EVALUATION OF TWO TECHNIQUES OF CANCELLOUS BONE GRAFTING OF EXPERIMENTAL SUBCHONDRAL BONE CYSTS IN THE MEDIAL FEMORAL CONDYLES OF HORSES

by

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(ABSTRACT)

Cylindric (10 mm diameter X 15 mm deep) osteochondral defects were created in the medial femoral condyles of 5 horses to mimic clinical cases of subchondral bone cysts after debridement. The defects were created with either a standard square ended drill bit or a compaction drill bit. The compaction drill bit compresses bone laterally and distally creating a dense wall and floor. Twelve-mm sternal cancellous cylinders were compressed to 9.25 mm and inserted into the femoral defects where they were presumed to expand and tighten the fit. The end result was sternal cancellous bone that exactly fit the femoral defects. Fluorochrome bone labels were used to confirm the origin of bone present in the defects at necropsy, which was performed after 6 months. Successful graft incorporation occurred in 3 of the compacted and 2 of the noncompacted defects. The surfaces of the successful cancellous bone grafts contained predominately fibrocartilage. The unsuccessful noncompacted defects expanded laterally and deeply into the parent bone epiphysis while the unsuccessful compacted defects remained confined to the originally created size.

TABLE OF CONTENTS

INTRODUCTION

Subchondral bone cysts (SBCs) are an important source of lameness in young horses.[1-9] They occur most commonly in the medial femoral condyle but have been reported in all joints of the equine limbs.[4,5,9,10] The pathogenesis of subchondral bone cysts remains a question. Currently most accepted theories include osteochondrosis,[2,5,10-13] traumatic disruption of articular cartilage[14,15] with extrusion of synovial fluid into the subchondral bone or both[1].

Conservative therapy has consisted of variable periods of stall rest.[4,5,7] Although some authors have been reported resolution of lameness,[4,7,16] some discrepancy exists between definitions of athletic soundness. Conservative therapy has not produced good overall results as lameness often returns at a critical level of work.[3,5,6,9,17]

Early surgical interventions used a supracondylar approach to access the cyst from outside the joint followed by introduction of a cancellous bone graft;[18] however, no results were reported. Success has been attained following curettage of the cysts via arthrotomy or arthroscopy.[3,6,9,17] The current recommendation is to evacuate the cyst contents, its fibrous lining, and to expand the opening to the margins of substantial subchondral bone.

Multiple drilling of the debrided cystic margins (forage) to expose the cavity to increased osteogenic potential has resulted in enlargement of the cyst or development of secondary cysts.[3] Synovial fluid hydraulic pressure through cartilage defects has been

incriminated in the pathogenesis of subchondral cystic defect, and it may play a role in these secondary postoperative changes.[14,15,19] Based on radiographs, subchondral cysts do not fill or only partially fill with bone postoperatively.[3,4,6,9,15,17] The defects are filled with fibrous connective tissue and fibrocartilage[9,20] which optimally eliminates synovitis and deeper bone pain.

Overall results following simple curettage of the lesions in athletic horses is not totally effective[3,9] increasing the need for an alternative treatment such as a predictable grafting procedure. Packing curetted defects with cancellous bone fragments has yielded success rates similar to those reported for debridement alone.[21,22] Because these were clinical cases, there was no control and only radiographic evaluation of healing. Three of the five horses destined to race were sound and back in full race training after 1 year.

A logical approach to achieve the best possible healing of debrided defects would be to use a single perfectly fitting autogenous cancellous bone graft. Such a graft could provide osteogenic potential and a stable scaffold for the ingrowth of new vessels.[23-27] Using such a graft, very good results were obtained when the graft healed primarily.[28] However, in some horses apparent leakage of synovial fluid between the graft and parent bone caused lysis of both surfaces and loss of the graft.[28] When the grafts healed well, a good quality cartilage formed on the surface of the grafted cancellous bone plugs. Clinical and experimental experience point to the importance of intact subchondral bone for the formation of an articular surface.[29,30]

Assuming the failure of some grafts to heal was due to leakage of synovial fluid

around the grafts, methods have been sought to seal the host-graft interface. Compaction drill bits (Compaction Drill Bits, Instrument Makar, Inc, Okemos, MI) are specialized orthopedic drills designed to displace the bone rather than remove it from the implant site. The displaced bone becomes densely compacted on the sides and floor of the defect. Compaction Pliers (Compaction Pliers, Instrument Makar, Inc, Okemos, MI) shape and compress the graft ensuring a circular perfectly sized graft for placement in the recipient site. The final compaction size is .75 mm smaller than the compaction hole permitting easy insertion of the graft and allows for bone memory (expansion) to tighten the fit postoperatively. We propose that this could be used in clinical cases by enlarging subchondral cystic lesions into a cylinder using the appropriate drill to receive the fitted autogenous cancellous plugs.

This project was designed to evaluate the healing of compacted cancellous bone cylinders when placed into experimentally induced medial femoral condylar defects which were created using conventional or compaction drilling. Hypothetically, the articular surface formed on a stable graft surface with active mesenchymal elements would be superior to that which forms after healing of a defect a centimeter or larger in diameter and depth.

LITERATURE REVIEW

SUBCHONDRAL BONE CYSTS

Types Of Bone Cysts

Three types of bone cysts have been described in the horse, and each of which represents a distinct clinical entity.[1] Subchondral bone cysts (SBCs), also referred to as subchondral cystic lesions or osseous cyst-like lesions, occur in multiple joints in horses;[1-9,11,13,17,18,21,22,31-45] primary aneurysmal and unicameral bone cysts are reported infrequently.[46-48] True bone cysts are defined as a closed cavity lined by epithelium.[49] Subchondral bone cysts which are not true bone cysts occur in all joints, but the weight bearing surface of the medial femoral condyle is the most common site.[1,2,5,7-9,17] The lesion contains synovial fluid or a mixture of semisolid detritus and loose fibrous tissue surrounded by a peripheral dense fibrous lining adjoining active fibroplasia and capillary proliferation.[1,9,20] Most subchondral bone cysts communicate with the joint through a cartilage defect.[5,7,9,20]

Aneurysmal bone cysts are not articular but are true bone cysts that usually affect the metaphysis of long bones. They are expansile lesions consisting of anastomosing cavernous spaces filled with unclotted blood and lined with fibrous walls.[50] These lesions develop secondary to a preexisting lesion such as a hematoma, bleeding disorder,

neoplasia or fibrous dysplasia.[1,48]

Unicameral bone cysts are solitary single chambered intraosseous cysts lined by thin connective tissue membranes.[51] Unicameral bone cysts may develop from encapsulation of focal intramedullary hemorrhage or from a defect in metaphyseal endochondral ossification.[52] Like aneurysmal bone cysts these lesions typically affect the metaphysis of long bones.

