

# THEPROGNOSISOFTHEARTHROSCOPICREMOVALOFOSTEOCHONDRAL FRAGMENTS (OCD,DOF, POF)

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## PREFACE

Ever since the start of my veterinary education, I am passionate about the horse's musculoskeletal system. Therefore, I am very grateful to have gotten the opportunity to write about the arthroscopic removal of osteochondral fragments, a very important topic in the current sport horse industry.

Writing this master's thesis would not have been possible without the guidance of my promotors Prof. Dr. Frederik Pille and Bram Van Mol. Thank you for taking the time to review my work and share your expertise regarding this topic. The whole process of creating this thesis has truly been a wonderful experience and sparked my interest in pursuing a career in research myself.

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# ABBREVIATIONS

DIRT	Distal intermediate ridge of the tibia
DOA	Developmental osteoarticular abnormality
DOD	Developmental orthopedic disease
DOF	Dorsal osteochondral fragment
LTR	Lateral trochlear ridge
MCP/MTP	Metacarpo-/metatarsophalangeal
OC	Osteochondrosis
OCD	Osteochondritis dissecans
P1	First phalanx
POF	Palmar/plantar osteochondral fragment
SB	Standardbreds
SSI	Surgical site infection
WB	Warmbloods

## 1. ABSTRACT

The arthroscopic removal of osteochondral fragments in clinically sound youngsters has become routine practice. One of the arguments for this elective procedure is prevention of clinical symptoms, which can arise once the horse starts training. Other important reasons are the need for clean X-rays and non-acceptance of joint distension in the current equine industry. Most existing studies investigating the outcome of arthroscopic fragment removal report a good prognosis for return to intended use, generally entailing that the horse is sound after surgery. Nevertheless, too little attention has been paid to the prognosis regarding the resolution of joint effusion. To date, no studies investigating the long-term effect of residual effusion on the osteoarticular health of the affected joint exist. However, this factor significantly influences the prognosis of arthroscopic fragment removal in Warmblood sport horses. As these horses have a much longer athletic career than racing breeds, only starting to reach peak performance capacity by the age of 8-10 years, a long-lasting optimal osteoarticular health is crucial in this population.

The research aspect of this master's thesis consists of 3 parts: the creation of a database including information about the orthopaedic examination and arthroscopic report from patients undergoing arthroscopic fragment removal at the faculty clinic between August 2020 and March 2023, the measurement of the volumes of these osteochondral fragments, and the evaluation of the correlation between the degree of joint effusion and the fragment volumes through statistical analysis.

The results of this thesis reveal joint effusion to be a common clinical sign in Warmbloods with OCD of the stifle or hock, but not in horses with fetlock osteochondral fragments. Furthermore, for both the femoropatellar and the tarsocrural joint, a significant positive correlation between the degree of effusion and volume of the fragment(s) was found. This thesis contributes to a larger study where the aim is to categorise OC(D) lesions and investigate the correlation between joint effusion, volume of the fragments and arthroscopic changes in the joint. This will be part of a research project in which the goal is to redefine the prognosis of the arthroscopic removal of osteochondral fragments (OCD, DOF, POF) in the Warmblood sport horse population.

## 2. SAMENVATTING

De artroscopische verwijdering van osteochondrale fragmenten bij klinisch gezonde jonge paarden is routine geworden. Eén van de argumenten voor deze electieve procedure is de preventie van klinische symptomen, welke kunnen ontstaan wanneer het paard begint te trainen. Andere belangrijke redenen zijn de nood aan 'cleane RX'en' en de non-acceptatie van gewrichtsopzetting in de huidige sportpaardenindustrie. De meeste bestaande studies die de uitkomst van de artroscopische verwijdering van fragmenten onderzoeken, rapporteren een goede prognose voor terugkeer naar het gebruiksdoel, wat meestal inhoudt dat het paard niet kreupel is. Desalniettemin, is er te weinig aandacht besteed aan de prognose omtrent het verdwijnen van gewrichtsopzetting op de osteo-articulaire gezondheid van het getroffen gewricht onderzoeken. Deze factor beïnvloedt echter significant de prognose van artroscopische verwijdering van fragmenten een veel langere atletische carrière hebben dan racepaarden en vaak pas maximale atletische capaciteiten bereiken op de leeftijd van 8-10 jaar, is een langdurige optimale osteo-articulaire gezondheid cruciaal in deze populatie.

Het onderzoeksaspect van deze masterproef bestaat uit 3 delen: het opstellen van een database met informatie uit de orthopedische onderzoeken en arthroscopieverslagen van patiënten die artroscopische verwijdering van fragmenten zijn ondergaan op de faculteitskliniek tussen augustus 2020 en maart 2023, het meten van de volumes van deze osteochondrale fragmenten, en de evaluatie van het verband tussen de graad van gewrichtsopzetting en de fragmentvolumes via statistische analyse.

De resultaten van deze masterproef onthullen dat gewrichtsopzetting een veelvoorkomend klinisch symptoom is bij Warmbloeden met OCD van de knie of sprong, maar niet bij paarden met osteochondrale fragmenten in de kogel. Bovendien was er voor zowel het femoropatellaire als het tarsocrurale gewricht, een significant positief verband tussen de graad van gewrichtsopzetting en het fragmentvolume. Deze masterproef draagt bij aan een grotere studie met als doel OC(D) letsels te categoriseren en het verband tussen gewrichtsopzetting, fragmentvolume en arthroscopische veranderingen in het gewricht te onderzoeken. Dit zal deel uitmaken van een onderzoeksproject met als doel de prognose van de artroscopische verwijdering van osteochondrale fragmenten (OCD, DOF, POF) bij Warmbloed sportpaarden te herdefiniëren.

# 3. LITERATURE STUDY

## **3.1. INTRODUCTION**

Lameness is one of the main reasons for the early retirement of sport horses (Seitzinger et al., 2000). Therefore, an optimal osteoarticular health is crucial for a long-lasting, successful athletic career. Consequently, the current equine industry attaches great value to the presales radiographic screening of sport horses. Also radiographic screening of youngsters for developmental orthopaedic diseases (DODs) is routine practice. These screenings can have major consequences for the economic value of the horse since certain radiographic findings can be a cause of lameness and thus could jeopardize their future sporting career. The results of these screenings also largely impact the genetic potential of the horse. As developmental orthopaedic diseases have a genetic background, studbooks do not accept candidate sires with radiographic signs of these disorders for breeding, and they advise the exclusion of broodmares with such radiographic abnormalities.

Osteochondrosis (OC) and fetlock osteochondral fragments are considered to be the most important developmental orthopaedic diseases (DODs) that can be detected during radiographic screening of youngsters. This is especially the case in the Warmblood population in north-western Europe, with studies reporting OC(D) prevalences as high as 44,3% to a staggering 70% combining results of X-ray screening of the femoropatellar, tarsocrural and metacarpo-/metatarsophalyngeal joints (Lepeule et al., 2008; Van Grevenhof et al., 2009; Vos, 2008).

To obtain a horse that is 'clean' on X-rays, meaning it has no (pathological) abnormalities on its radiographs, and to prevent further degenerative joint disease, the arthroscopic removal of these osteochondral fragments, regardless of whether clinical signs are present, has become common practice. The huge effect of DODs on the economics of the equine industry goes beyond the large devaluations and rejections by studbooks. Also the cost of the thousands of preventive surgical interventions performed each year accounts for a substantial part of the economic impact.

## 3.2. DEVELOPMENTAL ORTHOPAEDIC DISORDERS: TERMINOLOGY

The term 'developmental orthopaedic diseases' (DODs) encompasses all disorders of young, growing horses that involve the musculoskeletal system (Lepeule et al., 2008). 'Osteochondrosis' is the disorder in which the endochondral ossification of the articular-epiphyseal complex in the joint(s) is disturbed. Since this is a process that takes place in the young, growing horse, the development of OC is restricted to this period of life (van Weeren, 2019). 'Osteochondritis' is the inflammatory response to OC, and 'osteochondritis dissecans' (OCD) is used in case of cartilaginous or osteochondral separation (Poulos, 1986).

Different types of osteochondral fragments can be found in the metacarpo-/metatarsophalangeal (MCP/MTP) joints. Depending on their location, different aetiologies are assumed. Fragments originating from the sagittal ridge and/or parasagittal condylar regions of the metacarpus/metatarsus are classified as a manifestation of OC(D). Controversy still exists about the origin of fragments originating from the dorso- or palmaro-/plantaroproximal margin of the proximal phalanx (P1), often referred to as dorsoproximal osteochondral fragments (DOFs) or palmaro-/plantaroproximal osteochondral fragments (POFs). Formerly, these fragments were also believed to be a manifestation of OC(D). However, based on their histologic appearance inconsistent with osteochondrosis, they are now considered to be a form of developmental orthopaedic disease other than OC(D). Other types of fragments that can be detected in the MCP/MTP joints during radiographic screening of youngsters are those occurring in the dorsoproximal synovial pad (of which the origin still remains unclear) and those originating from fractures of the proximal sesamoid bones (due to trauma). An ununited palmaro-/plantaroproximal tuberosity of P1 is also possible but not very common ( Declercq et al., 2011).

This thesis will focus on the most important types of osteochondral fragments encountered in the current Warmblood population in North-Western Europe: OCD, DOF and POF.

## **3.3. PREVALENCE**

During the past years, many studies on the prevalence of OC have been conducted. Their results, however, must be interpreted with caution. Because of the dynamic character of OC and the different time windows during which lesions develop in different joints (this concept will be explained in a subsequent section), studies performed before the age of 12 months are not representative of a stable condition. Results from the age of 3-4 years, for screening by studbooks, may lead to underestimations due to preselection by breeders or spontaneous resolving of lesions at foal age. Even though unreliable and underestimated, the reported prevalences of OC are spectacular and mark the important breed differences (van Weeren, 2019).

OC is described in almost every diarthrodial joint but occurs most often in the tarsocrural, femoropatellar, and metacarpo-/metatarsophalangeal (MCP/MTP) joints (Ortved, 2017; van Weeren, 2019). Less frequently affected joints are the carpal joint, elbow joint, shoulder joint and the cervical articular facet joints (van Weeren & Barneveld, 1999). In Warmbloods and Standardbreds, tarsocrural OC is most frequent (Carlsten et al., 1993; Hoppe, 1984), whereas in racing Thoroughbreds femoropatellar OC is predominant (McIntosh and McIlwraith, 1993).

Any breed of horse can develop osteochondrosis; however, the reported incidence is greatest in Standardbreds (10.5%–35%), Thoroughbreds (20%), and Warmbloods (20%) (Alvarado et al., 1990; Hoppe and Philipsson, 1985; Lepeule et al., 2009; O'Donohue et al., 1992; Sandgren et al., 1993).

#### **3.4. PATHOGENESIS**

The endochondral ossification of the cartilaginous primordial skeleton starts during early foetal development and continues after birth at the physes of long bones, facilitating longitudinal growth (Ytrehus et al., 2007). During this process, chondrocytes of the epiphyseal growth cartilage through stages of proliferation, progress apoptosis calcification. hypertrophy, and Subsequently, blood vessels invade into the calcified cartilage, where chondroclasts remove cartilage after which osteoblasts deposit new bone (primary spongiosa) (figure 1). This is then remodelled into bony trabeculae (secondary spongiosa) to form the subchondral bone underlying the articular cartilage, a shaping process that continues into adulthood. The cartilage canals housing the blood vessels that supply nutrition to the deep cartilage, progress through chondrification and convert into bone along with the surrounding cartilage as the ossification front moves further toward the articular surface (Hurtig and Pool, 1996; Laverty and Girard, 2013; Semevolos, 2017).



Figure 1: Growth plate cartilage from a 3-day-old foal (hematoxylin). B. primary spongiosa; C.calcified cartilage zone; H. hypertrophic zone; P. proliferative zone; R. resting zone [H&E,original magnification x100] (From: Semevolos, 2017)

Development of osteochondrosis can occur at any location, but the most common site of clinical OC in horses is the articular-epiphyseal cartilage complex (i.e. the articular cartilage and underlying epiphyseal growth cartilage) (Hurtig and Pool, 1996; Laverty and Girard, 2013; Semevolos, 2017).

Different mechanisms have been proposed to play a role in the pathogenesis of osteochondrosis. One of the major factors in the development of OC is the obliteration of cartilage canals and the consequential premature disruption of the vascular supply to the epiphyseal growth cartilage. This leads to ischemia and chondrocyte death followed by cartilage matrix degeneration, ultimately resulting in focal areas of chondronecrosis and failure of the endochondral ossification in those regions (Laverty and Girard, 2013; Semevolos, 2017). This theory is supported by different studies. A study investigating ischemic necrosis of cartilage in pigs observed areas of chondronecrosis associated with such obliterated cartilage canals in spontaneous OC lesions. Furthermore, after experimental disruption of blood supply to the deep cartilage, similar areas of cartilage retention were present (Carlson et al., 1991). A more recent study investigating the effect of experimental devascularization of deep cartilage of the lateral trochlear ridge of the femur of foals also observed the development of areas of chondronecrosis, delayed endochondral ossification and OC lesions (Olstad et al., 2013).

Another important factor in the pathogenesis of OC is shearing of the osteochondral junction under the influence of biomechanical forces. Regions of high shear stress or impact are predisposed, and indeed these often correlate to the typical predilection sites for OC lesions. Primary separation along the osteochondral junction, a naturally biomechanically weak region, can be observed in foals, especially at 4 months of age (figure 2). Furthermore, shearing can occur as a secondary event after weakening of the cartilage matrix or obliteration of cartilage canals and subsequent chondronecrosis (Semevolos, 2017).



Figure 2: Photomicrograph of articular epiphyseal cartilage in a 4-month-old foal having separation along the osteochondral junction (arrow) [H&E, original magnification x40] (From: Semevolos, 2017)

Molecular alterations within the cartilage matrix and/or cell signalling pathways that occur during the endochondral ossification also play a role in the development of OC (Semevolos, 2017). Studies report changes in type II collagen synthesis (Lecocq et al., 2008) and gene expression of multiple growth factors and paracrine factors in chondrocytes, as well as increased expression of matrix metalloproteinases (Riddick et al., 2012). These alterations could all be responsible for weakening of the cartilage matrix near cartilage canals and along the osteochondral junction, making those areas more susceptible to injury under biomechanical stress. A dysregulation of growth factor and/or paracrine factor expression may also directly delay endochondral ossification. Dysfunction of signalling pathways regulating chondrocyte differentiation during cartilage maturation and endochondral ossification have been reported as well (Kinsley et al., 2015; Mirams et al., 2009; Semevolos et al., 2002). Additionally, dysfunction of mitochondria and endoplasmic reticulum stress have also been suggested as factors in the pathogenesis of OC (Desjardin et al., 2014).



