechanical and biochemical theories. There is good evidence (class I*) to support weight loss as an effective treatment for human knee OA. In the dog, the relationship is just beginning to be investigated. The results of one study in dogs (class IV evidence*) suggest that preventing the development of overweightness and obesity reduces the prevalence of hip dysplasia and OA of the hip and other joints. Three other studies (class III and IV evidence*) support weight loss as an effective treatment for OA in affected overweight and obese dogs. Further research could yield greater understanding of the pathophysiology of this relationship, perhaps identifying novel therapeutic targets. Confirmation and better understanding of the positive effect of treating and preventing obesity on symptoms and prevalence of OA is likely to be valuable in the campaign against canine obesity.

* Classes of evidence detailed in Table 1.

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Introduction

The World Health Organisation has declared obesity to be the most important health problem currently facing the Western world (1). In humans, body mass index (BMI) is calculated using the formula; body mass (kg) / height $(m)^2$, and obesity and overweightness are defined as a BMI = 30 and = 25 respectively (2) . In the United States in 2004, 33% of adults were considered obese and 17% of teenagers overweight (2). A study published in 1986 estimated the prevalence of canine obesity to be 24% and a similar study published in 2005 gave a figure of 41% (3, 4). It is widely suspected among veterinarians that the prevalence of obesity among populations of domestic dogs is increasing. Currently obesity and overweightness in the dog are arbitrarily defined as relative bodyweight = 120% and = 110% respectively (5). Relative bodyweight is calculated by dividing actual weight by an estimated ideal body weight and multiplying by 100%. Osteoarthritis (OA) is the most common cause of pain and physical disability in man, and is likely to be of similar significance in the dog, with an estimated 20% of adult dogs affected (6, 7). Obesity has consistently been identified as a risk factor for the development of OA in man, with the strongest evidence supporting a relationship with knee OA. It is now recognised that reducing the prevalence of OA and its associated burden on health services requires a commitment to tackling the obesity pandemic (8).

Aim of review

The purpose of this review is to summarise current theories surrounding the relationship between obesity and OA in man, and to systematically review the literature pertaining to a similar relationship in the dog. Two questions are raised in the systematic review: first – does prevention of canine overweightness and obesity reduce OA prevalence, and second – will weight loss alleviate clinical signs of pain and disability in overweight and obese dogs with OA?

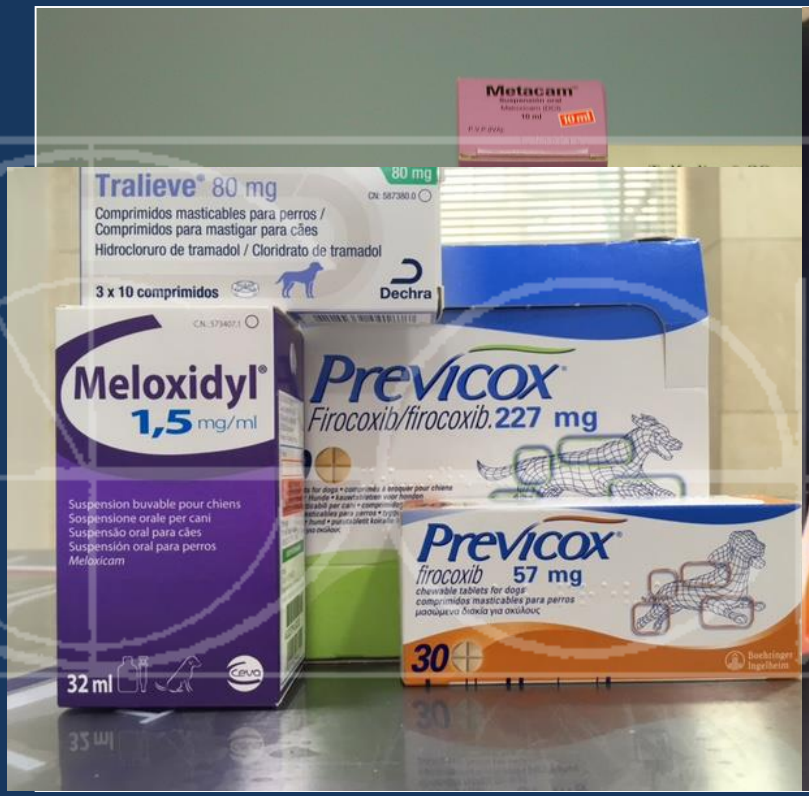
Obesity and osteoarthritis in man

Obesity has consistently been identified as a risk factor for development of knee and hand OA, and for the progression of knee OA (9–11). The relationship between obesity and hip OA is not so convincing: a meta-analysis performed in 2002 found only moderate evidence to support it, and two recent studies did

El tratamiento conservador de la osteoartritis se basará en:

1. Ejercicio controlado
2. Dieta
3. Medicación

Tramadol, Amantadina, Gabapentina, etc



Carprofeno, Mavacoxib, Cimicoxib, Deracoxib, Meloxicam, Firocoxib, Enflicoxib, Grapiprant

2022 AAHA Pain Management Guidelines for Dogs and Cats*

Margaret E. Gruen, DVM, MVPH, PhD, DACVB†,
B. Duncan X.Lascelles, BSc, BVSc, PhD, CertVA, DSAS(ST), DECVS, DACVS, FRCVS†,
Elizabeth Colleran, DVM, MS, ABVP feline specialty, Alison Gottlieb, BS, CVT, VTS (ECC),
Jennifer Johnson, VMD, CVPP, Peter Lotsikas, DVM, DACVS-SA, DACVSMR,
Denis Marcellin-Little, DEDV, DACVS, DACVSMR, Bonnie Wright, DVM, DACVAA

ABSTRACT

These updated guidelines present a practical and logical approach to the assessment and management of acute and chronic pain in canine and feline patients. Recognizing pain is fundamental to successful treatment, and diagnostic guides and algorithms are included for assessment of both acute and chronic pain. Particularly for chronic pain, capturing owner evaluation is important, and pain-assessment instruments for pet owners are described. Expert consensus emphasizes proactive, preemptive pain management rather than a reactive, "damage control" approach. The guidelines discuss treatment options centered on preemptive, multimodal analgesic therapies. There is an extensive variety of pharmacologic and nonpharmacologic therapeutic options for the management of acute and chronic pain in cats and dogs. The guidelines include a tiered decision tree that prioritizes the use of the most efficacious therapeutic modalities for the treatment of acute and chronic pain. (*J Am Anim Hosp Assoc* 2022; 58:55–76. DOI 10.5326/JAAHA-MS-7292)

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CONTRIBUTING REVIEWERS

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† M. Gruen and B. Lascelles were co-chairs of the Pain Management Guidelines Task Force.