Clinical Findings

Subchondral cystic lesions in the medial femoral condyle have been described in horses five months to15 years of age.[5,9] However, 60-70 % of affected horses are between the ages of 1-3 years.[3-5,9] The importance of these lesions as a cause of hindlimb lameness in young horses has been clearly established.[4,7-9,17,20,36] In one report of 86 cases of stifle lameness, 38% were attributed to subchondral bone cysts.[36] Of the 38%, the weight bearing surface of the medial femoral condyle accounted for 90%, and the lesions communicated with the joint.[36] Lameness varies from mild to severe and may be intermittent. In one study, the onset of lameness was associated with trauma in 30% of horses, and another 30% coincided with breaking-in or the commencement of training.[4] The most recent retrospective study reported bilateral radiographic lesions in 66% of the horses.[3]

Synovial effusion is not a feature, because the femorotibial joint has no room for expansion.[5] Approximately 65% of adult horses have communication between the medial

femorotibial and femoropatellar joints, and approximately 17.5% between the lateral femorotibial and femoropatellar joints.[53] However, femorotibial effusion does not always reach the femoropatellar joint.

The significance of cystic lesions must be based on localization of pain to the involved joint as not all cystic lesions cause pain. The effectiveness of diagnostic intraarticular anesthesia with cystic lesions is variable.[1,2,17] However, after injecting 20-30 ml of local anesthetic into the medial femorotibial joint a 50% or greater improvement should be considered significant.[1,17] Subchondral bone sensation may respond more slowly to local anesthetic and repeating the lameness examination one hour after administering intraarticular anesthesia has been suggested.[2]

Diagnosing subchondral bone cysts requires high quality radiographs.[1,5,11] Lesions in the medial femoral condyle are best demonstrated in the caudal to cranial view.[1,5,11] Flexed lateral and posterior to anterior lateral to medial (PALM) oblique views are also useful and easier to obtain using portable radiograph machines.[1,11] Condylar SBCs have been classified by size and radiographic appearance. [3,9] Type 1 lesions are ≤10 mm deep, and appear as shallow saucer or dome-shaped lucent areas confluent with a flattened joint surface. Type 2 lesions are >10 mm deep and are typically domed, conical or spherical lucent areas with a narrow radiographically lucent tract connecting the cyst to the articular surface. Type 1 lesions may progress to Type 2 lesions.[9,11] Type 3 lesions are a flattened or irregular contour of the subchondral bone [3] and are often seen contralateral to deeper SBCs.

Pathogenesis

Subchondral bone cysts have been reported in horses for almost 30 years, yet the exact cause remains unclear;[1,2,4,5,8,10,12,20,41,54] theories have implicated osteochondrosis, articular surface trauma or a combination of both.[1] In 1975 Rooney described subchondral bone cysts as one of two manifestations of osteochondrosis based on location in the joint.[13] He referred to SBCs as "nonmarginal osteochondrosis" because they occurred on the weight bearing surface of the joint and osteochondritis dissecans as "marginal" or nonweightbearing lesions.

Osteochondrosis is a general term for a syndrome in young animals, which results from a defect in endochondral ossification. In the developing fetus and neonate the cartilaginous epiphysis and growth plate are confluent, however, shortly after birth a distinct center of ossification develops in the epiphysis, separating the metaphyseal growth plate and the articular cartilage.[55] The growth cartilage of the epiphysis is interposed between the articular cartilage of the joint surface and the epiphyseal bone.[56] Therefore, the cartilage covering the end of a long bone in a growing animal is actually composed of both epiphyseal growth cartilage and articular cartilage and has been called the articular-epiphyseal (A-E) cartilage complex.[11,56] In immature animals the articular cartilage and epiphyseal growth cartilage are morphologically distinct, with the articular cartilage lacking cartilage canals.[56] The articular cartilage persists in the adult, while the epiphyseal cartilage gradually ossifies, resulting in a layer of calcified cartilage and underlying

subchondral bone. The first change observed in osteochondrosis is focal growth cartilage thickening from retention of the hypertrophied zone and failure of mineralization.[5,57] Rejno and Stromburg identified the four essential features of osteochondrosis as cartilage thickening, degeneration, necrosis, and disturbance of endochondral ossification, all of which have been identified in SBCs in the femoral condyle.[41]

Cartilage retention and thickening in the epiphyseal cartilage at weight bearing sites may lead to infolding of the cartilage.[5,6,58,59] Degeneration and necrosis may follow in the deeper layers of the thickened, retained cartilage.[58] As the epiphysis matures, disturbed ossification becomes radiographically evident when the defect becomes surrounded by normal bone formation. Supporting a role of osteochondrosis is the higher incidence of subchondral cystic lesions seen in young horses and the occasional presence of OCD lesions adjacent to the SBC or in the same or contralateral joint of some horses.[3]

Subchondral bone cysts have been diagnosed following known joint trauma which supports the possibility that SBCs may be acquired in adults suffering articular cartilage defects.[60] Mature adult horses in work have also been diagnosed with SBCs. However, since asymptomatic SBCs have been discovered, these late-developing cases may have carried the lesion without clinical signs for sometime. Clinical evidence that trauma plays a role in development of some SBCs includes their occurrence in weight bearing areas of affected joints, their diagnosis in older working horses, occasional enlargement after surgical debridement and cyst formation following documented joint trauma.[3,4,8,12,60]

Early theories implicating trauma were derived from human studies employing

injection of synovial fluid through an articular defect into the subchondral bone,[19] observation of bone necrosis secondary to direct trauma in the absence of normal articular cartilage,[61] direct trauma to normal cartilage,[62] regional overloading[63] and pressure necrosis of the osteochondral junction [64]. Landells hypothesized that synovial fluid pressure during weight bearing was sufficient to cause necrosis of the subchondral bone if a defect in the articular cartilage was present.[19] Direct trauma was proposed to produce a focal area of subchondral bone ischemia and necrosis with resorption of the necrotic bone.[8,64]

Medial femoral condylar subchondral cystic lesions have enlarged following arthroscopic debridement and drilling, and have developed in the glenoid cavity following debridement of OCD lesions.[3,32] These occurrences remain unexplained, but may involve focal damage (ischemia or pressure necrosis) to the subchondral bone together with the hydraulic influx of synovial fluid into cartilage and subchondral bone defects.[8,14]

Experimentally, subchondral cystic lesions have occurred following creation of focal articular cartilage and subchondral bone defects in horses.[14,15] In the first study, a cystic lesion developed in the medial femoral condyle in one of two ponies after creation of a full thickness incision in the articular cartilage.[15] Ray, *et al* simulated the study with a larger number of horses using arthroscopy to create the lesions[14] Either a 15-mm full thickness linear slit or a 15 X 3 mm X 5-mm-deep articular cartilage and subchondral bone lesion was created in the weight bearing articular cartilage[14] While none of the horses in the first group developed cysts, 5/6 of the horses in the second group developed

subchondral cystic lesions suggesting that linear defects which expose subchondral bone are at risk of cyst formation. [14]