Figure 3: Chronic histologic lesion of OC in pig. The AECC is visible. A focus of necrotic growth cartilage is present in the upper epiphysis and is partially surrounded by granulation tissue and bone. Numerous clusters of chondrocytes are present at the junction of the normal and necrotic cartilage. HPS staining (hematoxylin, phloxin and safranin) (From: Laverty and Girard, 2013)

Due to focal failure of the endochondral ossification, an area of necrotic growth cartilage will persist (figure 3). In a further stage, formation of a fissure extending from this site through the articular cartilage can occur. This may lead to separation of a cartilaginous, and later osteochondral, flap or fragment from the subchondral bone (Laverty and Girard, 2013; Semevolos, 2017). These can become completely detached, free-floating in the joint as true OCD fragments, also sometimes referred to as joint mouses (Ortved, 2017).

However, osteochondrosis has a dynamic character, meaning that early OC lesions can still regress or even heal completely in young animals (Dik et al., 1999). In case of resolution, the retained cartilage will still undergo endochondral ossification, resulting in a normal subchondral bone-articular cartilage interface. Nevertheless, healing is only possible during the early juvenile phase as the healing capacity of cartilage rapidly declines from the start of puberty at 6-12 months (Semevolos, 2017).

Some uncertainty still exists about the exact mechanisms involved in the pathogenesis of dorsoproximal and palmaro-/plantaroproximal osteochondral fragments originating from the proximal border of the first phalanx. Fragments at the dorsoproximal border of P1 diagnosed in racing Thoroughbreds and Standardbreds are considered to be chip fractures resulting from compression against the distal dorsal metacarpal/-tarsal bone during hyperextension of the MCP/MTP joint during racing. They are most often associated with lameness, pain upon palpation and effusion of the affected joint (Declercq et al., 2011). However, dorsoproximal osteochondral fragments are also diagnosed in yearling Thoroughbreds or Standardbreds (pre-training) and Warmblood youngsters. In these horses, DOFs are considered to be a manifestation of developmental orthopaedic disease other than OC(D), as histopathologic evaluation does not show the typical characteristics of osteochondrosis (cartilage retention) nor of a healing/reactive fracture (Declercq et al., 2009). Fragments at the palmaro-/ plantaroproximal border of P1 are assumed to be of traumatic origin rather than resulting from a developmental disorder. Morphologic and histologic examination of plantaroproximal osteochondral fragments is inconsistent with osteochondrosis as well and suggests that these are fractures that developed in the early postnatal period due to trauma to normal developing cartilage (and subchondral bone). Studies investigating POFs found that underlying lesions do not involve the articular-epiphyseal cartilage complex. Furthermore, these fragments consist of bone and fibrocartilage and are often adhered to the short sesamoidean ligaments. As POFs occur mostly at the medial side of the MTP joint, it is theorized that they are the result of an outwardly rotated hindlimb axis and subsequent point loading of the medial aspect of the MTP joint. High tensile stress on the medial short sesamoidean ligaments may cause ruptures at their site of attachment where osteogenic tissue can be brought into the ligament, later leading to the formation of osteochondral fragments (Dalin et al., 1993; Declercq et al., 2011; Nixon and Pool, 1995).

## 3.5. AETIOLOGY

The typical clinical signs and histological expression of OC are well-described by now, but the underlying predisposing factors of this multifactorial disease are still not completely clear. While genetics are undoubtedly involved, a substantial part of the aetiology is presumed to be environmental. Several factors influencing the development of OC and the balance between healing or progressing into OCD of early lesions have been proposed (Ortved, 2017; Semevolos, 2017).

The role of trauma and biomechanical forces is reflected by the consistent predilection sites of OC lesions in the specific joints (McIlwraith, 1993). Excessive/normal forces on respectively normal/abnormal tissues can cause disruption of vascular supply in cartilage going through endochondral ossification or may lead to tearing of cartilage flaps (Pool, 1993; Whitton, 1998). Other factors influencing biomechanical forces within joints are body size and conformation (Ortved, 2017). In 1993, Sandgren et al. discovered a significantly higher incidence of tarsocrural osteochondrosis in Standardbred foals with externally rotated tarsi than in those with a normal conformation. And as stated before, external hindlimb rotation also predisposes horses for the development of POFs in the MTP joints (Dalin et al., 1993; Nixon and Pool, 1995).

Although differing opinions exist about the importance of exercise, a general consensus exists that moderate exercise during early development is crucial for normal functional adaptation of the developing equine musculoskeletal system (Ortved, 2017). However, excessive exercise at a young age can lead to concentration of shear forces along the osteochondral junction, resulting in fissures of an abnormal cartilage matrix. Also a lack of exercise and most importantly irregular exercise has a negative impact on the development and progression of OC lesions (Barneveld & van Weeren, 1999; Semevolos, 2017; van Weeren & Denoix, 2013). In yearlings, intrinsic trauma from peak strains occurring at certain articular sites is the most important influence determining the osteochondral status. Pastures with rough surfaces and turn-out at daytime only have been identified as risk factors in this age group (J. Lepeule et al., 2009; Praud et al., 2013; van Weeren and Denoix, 2013).

Nutrition, specifically mineral concentration and digestible energy, can also contribute to the development of osteochondrosis. Because of the need for copper as a cofactor in lysyl oxidase, required for collagen cross-linking, copper deficiency was initially believed to lead to OC (Ortved, 2017). However, in 2003, a study by Weeren et al. investigating liver copper status of mare and newborn foal, found adequate copper levels to be beneficial for repair of OC lesions but not related to the pathogenesis. In regards to digestible energy, there is still discussion ongoing whether it is the fast growth rate that comes with it, or the high-energy feed itself, that leads to the development of osteochondrosis (van Weeren and Denoix, 2013). In 1993, Savage et al. discovered an increased prevalence of OC in foals fed high-energy diets. However, a few years later in 2006, Donabédian et al. observed an increase in OC lesions in foals with a fast growth rate, regardless of their diet. This indicates that a high growth rate is indeed a risk factor for the development of OC, regardless if it is caused by a higher food intake or if it is a characteristic of the individual animal. Interestingly, Vander Heyden et al. (2013) observed foals from mares receiving concentrates during gestation to be more likely to develop OC. Another recent study evaluating the impact of management factors on the development and evolution of OC lesions observed a higher probability of healing of early lesions in horses that did not receive concentrates in their diet (Mendoza et al., 2016). These findings suggest that high energy feeding does in fact play a role in the development of OC.

Although its share in the aetiology of osteochondrosis might be smaller than previously assumed, the genetic profile of the horse is also an important predisposing factor. A general consensus exists about osteochondrosis being a polygenic trait with complex inheritance (Distl, 2013; Hilla & Distl, 2014). Heritability estimates for osteochondrosis in Warmbloods have been described, ranging from 0.02 to 0.09 for the femoropatellar joints, from 0.19 to 0.37 for the tarsocrural joints and from 0.06 to 0.32 for the MCP/MTP joints (Distl, 2013). A variety of quantitative trait loci and candidate genes have been reported across breeds. Furthermore, OC(D) occurring in different joints, and even specific predilection sites within these joints, should be treated as distinct traits (Distl, 2013; Hilla & Distl, 2014). A recent study used genome-wide association analyses to identify specific genetic loci linked to osteochondrosis in Belgian Warmbloods. Single nucleotide polymorphisms on 3 chromosomes were significantly associated with the presence of OC at any joint (stifle and/or hock). Investigation of these regions revealed candidate genes related to cell differentiation and chondrocyte development (Drabbe et al., 2022).

The aetiology of dorsoproximal osteochondral fragments is much less extensively investigated to date. However, since DOFs are also considered to be form of a developmental orthopaedic disorder, similar factors as in osteochondrosis are presumed to be at cause. For palmaro-/plantaroproximal osteochondral fragments, biomechanical forces and trauma are the most important factors playing a role in the pathogenesis (Declercq et al., 2011).

#### 3.6. DIFFERENT WINDOWS-OF-TIME

This concept has first been established in 1999 in the EXOC-study (for exercise and osteochondrosis), evaluating the influence of exercise at an early age on the development of OC. In this large experimental study by van Weeren and Barneveld, the tarsocrural and femoropatellar joints of a group of Warmblood foals were radiographed on a monthly basis from 1 until 5 or 11 months of age. The results showed a possible spontaneous repair in not only small but also radiographically visible larger lesions and even fragments (Dik et al., 1999).



Figure 4: A. Schematic diagram of the early development of osteochondral lesions at the distal intermediate ridge of the tibia. At 1 month of age, there are several lesions that will heal. Only a few lesions originate after the age of 1 month, and at the age of 5 months, the situation remains stable. B. Schematic diagram of the early development of osteochondral lesions at the distal aspect of the lateral trochlear ridge of the talus. The same general pattern as in A is seen, but healing potential is better. C. Schematic diagram of the early development of osteochondral lesions of the lateral ridge of the femoral trochlea. The pattern is distinct from that of the hock. Lesions develop only after the age of 3 months, peak at about 6 months, and have resolved at the age of 8 months, although some lesions will remain (From: Dik et al., 1999) The ages at which lesions originated and before which they could still heal varied for each joint. In the femoropatellar joint, lesions on the lateral trochlear ridge (LTR) of the femur started to form from 3 months onward, with a peak at 6 months. Most lesions healed or reached a stable condition by approximately 8 months of age. In the tarsocrural joint, where the focus was laid on the distal intermediate ridge of the tibia (DIRT) and the LTR of the talus, epiphyseal maturation occurs earlier. In these sites most lesions had resolved at 5 months of age, and spontaneous healing of remaining lesions was unlikely after this age (figure 4) (Dik et al., 1999).

Some variation in timing and breed differences can be present. In a study in Lusitano foals, more lesions in the femoropatellar joint were found at 1 month of age (Baccarin et al., 2012). Additionally, the 'age of no return' was determined at 12 months, a safer age to consider for all joints after which no major OC lesions should be formed nor will resolve (van Weeren, 2019). Remarkably, the aforementioned time windows correspond with the age of cartilage blood supply loss, which is at 7 months of age for the distal aspect of the femur (Carlson et al., 1995) and 5 months for the distal aspect of the tibia (Olstad et al., 2008). These findings indicate the role of cartilage blood supply in the resolution of OC lesions (Jacquet et al., 2013).

A more recent longitudinal study investigating the evolution of radiological findings detected in the limbs of 321 young horses between the ages of 6 and 18 months observed similar findings. This study also included the MCP and MTP joints, where osteochondral fragments present at 6 months of age did not show a uniform trend in evolution. Furthermore, a large number of new lesions appeared between the ages of 6 and 18 months (Jacquet et al., 2013). Therefore, the 'age of no return' is presumed to be later for the MCP/MTP joints.

## **3.7. PREDILECTION SITES**

OC lesions occur at typical predilection sites and it is possible for more than one site to be affected in the same joint (McIlwraith, 2013). In the femoropatellar joint, the lateral trochlear ridge of the femur is most commonly involved (Ortved, 2017; van Weeren, 2019). Other possible sites are the medial trochlear ridge, trochlear groove and, less frequently, the articular surface of the patella (Bourzac et al., 2009). In the tarsocrural joint, the cranial end of the distal intermediate ridge of the tibia is most frequently affected, followed by the distal end of the lateral trochlear ridge of the talus and the medial malleolus of the distal tibia (McIlwraith et al., 1991).

As stated before, a variety of fragments can be found in the MCP/MTP joints. This thesis will focus on the most important fetlock osteochondral fragments: OCD, DOF and POF. OCD fragments typically originate from the dorsal aspect of the sagittal ridge, sometimes extending to the adjacent condyles of the third metacarpal/-tarsal bones. Dorsoproximal osteochondral fragments originate from the dorsoproximal border of the first phalanx and mostly occur at the medial side. Palmaro-/ plantaroproximal osteochondral fragments originate from the first phalanx, they are also most frequently found on the medial side and occur significantly more in the hindlimbs which is probably due to hindlimb external rotation and consequent loading of the medial aspect of the MTP joints (Dalin et al., 1993; Declercq et al., 2011).

Most OC lesions turn out to be bilateral in the tarsocrural and femoropatellar joints and osteochondral fragments often occur bilateral or even quadrilateral in the MCP/MTP joints upon further examination. For this reason, examination of the contralateral joint is advised in case of clinical signs. Screening of other joints is in theory not routinely needed, since concomitant occurrence in other joints is much less common, possibly because of the different 'windows of time' previously mentioned (van Weeren, 2019) and different genetic predisposition depending on the breed and affected joint (Distl, 2013). Nevertheless, it is routine practice to perform a complete radiographic screening of all predisposed joints, facilitating the arthroscopic removal of all fragments in one surgery. This prevents the horse from being put under general anaesthesia multiple times, minimising the risk for complications and lowering the costs for the owner.

#### 3.8. CLINICAL SIGNS

Although OCD lesions and fetlock osteochondral fragments are mostly detected during routine radiographic screenings of asymptomatic youngsters, some horses do present clinical signs. The disorder often becomes clinically evident once the joints become more biomechanically stressed during athletic activity. Nevertheless, large or unstable lesions might already provoke symptoms at a younger age. Foals can also present with transient clinical signs during the time windows when lesions can still evolve. Osteochondrosis can also be diagnosed in older horses, nevertheless, lesions will have been present from foal age (Ortved, 2017; van Weeren, 2019).

The most common clinical sign is effusion of a varying degree depending on the location and extent of the lesion(s) and the joint affected. The synovitis that leads to an increase in synovial fluid can be induced by the debris from the cartilage flap that is released into the joint when lesions form (McIlwraith, 2013). Synovial inflammation can also arise at a later stage due to intra-articular damage caused by the presence of the osteochondral fragments when the joint is put under more biomechanical stress (McIlwraith, 2016). Lameness is rare and if present generally mild, however, it can be severe in young foals with extensive lesions in the femoropatellar joints (Ortved, 2017; van Weeren, 2019).

Approximately 60% of horses with OC in the femoropatellar joint(s) develop clinical signs at less than 1 year of age. Femoropatellar OC patients generally present with a sudden onset of joint effusion (figure 5) and a variable degree of lameness, stiffness, a shortened stride and/or uneven gait. An increase in the exercise level during the preceding weeks could contribute to the clinical manifestation of the disorder (McIlwraith, 2013; Ortved, 2017). The earlier the condition becomes symptomatic, the more severe the changes within the joints can be expected. In some cases, radiographic lesions are found during X-ray screening of an older horse with no clinical signs (Foland et al., 1992).

The most common clinical sign of tarsocrural OC is joint effusion, which can be marked (figure 6). It was the presenting clinical sign in 86,1% of tarsocrural joints in a study evaluating the outcome of arthroscopic fragment removal (McIlwraith et al., 1991). Not correlated with this effusion, a variable degree of lameness (usually mild, except for large lesions of the LTR of the talus), can be observed (McIlwraith, 2013; Ortved, 2017). Gait abnormalities oftentimes arise or become more prevalent once the horse starts its athletic career (McIlwraith et al., 1991). Nonetheless, in the Warmblood population, most tarsocrural OC patients get presented as yearlings prior to training (McIlwraith, 2013).