These guidelines were prepared by a task force of experts convened by the American Animal Hospital Association. This document is intended as a guideline only, not an AAHA standard of care. These guidelines and recommendations should not be construed as dictating an exclusive protocol, course of treatment, or procedure. Variations in practice may be warranted based on the needs of the individual patient, resources, and limitations unique to each individual practice setting. Evidence-based support for specific recommendations has been cited whenever possible and appropriate. Other recommendations are based on practical clinical experience and a consensus of expert opinion. Further research is needed to document some of these recommendations. Because each case is different, veterinarians must base their decisions on the best available scientific evidence in conjunction with their own knowledge and experience.

*These guidelines are supported by generous educational grants from Arthrex Vet Systems, Boehringer Ingelheim Animal Health USA Inc., Elanco, and Zoetis.

AI (artificial intelligence); CBPI (Canine Brief Pain Inventory); CMIs (Clinical Metrology Instruments); COAST (Canine OsteoArthritis Staging Tool); COX (cyclooxygenase); CSOM (client-specific outcome measures); FMPI (Feline Musculoskeletal Pain Index); HRA (health risk assessment); HRQoL (health-related quality of life); IA (intra-articular); LOAD (Liverpool Osteoarthritis in Dogs); mAB (monoclonal antibody); MICAT-C (Montreal Instrument for Cat Arthritis Testing - Caretaker); MiPSC (Musculoskeletal Pain Screening Checklist); NGF (nerve growth factor); NSAID (nonsteroidal anti-inflammatory drug); OA (osteoarthritis); PRA (prostaglandin receptor antagonist); SNoRE (Sleep and Nighttime Restlessness Evaluation); TRPV1 (transient receptor potential cation channel subfamily V member 1)

Acetato de metil-prednisolona
+
Bupivacaina

Published online: 2020-04-21

Evaluation of a Single Intra-Articular Injection of Autologous Adipose Tissue for the Treatment of Osteoarthritis: A Prospective Clinical Study in Dogs

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Vet Comp Orthop Traumatol

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Abstract

Objective The aim of this study was to investigate the safety, feasibility and clinical efficacy of a single intra-articular injection of autologous and purified micro-fragmented adipose tissue for the treatment of osteoarthritis (OA) in dogs.

Study Design Twenty-one client-owned dogs with radiographically confirmed OA were recruited into this prospective study. Lameness and discomfort were evaluated by physical examination at day 0 and then 14, 30, 60 and 180 days after injection. Kinetic data and temporospatial parameters were obtained using a pressure-sensing walkway. Peak vertical force, vertical impulse and percentages of body weight distribution were determined. Owner perception data regarding their own dog's physical activity were also collected using the Canine Brief Pain Inventory.

Results Radiographic scores for OA from days 0 to 180 were similar, except in two dogs. No major side effects were noted after injection. Lameness and Canine Brief Pain Inventory scores were significantly lower at all time points compared with day 0. Post-injection results demonstrated gradual improvement of kinetic data up to day 180 compared with pre-treatment values: vertical impulse (>2.25%), peak vertical force (>5.32%) and percentages of body weight distribution (>3.6%). In dogs with elbow OA, gait analysis values significantly increased at all time points compared with day 0.

Conclusion Regenerative autologous adipose tissue injection therapy is a promising alternative to traditional analgesics treatment in patients with OA, associated with significant reductions in pain and lameness, delayed disease progression and improved quality of life.

Keywords
- dogs
- osteoarthritis
- kinetic gait analysis
- regenerative therapies
- autologous adipose tissue

Introduction

Osteoarthritis is a chronic degenerative disease affecting the articular cartilage and subchondral bone; it causes pain and inhibits movement.[1] Approximately 20% of the canine population develops osteoarthritis.[2] Since the discovery in 2001 by Zuk and colleagues[3] that adipose tissue is a major source of mesenchymal stem cells, scientists have been studying the use of adipose-derived stem cells for the treatment of various disorders including osteoarthritis.[4,5] Adipose tissue provides an advantage over other sources of adult stem cells due to its abundant availability, easy isolation of cells and minimal patient discomfort.[3]

The isolation of adipose-derived stem cells, contained within the adipose tissue-derived stromal vascular fraction, requires an in vitro selection and expansion process that

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February 28, 2019
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January 26, 2020