A combined theory is that the disturbance in endochondral ossification leads to thickening and weakening of the epiphyseal cartilage which eventually cracks leaving a narrow opening for synovial fluid to be pumped into the subchondral bone during normal joint motion.[1,2] The mechanical effect of the synovial fluid being hydraulically pumped through the cartilage defect against the subchondral bone during weight bearing may cause bone resorption leading to formation of a subchondral cystic lesion.[1,2,19] The joint fluid mechanics dictate the eventual size of the lesion, which enlarges until equilibrium is reached between the synovial fluid pressure and bone resistance.[1,2] Surrounding bone appears sclerotic radiographically, however it is woven bone that is often soft and poorly organized.[2]

Pain associated with SBCs may originate from synovitis, the subchondral bone or both.[1] It has been proposed that subchondral cystic lesions do not cause pain until they rupture into the joint.[2] This situation would apply only if osteochondrosis developing beneath intact articular cartilage is the primary cause of the lesion. Cysts produced secondarily to articular cartilage disruption would be expected to always communicate with the joint, making rupture into the joint an unlikely cause of the clinical signs.[1]

Alternatively, lameness may be due to increased intraosseous pressure [1,2] which causes severe pain in people.[65] Variation in subchondral bone sensory innervation may explain the variable lameness, as well as the failure to completely eliminate lameness by

use of intra-articular anesthesia.[38] The actual source of pain may vary depending on the pathogenesis of the individual lesion and in the horse remains unknown.

Treatment

Conservative treatment consists of rest with or without intra-articular medications or nonsteroidal anti-inflammatory drugs. Early success rates of 50-64% were reported,[4,7,16] others reported less favorable results for athletic horses.[5,6,9,10,17]

Surgical treatment consists of complete evacuation of the cystic contents and enlargement of the articular margins to substantial adjacent bone. Extra-articular [18] and femorotibial arthrotomy [9] and arthroscopic [3,17] approaches have been described. Debridement alone or with an autogenous cancellous bone graft,[21,22] bone cement,[66] or drilling of the parent bone [3,9,17] have been used. Surgical debridement via arthroscopy or arthrotomy has been reported to results in improvement so 70-80 % of horses are able to return to their intended use. [3,9,17] Only one report provided follow-up on horses intended for racing, 61% returned to racing, however, only 50% remained sound.[17] Drilling of the parent bone is no longer recommended due to subsequent cystic enlargement or secondary cyst formation.[1,3]

Horses should improve within 6-12 months after surgery; however, the range is 4-22 months and the average is 6-7.5 months.[1,3,17] Pain after surgery may not resolve until the sclerosis disappears from the subchondral bone.[2] Unfortunately, no specific preoperative findings predict which horses will respond to treatment, and there is often no

explanation for those that do not respond to surgery. Subchondral cystic lesions rarely resolve radiographically despite improvement or resolution of the lameness, and there does not appear to be any relationship between radiographic density of the lesion and eventual soundness of the horse.[1,6,9]

Addition of a cancellous bone graft after debridement of the cyst resulted in a success rate similar to debridement alone.[22] Grafting of experimentally created subchondral cystic lesions has resulted in successful graft incorporation and joint resurfacing.[28] However, some of the horses experienced graft resorption and cyst enlargement 6 to 12 months later.[28]

BONE GRAFTS

Microanatomy and Physiology of Bone

Bone is a complex tissue containing four cell types, the osteoprogenitor (osteogenic) cell, the osteoblast, the osteocyte and the osteoclast.[67] Osteoprogenitor cells consist of determined osteoprogenitor cells that will become osteoblasts and inducible osteoprogenitor cells that require exposure to an inducing agent.[68] Determined osteoprogenitor cells in their resting state are flattened, relatively undifferentiated mesenchymal cells located on or near all free surfaces of bone including the cambium layer of the periosteum, the endosteum and in the Haversian canals and bone marrow.[68,69] Determined osteoprogenitor cells are involved in normal bone growth and become activated in bone healing. Inducible osteoprogenitor cells are pluripotent mesenchymal

cells which are widely dispersed throughout the body, but are more concentrated in muscle and connective tissue.[68] Osteoblasts are uninucleate cells which contribute to bone formation by secreting the collagenous bone matrix, osteoid. Osteocytes are osteoblasts that have become surrounded by bone matrix. Osteoclasts are multinucleate cells thought to be formed from the fusion of circulating monocytes[70,71] and are responsible for bone resorption effected through the release of collagenase and other proteolytic enzymes.

The two morphologic forms of bone are woven and lamellar . Woven bone is an immature form characterized by random organization of its fibrous elements and is usually found in early fracture repair. Lamellar bone is mature bone composed of successive layers, each of which has a highly organized infrastructure. Lamellar bone may exist as a solid mass of compact bone such as cortical bone, or it may be a spongy trabecular mass such as cancellous bone where the spaces contain bone marrow. Cancellous bone does not usually contain Haversian systems, so the osteocytes exchange metabolites via canaliculi to sinusoids in the marrow.[72]

Biology of Bone Grafting

In 1668, the first recorded bone graft procedure was performed by Job van Meekeren, a Dutch surgeon.[73,74] The transplantation of bone or biosynthetic materials to repair skeletal defects is now an accepted surgical technique. Materials reported to have been used include cancellous,[21,23,25-27,75-85] cortical,[24,80,86-91] and corticocancellous bone,[24,26,75,76,80,87,89,90,92-96] osteochondral plugs [97-101] or synthetic material. [26,88,90,102-106]

Intraarticular grafts have been reported using cancellous bone [21,22] and osteochondral grafts. [98,107-109]

Bone grafts provide osteogenic potential (primary osteogenesis or osteoinduction) and scaffolding for ingrowth of new elements (osteoconduction). Primary osteogenesis is the formation of new bone stemming from transferred living cells[110] which may survive up to 1 mm from the implant surface. Osteoinduction is the phenotypic conversion of host mesenchymal cells to osteogenic cells in response to biochemical factors from the graft such as bone morphogenetic proteins (BMP's).[26,70,111] Osteoconduction is the process by which the bone graft provides passive support for host neovascularization and osteogenic elements orienting the structure of the newly forming bone.[110] Osseointegration is the formation of a direct, intimate and lasting connection between the host bone and graft.[70] An additional purpose of a graft may be structural support [27,112] which is mainly derived from cortical, corticocancellous or osteochondral bone grafts.[76]

Bone has the unique ability to heal completely and regain its original structure and mechanical properties; repair tissue consists of new bone rather than scar tissue.[70,79,113] The net biologic activity of the graft is the sum of its inherent biologic activity, its capacity to activate surrounding host tissues and its ability to support the ingrowth of host osteogenic tissue.[87]