Figure 5: Severe effusion of the femoropatellar joint (Source: Bram Van Mol)



Figure 6: Severe effusion of the tarsocrural joint (Source: Bram Van Mol)

Horses affected with OCD of the sagittal ridge of the metacarpus/-tarsus are often asymptomatic. Effusion of the MCP/MTP joint is the most common clinical sign and the horse is often not lame, unless lesions are large or unstable. Flexion of the affected fetlock joint will provoke lameness in most patients, especially if joint effusion is present (Declercq et al., 2011; McIlwraith, 2013). Dorsoproximal osteochondral fragments are often detected during radiographic screening of clinically sound horses as well, nevertheless some patients might present with effusion of the affected joint and lameness of a variable degree. Palmaro-/plantaroproximal osteochondral fragments are also generally found in horses without clinical abnormalities. In racing breeds POFs can be related to lameness of a varying degree during training at high speed (Declercq et al., 2011).

#### 3.9. DIAGNOSIS

As stated before, radiographic OC screening in young horses is a well-established concept in the modern horse world. In Warmbloods, screening of the most affected joints typically happens at the age of 1-2 years but is in any case possible from 12 months of age, since OC lesions still detectable after this age are not likely to resolve spontaneously. In recent years, with the upcoming of polydioxanone pinning for large femoropatellar OC fragments, an extra screening moment at the age of 6 months was introduced. Early detection and application of this relatively new surgical technique can still facilitate reattachment and resolution of these OC lesions in the stifle (Nixon et al., 2004; Sparks et al., 2011).

The gold standard for diagnosing OC is radiographic examination. In general, findings can vary from the clear presence of one or more fragments to less severe signs, such as minor irregularities in or only a flattening of the articular contour of the subchondral bone (van Weeren, 2019). Radiographic signs of joint effusion like soft tissue swelling can also be observed.

Table 1 contains the radiographic views that are generally used during OC screening of youngsters to highlight the most common predilection sites:

Joint	Radiographic view	Predilection site(s)
Stifle	Caudo60°lateral-craniomedial oblique (Cd60L-CrMO)	Lateral trochlear ridge of the femur
	Extra: cranioproximal-craniodistal	(Articular surface of) patella, trochlear ridges and
	oblique (CrPr-CrDiO) (skyline)	intertrochlear groove of the femur
	Extra: flexed lateromedial (LM)	(Articular surface of) patella, proximal aspect of trochlear ridges of the femur
Hock	Lateromedial (LM)	Distal intermediate ridge of the tibia
	Plantaro45°lateral-dorsomedial	Distal intermediate ridge of the tibia, lateral
	oblique (Pl45L-DMO) or	trochlear ridge of the talus
	dorso45° medial-plantarolateral	
	oblique (D45M-PILO)	
	Dorso45°lateral-plantaromedial	Medial malleolus
	oblique (D45L-PIMO)	
	Variant: dorso30°lateral-	Preferred view for medial malleolus because no
	plantaromedial oblique (D30L- PIMO)	overlap of medial trochlear ridge of the talus
Fetlock	Lateromedial (LM)	Sagittal ridge of MCPIII/MTPIII (OC)
		Proximal aspect of P1 (DOF/POF)
	Extra: flexed lateromedial (LM)	Distal aspect sagittal ridge of the MCP/MTP (OC)
	Extra: dorso45°medial-	To determine exact location of fragment(s)
	palmaro-/plantarolateral oblique	
	(D45M-Pa/PILO) and/or	
	dorso45°lateral-	
	palmaro-/plantaromedial oblique	
	(D45L-Pa/PIMO)	

Table 1: Radiographic views that highlight the most common predilection sites.

For the femoropatellar joint, a caudo60°lateralcraniomedial oblique (Cd60L-CrMO) view is preferred since this highlights the lateral trochlear ridge of the femur and provides the most useful information regarding size and location of the OC lesions (figure 7). Radiographic signs of OC vary from flattening or irregularity of the LTR to deep contour defects with or without the presence of osteochondral fragments (Butler et al., 2000; McIlwraith, 2013).



Figure 7: Cd60L-CrMO view of stifle showing a large OCD lesion of the lateral trochlear ridge of the femur (Source: Medical Imaging Department, Faculty of Veterinary Medicine UGent)

For a complete evaluation of the tarsocrural joint, different radiographic views are necessary. A general radiographic screening consists of the 4 standard views. This includes the lateromedial (LM) and dorsoplantar (DPI) view, however, the oblique views are the most important and enable the best visualization of the tarsocrural predilection sites. The plantaro45°lateral-dorsomedial oblique (Pl45L-DMO) or dorso45°medial-plantarolateral oblique (D45M-PILO) view highlights the distal intermediate ridge of the tibia and the lateral trochlear ridge of the talus (figure 8 and 9). The dorso45°lateral-plantaromedial oblique (D45L-PIMO) view highlights the medial malleolus. Nevertheless, for evaluation of the medial malleolus the dorso30°lateral-plantaromedial oblique (D30L-PIMO) view is often preferred because there is no overlap of the medial malleolus with the medial trochlear ridge of the talus (figure 10) (McIlwraith, 2013; Ortved, 2017). OC lesions in the tarsocrural joint range from subtle irregularities to large defects containing fragments (Hoppe, 1984).



Figure 8: D45M-PILO view of tarsus showing an OCD lesion of the distal intermediate ridge of the tibia (Source: Medical Imaging Department, Faculty of Veterinary Medicine, UGent)



Figure 9: D45M-PILO view of tarsus showing an OCD lesion of the lateral trochlear ridge of the talus (Source: Medical Imaging Department, Faculty of Veterinary Medicine, UGent)



Figure 10: D30L-PIMO view of tarsus showing an OCD lesion of the medial malleolus (Source: Medical Imaging Department, Faculty of Veterinary Medicine, UGent)

A lateromedial (LM) view is the most common radiographic projection used for screening of the MCP/MTP joints for osteochondral fragments (figure 11). A flexed LM can help visualize the distal part of the dorsal sagittal ridge of the third metacarpal/-tarsal bone (Ortved, 2017). Additional oblique projections, such as the dorso45°medial-palmaro-/plantarolateral oblique (D45M-Pa/PILO) and dorso45°lateral-palmaro-/plantaromedial oblique (D45L-Pa/PIMO) view, and ultrasonography are often necessary to determine the exact location of the fragments.

Three types of sagittal ridge osteochondrosis have been described (McIlwraith & Vorhees, 1990):

- Type I: flattening or defect of the sagittal ridge
- Type II: discrete fragmentation within area of flattening
- Type III: flattening with or without a discrete fragmentation and loose fragments within the joint



Figure 11: LM view of fetlock showing OCD lesion of the sagittal ridge of the metacarpus/ metatarsus (Source: Medical Imaging Department, Faculty of Veterinary Medicine, UGent)







Figure 13: LM view of fetlock showing palmaro-/ plantaroproximal osteochondral fragment (Source: Medical Imaging Department, Faculty of Veterinary Medicine, UGent)

On radiographs, dorsoproximal osteochondral fragments are often visible as smooth and rather small fragments located on the proximal dorsal aspect of the first phalanx (figure 12). Palmaro-/ plantaroproximal osteochondral fragments are also mostly seen as rounded fragments (with a varying radiopacity) (figure 13). They are generally located between the base of the proximal sesamoid bone and adjacent first phalanx; halfway between the sagittal groove and the lateral or medial palmar/plantar eminence of P1. POFs can be associated with a defined defect in the proximal palmar/plantar aspect of P1, but this is not always the case (McIlwraith et al., 2015a).

It has to be noted that X-ray screening has several shortcomings. Although atypical, it is possible for lesions to only involve the articular cartilage layer. This manifestation of OC(D) lacks the classical subchondral bone defect and can therefore not be detected on radiographs. Also subtle bony lesions can be missed due to superimposition. Not uncommonly, radiographically silent lesions (i.e. lesions that can be visualized during arthroscopy but not on X-rays) can be present, this is something to be thoughtful of in case of persistent effusion (McIlwraith, 2013). Furthermore, it's important to be mindful of the fact that primary changes in the cartilage can be more or less severe than they would appear from the subchondral bone lesions visible on radiographs (McIlwraith, 2013; van Weeren, 2019). Additionally, secondary lesions such as cartilage erosion or wear lines can develop due to the presence of synovitis and/or intra-articular fragments. For this reason, the lesions and damage to the joint are often discovered to be more severe and extensive during arthroscopy than would be suspected from radiographic evaluation. This is especially the case for the femoropatellar and tarsocrural joints (McIlwraith, 2013; Steinheimer et al., 1995).

## 3.10. TREATMENT

In 2003 McIlwraith described three types of presentations of OC lesions:

- 1. Patients showing both clinical and radiographic signs.
- 2. Patients showing clinical signs without radiographic (but arthroscopic) signs.
- 3. Patients showing radiographic, but no clinical signs.

For patients presenting with clinical signs, treatment (most often under the form of arthroscopic debridement and fragment removal) is recommended. When lameness is present, treatment is necessary to obtain functional recovery. Also in case of effusion of the affected joint, treatment is advised to prevent further joint degeneration. However, many asymptomatic OC patients are treated preventively since clinical signs can become manifest when the horse is put into training. Generally, if one or more fragments are found during radiographic screening, an arthroscopic removal will be performed. The aim of these elective surgeries is to obtain a horse with no joint effusion or lameness at a later age, when surgical treatment and the associated rehabilitation period would keep the horse out of training for several months. Another explanation for arthroscopic fragment removal in asymptomatic patients is the need for 'clean' X- rays when going on the market at the age of 3-4 years or later. So arguments for surgical treatment also include aesthetic and/or economic reasons, due to the lower prices that horses with osteochondral fragments sell at (van Weeren, 2019).

For OC lesions detected in older horses that are already in training and do not present any clinical signs, treatment is generally not deemed necessary. Especially in these asymptomatic cases, the age of the patient, training level and commercial goals should be brought into consideration by owners and veterinarians when deciding if the arthroscopic removal of the fragments is worth the long revalidation period and possible risk of developing joint effusion or visible scar tissue after surgery.

## 3.10.1. Conservative treatment

If lesions are small, clinical signs are absent, and at an age when lesions can still resolve, a conservative treatment can be implemented. As previously mentioned, spontaneous healing of lesions in the femoropatellar joint is generally possible until 8 months of age. Small lesions (<2 cm long, <5mm deep) without radiographic fragmentation can therefore respond to conservative treatment in young horses (Dik et al., 1999; McIntosh & McIlwraith, 1993). For the tarsocrural joint, lesions of the DIRT and LTR of the talus can still heal spontaneously until the age of 5 months (Dik et al., 1999). After this age, a spontaneous resolution is unlikely and an arthroscopic fragment removal and debridement is advised. Nevertheless, in some horses with small lesions, minimal clinical signs and no future career in the sports planned; a conservative treatment might suffice (McIlwraith, 2013).

Controversy still exists on the clinical relevance and need for removal of all types of fetlock osteochondral fragments, especially when they are incidental findings during radiographic examination of horses without lameness or joint effusion (Declercq et al., 2011). For type I lesions of the sagittal ridge (OCD), conservative treatment is advised, unless clinical signs persist past 1 year of age (C. McIlwraith & Vorhees, 1990).

The two main pointers of the conservative treatment of an OC patient are restriction of movement, meaning confinement in a stall or small paddock, and provision of the appropriate nutrition, most often entailing dietary restriction and elimination of high-energy, sugary foods such as concentrates from the diet (Ortved, 2017). The administration of systemic nonsteroidal anti-inflammatory drugs and/or intra-articular medication with corticosteroids and/or hyaluronic acid might facilitate the resolution of joint effusion, if present (Carmona et al., 2009). Recently, the use of biologics like mesenchymal stem cells and platelet-rich plasma is gaining popularity and these could be implemented in certain types of OC lesions (Seo et al., 2013).

It is of utmost importance to closely monitor the patient for an increase in lameness and/or degree of effusion of the affected joint(s). Additionally, radiographic assessment on a regular basis is recommended to keep track of the evaluation of the lesion(s). It is worth considering a conservative treatment if possible. This could result in spontaneous healing of lesions, which is superior for the osteoarticular health of the affected joint compared to healing after arthroscopic debridement (i.e. the remaining defect is filled with fibrocartilage). Furthermore, it can lead to a complete radiographic resolution without any remaining defects of the subchondral bone visible (Ortved, 2017). In all OC cases, no spontaneous healing should be expected after 12 months of age.

## 3.10.2. The arthroscopic removal of osteochondral fragments

Although conservative treatment is possible in certain cases, most osteochondral fragments are removed via arthroscopy. The surgical intervention is often preferred since this eliminates the risk of loose flaps or free fragments causing joint damage in the future and usually entails a resolution of clinical signs, if present. The procedure should not be postponed for too long after the development of joint effusion: the longer the joint capsule is stretched, the less likely will the joint effusion resolve post-operatively (McIlwraith, 2013).

The standard arthroscopic procedure for removal of osteochondral fragments consists of extensive exploration and inspection of the affected joint. After careful probing of the cartilage to detect cartilage flaps or defects, loose fragments, detached tissue and/or cartilaginous and osteochondral flaps are removed. Afterwards, the remaining surrounding tissue is debrided down to healthy subchondral bone. During this last step, special care needs to be taken of the soft subchondral bone in young horses (McIlwraith, 2013; Ortved, 2017; van Weeren, 2019).

The postoperative plan that is implemented at our faculty follows the standard post-arthroscopy guidelines that are generally advised. Sutures are protected under a sterile nonadherent absorbent dressing and standard bandage for the fetlock and hock, and an adhesive dressing for the stifle. Most horses receive flunixine meglumine (Finadyne ®) orally for 2 days after surgery. Bandages are changed every 3-5 days (or earlier if needed) and the surgical sites should stay covered for 2 more days after the removal of the sutures at 14 days post-op. The rehabilitation program consists of 2 weeks of box rest, followed by 3-4 weeks of controlled movement: starting with daily hand walking for 5-10 minutes and slowly progressing to 30-40 minutes per day. After this period, the horse can be turned out in a small paddock for 2 weeks and eventually normal training and pasture turnout can be gradually built up again. For larger lesions of the femoropatellar and tarsocrural joints, a longer resting period and a slower, more gradual build-up of training intensity is recommended.

Fragments at the dorsal aspect of the fetlock joint are more often than those at the palmar/plantar aspect, associated with synovitis, cartilage erosions or wear lines, and the MCP joints are more susceptible to osteoarthritis than other joints (McIlwraith, 2016). Therefore, arthroscopic fragment removal is often advised for OC of the sagittal ridge of the metacarpus/-tarsus (type II and III) and DOFs, even if no clinical signs are present, to prevent the development of joint degeneration. POFs are assumed to have little clinical relevance in Warmbloods and arthroscopic fragment removal is generally not deemed necessary in cases without clinical signs. Nevertheless, they often get removed preventively with the goal of obtaining clean X-rays (Declercq et al., 2011).