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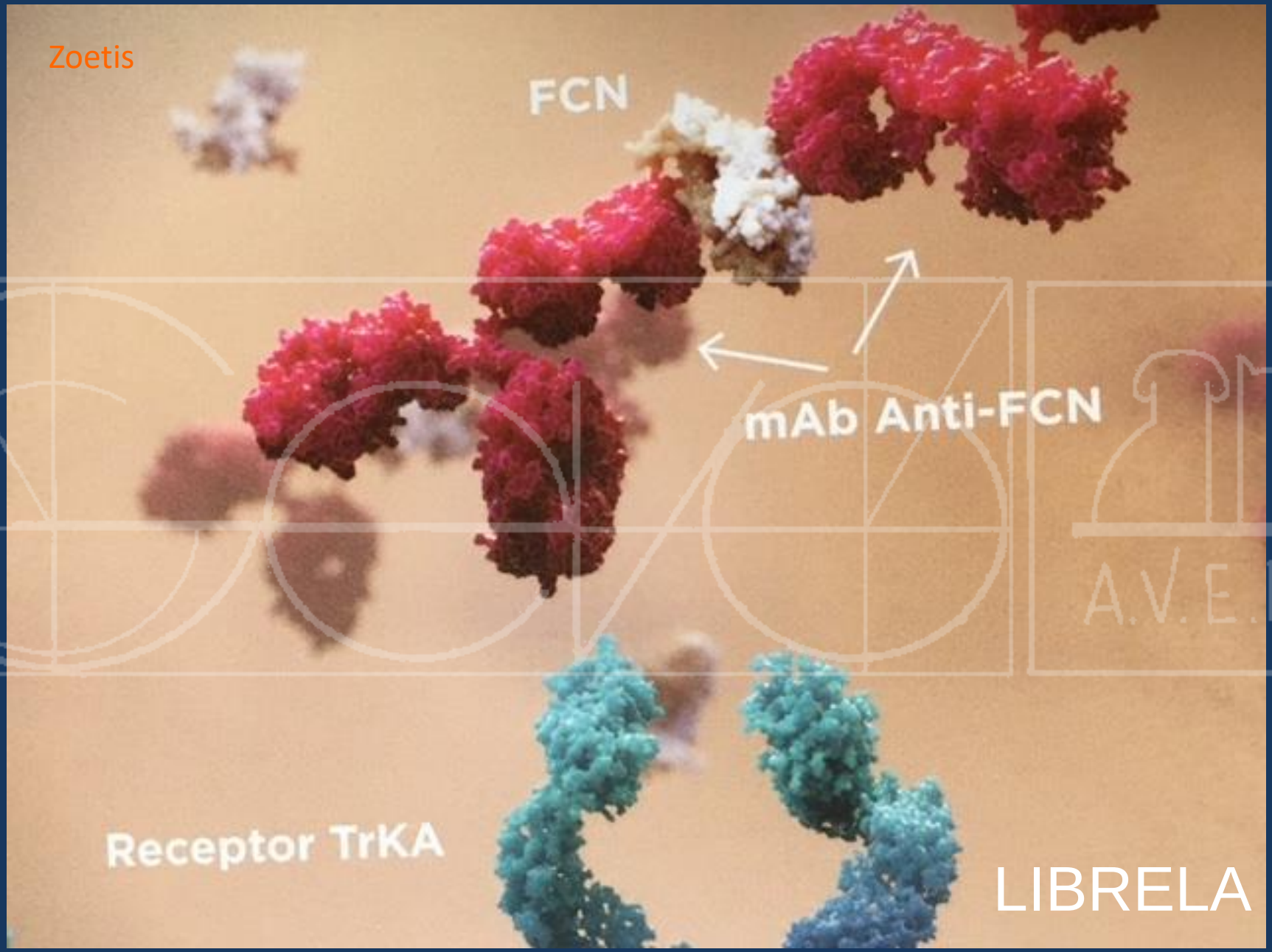
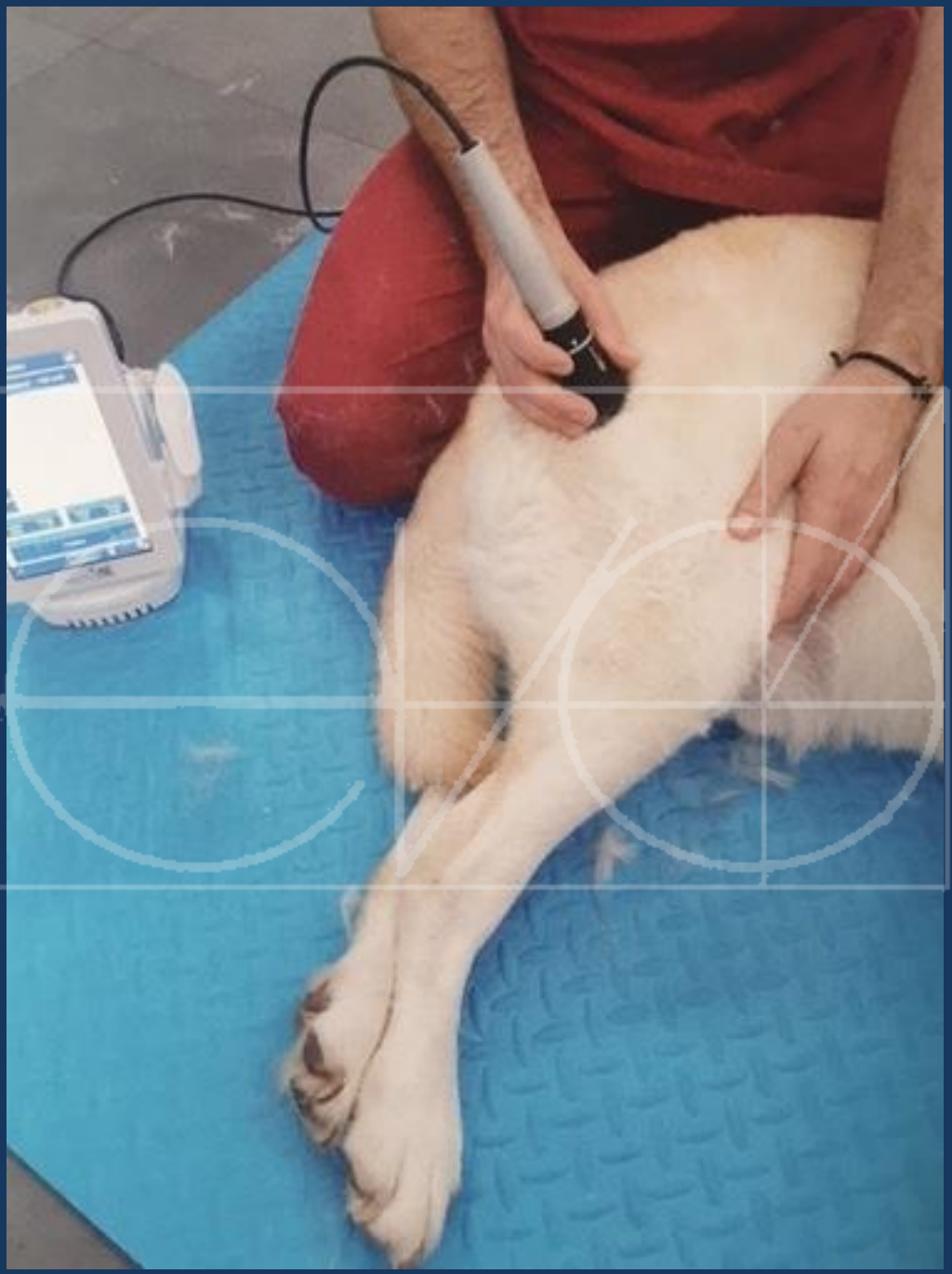
ISSN 0932-0814.