During incorporation the graft site goes through several concurrent phases.[26] Within minutes platelet aggregation and degranulation initiates the release of cytokines and growth factors causing inflammation.[112,114] Neutrophils, macrophages and fibroblasts

are recruited via chemical messengers such as kinins, complement, histamine, serotonin, prostaglandins and leukotrienes.[26] Macrophages and giant cells debride the wound of devitalized protein while osteoclasts begin removing dead bone. Ischemic death of lacunar osteocytes and subsequent release of lysosomal enzymes results in osteoid destruction.[115] The inflammatory phase lasts up to one week in cancellous autografts.[27]

Centripetal vascularization begins as early as the second day.[25,85,116-118] In cancellous bone, vascularity advances at a rate of 0.2-1.0 mm/day [75,85] and may be completed within 1-3 weeks.[119] Cortical grafts become revascularized much more slowly requiring 4-8 weeks for fresh autogenous grafts and greater than 4 months for frozen grafts or allografts with a histocompatibility antigen difference.[87]

Mesenchymal cells begin to proliferate by day 3, differentiate into chondroblasts by day 5, and osteoblasts by day 10.[80] The osteoinduction of inducible pluripotent stem cells by BMP and transforming growth factor-β is complete within the first one to two weeks.[26] Vascular ingrowth also brings osteoclastic activity initiating graft resorption. Osteoclasts resorb the dead bone, while osteoblasts deposit an osteoid seam along the remnants of the dead trabeculae. The osteoid is then mineralized into new host bone.[24] During this phase the installed graft trabeculae are gradually replaced by new host bone either by 'creeping substitution' in cortical bone or by surface resorption in cancellous bone. Osteoconduction lasts several months in cancellous grafts and may take years in cortical bone.[27]

Bone graft success depends on the host recipient site, local growth factors of the host bed, bone graft viability, the volume of bone grafted and the structural function of the

bone graft.[76] The host recipient site influences the graft physiologically and mechanically. The number of host osteoprogenitor cells and the quality of the perivascular connective tissue determine hosts ability to respond to graft BMP's and other growth factors.[76] Factors adversely affecting graft incorporation include trauma, infection, insufficient vascular supply and graft or fracture instability. Motion at the graft/host bone or soft tissue interface will impede or prevent revascularization.[87,112]

Growth factors are polypeptides that bind to specific cell membrane receptors and stimulate or inhibit certain cell functions.[120] Five important growth factors that have been identified include platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-β), insulin-like growth factor, basic fibroblastic growth factor and epidermal growth factor.[104] Within the TGF-β super family, the BMP's which are secreted by osteoblasts have emerged as the trigger needed to stimulate bone formation.[70,104] BMP-2 appears to be the most potent member of the family inducing heterotopic bone formation as early as 5 days. [68,121] BMP-2 induces bone formation at heterotopic as well as orthotopic sites[122] and has been evaluated in segmental bone defects in rats,[123,124] dogs,[125] rabbits[126-131] and monkeys[132,133] with similar results.

BMP induced bone formation is dose dependent and exposure to an immediate source of osteoprogenitor cells is crucial.[68,120,122,134] BMP plus bone marrow yields the highest bone production and may be 3 times as effective as autogenous cancellous grafts.[135] BMP increases the recruitment of bone forming cells but does not increase individual cell activity.[136] Bone morphogenetic protein activity of the bone graft and host

bed induce proliferation of perivascular connective tissue and facilitate osteogenesis.

Because graft viability improves success, the graft should be transferred directly or wrapped in a blood-soaked sponge.[26] Air, saline or antibiotics damage the grafted cells.[137] Even with ideal handling, up to 90% of living cells die after bone graft transfer,[76] but those up to 1.0 mm from the bone surface usually survive. The open structure of a cancellous graft allows diffusion of nutrients and limited capillary microanastomosis, whereas denser cortical bone is a greater barrier.[138] Autogenous cancellous and vascularized corticocancellous bone grafts have a greater chance of survival than do allografts and xenografts which lack early vascularization and elicit an immune response.[76] Larger grafts require longer for complete incorporation which increases the likelihood of complications and failure. Conversely, transferring more cells means that more osteogenic cells survive.[76]

Bone Grafts and Bone Graft Substitutes

Cancellous Bone Grafts

Cancellous bone is a 3-dimensional coarse trabecular lattice that creates a network of intercommunicating spaces occupied by bone marrow.[67,139] Complete osteons are present only in thicker trabeculae.[70] The majority of cancellous bone is present in the epiphysis and metaphysis of long bones where it transmits multidirectional forces to the diaphysis.[139] Fresh cancellous autografts have long been considered the optimal graft,

because under ideal conditions they contain a maximum number of surviving cells and have a loose framework which allows rapid vascular ingrowth from the host tissues.[25,118] The goal of autogenous cancellous chip grafting is to seed the recipient site with viable mesenchymal cells that will produce new bone rather than to transplant viable morsels of bone.[85]

Cancellous bone can be readily obtained from sternebrae, tuber coxae, proximal tibia, proximal humerus and ribs.[24-27,76,83,84,89,110,112,140] The sternum is suitable for harvesting cancellous bone when horses are placed in dorsal recumbency.[140] Histologically, sternal cancellous bone is comparable to cancellous bone from the tuber coxae.[140] The caudal sternebrae provide relatively more cancellous bone compared to the cranial sternebrae.[140]

Sullins *et al* transplanted intact cancellous bone cylinders into experimentally created osteochondral defects in equine stifle joints.[141] In this study, the successfully grafted defects were covered with a uniform cartilage covering, compared with the nongrafted lesions.[141]

Cortical Bone Grafts

Cortical bone has a porosity of 5 to 30% compared to the 30 to 90% porosity of cancellous bone, and is most commonly used to provide stability at a fracture site.[67] Autogenous cortical grafts provide limited osteogenic potential as only surface cells survive, and osteoinduction is limited by the small quantity of BMP. Cortical graft

incorporation is significantly delayed compared with cancellous grafts.[27]

In contrast to cancellous grafts, incorporation of cortical grafts is initiated by osteoclasts rather than osteoblasts.[26] Vascularization is primarily the result of peripheral resorption and vascular infiltration of the Haversian canals,[142] and takes at least twice as long than for similar sized cancellous grafts.[143] Osteoclasts resorb bone at a relatively high rate (50 μ/day) while osteoblasts form bone at a rate of only 1 μ/day. The result is increased porosity [26] and significant weakening of the graft beginning 2-6 weeks after and persisting for at least 6 months.[27] Large segmental grafts are subject to fatigue microdamage due to cyclic loading and require protection until replaced by new host bone.[112] Matrix in dead bone is as strong as that in viable bone, and a combination of graft lamella and new host osteons make the graft as strong as control segments at one year.[27]