## 3.10.3. Risks and complications

Although these elective surgical procedures have become common practice throughout the past decades, the arthroscopic removal of osteochondral fragments is not without risk. Despite the fact that complications rarely occur, they can have major consequences for the horse's general health and future athletic career.

OC patients undergoing arthroscopic fragment removal are generally young and healthy horses, nevertheless general anaesthesia is never without risk. The anaesthesia-related mortality risk for horses undergoing elective surgery is ca. 1%. Anaesthesia-associated death can be related to malfunction of the respiratory or cardiovascular system, however noncardiorespiratory causes like fractures during recovery or fatal postanaesthetic myopathy might be even more frequent in horses (Senior, 2013). Nonfatal perioperative complications have been reported at incidences as high as 13,7% (Senior et al., 2007). The most common anaesthetic-related morbidities in horses are postanaesthetic colic and postanaesthetic myopathy (Senior, 2013).

Indeed, arthroscopy is a minimally invasive surgical technique, however there is always a certain risk for iatrogenic damage to the articular cartilage, joint capsule or peri-synovial structures. This appears to be the most frequently unreported complication of all arthroscopic surgeries. Small cartilage lesions do not seem to be of major significance, however, partial- or full-thickness lesions can strongly influence the patient's osteoarticular health on short and long term (McIlwraith et al., 2015). Adequate fluid distension of the joint prior to insertion of the arthroscopic sleeve and the use of a conical (instead of a sharp) obturator angled away from the articular cartilage while avoiding the use of excessive force, can help prevent or minimize this damage. It is also strongly recommended to only use arthroscopic instruments under direct visualization with the arthroscope. Synovial resection might be necessary to allow complete and clear visualization. However, as synovium does not regenerate, excessive resection could lead to joint capsule fibrosis and trauma to the fibrous joint capsule can lead to postoperative capsular mineralisation (Goodrich and McIlwraith, 2008).

Femoral neuropathy or neuromyopathy of the quadriceps muscles presenting as failure to extend hindlimb joints can occur as a (transient) complication after arthroscopy when both hind limbs are in extension during a surgery where the horse is put in dorsal recumbency. To prevent this, the contralateral leg that is not operated on, should always remain in a flexed position (Goodrich and McIlwraith, 2008).

Studies investigating the occurrence of surgical site infection (SSI) and septic arthritis after elective arthroscopy reported incidences of 0,65% for portal infection and 0,7-1,11% for septic arthritis (Borg and Carmalt, 2013; Brunsting et al., 2018; Olds et al., 2006). One of the risk factors for development of SSI is lesion size, with lesions exceeding 40mm in length being at significant higher risk. The removal of larger fragments requires more extensive manipulation and debridement, as well as repetitive introduction of instruments. This inevitably leads to more soft tissue damage and subcutaneous fluid accumulation, which are proposed to be predisposing factors for the development of postoperative infection. Younger horses seem to be more susceptible to the development of septic arthritis after elective arthroscopy. This might be related to external contamination of the portals, since youngsters generally spend a longer time in recumbency and appropriate protection of the surgical sites can be more difficult if the horse is not used to being handled. Nevertheless, the causative agent probably originates from the skin rather than the environment, with Staphylococcus and Streptococcus being isolated most commonly from infected joints. Septic arthritis mostly occurs secondary to surgical site infection. It is therefore strongly recommended to treat cases of SSI with joint lavage, local debridement, and a combination of local and systemic antimicrobial treatment, to prevent progression to septic arthritis. Even though the risk of developing septic arthritis after arthroscopic fragment removal can be considered low, it is a serious and potentially life threatening complication (Brunsting et al., 2018).

Postoperative joint effusion due to (persistent) synovitis is another important complication that can occur after the arthroscopic removal of osteochondral fragments. Joint distension can persist or arise postoperatively due to ongoing disease or failure to remove all fragments. In this case, medical imaging modalities should be used to re-evaluate the affected joint. When the affected joint was already distended preoperatively, as a result of synovial inflammation due to the presence of cartilage flaps and/or osteochondral fragments, the risk of persistence of effusion after surgery increases. To determine the probability of resolution, the duration of preoperative distension of the joint capsule plays a role. Some veterinarians use 6-8 weeks as the timeframe within which resolution can be assumed in most cases. Ongoing mild effusion can persist without immediate clinical significance. However, this can still indirectly influence the horse's athletic career since joint distension is not accepted in the sport horse industry (Goodrich and Mcllwraith, 2008). Furthermore, the long-term effects of residual effusion on the osteoarticular health of the affected joints have not been investigated yet. This concern will be further elaborated in the discussion of this thesis.

Although the incisions made to create the arthroscope and instrument portals generally do not exceed 1-2cm, the formation of scar tissue and regrowth of lighter hairs after surgery is possible. Of course this complication does not limit the performance of the horse, nevertheless it could lead to devaluation, since the cosmetic aspect is of large importance in the current sport horse industry.

## 3.11. PROGNOSIS

Several studies investigating the outcome of conservative and/or surgical treatment for osteochondral fragments in different populations exist. Table 2 contains a summary of some of the most important studies conducted during the past years.

Study	Population	Joints investigated	Follow-up	Outcome	Factors influencing outcome/ prognosis
Arthroscopic surgery for osteochondritis dissecans of the femoropatellar joint of the horse (Foland et al., 1992)	161 horses: 82 Thoroughbreds, 39 Quarters, 16 Arabians, 9 Warmbloods and 15 others of various breeds	Femoro- patellar joint	No time period mentioned	Follow-up of 134 horses (79 racehorses and 55 non- racehorses): 86 horses (64%) returned to their intended use, 9 were sound in training, 21 were unsuccessful and 18 were unsuccessful due to other defined reasons.	Grade I lesions (<2 cm in length) had a significantly higher success rate (78%) than grade 2 (2-4 cm) or grade 3 (>4 cm) lesions (63% and 54% success rates respectively). A significantly higher success rate was seen in horses undergoing surgical treatment as 3-year- olds, while a lower success rate was observed for yearlings. However, this age factor is probably due to the younger horses presenting with more severe lesions. <i>No significant effect of sex, racehorse</i> <i>versus non-racehorse, lesion location,</i> <i>limb involvement, presence or absence</i> <i>of patellar or trochlear groove lesions, or</i> <i>presence or absence of loose bodies on</i> <i>outcome was found.</i>
Outcome after arthroscopic treatment of lateral femoral trochlear ridge osteochondrosis in sport horses (Uprichard et al., 2013)	37 horses: 34 Warmbloods, 2 Thoroughbreds, and 1 Friesian	Femoro- patellar joint	6 months – 9 years	Short-term follow-up: 27 horses (73%) had no complications, 7 horses had persistent articular distension with lameness during the first month after surgery, which eventually resolved in 4 horses. One horse developed an extra-articular abscess after incomplete removal of suture material. Two horses were euthanatized: 1 at the time of surgery due to the severity of articular lesions, and the other due to colic	An increasing lesion depth significantly increased the likelihood of persistent lameness, joint effusion or complications related to the surgical procedure within the first 6 months post-op.

Table 2: A summary of some of the most important studies on the outcome of surgical (and conservative) treatment of different types of osteochondral fragments conducted during the past years

				three weeks after leaving the	
				hospital.	
				Long-term follow-up of 29 horses:	The odds of a negative long-term
				19 horses (66%) returned to their	outcome increased ten-fold if additional
				intended use, 5 were working at a	locations, such as the medial trochlear
				lower level than anticipated, 2 were	ridge of the femur or the patella, were
				not in use due to persistent lameness	also affected. Furthermore, a trend for
				(directly related to the stifle), 1 was	decreasing prognosis with increasing
				not in use for reasons unrelated to	age was observed.
				the surgery, and 2 were euthanatized	No significant effect of sex, limb
				(1 due to chronic lameness and the	involvement or lesion depth on long-
				other for unrelated reasons).	term outcome was found.
Osteochondritis	225 horses:	Tarsocrural	No time	Follow-up of 183 horses: 140 orses	There was a significantly inferior
dissecans of the	154 racehorses	joint	period	(76,5%) returned to their intended	outcome in racehorses with articular
tarsocrural joint:	(106 Standardbreds,		mentioned	use (90/124 (72,6%) racehorses and	cartilage degeneration or erosion.
results of	30 Thoroughbreds			50/59 (83,1%) non-racehorses).	No significant effect of age, sex, limb
treatment with	and 18 Quarters)			34 racehorses were unsuccessful:	involvement and lesion location or size
arthroscopic	and 71 non-			6 had a remaining tarsocrural joint	on outcome was found.
surgery	racehorses			problem, 8 were poor race horses,	
(Mcllwraith et al.,	(20 Arabians,			15 developed other problems,	
1991)	18 Quarters,			3 developed septic arthritis that was	
	13 Warmbloods			not resolved and 2 died from other	
	and 20 horses from			causes.	
	a variety of breeds)			9 non-racehorses were unsuccessful:	
				5 had remaining tarsocrural joint	
				problems and 4 developed other	
				problems.	
				Follow-up on the degree of synovial	Lesions of the lateral trochlear ridge of
				effusion of 217 preoperatively	the talus or medial malleolus of the tibia
				distended joints: synovial effusion	showed a significantly inferior outcome
				resolved in 117/131 (89,3%)	for resolution of synovial effusion in
				racehorse joints and 64/86 (74,4%)	comparison with lesions of the distal
				non-racehorse joints.	intermediate ridge of the tibia.

					No relationship between post-operative performance and resolution of effusion was found.
Racing performance of Standardbreds after conservative and surgical treatment for tarsocrural osteochondrosis (Laws et al., 1993)	114 Standardbreds diagnosed with osteochondrosis of the intermediate ridge of the tibia and 456 control horses (matched by age, sex and sire)	Tarsocrural joint	No time period mentioned	90/114 (78,9%) OC horses made at least one racing start: 44/58 (76%) surgically treated horses and 42/56 (75%) conservatively treated horses	Horses were not randomly assigned, making comparison between surgical and conservative treatment impossible (horses presenting with joint effusion or bilateral OC were more likely to be treated surgically). Control horses made significantly more starts and had a longer racing career than did OC-affected horses. No significant difference in total lifetime earnings or record time between OC- affected horses and controls was found.
Postoperative racing performance in standardbreds and thoroughbreds with osteochondrosis of the tarsocrural joint: 109 cases (1984-1990) (Beard et al., 1994)	109 horses: 64 Thoroughbreds and 45 Standardbreds, controls: 207 Standardbred and 277 Thoroughbred siblings (other foals from same mare)	Tarsocrural joint	No time period mentioned	Thoroughbreds: 43% of surgically treated horses raced as 2-year-olds and 78% as 3-year-olds, compared with 48 and 73% of the siblings respectively . Standardbreds: 22% of surgically treated horses raced as 2-year-olds and 43% as 3-year-olds, compared with 42 and 50% of the siblings respectively.	Median number of starts was significantly less for surgically treated horses than that for siblings, except for the 3-year-old Thoroughbreds. The distribution of starts was different between surgically treated and sibling horses only for 2-year-old Standardbreds. Affected horses were less likely to start a race as 2-year-olds than were their siblings. Horses with multiple lesions seemed to be less likely to start a race than horses with only 1 lesion. Median earnings were lower for affected horses than for siblings. <i>No significant effect of lesion location or unilateral vs bilateral involvement on the ability to start at least 1 race.</i>

Results of	146 horses	Tarsocrural	6 months –	Follow-up of 96 horses: 60% of the	Standardbreds had a significantly worse
conservative	(71 Warmbloods	joint	5 years	Warmbloods (WB) and 18% of	outcome after conservative treatment
treatment of	and	-	-	Standardbreds (SB) returned to their	(consisting of box rest and dietary
osteochondrosis	75 Standardbreds)			intended use, 10% of WB and 18% of	restrictions) than Warmbloods.
of the tibiotarsal				SB had to work lighter than expected,	
joint in the horse				7% of WB and 41% of SB was	
(Peremans and				euthanized due to poor performance	
Verschooten,				and 23% of both WB and SB could not	
1997)				perform due to other problems	
				Follow-up of joint distension was	
				available for 37 horses: effusion	
				disappeared in 8 WB and 5 SB,	
				effusion persisted in 14 WB and 10 SB	
Lameness and	79 horses:	Tarsocrural	6 weeks –	Follow-up of 113 joints (79 horses):	The quantitative effect of surgical
effusion of the	30 Standarbreds,	joint	20 months	25 of 79 (31,6%) horses were lame	treatment of OCD of the intertrochlear
tarsocrural joints	46 Warmbloods,			before surgery, 2 horses remained	ridge of the tibia was found to be a
after arthroscopy	1 Andalusian,			lame (1 of them related to the	major reduction in lameness and
of	1 Friesian and			tarsocrural joint), 1 horse developed	reaction to flexion test, but only a
osteochondritis	1 American Paint			lameness after surgery.	moderate reduction of joint effusion.
dissecans in				Whereas 38 of 111 (34,2%) tested	A significantly greater reduction in
horses (Brink et				hindlimbs had a positive reaction to	response to flexion was observed in
al., 2009)				flexion preoperatively, 11 of 112	older than younger horses. A significant
				(10%) showed a reaction after	correlation between joint effusion and
				surgery, 4 horses developed reaction	time to follow-up was observed, with
				to flexion after surgery.	joint effusion decreasing with increasing
				98 of 113 (86,7%) affected joints were	time.
				distended preoperatively and	No significant effect of age on the
				72 of 113 (63%) showed effusion after	outcome and no significant correlation
				surgery, 5 horses developed joint	between time to follow-up and the effect
				distension after surgery, the degree	of surgery on lameness or the reaction
				of effusion generally decreased (only	to flexion was found.
				1 horse developed increased joint	
				distension).	