Zoetis
FCN
mAb Anti-FCN
Receptor TrKA
LIBRELA

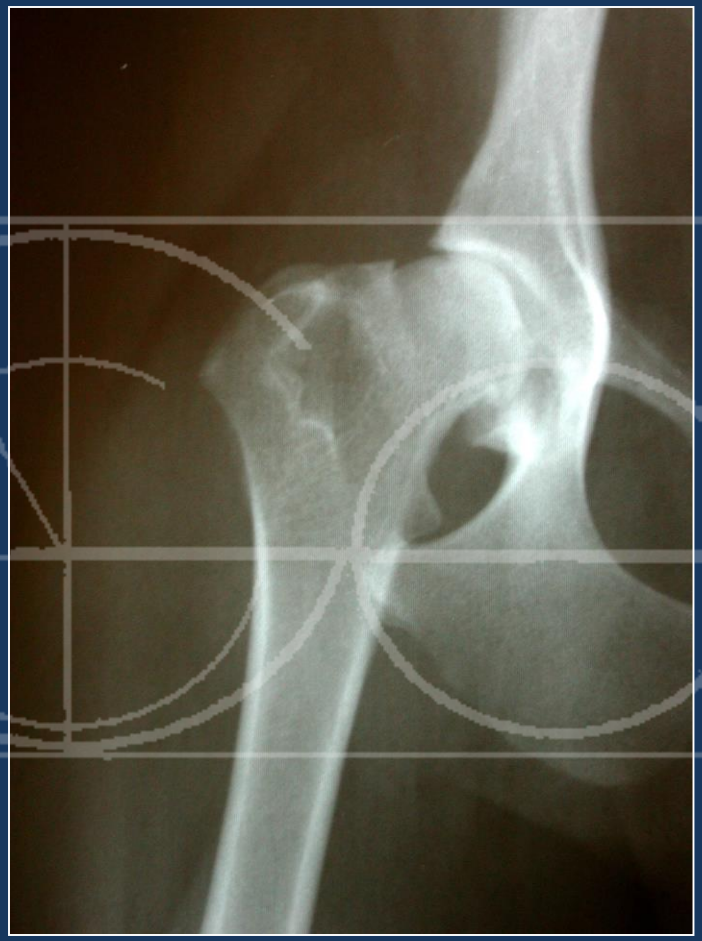
El tratamiento conservador de la osteoartritis se basará en:

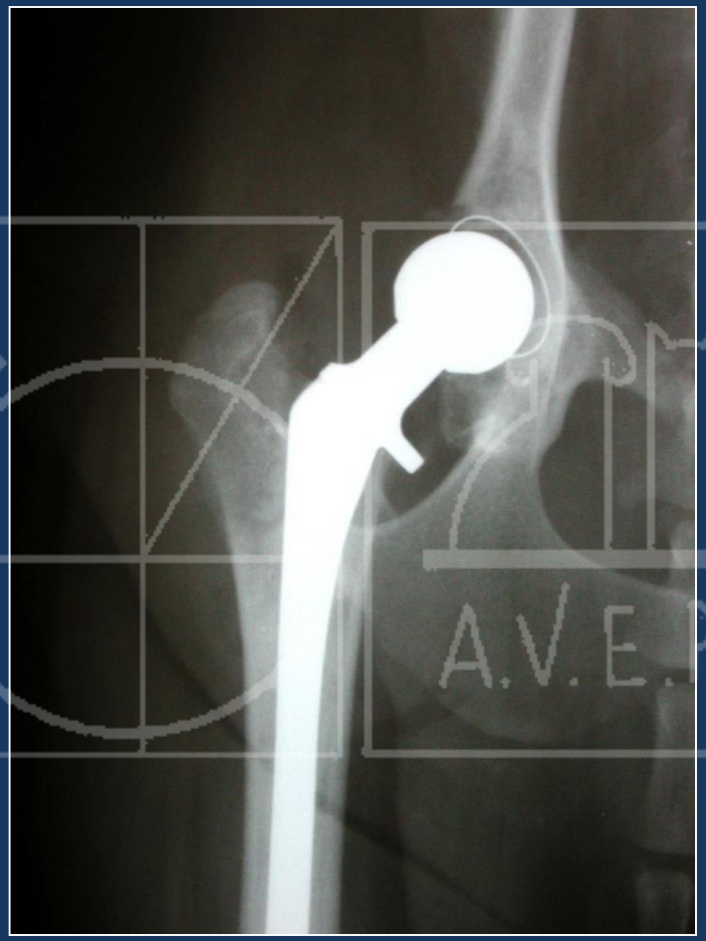
1. Ejercicio controlado
2. Dieta
3. Medicación
4. Fisioterapia



El tratamiento quirúrgico de la osteoartritis se basará en:

1. Reemplazo articular: Prótesis
2. Fusión articular: Artrodesis
3. Escisión de la cabeza femoral

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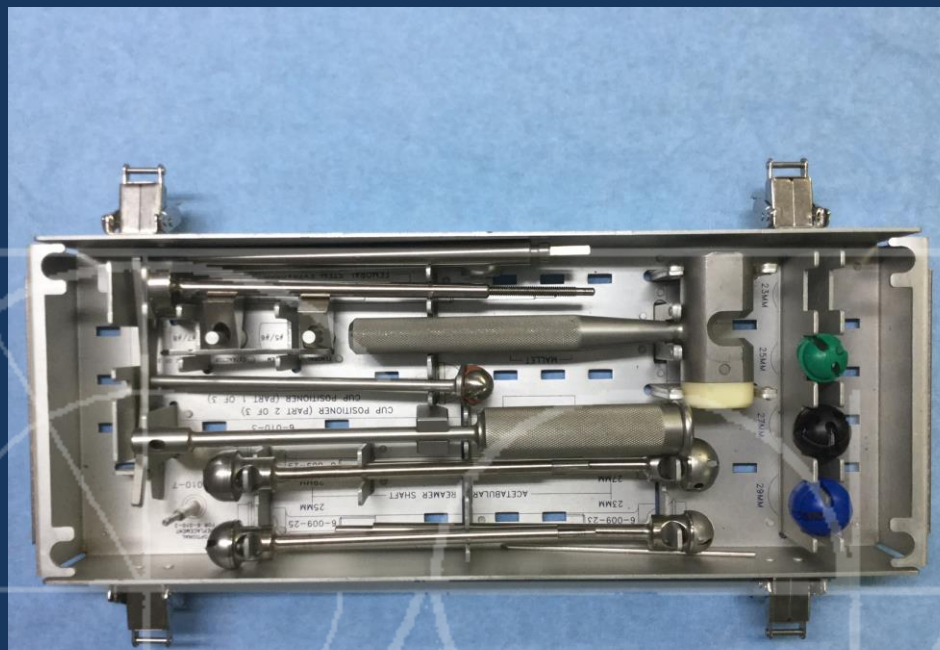
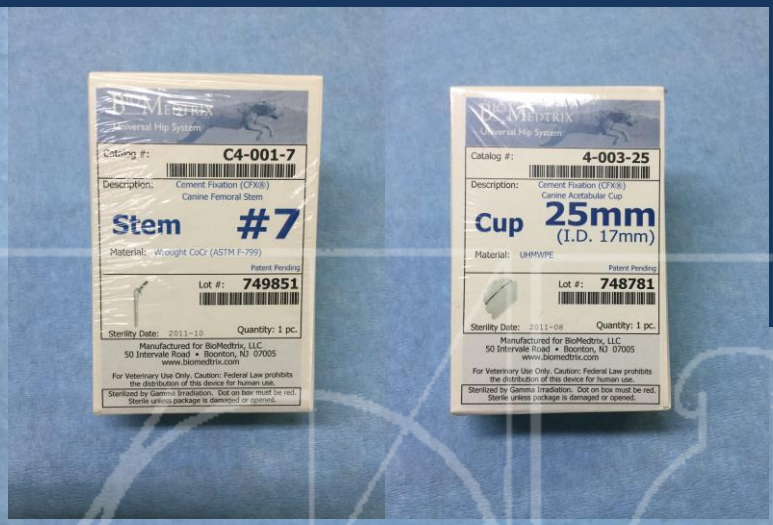
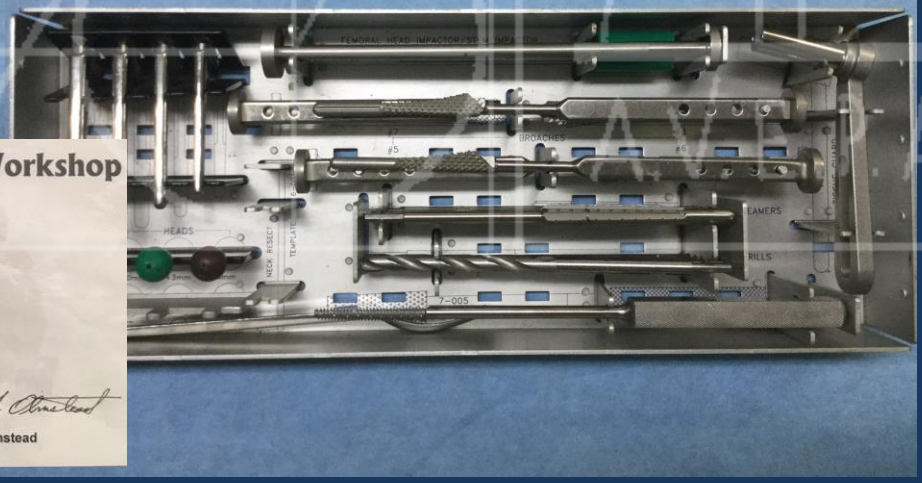