Osteochondral Grafts

In humans and animals osteochondral grafts have been used clinically and experimentally to treat highly comminuted, intra-articular fractures, severely osteoarthritic joints, osteochondrosis and malignant bone tumors.[28,67,97,98,107,108,138,144,145] Cartilage and subchondral bone have a unique biomechanical interdependence because they are joined by a semisolid layer of calcified cartilage[97] which minimizes the stiffness gradient between cartilage and bone.[146]

There are two major types of osteochondral grafts, shell grafts and massive grafts. Shell grafts consist of articular cartilage and 2-8 mm of underlying bone, which is set in

fitted apposition to normal articular cartilage. Shell grafts have been used successfully in defects up to 30 mm in diameter.[98] The small volume of bone in shell grafts is efficiently remodeled, preserving the structural support for cartilage.[97,107] Cartilage survival has been documented following osteochondral allografts used to resurface the tibial condyle in humans.[147,148]

Hurtig successfully used autogenous cancellous grafts harvested from the contralateral limb to resurface experimental small weight bearing defects on the third carpal bone.[144] In the same study allografts were unsuccessful due to pannus formation and host rejection. Desjardins successfully transferred fresh and frozen autogenous osteochondral grafts from the tibiotarsal joint to the femoropatellar joint.[97] Both studies reported limitations of this procedure due to donor site morbidity and the size, shape and quantity of tissue available.[97,144]

Sullins, *et al* used sternal osteochondral autografts to fill experimentally created subchondral cystic lesions in the medial femoral condyle.[28] In this study when graft incorporation was successful, the cartilage of the graft remained attached and viable. However, graft incorporation was unsuccessful in four of nine cases.[28] It was hypothesized that the bone lysis that occurred was due to influx of synovial fluid around the graft.

Allografts

Allograft bone is an important source of graft material in small animal[24,27,89] and

human orthopedic surgery[26,93,94,107]. Allografts have consisted of cancellous bone chips, cortical bone and osteochondral bone. Approximately 2 weeks post grafting fresh allografts invoke a host immune response.[67,110] Allografts may be sufficiently immunogenic to slow or prevent incorporation.[27] Antigenicity may be reduced by freezing, freeze-drying, or irradiation. These preservation techniques diminish the mechanical properties of the graft but when done properly preserve the bone morphogenetic proteins and thus the osteoinductive capabilities of the graft.[112] Allografts have been used successfully in experimental fractures of the equine third carpal bone [144] but have found little use in clinical procedures primarily due to the prolonged incorporation time.

Demineralized Bone Matrix

Demineralized bone grafts are potent osteoinductive stimulants, but have no osteoconductive properties.[26] Demineralized bone matrix (DBM) is produced by carefully leaching mineral from the bone without destroying the bone morphogenetic proteins and other growth factors.[149] Demineralized bone grafts are available as powder, chips or segmental blocks and are widely used in human orthopedics.[150] Demineralized bone matrix induces heterotopic and orthotopic bone formation in the horse similar to other species but is not currently used in equine orthopedics.[92,96]

Xenografts

Commercially available processed bovine cancellous xenografts have been used for

cervical vertebral stabilization in humans [151] and horses.[152] Vascularization of xenogenic cancellous bone is markedly delayed compared to autogenous or allogeneic grafts.[85] Xenografts have not become popular due to prolonged incorporation time and histopathologic evidence that fibrous, rather than osseous fusion may occur. [152,153]

Hydroxyapatite

Hydroxyapatite is a nonbiodegradable ceramic used for bone replacement that is well tolerated and augments healing by osteoconduction.[76] These implants have a network of interconnecting pores and channels similar to the mineralized inorganic structure of bone and are available in cylinders, blocks or granules. Formation of bone is preceded by vascular ingrowth into the gaps and pores of the hydroxyapatite.[76] Initially, hydroxyapatite has 55% of the strength of trabecular bone, which increases to three times the strength of cancellous bone at 6 months.[103] When placed into experimental subchondral defects in the medial femoral condyle, the deeper central portion of the implant was filled with new bone at six months, but the superficial area did not fill with bone nor was there any cartilage covering the defect.[34]

Tricalcium Phosphate

Tricalcium phosphate (TCP) is a biodegradable product with exceptional tissue acceptance which is slowly replaced by bone.[76,154] TCP has been placed experimentally into osteochondral defects of the distal third metacarpal/third metatarsal bones of horses

with good results.[154] Interestingly the tricalcium phosphate cylinders remodeled into cortical bone in the region of the cortex and into cancellous bone in the region of the medullary cavity. A new subchondral bone plate developed over the TCP toward the surface and was covered by a calcified cartilage layer a fibrocartilage layer.[154]

LITERATURE CITED

1. Baxter G: Subchondral Cystic Lesions in Horses, in McIlwraith W, Trotter G (eds): Joint Disease in the Horse. Philadelphia, W.B. Saunders, 1996, pp 384-397
2. Bramlage L: Osteochondrosis Related Bone Cysts. Proc American Association of Equine Practitioners. 39, pp 83-85, 1993
3. Howard RD, McIlwraith CW, Trotter GW: Arthroscopic surgery for subchondral cystic lesions of the medial femoral condyle in horses: 41 cases (1988-1991). J Am Vet Med Assoc 206:842-850, 1995
4. Jeffcott LB, Kold SE: Clinical and radiological aspects of stifle bone cysts in the horse. Equine Vet J 14:40-46, 1982
5. McIlwraith CW: Subchondral cystic lesions (osteochondrosis) in the horse. Comp Cont Educ Pract Vet 4:S394, 1982
6. McIlwraith W: Subchondral cystic lesion in the horse- the indications, methods, and results of surgery. Equine Vet Educ 2:75-80, 1990
7. Stewart B, Reid CF: Osseous cyst-like lesions of the medial femoral condyle in the horse. J Am Vet Med Assoc 180:254-257, 1982
8. Verschooten F, De Moor A: Subchondral cystic and related lesions affecting the equine pedal bone and stifle. Equine Vet J 14:47-54, 1982
9. White NA, McIlwraith CW, Allen D: Curettage of subchondral bone cysts in medial femoral condyles of the horse. Equine Vet J Suppl 6:120, 1988
10. Trotter GW, McIlwraith CW: Osteochondritis dissecans and subchondral cystic lesions and their relationship to osteochondrosis in the horse. J Equine Vet Sci 1:157-162, 1981
11. Watkins J: Osteochondrosis, in Auer J (ed): Equine Surgery. Philadelphia, W.B. Saunders, 1992, pp 971-984
12. Nixon AJ: Osteochondrosis and osteochondritis dissecans of the equine fetlock. Compend Contin Educ Pract Vet 12:1463-1475, 1990
13. Rooney J: Osteochondrosis in the horse. Mod Vet Pract 56:41-43 113-116, 1975
14. Ray C, Baxter G, McIlwraith C: Development of subchondral cystic lesions following articular cartilage and subchondral bone damage in young horses. Equine Vet J 28:225-232, 1996
15. Kold SE, Hickman J, Melsen F: An experimental study of the healing process of equine chondral and osteochondral defects. Equine Vet J 18:18-24, 1986
16. Rose JA, Sande RD, Rose EM: Results of conservative management of osteochondrosis in the horse. Proc Am Assoc Equine Pract 31:617-626, 1985
17. Lewis R: A retrospective study of diagnostic and surgical arthroscopy of the equine femorotibial joint. Proc Assoc Am Equine Pract. 33, pp 887-893, 1987