Management of osteochondritis dissecans of the dorsal aspect of the distal metacarpus and metatarsus (McIlwraith & Vorhees, 1990)	Number of horses and breed distribution not mentioned	Metacarpal/ -tarsal joints (OC(D))	No time period mentioned	Conservative treatment of 15 type 1 lesions: 12 resolved clinically of which 8 improved radiographically, in 3 cases clinical signs persisted of which 2 showed no change on radiographs and 1 progressed Conservative treatment of 8 type 2 lesions: in only 1 horse clinical signs improved, in the others clinical signs persisted and often fragmentation progressed on radiographs Surgical treatment of mostly type 2 or 3 lesions (number of horses not mentioned): 57% returned to their intended use, 25% had persistent fatlack problems 18% was	Conservative treatment of type 1 lesions can lead to clinical and radiographic improvement. Restriction of the energy intake seems to reduce problems. The success rate was higher for surgery in the hind- than in the forelimbs, and in type 2 than in type 3 lesions. The presence of osteophytes, subchondral hence defects or wear lines or ergriges of
				unsuccessful due to other reasons	the articular cartilage significantly decreased the outcome.
Identification and treatment of osteochondritis dissecans of the distal sagittal ridge of the third metacarpal bone (Wright and Minshall, 2014)	16 horses: 14 Thoroughbreds and 2 Warmbloods	Metacarpal/ -tarsal joints (OC(D))	≥12 months	Follow-up of 12 Thoroughbreds: 11 horses raced after surgery, 1 horse had periarticular new bone formation of P1 and the proximal sesamoid bones and remained lame post-op. Follow-up of 2 Warmbloods: both horses returned to their intended use Effusion resolved in all 13 preoperatively distended joints.	The presence of additional intra- articular lesions decreases the prognosis for a functional recovery.
Proximodorsal first phalanx osteochondral chip fragmentation in 336 horses	<ul> <li>336 horses:</li> <li>194 Thoroughbred</li> <li>s, 127 Quarters,</li> <li>5 Warmbloods,</li> <li>2 Standardbreds,</li> </ul>	Metacarpal/ -tarsal joints (DOF)	No time period mentioned	Follow-up of 270 racehorses: 196 of 270 (73%) returned to racing and 141 of these (52%) raced at the same or higher level; 18 (6,7%) developed another fragment and 56 (21%) failed to return to previous use.	The presence of additional intra- articular lesions (or concurrent carpal lesions) significantly decreased the outcome for the Thoroughbred racehorses.

(Kawcak and McIlwraith, 1994)	and 7 horses of other breeds			Follow-up of 16 non-racehorses: 12 of 16 (75%) returned to their intended use and 4 (25%) failed to return to previous use.	No significant effect of racehorse vs non- racehorse, single vs multiple limb involvement, single vs multiple fragmentation or fragment location on outcome was observed.
Arthroscopic removal of axial osteochondral fragments of the plantar/palmar proximal aspect of the proximal phalanx in horses: 119 cases (1988-1992). (Fortier et al., 1995)	119 horses: 109 (92%) Standardbreds, 2 Warmbloods, 3 Thoroughbreds and 4 horses of other breeds	Metacarpal/ -tarsal joints (POF)	No time period mentioned	Follow-up of 117 horses: 20 Standarbreds had not (yet) started a race: 6 were in training, 8 were too slow, 5 had unrelated problems and 1 showed persistent lameness from the affected joint. Of the 87 Standardbreds that started a race, 55 (63%) raced at or above the preoperative level. All 9 (100%) nonracehorses returned to their intended use. Joint effusion persisted in only 1 horse (without associated lameness).	The presence of articular cartilage fibrillation or synovial proliferation significantly decreased the outcome. <i>No significant effect of concurrent DIRT</i> <i>lesions or tarsal osteoarthritis, nor</i> <i>location and amount of fragments was</i> <i>found.</i>
Racing performance in standardbred trotting horses with proximal palmar/plantar first phalangeal fragments relative to the timing of surgery: proximal palmar/plantar first phalangeal fragment (Carmalt et al., 2014)	193 Swedish Standardbred trotters	Metacarpal/ -tarsal joints (POF)	No time period mentioned	158 of 193 (82%) horses raced after surgery.	Horses with 3 legs affected ran slower than those with 1 or 2 legs affected. No significant difference in race speed between preraced and never raced horses; no association between timing of surgery and race speed or career longevity and no significant effect of concurrent DIRT lesions or location and amount of fragments on outcome was found.

## 3.11.1. Femoropatellar joint

With studies reporting approximately 65% of horses performing at their intended level after arthroscopic debridement of LTR lesions (Foland et al., 1992; Uprichard et al., 2013), the prognosis for surgical treatment of femoropatellar OC is generally considered to be fair to good, and depends on the severity of the lesions (Ortved, 2017). When further categorizing lesions in a mixed study population mainly including Thoroughbreds and Quarters, Foland et al. (1992) observed grade I lesions(<2 cm) to have a significantly better prognosis (78%) than grade II (2-4 cm; 63%) or grade III lesions (>4 cm; 54%). In this same study, a higher success rate was seen in horses undergoing surgical treatment as 3-yearolds, while a lower success rate was observed for yearlings. The explanation behind this age factor was the fact that horses presenting with clinical signs at a younger age generally have more severe lesions than those who only develop symptoms after starting training or those who remain asymptomatic and in which lesions get detected during radiographic screening. These older horses often have already started their athletic career, meaning they automatically have a higher chance of having a successful outcome compared to unproven youngsters. No significant difference in outcome was observed in racehorses versus non-racehorses, and also the presence of additional lesions did not significantly influence outcome. Nevertheless, a later study investigating a population involving mostly Warmbloods, found the prognosis to be ten-fold worse when additional structures other than the LTR, such as the patella and medial trochlear ridge, are involved. Furthermore, these authors also observed a trend for decreasing prognosis with increasing age (Uprichard et al., 2013).

## 3.11.2. Tarsocrural joint

For the tarsocrural joint, the prognosis of surgical treatment of OC lesions located at the cranial intermediate ridge of the distal tibia or LTR of the talus has been reported to be good (Beard et al., 1994; Brink et al., 2009). In 1991, McIlwraith et al., investigated the outcome after arthroscopic treatment of tarsocrural OC in a population consisting of 154 racehorses (mainly Standardbreds and Thoroughbreds) and 71 non-racehorses (mainly Arabians, Quarters and Warmbloods). After surgery, 76,5% of horses (72,6% of racehorses and 83,1% of non-racehorses) returned to their intended use. A significantly inferior outcome was observed in racehorses with articular cartilage degeneration. Follow-up of the degree of synovial effusion revealed resolution in 89,3% of racehorses and in 74,4% of non-racehorses. A significantly inferior outcome for resolution of effusion was observed in lesions of the LTR of the talus or medial malleolus in comparison to lesions of the distal intermediate ridge of the tibia. Regardless, no relationship was found between post-operative performance and persistence of effusion.

Laws et al. (1993) and investigated the racing performance of Standardbreds and their matched controls after conservative and surgical treatment of the intermediate ridge of the tibia. Approximately 75% of surgically as well as 75% of conservatively treated horses made at least one racing start. However, as treatment was not randomly assigned (more extensive lesions or severe clinical signs lead to surgical treatment), no conclusions regarding the comparison of surgical versus conservative treatment could be made. Although, the authors found no difference in performance (evaluated through lifetime earnings and record time), control horses made significantly more starts and had longer racing careers than OC-affected horses. Beard et al., 1994 compared the postoperative racing performance of Thoroughbreds and Standardbreds with that of their siblings. Even though they reported similar findings regarding the lower number of starts, they observed median earnings to be lower as well for surgically treated horses. Additionally, horses with multiple lesions seemed to be less likely to start a race than horses with only 1 lesion.

A more recent study, conducted by Brink et al. in 2009, investigated a mixed population of mainly Standardbreds and Warmbloods. The authors reported a major reduction in lameness and reaction to flexion tests after surgery. However, the follow-up of joint effusion revealed rather disappointing results, as 63% of horses still showed distension of the affected joint postoperatively compared to

86,7% before surgery and 5 horses even developed distension after the procedure. Even though a significant correlation between joint effusion and time to follow-up was observed, with joint effusion decreasing with increasing time, the effect of surgical treatment on resolution of effusion was only moderate.

Peremans and Verschooten (1997) found the outcome after conservative treatment of tarsocrural OC, consisting of box rest and dietary restriction, to be significantly worse in Standardbreds (18% of horses returned to their intended use) compared to Warmbloods (60% of horses returned to their intended use). For a small part of the study population follow-up on the degree of joint distension was available, showing rather disappointing results for both Standardbreds and Thoroughbreds as effusion remained present in 65% (24/37) of horses.

## 3.11.3. MCP/MTP joint

In the MCP/MTP joints, the prognosis for conservative treatment of type I lesions of the sagittal ridge (OC(D)) is good. In type II and III lesions however, surgical treatment is generally advised and has a reported success rate of 57% (return to intended use). A higher success rate was observed in the hindlimbs compared to the frontlimbs and in type 2 versus type 3 lesions. Furthermore, detection of osteophytes, erosions or wear lines in the articular cartilage or subchondral bone defects during arthroscopy significantly decreased the outcome (McIlwraith and Vorhees, 1990). A more recent study investigated a small population of 14 Thoroughbreds and 2 Warmbloods diagnosed with OC of the distal sagittal ridge, all presenting with lameness (Wright and Minshall, 2014). After surgery, follow-up of 14 horses revealed 13 of them returning to their intended use. Furthermore, effusion resolved in all 13 preoperatively distended joints. Also in this study, the presence of additional intra-articular lesions decreased the outcome.

Currently not many studies investigating the prognosis of arthroscopic fragment removal of dorsoproximal osteochondral fragments, diagnosed in young horses and categorised as manifestations of a developmental orthopaedic disease, exist. Kawcak and McIlwraith (1994) investigated the outcome after surgical treatment of proximodorsal first phalanx osteochondral chip fragmentation in a large population mainly consisting of Thoroughbreds and Quarters. The authors reported a success rate of approximately 74% for racehorses as well as non-racehorses. The presence of additional intra-articular lesions (or concurrent carpal lesions) significantly decreased the outcome for the Thoroughbred racehorses. However, insignificant through statistical analysis, this trend was also observed in the other breed groups.

The prognosis of the arthroscopic removal of palmaro-/plantaroproximal osteochondral fragments has been reported to be good. Fortier et al., 1995 investigated a population mainly consisting of Standardbreds and found 63% of racehorses racing at or above the preoperative level and 100% of non-racehorses returning to their intended use. The presence of articular cartilage fibrillation or synovial proliferation significantly decreased the outcome. More recently, Carmalt et al. (2014) reported a higher success rate in a Standardbred study population, with 82% of horses racing after surgery.

## 3.11.4. Summary

For the femoropatellar joint, a success rate of approximately 65% has been reported in both racing breed and Warmblood study populations (Foland et al., 1992; Uprichard et al., 2013). The outcome decreased significantly with increasing lesion length (Foland et al., 1992) and the prognosis was observed to be worse when additional structures other than the lateral trochlear ridge of the femur, such as the patella or the medial trochlear ridge, were involved (Uprichard et al., 2013).

For the tarsocrural joint, approximately 75% of horses returned to their intended use combining results of different studies investigating study populations mainly consisting of racing breeds (Beard et al., 1994; Laws et al., 1993; McIlwraith et al., 1991). The presence of articular cartilage degeneration significantly decreased the outcome in racehorses (McIlwraith et al., 1991). This same study reported a relatively good prognosis for resolution of effusion in joints where the intermediate ridge of the tibia was affected, however an inferior outcome regarding resolution of effusion was observed in joints with lesions of the lateral trochlear ridge of the tibia or medial malleolus. Remarkably, a more recent study that also included Warmbloods found effusion to remain present in 73% of preoperatively distended joints. With 5 horses even only developing effusion of the affected joint after surgery, the prognosis of arthroscopic fragment removal regarding the resolution of joint effusion was considered to be bad in this population (Brink et al., 2009). Peremans & Verschooten (1997) found conservative treatment of tarsocrural OC (consisting of box rest and dietary restrictions) to have a significantly better outcome in Warmbloods compared to Standardbreds, however, the prognosis regarding resolution of joint effusion was bad in both breed groups (Peremans and Verschooten, 1997).

For the metacarpo-/metatarsophalangeal joints, the success rate for return to the intended use after arthroscopic removal of fetlock osteochondral fragments (OCD, DOF, POF) is approximately 75% combining results from different studies investigating study populations mainly consisting of racing breeds. In the MCP/MTP joints, the presence of additional intra-articular lesions such as osteophytes, cartilage erosions, wear lines or subchondral bone defects, significantly decreased the outcome (Carmalt et al., 2014; Fortier et al., 1995; Kawcak and McIlwraith, 1994; McIlwraith and Vorhees, 1990; Wright and Minshall, 2014).

In conclusion, the prognosis of the arthroscopic removal of osteochondral fragments for a return to the intended use is considered to be fair to good for the femoropatellar joint and good for the tarsocrural and MCP/MTP joints (Beard et al., 1994; Carmalt et al., 2014; Foland et al., 1992; Fortier et al., 1995; Kawcak & McIlwraith, 1994; Laws et al., 1993; C. McIlwraith & Vorhees, 1990; C. W. McIlwraith et al., 1991; Uprichard et al., 2013; Wright & Minshall, 2014). The presence of additional intra-articular lesions significantly decreases the outcome in all joints (Fortier et al., 1995; Kawcak & McIlwraith et al., 1991; C. McIlwraith & Vorhees, 1990; Uprichard et al., 2013). Not many studies include follow-up information regarding resolution of joint effusion. Nevertheless, those who do, report rather disappointing results, especially for the tarsocrural joint (Brink et al., 2009). Most existing studies investigate mainly racing breeds and only a few include a significant proportion of Warmbloods in the study population (Brink et al., 2009; Peremans & Verschooten, 1997; Uprichard et al., 2013).

## 4. PROBLEM STATEMENT

Further research on the importance of OC and the prognosis of arthroscopic removal of osteochondral fragments in the Warmblood population is needed. Currently, most literature and studies have investigated OC in the Thoroughbred and Standardbred population. Nonetheless, large breed differences in training intensity and structure and duration of the sports career need to be considered. In most OC patients, clinical symptoms and gait alterations are minimal (Gorissen et al., 2017). In a study conducted by Verwilghen et al. in 2013, no differences in future jumping performance between Warmblood stallions with tarsocrural OC, or DOFs or POFs in the MCP/MTP joints and controls were reported. Nevertheless, OC of the femoropatellar and MCP/MTP joints significantly decreased performance.

As mentioned before, in the show horse industry the focus lies on the cosmetic appearance: looking 'clean' on radiographs, with no joint effusion. However, it is not always guaranteed that preoperatively present joint effusion will resolve after the surgical treatment (Brink et al., 2009). Sometimes joint effusion will only arise or become worse postoperatively, and in the long term, chronic synovitis can lead to more cartilage damage and osteoarthritis. Since Thoroughbreds only race for 3-4 years before retiring, the importance of OC and its effect on performance will be smaller compared to Warmbloods; which can be in training for over 10 years, meaning that development of these arthropathies associated with persistent effusion and chronic synovitis, can cause problems during this time period. When considering the aesthetic aspect, awareness needs to be brought to the possibility of scar tissue remaining visible on the incision sites.

In conclusion, the prognosis for arthroscopic removal of osteochondral fragments in Warmbloods might not be as good as it is assumed to be in literature.

## 5. **OBJECTIVE**

This master's thesis will contribute to a larger study where the aim is to categorise OC lesions and investigate the correlation between joint effusion, volume of the fragments and arthroscopic changes in the joint. The results of this study will be part of a research project in which the goal is to determine the prognosis of the arthroscopic removal of osteochondral fragments (OCD, DOF, POF) in the Warmblood sport horse population.