1997

97 Canine Total Hip Replacement Workshop

presented by

The Ohio State University

College of Veterinary Medicine

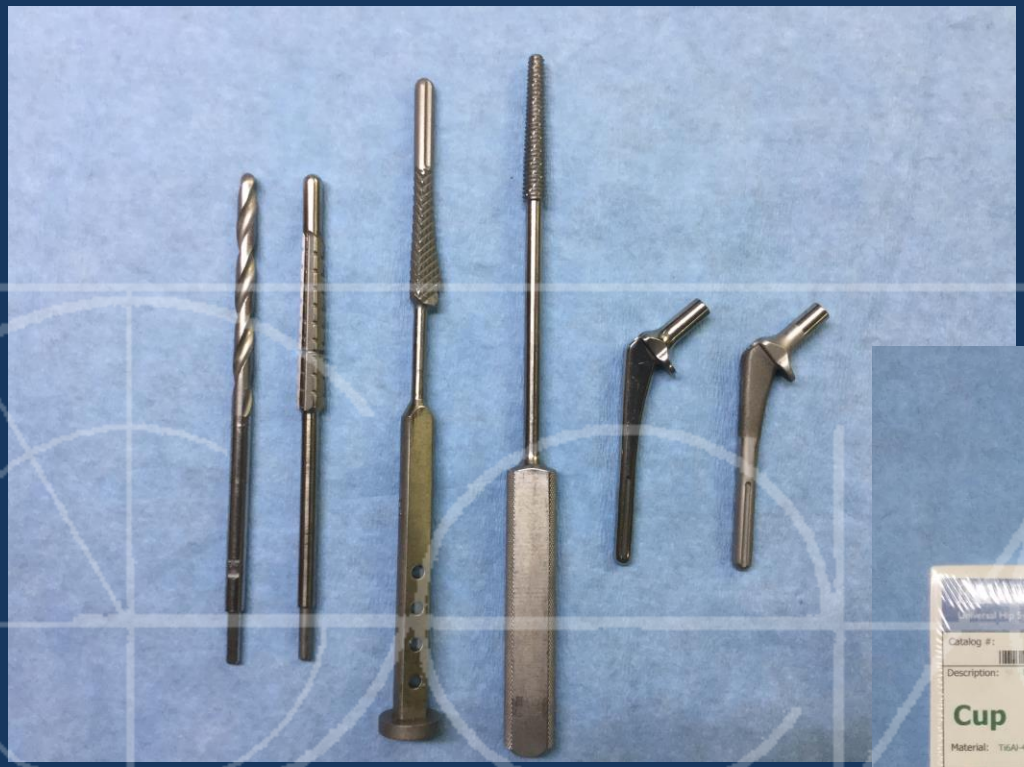
June 20, 1997

CE Credit: 14 Hours

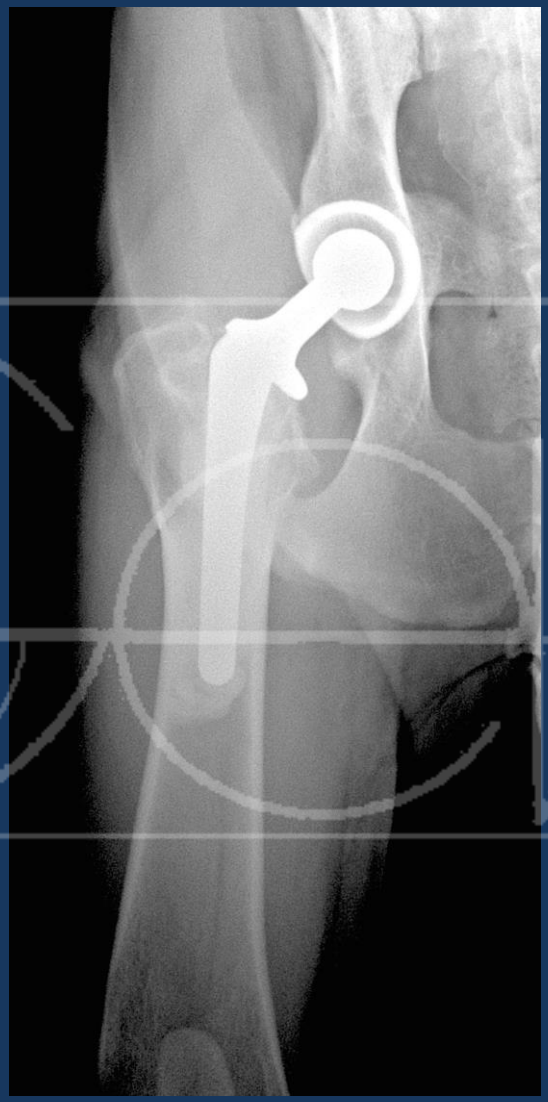
Glen F. Hoffsis, D.V.M.
Dean

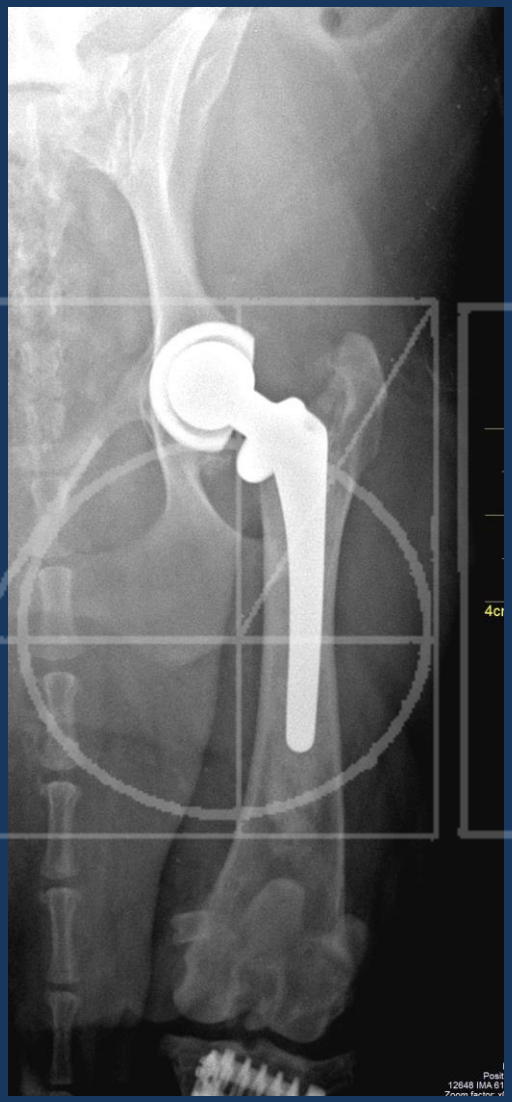
Marvin L. Olmstead
Chairperson

Tomás Fernández González DVM PhD



2011





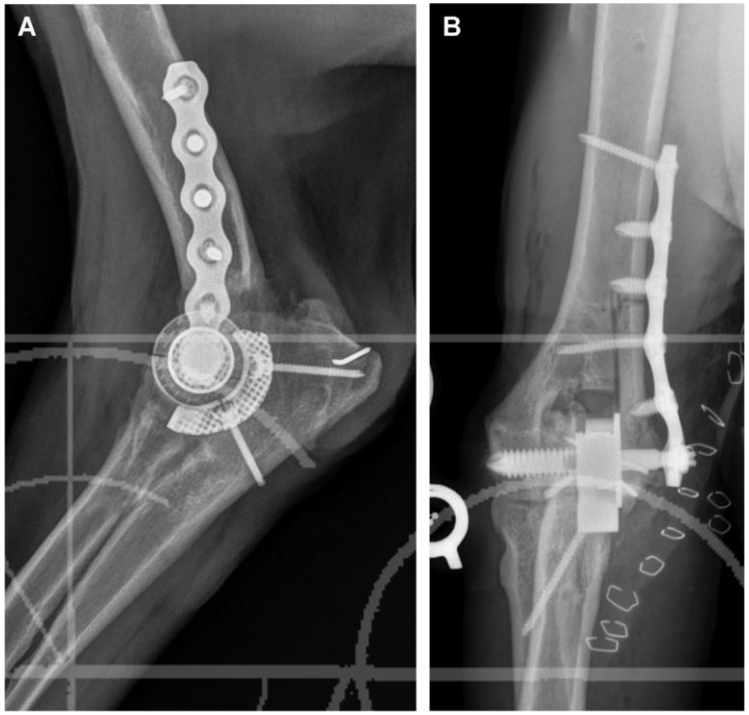


Fig. 27. Immediate postoperative orthogonal view radiographs [*A*] ML view; [*B*] CrCd view of patient with BANC PER. (*Courtesy of* KYON Veterinary Surgical Products, Boston, MA.)

growing interest in TER and PER. Clinical use of a TER first was reported by Whittick and colleagues,[23] who in 1964, used a custom-made spherical hinged prosthesis to treat a gunshot-induced comminuted elbow fracture in a cat. To the authors' knowledge, the first clinical canine TER was implanted in 1989 by Chancrin, who used a prototype cemented hinged prosthesis to treat a Labrador retriever affected with end-stage OA (Chancrin J, personal communication, 2008). Subsequently in 1996, Lewis[24] reported on the first clinical results of a hinged TER implanted in 10 dogs. In these first-generation systems, Chancrin and Lewis used cemented, fully constrained hinged designs (linked systems). Because of the rigid mechanical link between the humeral and RU components, most of the forces across the joint were transmitted through the implant to the cement and its interfaces.[25] The high complication rates encountered with these initial designs quickly led to a paradigm shift to unlinked TER designs. Vasseur (Sidebotham CG, personal communication, 2008), Lewis (second and third generations),[24] Cook,[26] and Conzemius[27,28] developed the first unlinked designs in the late 1990s. All encountered unacceptable postoperative morbidity that led either to termination or further refinement of the respective designs. Following iterations of his earlier designs, Conzemius and colleagues[29,30] reported encouraging results after TER in 6 normal dogs, then 2 years later, in 20 dogs afflicted