18. Evans H, Jenny J: Surgical and clinical management of subchondral "bone cysts". Proc Am Assoc Equine Pract. 16, pp 195-197, 1970
19. Landells J: The bone cysts of osteoarthritis. J. Bone Joint Surg 35-B: 643, 1953
20. Jeffcott LB, Kold SE, Melsen F: Aspects of the pathology of stifle bone cysts in the horse. Equine Vet J 15:304-311, 1983
21. Kold SE, Hickman J: Use of an autogenous cancellous bone graft in the treatment of subchondral bone cysts in the medial femoral condyle of the horse. Equine Vet J 15:312-316, 1983
22. Kold SE, Hickman J: Results of treatment of subchondral bone cysts in the medial condyle of the equine femur with an autogenous cancellous bone graft. Equine Vet J 16:414-418, 1984
23. Gross TP, Jinnah RH, Clarke HJ, et al: The biology of bone grafting. Orthopedics 14:563-568, 1991
24. Johnson AL: Principles of bone grafting. Semin Vet Med Surg (Small Anim) 6:90-99, 1991
25. Kold SE, Hickman J, Melsen F: Qualitative aspects of the incorporation of equine cancellous bone grafts. Equine Vet J 19:111-119, 1987
26. Markel M: Bone Grafts and Bone Substitutes, in Nixon A (ed): Equine Fracture Repair. Philadelphia, W.B. Saunders, 1996, pp 87-92
27. Stevenson S: Bone Grafting, in Slatter DB (ed): Small Animal Surgery. Philadelphia, W.B. Saunders, 1985, pp 2035-2048
28. Sullins K, Veit H, McIlwraith C: Osteochondral grafts to fill large articular defects in horses. Vet Surg 18, 1989
29. Sullins K, McIlwraith C, Powers B, et al: Evaluation of periosteal grafts in articular cartilage repair in horses. Vet Surg 14:66, 1985
30. Vachon AM, McIlwraith CW, Powers BE, et al: Morphologic and biochemical study of sternal cartilage autografts for resurfacing induced osteochondral defects in horses. Am J Vet Res 53:1038-1047, 1992
31. Bertone AL, McIlwraith CW, Powers BE, et al: Subchondral osseous cystic lesions of the elbow of horses: conservative versus surgical treatment. J Am Vet Med Assoc 189:540-546, 1986
32. Bertone AL, McIlwraith CW, Powers BE, et al: Arthroscopic surgery for the treatment of osteochondrosis in the equine shoulder joint. Vet Surg 16:303-311, 1987
33. Dyson S: Shoulder Lameness in horses: An analysis of 58 suspected cases. Equine Vet J 18:29-36, 1986
34. Gillis J, Auer J, Holmes R: The use of a porous bioimplant in the treatment of subchondral bone cysts in the horse. Vet Surg 16:90, 1987
35. Hopen LA, Colahan PT, Turner TA, et al: Nonsurgical treatment of cubital subchondral cyst-like lesions in horses: seven cases (1983-1987). J Am Vet Med Assoc 200:527-530, 1992

36. Jeffcott L, Kold S: Stifle lameness in the horse: a survey of 86 referral cases. Equine Vet J 14:31-39, 1982
37. McIlwraith W, Goodman N: Conditions of the Interphalangeal Joint, in Yovich J (ed): Veterinary Clinics of North America, Equine Practice. Philadelphia, W.B. Saunders, 1989, pp 161-178
38. Nixon AJ, Adams RM, Teigland MB: Subchondral cystic lesions (osteochondrosis) of the femoral heads in a horse. J Am Vet Med Assoc 192:360-362, 1988
39. Petterson H, Sevellus F: Subchondral bone cysts in the horse: A clinical study. Equine Vet J 1:75-82, 1968
40. Petterson H, Reiland F: Periarticular subchondral "bone cysts" in horses. Clin Orthop 62:95-103, 1969
41. Rejno S, Stromberg B: Osteochondrosis in the horse. II. Pathology. Acta Radiol Suppl (Stockh) 358:153-178, 1978
42. Specht TE, Nixon AJ, Colahan PT, et al: Subchondral cyst-like lesions in the distal portion of the radius of four horses. J Am Vet Med Assoc 193:949-952, 1988
43. Trotter GW, McIlwraith CW, Norrdin RW, et al: Degenerative joint disease with osteochondrosis of the proximal interphalangeal joint in young horses. J Am Vet Med Assoc 180:1312-1318, 1982
44. Verschooten F: Post-traumatic subchondral bone cysts and subchondral bone necrosis in the horse. Vlaams Diergeneeskd Tijdschr 49:237-264, 1980
45. Watson DE, Selcer BA: Radiographic signs of osteochondrosis in horses. Compend Contin Educ Pract Vet 14:809-815, 819, 1992
46. Blackwell J, Griffith A, Crosby W: Surgical correction of an alveolar mandibular bone cyst. Equine Pract 7:70-73, 1985
47. Jackman BR, Baxter GM: Treatment of a mandibular bone cyst by use of a corticocancellous bone graft in a horse. J Am Vet Med Assoc 201:892-894, 1992
48. Steiner JV, Rendano VT, Jr.: Aneurysmal bone cyst in the horse. Cornell Vet 72:57-63, 1982
49. Taylor E, Anderson D, Patwell J, et al: Dorland's Illustrated Medical Dictionary (ed 27). Philadelphia, W.B. Saunders, 1988
50. Malghem J, Maldague B, Esselinckx W: Spontaneous healing of aneurysmal bone cyst. J Bone Joint Surg Br 71B:645-650, 1989
51. Campanacci M, Capanna R, Picci P: Unicameral and aneurysmal bone cysts. Clin Orthop 204:25-36, 1986
52. Schrader S, Burk R, Si-kwang L: Bone cysts in two dogs and a review of similar cystic bone lesion in the dog. J Am Vet Med Assoc 182:490-495, 1983
53. Reeves M, Trotter G, Kainer R: Anatomical and functional communications between the synovial sacs of the equine stifle joint. Equine Vet J 23:215-218, 1991