The first aspect of this thesis is the creation of a database where data is extracted from the Filemaker<sup>®</sup> application containing all patient files from the faculty clinic. The data includes information about the orthopaedic examination and arthroscopic report from OC patients undergoing arthroscopic fragment removal at the faculty clinic between August 2020 and March 2023. The focus is on osteochondral fragments in the femoropatellar, tarsocrural and MCP/MTP joints. The second aspect is the measurement of the volumes of the osteochondral fragments elevated during arthroscopy, which will also be included in the database. Lastly, as a first step in the larger research project, statistical analysis will be used to evaluate the correlation between the degree of joint effusion and the volume of the fragment(s).

## 6. MATERIALS & METHODS

#### 6.1. CREATION OF DATABASE

The study population consists of 213 horses, mainly Warmbloods, presented at the faculty clinic between August 2020 and March 2023 for arthroscopic removal of one or more osteochondral fragments from the femoropatellar, tarsocrural and/or MCP/MTP joints.

A database is created using Microsoft Office Excel<sup>®</sup>. Data is collected from the patient files in the Filemaker<sup>®</sup> application of the faculty clinic, the focus lies on the findings on orthopaedic examination and during arthroscopy.

The following information is included:

- a) Patient data
  - o File number
  - Date of orthopaedic examination
  - o Date of birth
  - o Name
  - o Weight
  - Height at the withers
- b) Orthopaedic examination

#### Degree of joint effusion:

The stifles, hocks and fetlocks are inspected and palpated (if possible) after which the joint effusion is graded as in table 3:

Table 3: Grading scale for the degree of joint effusion

0	No joint effusion
1	Mild joint effusion
2	Moderate Joint effusion
3	Severe joint effusion

#### Lameness:

The horse is walked and trotted in a straight line on a hard surface. If it is comfortable with being lunged, the walk and trot is also inspected in a circle on a hard and soft surface. The lameness is graded following the American Association of Equine Practitioners Lameness Scale<sup>1</sup> (table 4):

Table 4: AAEP Lameness Scale

0	Lameness not perceptible under any circumstances.
1	Lameness is difficult to observe and is not consistently apparent, regardless of circumstances (e.g. under saddle, circling, inclines, hard surface, etc.).
2	Lameness is difficult to observe at a walk or when trotting in a straight line but consistently apparent under certain circumstances (e.g. weight-carrying, circling, inclines, hard surface, etc.).
3	Lameness is consistently observable at a trot under all circumstances.
4	Lameness is obvious at a walk.
5	Lameness produces minimal weight bearing in motion and/or at rest or a complete inability
	to move.

<sup>&</sup>lt;sup>1</sup> American Association of Equine Practitioners, 2023. LAMENESS EXAMS: Evaluating the Lame Horse. [WWW Document]. URL https://aaep.org/horsehealth/lameness-exams-evaluating-lame-horse/ (accessed 19.05.23).

#### Flexion tests:

If lameness is visible or suspected, and if possible, flexion tests are performed. The change in gait is evaluated using the following grading scale developed by Kaneps in 2014 (table 5):

#### Table 5: Grading scale for outcome of flexion test (Kaneps, 2014)

0	Negative: no change in lameness
1	Slight positive: slight exacerbation of lameness following flexion that is noticed during only a
	portion of the trotting course
2	Moderate positive: lameness is exacerbated while the horse is trotting away from the
	examiner, but not on the return
3	Severe positive: marked exacerbation of lameness during the outbound and return portions
	of the trotting course.

c) Arthroscopy report

The exact location of the fragment, degree of synovitis of the affected joint and changes in the articular cartilage (e.g. wear lines) are obtained from the arthroscopy report. These parameters are graded by the surgeon during the procedure using predetermined scales.

## 6.2. MEASUREMENT OF THE VOLUME OF THE FRAGMENT(S)

The osteochondral fragments are removed from the joint(s) during the arthroscopy and collected on the sterile table. At the end of the surgery, any redundant surrounding soft tissue is removed and the fragments are transferred into a small plastic container, on which the patient's file number and the joint in question is written. The plastic container is then stored in the fridge until the fragments are measured.

The osteochondral fragments elevated during arthroscopy can be considered irregular objects, meaning there is no standard formula to calculate their volumes. Therefore the method of water displacement is used, where the volume is measured by submerging the fragments in a known volume of water. For this, a graduated cylinder is filled with a certain amount of water and placed on a flat surface. Next, all the fragments collected from one joint are transferred to the cylinder and submerged. The increase in the level of the water is read at the bottom of the meniscus at eye level. This indicates the amount of water displaced, which equals the volume of the submerged fragment. A measuring cylinder of 10mL with 0,1mL grading divisions is used. The volumes are classified into different categories (table 6) and included in the database:

Increase in water	Volume in database
0,00-0,04	0,05
0,05-0,14	0,10
0,15-0,24	0,20
0,25-0,34	0,30
0,35-0,44	0,40
0,45-0,54	0,50
0,55-0,64	0,60
0,65-0,74	0,70
0,75-0,84	0,80
0,85-0,94	0,90
0,95-1,04	1,00
Etc.	Etc.

Table 6: Categories of fragment volumes

## **6.3. STATISTICAL ANALYSIS**

Statistical analysis is performed using IMB SPSS Statistics<sup>®</sup>. The correlation between the effusion of the affected joint and the volume of the fragment(s) elevated during the arthroscopy is evaluated through statistical analyses using the Kendall's coefficient of rank correlation tau-sub-b,  $\varphi_b$  and the Spearman rank correlation coefficient,  $r_s$  (Khamis, 2008).

# 7. RESULTS

Assessment of the degree of joint effusion and measurement of the volume of the osteochondral fragments was available for 162 horses.

Table 7: Total number of joints and mean fragment volume for each joint

	Femoropatellar joint	Tarsocrural joint	MCP/MTP joint
Total number of joints	43	96	132
Mean fragment volume	ca. 0,68 mL	ca. 0,48 mL	ca. 0,12 mL

Table 8: Number of joints (%), median fragment volume and mean fragment volume per categoryof joint effusion for each joint

Femoropatellar joint					
Degree of joint effusion	Number of joints (%)	Median fragment volume	Mean fragment volume		
No effusion	18 (42)	0,20 mL	ca. 0,44 mL		
Mild effusion	5 (12)	0,60 mL	ca. 0,56 mL		
Moderate effusion	7 (16)	0,90 mL	ca. 1,12 mL		
Severe effusion	13 (30)	0,40 mL	ca. 0,84 mL		
	Tarsocrura	l joint			
Degree of joint effusion	Number of joints	Median fragment	Mean fragment		
	(%)	volume	volume		
No effusion	30 (31)	0,15 mL	ca. 0,23 mL		
Mild effusion	13 (14)	0,30 mL	ca. 0,59 mL		
Moderate effusion	23 (24)	0,30 mL	ca. 0,41 mL		
Severe effusion	30 (31)	0,65 mL	ca. 0,73 mL		
MCP/MTP joint					
Degree of joint effusion	Number of joints	Median fragment	Mean fragment		
	(%)	volume	volume		
No effusion	fusion 112 (85) 0,10 mL		ca. 0,12 mL		
Mild effusion	6 (5)	0,10 mL	ca. 0,14 mL		
Moderate effusion	11 (8)	0,10 mL	ca. 0,16 mL		
Severe effusion	3 (2)	0,10 mL	ca. 0,15 mL		

#### 7.1. FEMOROPATELLAR JOINT

Data included 43 femoropatellar joints. The mean volume of the fragments elevated from the femoropatellar joint was ca. 0,68 mL. 18 of 43 (42%) femoropatellar joints showed no effusion. Median fragment volume for this category was 0,20 mL and mean fragment volume was ca. 0,44 mL. 5 of 43 (12%) femoropatellar joints showed mild effusion. Median fragment volume for this category was 0,60 mL and mean fragment volume was ca. 0,56 mL. 7 of 43 (16%) femoropatellar joints showed moderate effusion. Median fragment volume for this category was 0,90 mL and mean fragment volume was ca. 1,12 mL. 13 of 43 (30%) femoropatellar joints showed severe effusion. Median fragment volume was ca. 0,84 mL.



*Figure 14: Violin plot of the fragment volume for the 4 categories of effusion of the femoropatellar joint. Number of joints, as well as mean and median fragment volumes for each category are provided in table 8.* 

Table 9: Results of the statistical analyses evaluating the correlation between fragment volume and degree of joint effusion for the femoropatellar joint

Contractione				
			VOLstifle	EFFstifle
Kendall's tau_b	VOLstifle	Correlation Coefficient	1,000	,249 <sup>*</sup>
		Sig. (2-tailed)		,042
		N	43	43
	EFFstifle	Correlation Coefficient	,249 <sup>*</sup>	1,000
		Sig. (2-tailed)	,042	-
		N	43	43
Spearman's rho	VOLstifle	Correlation Coefficient	1,000	,305*
		Sig. (2-tailed)		,047
		N	43	43
	EFFstifle	Correlation Coefficient	,305 <sup>*</sup>	1,000
		Sig. (2-tailed)	,047	-
		N	43	43

#### Correlations

\*. Correlation is significant at the 0.05 level (2-tailed).

The results of the statistical analyses (table 9) revealed a significant positive correlation between the volume of the fragments and the effusion of the femoropatellar joint:  $\phi_b=0,25$ ; p=0,042 and r<sub>s</sub> (41)= 0,31; p= 0,047.

#### 7.2. TARSOCRURAL JOINT

Data included 96 tarsocrural joints. The mean volume of the fragments elevated from the tarsocrural joint was ca. 0,48 mL. 30 of 96 (31%) tarsocrural joints showed no effusion. Median fragment volume for this category was 0,15 mL and mean fragment volume was ca. 0,23 mL. 13 of 96 (14%) tarsocrural joints showed mild effusion. Median fragment volume for this category was 0,30 mL and mean fragment volume was ca. 0,59 mL. 23 of 96 (24%) tarsocrural joints showed moderate effusion. Median fragment volume for this category was 0,30 mL and mean fragment volume was ca. 0,41 mL. 30 of 96 (31%) tarsocrural joints showed severe effusion. Median fragment volume for this category was 0,65 mL and mean fragment volume was ca. 0,73 mL.



Figure 15: Violin plot of the fragment volume for the 4 categories of effusion of the tarsocrural joint. Number of joints, as well as mean and median fragment volumes for each category are provided in table 8.

Table 10: Results of the statistical analyses evaluating the correlation between fragmen	nt
volume and degree of joint effusion for the tarsocrural joint	

Correlations				
			VOLhock	EFFhock
Kendall's tau_b	VOLhock	Correlation Coefficient	1,000	,314**
		Sig. (2-tailed)	<u>.</u>	<,001
		N	96	96
	EFFhock	Correlation Coefficient	,314**	1,000
		Sig. (2-tailed)	<,001	
		N	96	96
Spearman's rho	VOLhock	Correlation Coefficient	1,000	,385**
		Sig. (2-tailed)		<,001
		N	96	96
	EFFhock	Correlation Coefficient	,385**	1,000
		Sig. (2-tailed)	<,001	
		N	96	96

\*\*. Correlation is significant at the 0.01 level (2-tailed).

The results of the statistical analyses (table 10) revealed a significant positive correlation between the volume of the fragments and the effusion of the tarsocrural joint:  $\varphi_{b}=0,31$ ; p<0,001 and r<sub>s</sub> (94)= 0,39; p< 0,001.

## 7.3. MCP/MTP JOINT

Data included 132 MCP/MTP joints. No distinction between front- and hindlimbs or type of fragment (OCD/DOF/POF) was made. The mean volume of the fragments elevated from the MCP/MTP joints was ca. 0,10 mL. 112 of 132 (85%) MCP/MTP joints showed no effusion an mean fragment volume for this category was ca. 0,12 mL. 6 of 132 (5%) MCP/MTP joints showed mild effusion and mean fragment volume for this category was ca. 0,14 mL. 11 of 132 (8%) MCP/MTP joints showed moderate effusion and mean fragment volume for this category was ca. 0,14 mL. 11 of 132 (8%) MCP/MTP joints showed moderate effusion and mean fragment volume for this category was ca. 0,16 mL. 3 of 132 (2%) MCP/MTP joints showed severe effusion and mean fragment volume for this category was ca. 0,15 mL. Median fragment volume for all degrees of effusion of the MCP/MPT joints was 0,10 mL.



Figure 16: Violin plot of the fragment volume for the 4 categories of effusion of the MCP/MTP joints. Number of joints, as well as mean and median fragment volumes for each category are provided in table 8

Table 11: Results of the statistical analyses evaluating the correlation between fragment volume and degree of joint effusion for the MCP/MTP joints

Correlations					
			VOLfetlock	EFFfetlock	
Kendall's tau_b	VOLfetlock	Correlation Coefficient	1,000	,112	
		Sig. (2-tailed)	-	,159	
		N	132	132	
	EFFfetlock	Correlation Coefficient	,112	1,000	
		Sig. (2-tailed)	,159		
		N	132	132	
Spearman's rho	VOLfetlock	Correlation Coefficient	1,000	,122	
		Sig. (2-tailed)	-	,163	
		N	132	132	
	EFFfetlock	Correlation Coefficient	,122	1,000	
		Sig. (2-tailed)	,163		
		Ν	132	132	

The results of the statistical analyses (table 11) revealed no significant correlation between the volume of the fragments and the effusion of the MCP/MTP joint:  $\phi_b=0,11$ ; p=0,159 and r<sub>s</sub> (130)= 0,12; p=0,163.

## 8. DISCUSSION

## **8.1. CURRENT LITERATURE**

Several considerations can be made when reviewing current literature regarding the prognosis of the arthroscopic removal of osteochondral fragments.

## 8.1.1. Breed differences

Most studies evaluating the prognosis of arthroscopic fragment removal define a successful outcome as the ability to return to the original or intended use after surgery. Nevertheless, it is important to note that the definition of a favourable outcome might be different depending on the discipline and training level of the horse. In racing breeds, like Thoroughbreds and Standardbreds, soundness is of greatest importance, as the focus lays on the ability of the horse to compete at its maximal athletic capacity. In the sport horse sales industry, where the population mainly consists of Warmbloods, the cosmetic appearance is crucial as well. The horse must look 'clean' on radiographs and in real life; residual joint effusion, visible scars etc. are not accepted.

Another critical concern in terms of differences between breeds and disciplines can be raised when evaluating the long-term prognosis. Thoroughbreds and Standardbreds have a relatively short sports career that starts and ends at a relatively young age. Warmbloods, on the other hand, have a much longer athletic career, with horses often only starting to reach their maximal capacity towards the age of 8-10 years. Therefore, a long-lasting optimal osteoarticular health is of utmost importance in this population.