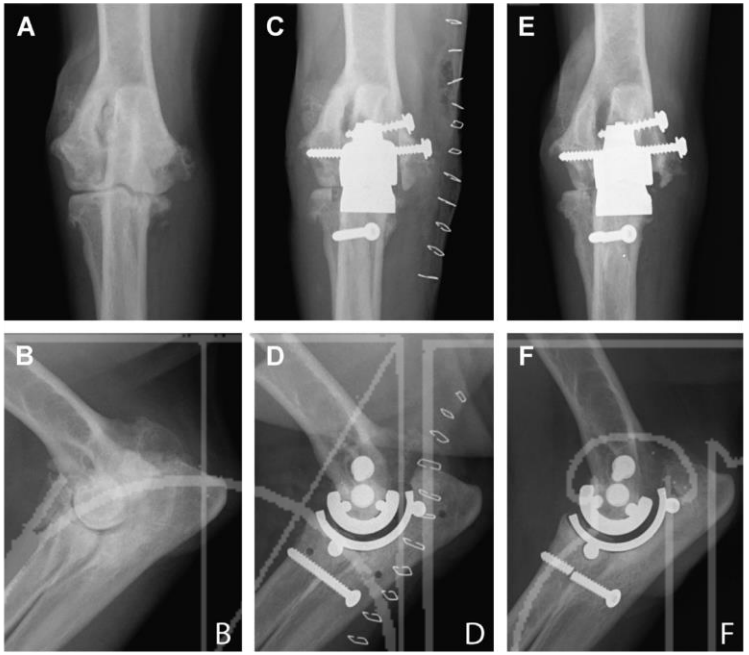


Fig. 29. Preoperative (*A*, *B*) and postoperative (*C*, *D*) radiographs showing proper positioning of a TATE prosthesis as well as the bone implant interface 21 months later (*E*, *F*). The proximal screws are used to stabilize the medial epicondyle. Lagged between the radius and the ulna, the distal screw is used to maintain stability during healing of a surgical RU synostosis. Note the fractured RU screw and the mild local bone resorption around the ulnar post. (*Courtesy of* L. Déjardin, DVM, East Lansing, MI. and BioMedtrix, Whippany, NJ.)

how might the compilation of a few cases from numerous sources affect the interpretation of the findings of the clinical reports?.

What follows is a synthesis of subjective data provided by fellow surgeons who have performed at least 5 procedures. The authors strongly emphasize that this information is anecdotal in nature and, therefore, should be assessed cautiously.

It is estimated that the TATE prosthesis has been implanted in approximately 250 cases worldwide since July 2007. In 2009[38] and then 2010,[39] the authors reported subjective data compiled through feedback from the 6 centers, where more than 5 cases had been performed (total of 73 elbows at the time). Three severe complications, consisting of 2 humeral fractures and 1 implant loosening, were recorded, all within 5 weeks postoperatively (rate of 4%). Of these, 2 cases were associated with secondary infection and 1 case with secondary ulnar fracture. Two cases were euthanized by the referring veterinarian without reevaluation by the primary surgeon, and 1 was amputated because of concomitant deep infection (**Fig. 30**).

Although recent biomechanical studies have demonstrated that implant intrinsic stability is lower in both TATE Elbow generations than in the Iowa State system (first

El tratamiento quirúrgico de la osteoartritis se basará en:

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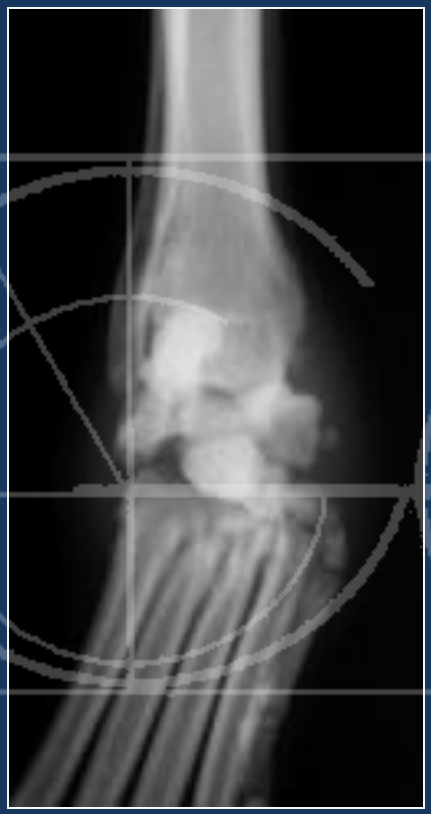
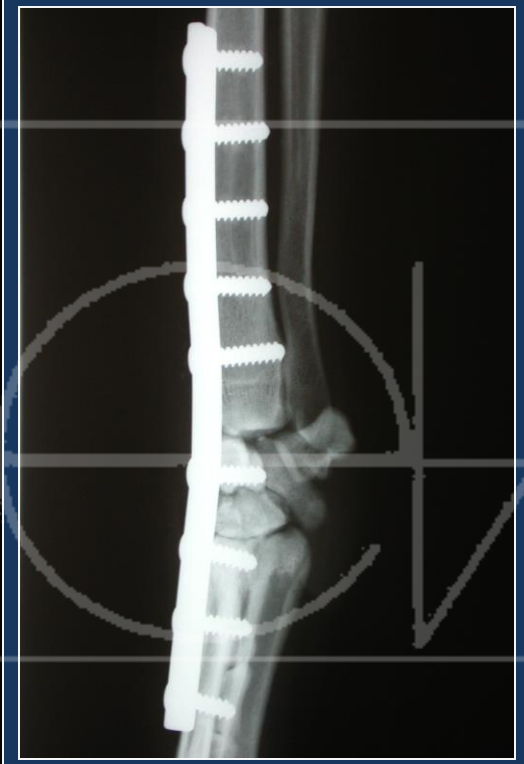
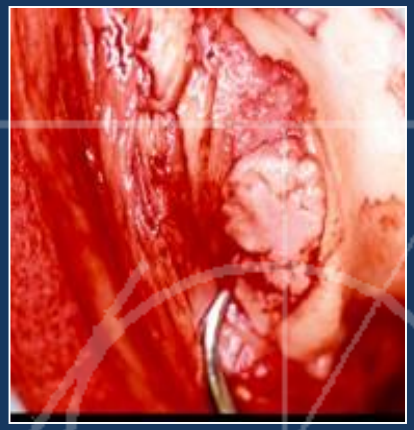
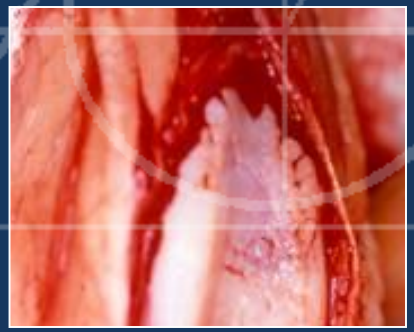
3. Escisión de la cabeza femoral