54. McIlwraith W: Clinical Aspects of Osteochondritis Dissecans, in McIlwraith W, Trotter G (eds): Joint Disease in the Horse. Philadelphia, W. B. Saunders, 1996, pp 362-383
55. Hurtig M, Pool R: Pathogenesis of equine osteochondrosis, in McIlwraith C, Trotter G (eds): Joint Disease In The Horse. Philadelphia, W.B. Saunders, 1996, pp 335-358
56. Carlson C, Hilley H, Henrikson C: Ultrastructure of normal epiphyseal cartilage of the articular-epiphyseal complex in growing swine. Am J Vet Res 46:306-313, 1985
57. Reiland S: Morphology of osteochondrosis and sequelae in pigs. Acta Radiol Suppl 358:45-90, 1978
58. Stromberg B: A review of the salient features of osteochondrosis in the horse. Equine Vet J 11:211-214, 1979
59. McIlwraith CW: Diseases of joints, tendons, ligaments, and related structures. Adams' Lameness in horses / Ted S. Stashak. 4th ed. Philadelphia : Lea & Febiger 1987, pp 339-485
60. Yovich JV, Stashak TS: Subchondral osseous cyst formation after an intra-articular fracture in a filly. Equine Vet J 21:72-74, 1989
61. Rhaney K, Lamb D: The cyst of osteoarthritis of the hip. J. Bone Joint Surg 37-B:663, 1955
62. Dashefsky J: Post-traumatic subarticular cyst of bone- a case report. J. Bone Joint Surg 53-A:154, 1971
63. Ondrouch A: Cyst formation in osteoarthritis. J. Bone Joint Surg 45-B:755, 1963
64. Rooney J: Biomechanics in Lameness in Horses. Baltimore, Williams & Williams, 1969
65. Bunger C, Sorensen S, JC D, et al: Intraosseous pressures in the knee in relation to simulated joint effusion, joint position, and venous obstruction. Scand J Rheumatol 10, 1981
66. Hickman J, Kold S, Ellis D: Use of bone cement in two equine orthopaedic cases. Equine Vet J 16:543-545, 1984
67. Binnington A: Bone Remodeling and Transplantation, in Whittick W (ed): Canine Orthopedics. Philadelphia, Lea & Febiger, 1990, pp 166-189
68. Kirker-Head C: Novel Biological Agents to Enhance Fracture Healing, in Nixon A (ed): Equine Fracture Repair. Philadelphia, W.B. Saunders, 1996, pp 93-103
69. Harris R: Fracture Healing, in Whittick W (ed): Canine Orthopedics. Philadelphia, Lea & Febiger, 1990, pp 158-165
70. DeLacure MD: Physiology of bone healing and bone grafts. Otolaryngol Clin North Am 27:859-873, 1994
71. Ham A, Cormack D: Histology (ed 8). Philadelphia, J.B. Lippincott, 1979
72. Wheater P, Burkitt H, Daniels V: Functional Histology (ed 1). Edinburgh, Churchill Livingstone, 1979

73. van Meekeren j: Heel-en Geneeskonstige Aanmerkingen: Commelijn. Amsterdam, 1668
74. Blitch EL, Ricotta PJ: Introduction to bone grafting. J Foot Ankle Surg 35:458-462, 1996
75. Albrektsson T: Healing of bone grafts. In vivo studies of tissue reactions at autografting of bone in the rabbit tibia., Goteborg, 1979
76. Auer J: Principles in Fracture Treatment, in Auer J (ed): Equine Surgery. Philadelphia, W.B. Saunders, 1992, pp 812-832
77. Bramlage L: Autologous cancellous bone grafting in the horse. Proc American Association of Equine Practitioners. 27, pp 243-247, 1981
78. Catanzariti AR: Graft-enhanced arthrodesis. J Foot Ankle Surg 35:463-473, 1996
79. Friedlaender G: Bone Grafts. J Bone Joint Surg 69-A:786-789, 1987
80. Goldburg V: Bone Transplantation, in Evarts C (ed): Surgery of the Musculoskeletal System. New York, Churchill Livingston, 1989, pp 54-81
81. Hanie EA, Sullins KE, Powers BE, et al: Comparison of two grafting methods in 4.0-mm drill defects in the third metacarpal bone of horses [see comments]. Equine Vet J 24:387-390, 1992
82. Mendicino SS, Rockett A, Wilber MR: The use of bone grafts in the management of nonunions. J Foot Ankle Surg 35:452-457, 1996
83. Nunamaker DM, Rhinelander FW: Bone grafting, in Charles D. Newton DMN (ed): Textbook of small animal orthopaedics. Philadelphia, Lippincott, 1985
84. Olds RB, Sinibaldi KR, DeAngelis MP, et al: Autogenous cancellous bone grafting in small animals. J Am Anim Hosp Assoc, Sept/Oct 9:454-457, 1973
85. Wilson JW, Rhinelander FW, Stewart CL: Vascularization of cancellous chip bone grafts. Am J Vet Res 46:1691-1699, 1985
86. Whittick WG: Bone grafting: collection and storage, indications, and methods. Curr Tech Small Anim Surg 1:554-562, 1975
87. Stevenson S, Emery SE, Goldberg VM: Factors affecting bone graft incorporation. Clin Orthop :66-74, 1996
88. Smiler DG: Bone grafting: materials and modes of action. Pract Periodontics Aesthet Dent 8:413-416, 1996
89. Kerwin SC, Lewis DD, Elkins AD: Bone grafting and banking. Compend Contin Educ Pract Vet 13:1558-1562, 1564, 1566-1567, 1991
90. Habal MB, Reddi AH: Bone grafts & bone substitutes, in Habal MB, Reddi AH (eds): Bone grafts & bone substitutes. Philadelphia, Saunders, 1992, pp 461
91. Goldberg VM, Stevenson S: Bone graft options: fact and fancy. Orthopedics 17:809-810, 821, 1994
92. Fackelman GE, von Rechenberg B, Fetter AW: Decalcified bone grafts in the horse. Am J Vet Res 42:943-948, 1981
93. Aro HT, Aho AJ: Clinical use of bone allografts. Ann Med 25:403-412, 1993