Existing studies report follow-up time periods ranging from several months to a few years. Given the longer duration of the athletic career of Warmblood sport horses, it is important to implement a more extended follow-up time period in order to evaluate the long-term prognosis for when the horse will reach its maximum athletic capacity. Currently, such longitudinal studies are lacking.

## 8.1.2. Prognosis regarding residual effusion in long term

A second consideration that has to be made is the prognosis of the arthroscopic removal of osteochondral fragments regarding the resolution of effusion of the affected joint(s). Only a few existing studies evaluate the degree of joint distension before the surgery and as a part of the follow-up. In 2009, Brink et al. reported a general decrease in the degree of effusion after arthroscopic removal of OCD fragments in the tarsocrural joint. However, in only 26 of 98 (27%) preoperatively distended joints, a complete resolution was observed, with 5 horses even developing effusion after surgery.

In OC patients, joint effusion is assumed to be caused by synovitis arising due to the presence of the cartilage flaps and/or osteochondral fragments. Joint distension can persist after the arthroscopic fragment removal due to continuing of this primary process and/or as a result of surgical manipulation. Synovial inflammation is associated with an increase in certain enzymes, inflammatory mediators and cytokines. These changes in the synovial fluid influence the nutrition of the articular cartilage, initiate degenerative processes and contribute to physical and biomechanical damage to the articular cartilage (and subchondral bone). If this cycle is not interrupted, it will ultimately lead to the development of osteoarthritis, which can result in further joint degeneration and osteoarthrosis in the long term (McIlwraith, 2016). This implies any residual effusion to play an important role in the prognosis of arthroscopic fragment removal in the Warmblood population, as these sport horses have a long athletic career.

Brink et al. (2009) found joint distension to significantly decrease with increasing time to follow-up and complete resolution could take up to six months following surgery. Nevertheless, most clinicians prefer to not postpone treatment of the remaining effusion with intra-articular corticosteroids more

than 6 weeks after surgery to stop the inflammatory process in the affected joint. Additionally, the duration of the period of joint effusion prior to surgery has been suggested to play a role in the probability of post-operative reduction of effusion, with increasing time decreasing the likelihood of a complete resolution. This is an argument to not delay treatment for more than 6-8 weeks after the first signs of joint distension (Goodrich and McIlwraith, 2008).

## 8.1.3. Problems existing studies

Most existing studies base their success rates on patients returning to the original or intended use after surgery. However, it has to be noted that a lot of horses undergo arthroscopic fragment removal before the age of 3 years old, meaning they are still unbroken and have yet to start training. Inevitably, a certain percentage of these youngsters will not be able to perform on the level they are intended to, based on their genetic profile, regardless of the presence of OC lesions. A lack of natural athletic ability or development of other (orthopaedic) problems making the horse unsuitable for the sport can be reasons behind this inability to perform at the intended level. The variety of competition types and levels are an additional factor complicating the comparison of performance in sport horses (Uprichard et al., 2013).

As the arthroscopic removal of osteochondral fragments has become routine practice during the past decades, not many recent studies investigating the prognosis of surgical treatment exist. The non-acceptance of fragments on the X-rays of sport horses makes it difficult to conduct studies evaluating the prognosis of conservative treatment or compare cases that undergo arthroscopic fragment removal to controls with similar OC lesions that do not undergo surgery (Brink et al., 2009).

## 8.2. RESULTS STATISTICAL ANALYSES

## 8.2.1. Degree of joint effusion

For the femoropatellar and tarsocrural joint, effusion of a variable degree was present in more than half of the affected joints (58% and 69% respectively). Distribution of joints across the different categories of effusion was similar for both of these joints. While smaller numbers of femoropatellar and tarsocrural joints showed mild (12% and 14% respectively) or moderate (16% and 24% respectively) distension, a significant proportion showed severe effusion (30% and 31% respectively). Not many past studies report the presence and degree of effusion, nor include a significant proportion of Warmbloods in the study population. This makes it difficult to compare the results of this thesis with those reported in existing literature. Brink et al. (2009) investigated the outcome of arthroscopic fragment removal for tarsocrural OC in a mixed study population mainly consisting of Warmbloods and Standardbreds. This study reported different numbers regarding the preoperative effusion compared to our findings. The author found 87% of tarsocrural joints to show effusion of a varying degree. Most of these joints showed mild (45%) or moderate (26%) effusion, and a smaller number of joints (16%) showed severe effusion. Regardless of these differences in distribution across the different categories of effusion, it is undeniable that joint effusion is a common clinical sign of OC(D) of the femoropatellar and tarsocrural joints in the Warmblood sport horse population.

For the metacarpo-/metatarsophalangeal joints on the other hand, the majority of affected joints (85%) showed no effusion. If distension was present, it was mostly of a mild or moderate degree, and only 2% of MCP/MTP joints showed severe effusion. Our results confirm the general assumption that joint effusion is much less common in the MCP/MTP joints and fetlock osteochondral fragments are often only detected on radiographic screening of asymptomatic horses (Declercq et al., 2011).

## 8.2.2. Correlation between fragment volume and degree of joint effusion

The mean fragment volume was largest in the femoropatellar and tarsocrural joint (ca. 0,68 mL and ca. 0,48 mL respectively). Additionally, for both of these joints, a significant positive correlation between the volume of the fragments and the degree of effusion was found; i.e. an increasing volume was measured from fragments elevated from joints presenting with a more severe degree of effusion. This implies larger fragments to be associated with larger underlying lesions and/or more extensive intra-articular damage, leading to more synovial inflammation. For the MCP/MPT joints, the mean fragment volume was much smaller (ca. 0,12 mL) and the fragment volumes were less spread. This could explain the fact that joint effusion was much less common in the MCP/MTP joints. Unsurprisingly, no significant correlation between the volume of the fragments and the degree of effusion of the MCP/MTP joints was found.

To date, the volume of the fragments elevated from joints affected with OCD and associated correlations have not been investigated yet. Most existing studies evaluate the underlying lesion in the articular cartilage and subchondral bone rather than the fragment(s) elevated from the affected joint (Foland et al., 1992; McIlwraith et al., 1991; Uprichard et al., 2013). It would have been better to make a distinction between the different types of fetlock osteochondral fragments. This would allow for comparison between fetlock OCD, DOF and POF.

## 8.2.3. Considerations

Several concerns can be made when evaluating the design of the present study. To provide consistency in the patient assessment and avoid interobserver variation (Keegan et al., 2010), the orthopaedic examination of all horses included in the study population was performed by the same clinician. As most patients presenting for arthroscopic removal of osteochondral fragments are young and sometimes not used to being handled, a complete and standardized orthopaedic examination including flexion tests was not possible in all cases. A second consideration that has to be taken into account is the possibility of mistakes being made during the acquisition of the fragments. Although the protocol was very simple, it could have happened that (parts of) fragments were lost or that the plastic containers used to store the fragments were labeled incorrectly.

## 9. CONCLUSION

Current literature reports the prognosis for the arthroscopic removal of osteochondral fragments (OCD, DOF, POF) to be fair to good, depending on the affected joint and the extent of the lesions (McIlwraith, 2013). Nevertheless, most existing studies investigate populations mainly consisting of Thoroughbreds and Standardbreds, and define a successful outcome as the ability to perform at the intended level. However, in the Warmblood sport horse industry, the cosmetic aspect is important as well and the duration of the horse's athletic career is much longer compared to that of the racing breeds. Therefore, resolution of joint effusion plays a crucial role in determining the prognosis of the arthroscopic fragment removal in this population. Additionally, there is a need for longitudinal studies investigating the implications of residual effusion on the horse's joint health in the long term.

The results of this thesis reveal joint effusion to be present in 58% of femoropatellar and 69% of tarsocrural joints affected with OCD in a study population mainly consisting of Warmbloods. For the MCP/MTP joints on the other hand, only 15% of joints containing (an) osteochondral fragment(s) were distended upon orthopaedic examination. The fragment volumes were more spread and the mean fragment volume was larger in the femoropatellar and tarsocrural joints compared to the MCP/MTP joints. Furthermore, for both the femoropatellar and the tarsocrural joints, a significant positive correlation between the volume of the fragments and the degree of effusion was found, implying larger fragments to lead to more synovial inflammation.

This thesis will contribute to a larger study where the aim is to categorise OC lesions and investigate the correlation between joint effusion, volume of the fragments and arthroscopic changes in the joint. The results of this study will be part of a research project in which the goal is to redefine the prognosis of the arthroscopic removal of osteochondral fragments (OCD, DOF, POF) in the Warmblood sport horse population.

## **10. REFERENCES**

- Alvarado, A.F., Marcoux, M., Breton, L., 1990. The incidence of osteochondrosis in a Standardbred breeding farm in Quebec. Proceedings of the Annual Convention of the American Association of Equine Practitioners, 293–307.
- Baccarin, R.Y.A., Pereira, M.A., Roncati, N.V., Bergamaschi, R.R.C., Hagen, S.C.F., 2012. Development of osteochondrosis in Lusitano foals: A radiographic study. Canadian Veterinary Journal 53, 1079-1084.
- Beard, W.L., Bramlage, L.R., Schneider, R.K., Embertson, R.M., 1994. Postoperative racing performance in standardbreds and thoroughbreds with osteochondrosis of the tarsocrural joint: 109 cases (1984-1990). Journal of the American Veterinary Medical Association 204, 1655-1659.
- Borg, H., Carmalt, J.L., 2013. Postoperative septic arthritis after elective equine arthroscopy without antimicrobial prophylaxis. Veterinary Surgery 42, 262–266.
- Bourzac, C., Alexander, K., Rossier, Y., Laverty, S., 2009. Comparison of radiography and ultrasonography for the diagnosis of osteochondritis dissecans in the equine femoropatellar joint. Equine Veterinary Journal 41, 685-692.
- Brink, P., Dolvik, N.I., Tverdal, A., 2009. Lameness and effusion of the talocrural joints after arthroscopy of osteochondritis dissecans in horses. Veterinary Record 165, 709-712.
- Brunsting, J.Y., Pille, F.J., Oosterlinck, M., Haspeslagh, M., Wilderjans, H.C., 2018. Incidence and risk factors of surgical site infection and septic arthritis after elective arthroscopy in horses. Veterinary Surgery 47, 52–59.
- Butler, J.A., Colles, C.M., Dyson, S.J., Kold, S.E., Poulos, P.W., 2000. The stifle and tibia. Clinical radiology of the horse 3, 363–412.
- Carlson, C.S., Cullins, L.D., Meuten, D.J., 1995. Osteochondrosis of the articular-epiphyseal cartilage complex in young horses: evidence for a defect in cartilage canal blood supply. Veterinary Pathology 32, 641–647.
- Carlson, C.S., Meuten, D.J., Richardson, D.C., 1991. Ischemic necrosis of cartilage in spontaneous and experimental lesions of osteochondrosis. Journal of Orthopaedic Research 9, 317–329.
- Carlsten, J., Sandgren, B., Dalin, G., 1993. Development of osteochondrosis in the tarsocrural joint and osteochondral fragments in the fetlock joints of Standardbred trotters. I. A radiological survey. Equine Veterinary Journal 25, 42-47.
- Carmalt, J.L., Borg, H., Näslund, H., Waldner, C., 2014. Racing performance of Swedish standardbred trotting horses with proximal palmar/plantar first phalangeal (Birkeland) fragments compared to fragment free controls. Veterinary Journal 202, 43–47.
- Carmona, J.U., Argüelles, D., Deulofeu, R., Martínez-Puig, D., Prades, M., 2009. Effect of the administration of an oral hyaluronan formulation on clinical and biochemical parameters in young horses with osteochondrosis. Veterinary and Comparative Orthopaedics and Traumatology 22, 455-459.
- Clarke, K.L., Reardon, R., Russell, T., 2015. Treatment of osteochondrosis dissecans in the stifle and tarsus of juvenile thoroughbred horses. Veterinary Surgery 44, 297–303.

- Colón, J.L., Bramlage, L.R., Hance, S.R., Embertson, R.M., 2000. Qualitative quantitative documentation of the racing performance of 461 Thoroughbred racehorses after arthroscopic removal of dorsoproximal first phalanx osteochondral fractures (1986-1995). Equine Veterinary Journal 32, 475–481.
- Dalin, G., Sandgren, B., Carlsten, J., 1993. Plantar osteochondral fragments in the metatarsophalangeal joints in Standardbred trotters; result of osteochondrosis or trauma? Equine Veterinary Journal 25, 62–65.
- Declercq, J., Hauspie, S., Saunders, J., Martens, A., 2011. Osteochondral fragments in the metacarpoand metatarsophalangeal joint and their clinical importance. Vlaams Diergeneeskundig Tijdschrift 80, 271-280.
- Declercq, J., Martens, A., Maes, D., Boussauw, B., Forsyth, R., Boening, K.J., 2009. Dorsoproximal proximal phalanx osteochondral fragmentation in 117 Warmblood horses. Veterinary and Comparative Orthopaedics and Traumatology 22, 1–6.
- Desjardin, C., Chat, S., Gilles, M., Legendre, R., Riviere, J., Mata, X., Balliau, T., Esquerré, D., Cribiu, E.P., Betch, J.M. et al., 2014. Involvement of mitochondrial dysfunction and ER-stress in the physiopathology of equine osteochondritis dissecans (OCD). Experimental and Molecular Pathology 96, 328–338.
- Dik, K.J., Enzerink, E., van Weeren, P.R., 1999. Radiographic development of osteochondral abnormalities, in the hock and stifle of Dutch Warmblood foals, from age 1 to 11 months. Equine Veterinary Journal Supplement, 9-15.
- Distl, O., 2013. The genetics of equine osteochondrosis. Veterinary journal 197, 13–18.
- Donabédian, M., Fleurance, G., Perona, G., Robert, C., Lepage, O., Trillaud-Geyl, C., Leger, S., Ricard, A., Bergero, D., Martin-Rosset, W., 2006. Effect of fast vs. moderate growth rate related to nutrient intake on developmental orthopaedic disease in the horse. Animal research 55, 471–486.
- Drabbe, A., Janssens, S., Blott, S., Ducro, B.J., Fontanel, M., Francois, L., Schurink, A., Stinckens, A., Lindgren, G., Van Mol, B. et al., 2022. Genome-wide association analyses of osteochondrosis in Belgian Warmbloods reveal candidate genes associated with chondrocyte development. Journal of Equine Veterinary Science 111. https://doi.org/10.1016/j.jevs.2022.103870
- Foland, J.W., McIlwraith, C.W., Trotter, G.W., 1992. Arthroscopic surgery for osteochondritis dissecans of the femoropatellar joint of the horse. Equine Veterinary Journal 24, 419–423.
- Fortier, L.A., Foerner, J.J., Nixon, A.J., 1995. Arthroscopic removal of axial osteochondral fragments of the plantar/palmar proximal aspect of the proximal phalanx in horses: 119 cases (1988-1992). Journal of the American Veterinary Medical Association 206, 71–74.
- Goodrich, L.R., McIlwraith, C.W., 2008. Complications associated with equine arthroscopy. Veterinary Clinics of North America Equine Practice 24, 573-589.
- Gorissen, B.M.C., Wolschrijn, C.F., Serra Bragança, F.M., Geerts, A.A.J., Leenders, W.O.J.L., Back, W., van Weeren, P.R., 2017. The development of locomotor kinetics in the foal and the effect of osteochondrosis. Equine Veterinary Journal 49, 467-474.