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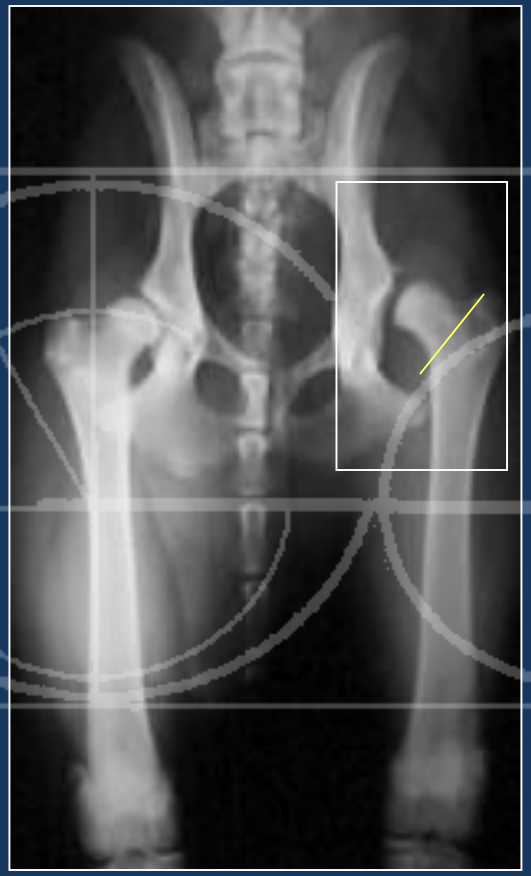

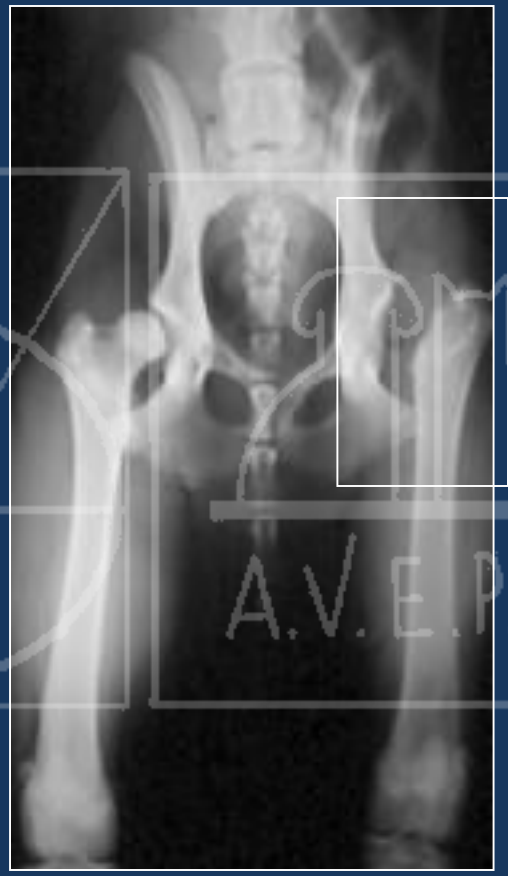


Muchas Gracias
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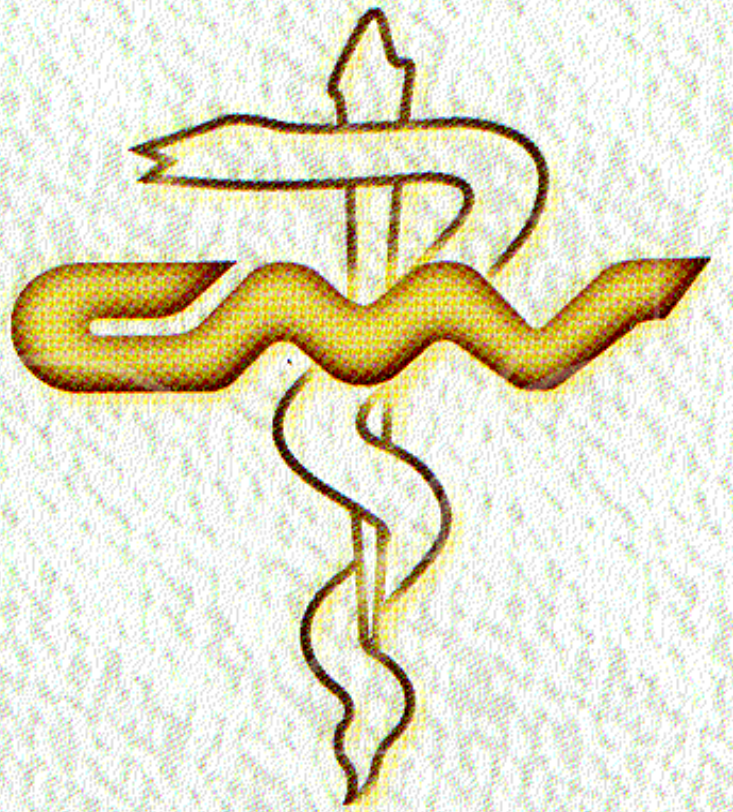

CENTRO MEDICO VETERINARIO