- Hilla, D., Distl, O., 2014. Heritabilities and genetic correlations between fetlock, hock and stifle osteochondrosis and fetlock osteochondral fragments in Hanoverian Warmblood horses. Journal of Animal Breeding and Genetics 131, 71–81.
- Hoppe, F., 1984. Radiological investigations of osteochondrosis dissecans in Standardbred Trotters and Swedish Warmblood horses. Equine Veterinary Journal 16, 425-429.
- Hoppe, F., Philipsson, J., 1985. A genetic study of osteochondrosis dissecans in Swedish horses. Equine practice (USA) 7, 7-15.
- Hurtig, M., Pool, R., 1996. Pathogenesis of equine osteochondrosis. In: Joint Disease in the Horse, Second Edn., W.B. Saunders Co., Philadelphia, pp. 335–358.
- Jacquet, S., Robert, C., Valette, J.P., Denoix, J.M., 2013. Evolution of radiological findings detected in the limbs of 321 young horses between the ages of 6 and 18 months. Veterinary Journal 197, 58–64.
- Kaneps, A.J., 2014. 13 Diagnosis of lameness. In: Equine Sports Medicine and Surgery, Second Edn., W.B. Saunders Co., Philadelphia, pp. 239–251.
- Kawcak, C.E., McIlwraith, C.W., 1994. Proximodorsal first phalanx osteochondral chip fragmentation in 336 horses. Equine Veterinary Journal 26, 392–396.
- Keegan, K.G., Dent, E. V., Wilson, D.A., Janicek, J., Kramer, J., Lacarrubba, A., Walsh, D.M., Cassells, M.W., Esther, T.M., Schiltz, P. et al., 2010. Repeatability of subjective evaluation of lameness in horses. Equine Veterinary Journal 42, 92–97.
- Khamis, H., 2008. Measures of association: How to choose? Journal of Diagnostic Medical Sonography 24, 155–162.
- Kinsley, M.A., Semevolos, S.A., Duesterdieck-Zellmer, K.F., 2015. Wnt/β-catenin signaling of cartilage canal and osteochondral junction chondrocytes and full thickness cartilage in early equine osteochondrosis. Journal of Orthopaedic Research 33, 1433–1438.
- Laverty, S., Girard, C., 2013. Pathogenesis of epiphyseal osteochondrosis. Veterinary Journal 197, 3-12.
- Laws, E.G., Richardson, D.W., Ross, M.W., Moyer, W., 1993. Racing performance of Standardbreds after conservative and surgical treatment for tarsocrural osteochondrosis. Equine Veterinary Journal 25, 199-202.
- Lecocq, M., Girard, C.A., Fogarty, U., Beauchamp, G., Richard, H., Laverty, S., 2008. Cartilage matrix changes in the developing epiphysis: Early events on the pathway to equine osteochondrosis? Equine Veterinary Journal 40, 442–454.
- Lepeule, J., Bareille, N., Robert, C., Ezanno, P., Valette, J. P., Jacquet, S., Blanchard, G., Denoix, J. M., Seegers, H., 2009. Association of growth, feeding practices and exercise conditions with the prevalence of Developmental Orthopaedic Disease in limbs of French foals at weaning. Preventive Veterinary Medicine 89, 167–177.
- Lepeule, J., Bareille, N., Valette, J.P., Seegers, H., Jacquet, S., Denoix, J.M., Robert, C., 2008. Developmental orthopaedic disease in limbs of foals: Between-breed variations in the prevalence, location and severity at weaning. Animal 2, 284–291.

- McIlwraith, C. W., 2016. 3 Traumatic Arthritis and Posttraumatic Osteoarthritis in the Horse. In: Joint Disease in the Horse, Second Edn. W.B. Saunders, Edinburgh, pp. 33–48.
- McIlwraith, C.W., 2013. Surgical versus conservative management of osteochondrosis. Veterinary Journal 197, 19-28.
- McIlwraith, C.W., 1993. Inferences from referred clinical cases of osteochondritis dissecans. Equine Veterinary Journal 25, 27–30.
- McIlwraith, C.W., Vorhees, M., 1990. Management of osteochondritis dissecans of the dorsal aspect of the distal metacarpus and metatarsus. Proceedings of the American Association of Equine Practitioners. Annual Meeting, 547–550.
- McIlwraith, C W., Nixon, A.J., Wright, I.M., 2015. Chapter 5 Diagnostic and Surgical Arthroscopy of the Metacarpophalangeal and Metatarsophalangeal Joints. In: Diagnostic and Surgical Arthroscopy in the Horse, Fourth Edn. Mosby, pp. 111–174.
- McIlwraith, C. Wayne, Nixon, A.J., Wright, I.M., 2015. Problems and Complications of Diagnostic and Surgical Arthroscopy. In: Diagnostic and Surgical Arthroscopy in the Horse, Elsevier, pp. 419–425.
- McIlwraith, Foerner, J.J., Davis, D.M., 1991. Osteochondritis dissecans of the tarsocrural joint: results of treatment with arthroscopic surgery. Equine Veterinary Journal 23, 155-162.
- McIntosh, S.C., McIlwraith, C.W., 1993. Natural history of femoropatellar osteochondrosis in three crops of Thoroughbreds. Equine Veterinary Journal 25, 54-61.
- Mendoza, L., Lejeune, J.P., Caudron, I., Detilleux, J., Sandersen, C., Deliège, B., Serteyn, D., 2016.
   Impact of feeding and housing on the development of osteochondrosis in foals-A longitudinal study. Preventive Veterinary Medicine 127, 10–14.
- Mirams, M., Tatarczuch, L., Ahmed, Y.A., Pagel, C.N., Jeffcott, L.B., Davies, H.M.S., Mackie, E.J., 2009. Altered gene expression in early osteochondrosis lesions. Journal of Orthopaedic Research 27, 452–457.
- Nixon, A.J., Fortier, L.A., Goodrich, L.R., Ducharme, N.G., 2004. Arthroscopic reattachment of osteochondritis dissecans lesions using resorbable polydioxanone pins. Equine Veterinary Journal 36, 376-383.
- Nixon, A.J., Pool, R.R., 1995. Histologic appearance of axial osteochondral fragments from the proximoplantar/proximopalmar aspect of the proximal phalanx in horses. Journal of the American Veterinary Medical Association 207, 1076—1080.
- O'Donohue, D.D., Smith, F.H., Strickland, K.L., 1992. The incidence of abnormal limb development in the Irish Thoroughbred from birth to 18 months. Equine Veterinary Journal 24, 305–309.
- Olds, A.M., Stewart, A.A., Freeman, D.E., Schaeffer, D.J., 2006. Evaluation of the rate of development of septic arthritis after elective arthroscopy in horses: 7 cases (1994–2003). . Journal of the American Veterinary Medical Association 229, 1949–1954.
  - Olstad, K., Cnudde, V., Masschaele, B., Thomassen, R., Dolvik, N.I., 2008. Micro-computed tomography of early lesions of osteochondrosis in the tarsus of foals. Bone 43, 574–583.

- Olstad, K., Hendrickson, E.H.S., Carlson, C.S., Ekman, S., Dolvik, N.I., 2013. Transection of vessels in epiphyseal cartilage canals leads to osteochondrosis and osteochondrosis dissecans in the femoro-patellar joint of foals; a potential model of juvenile osteochondritis dissecans. Osteoarthritis Cartilage 21, 730–738.
- Ortved, K.F., 2017. Surgical Management of Osteochondrosis in Foals. Veterinary Clinics of North America - Equine Practice 33, 379-396.
- Peremans, K., Verschooten, F., 1997. Results of conservative treatment of osteochondrosis of the tibiotarsal joint in the horse. Journal of Equine Veterinary Science 17, 322–326.
- Pool, R.R., 1993. Difficulties in definition of equine osteochondrosis; differentiation of developmental and acquired lesions. Equine Veterinary Journal 25, 5–12.
- Poulos, P., 1986. Radiologic manifestations of developmental problems. In: AQHA Developmental Orthopedic Disease Symposium, American Quarter Horse Association Amarillo, TX, pp. 1–2.
- Praud, A., Dufour, B., Robert, C., Valette, J.P., Denoix, J.M., Crevier-Denoix, N., 2013. Effects of management practices as risk factors for juvenile osteochondral conditions in 259 French yearlings. Veterinary Journal 197, 72–76.
- Riddick, T.L., Duesterdieck-Zellmer, K., Semevolos, S.A., 2012. Gene and protein expression of cartilage canal and osteochondral junction chondrocytes and full-thickness cartilage in early equine osteochondrosis. Veterinary Journal 194, 319–325.
- Sandgren, B., Dalin, G., Carlsten, J., 1993. Osteochondrosis in the tarsocrural joint and osteochondral fragments in the fetlock joints in Standardbred trotters. I. Epidemiology. Equine Veterinary Journal 25, 31-37.
- Savage, C.J., McCarthy, R.N., Jeffcott, L.B., 1993. Effects of dietary energy and protein on induction of dyschondroplasia in foals. Equine Veterinary Journal 25, 74–79.
- Schougaard, H., Ronne, J.F., Phillipson, J., 1990. A radiographic survey of tibiotarsal osteochondrosis in a selected population of trotting horses in Denmark and its possible genetic significance. Equine Veterinary Journal 22, 288-289.
- Seitzinger, A.H., Traub-Dargatz, J.L., Kane, A.J., Kopral, C.A., Morley, P.S., Garber, L.P., Wc, L., Hill, G.W., 2000. A comparison of the economic costs of equine lameness, colic, and equine protozoal myeloencephalitis (EPM). In Proceedings of the 9th International Symposium on Veterinary Epidemiology and Economics, Breckenridge, CO, USA, pp. 1–4.
- Semevolos, S.A., 2017. Osteochondritis Dissecans Development. Veterinary Clinics of North America Equine Practice 33, 367–378.
- Semevolos, S.A., Brower-Toland, B.D., Bent, S.J., Nixon, A.J., 2002. Parathyroid hormone-related peptide and Indian hedgehog expression patterns in naturally acquired equine osteochondrosis. Journal of Orthopaedic Research 20, 1290–1297.
- Senior, J.M., 2013. Morbidity, Mortality, and Risk of General Anesthesia in Horses. Veterinary Clinics of North America: Equine Practice 29, 1–18.
- Senior, J.M., Pinchbeck, G.L., Allister, R., Dugdale, A.H.A., Clark, L., Clutton, R.E., Coumbe, K., Dyson,
   S., Clegg, P.D., 2007. Reported morbidities following 861 anaesthetics given at four equine hospitals. Veterinary Record 160, 407–408.

- Seo, J., Tanabe, T., Tsuzuki, N., Haneda, S., Yamada, K., Furuoka, H., Tabata, Y., Sasaki, N., 2013.
   Effects of bilayer gelatin/β-tricalcium phosphate sponges loaded with mesenchymal stem cells, chondrocytes, bone morphogenetic protein-2, and platelet rich plasma on osteochondral defects of the talus in horses. Research in Veterinary Science 95, 1210–1216.
- Sparks, H.D., Nixon, A.J., Fortier, L.A., Mohammed, H.O., 2011. Arthroscopic reattachment of osteochondritis dissecans cartilage flaps of the femoropatellar joint: Long-term results. Equine Veterinary Journal 43, 650-659.
- Steinheimer, D.N., McIlwraith, C.W., Park, R.D., Steyn, P.F., 1995. Comparison of radiographic subchondral bone changes with arthroscopic findings in the equine femoropatellar and femorotibial joints: a retrospective study of 72 joints (50 horses). Veterinary Radiology & Ultrasound 36, 478–484.
- Uprichard, K., Elce, Y.A., Piat, P., Beauchamp, G., Laverty, S., Yvonne, A., 2013. Outcome after arthroscopic treatment of lateral femoral trochlear ridge osteochondrosis in sport horses;a retrospective study of 37 horses. Veterinary and Comparative Orthopaedics and Traumatology 26, 105-109.
- Van Grevenhof, E.M., Ducro, B.J., Van Weeren, P.R., Van Tartwijk, J.M.F.M., Van Den Belt, A.J., Bijma,
   P., 2009. Prevalence of various radiographic manifestations of osteochondrosis and their correlations between and within joints in Dutch Warmblood horses. Equine Veterinary Journal 41, 11-16.
- van Weeren, P R., 2019. Chapter 89 Osteochondritis Dissecans. In: Equine Surgery, Fifth Edn. W.B. Saunders, pp. 1509–1528.
- van Weeren, P.R., Barneveld, A., 1999. Study design to evaluate the influence of exercise on the development of the musculoskeletal system of foals up to age 11 months. Equine Veterinary Journal Supplement, 4-8.
- van Weeren, P.R., Barneveld, A., 1999. The effect of exercise on the distribution and manifestation of osteochondrotic lesions in the Warmblood foal. Equine Veterinary Journal 31, 16–25.
- van Weeren, P.R., Denoix, J.M., 2013. The Normandy field study on juvenile osteochondral conditions: Conclusions regarding the influence of genetics, environmental conditions and management, and the effect on performance. Veterinary Journal 197, 90–95.
- van Weeren, P.R., Knaap, J., Firth, E.C., 2003. Influence of liver copper status of mare and newborn foal on the development of osteochondrotic lesions. Equine Veterinary Journal 35, 67–71.
- Vander Heyden, L., Lejeune, J.P., Caudron, I., Detilleux, J., Sandersen, C., Chavatte, P., Paris, J., Deliège, B., Serteyn, D., 2013. Association of breeding conditions with prevalence of osteochondrosis in foals. Veterinary Record 172, 68
- Verwilghen, D.R., Janssens, S., Busoni, V., Pille, F., Johnston, C., Serteyn, D., 2013. Do developmental orthopaedic disorders influence future jumping performances in Warmblood stallions? Equine Veterinary Journal 45, 578-581.
- Vos, N.J., 2008. Incidence of osteochondrosis (dissecans) in Dutch Warmblood horses presented for pre-purchase examination. Irish Veterinary Journal 61, 33-37.
- Whitton, R.C., 1998. Equine developmental osteochondral lesions: the role of biomechanics. Veterinary Journal 156, 167–168.

- Wright, I.M., Minshall, G.J., 2014. Identification and treatment of osteochondritis dissecans of the distal sagittal ridge of the third metacarpal bone. Equine Veterinary Journal 46, 585–588.
- Ytrehus, B., Carlson, C.S., Ekman, S., 2007. Etiology and pathogenesis of osteochondrosis. Veterinary Pathology 44, 429-448.