94. Gross TP, Cox QG, Jinnah RH: History and current application of bone transplantation. Orthopedics 16:895-900, 1993
95. Springfield DS: Allograft reconstructions. Semin Surg Oncol 13:11-17, 1997
96. Vail TB, Trotter GW, Powers BE: Equine demineralized bone matrix: relationship between particle size and osteoinduction [see comments]. Vet Surg 23:386-395, 1994
97. Desjardins MR, Hurtig MB, Palmer NC: Incorporation of fresh and cryopreserved bone in osteochondral autografts in the horse. Vet Surg 20:446-452, 1991
98. Garrett JC: Fresh osteochondral allografts for treatment of articular defects in osteochondritis dissecans of the lateral femoral condyle in adults. Clin Orthop 303:33-37, 1994
99. Gerhart T, Kirker-Head C, Henning G: Healing segmental femoral defects in sheep using recombinant human bone morphogenetic protein (BMP-2). Clin Orthop 293:317-326, 1993
100. Meyers MH, Akeson W, Convery FR: Resurfacing of the knee with fresh osteochondral allograft. J Bone Joint Surg [Am] 71:704-713, 1989
101. Outerbridge HK, Outerbridge AR, Outerbridge RE: The use of a lateral patellar autologous graft for the repair of a large osteochondral defect in the knee. J Bone Joint Surg Am 77:65-72, 1995
102. Misch CE, Dietsh F: Bone-grafting materials in implant dentistry. Implant Dent 2:158-167, 1993
103. Holmes R, Mooney V, Bucholz R, et al: A corraline hydroxyapatite bone graft substitute. Clin. Orthop 188:252-262, 1984
104. Hollinger JO, Seyfer AE: Bioactive factors and biosynthetic materials in bone grafting. Clin Plast Surg 21:415-418, 1994
105. Elsinger EC, Leal L: Coralline hydroxyapatite bone graft substitutes. J Foot Ankle Surg 35:396-399, 1996
106. Costantino PD, Friedman CD: Synthetic bone graft substitutes. Otolaryngol Clin North Am 27:1037-1074, 1994
107. Garrett JC: Osteochondral allografts. Instr Course Lect 42:355-358, 1993
108. Helphrey ML, Stevenson S: Osteoarticular allogeneic bone grafting in dogs. Vet Surg 9:83-87, 1980
109. Marco F, Lopez-Oliva F, Fernandez Fernandez-Arroyo JM, et al: Osteochondral allografts for osteochondritis dissecans and osteonecrosis of the femoral condyles. Int Orthop 17:104-108, 1993
110. Alexander JW: Bone grafting. Vet Clin North Am Small Anim Pract 17:811-819, 1987
111. Urist M: Bone: Formation by autoinduction. Science 150:893-899, 1965
112. Stevenson S: Bone Grafting, in Bojrab J (ed): Current Techniques in Small Animal Surgery. Philadelphia, Lea & Febiger, 1990, pp 836-844

113. Markel M: Fracture Healing and Its Noninvasive Assessment, in Nixon A (ed): Equine Fracture Repair. Philadelphia, Saunders, 1996, pp 19-29
114. Mast BA: Healing in other tissues. Surg Clin North Am 77:529-547, 1997
115. Wornom I, Buchman S: Bone and cartilaginous tissue., in Cohen I, Diegelmann R, Lindblad W (eds): Wound Healing : Biochemical and Clinical Aspects. Philadelphia, WB Saunders, 1992, pp 356-383
116. Albrektsson B: Repair of diaphyseal defects. Experimental studies on the role of bone grafts in reconstruction of circumferential defects in long bones, Goteborg, 1971
117. Ray R: Vascularization of bone grafts and implants. Clin. Orthop. rel. Res. 87:43-48, 1972
118. Stringa B: Studies of the vascularization of bone grafts. J. Bone Jt. Surg. 39-B:295-420, 1957
119. Burwell R: The fate of bone grafts, in Apley A (ed): Recent Advances in Orthopaedics. , Churchhill, 1969
120. Riley E, Lane J, Urist M, et al: Bone Morphogenetic Protein-2 Biology and Applications. Clin. Orthop. Rel. Res. 324:39-46, 1996
121. Wozney J: Bone morphogenetic proteins. Prog Growth Factor Res. :267-280, 1989
122. Yasko A, Lane J, Fellinger E: The healing of segmental defects, induced by recombinant human bone morphogenetic protein (rhBMP-2). A radiographic, histologic, and biomechanical study in rats. J Bone Joint Surg 74A:659-670, 1992
123. Yasko AW, Lane JM, Fellinger EJ, et al: The healing of segmental bone defects, induced by recombinant human bone morphogenetic protein (rhBMP-2). A radiographic, histological, and biomechanical study in rats [published erratum appears in J Bone Joint Surg Am 1992 Aug;74(7):1111]. J Bone Joint Surg Am 74:659-670, 1992
124. Linde A, Hedner E: Recombinant bone morphogenetic protein-2 enhances bone healing, guided by osteopromotive e-PTFE membranes: an experimental study in rats. Calcif Tissue Int 56:549-553, 1995
125. Toriumi DM, Kotler HS, Luxenberg DP, et al: Mandibular reconstruction with a recombinant bone-inducing factor. Functional, histologic, and biomechanical evaluation. Arch Otolaryngol Head Neck Surg 117:1101-1112, 1991
126. Bostrom M, Lane JM, Tomin E, et al: Use of bone morphogenetic protein-2 in the rabbit ulnar nonunion model. Clin Orthop :272-282, 1996
127. Cook SD, Baffes GC, Wolfe MW, et al: The effect of recombinant human osteogenic protein-1 on healing of large segmental bone defects. J Bone Joint Surg Am 76:827-838, 1994

128. Kim NH, Yang KH, Lee HM, et al: Effect of porcine bone morphogenetic protein on healing of bone defect in the rabbit radius. Yonsei Med J 33:54-63, 1992
129. Zellin G, Linde A: Treatment of segmental defects in long bones using osteopromotive membranes and recombinant human bone morphogenetic protein-2. An experimental study in rabbits. Scand J Plast Reconstr Surg Hand Surg 31:97-104, 1997
130. Reid CA, McCarthy JG, Kolber AB: A study of regeneration in parietal bone defects in rabbits. Plast Reconstr Surg 67:591-596, 1981
131. Lu S, Zhang Z, Wang J: Guided bone regeneration in long bone. An experimental study. Chin Med J (Engl) 109:551-554, 1996
132. Aspenberg P, Turek T: BMP-2 for intramuscular bone induction: effect in squirrel monkeys is dependent on implantation site. Acta Orthop Scand 67:3-6, 1996
133. Boyne PJ: Animal studies of application of rhBMP-2 in maxillofacial reconstruction. Bone 19:83S-92S, 1996
134. Wozney J: BMPS: Roles in Bone Development and Repair. ACVS Symposium. 6, pp 133-135, 1996
135. Yasko A, Lane J, Fellinger E: The healing of segmental bone defects, induced by recombinant human bone morphogenetic protein (rhBMP-2). J Bone Joint Surg 74-A:659-671, 1992
136. Welch R: Effect of rhBMP-2 in Tibial Fracture Models. ACVS Symposium, pp 136-137, 1996
137. Bassett C: Clinical implications of cell function in bone grafting. Clin Orthop Rel Res 87:49, 1972
138. Brown K, Cruess R: Bone and cartilage transplantation in orthopaedic surgery. J Bone Joint Surg 64-A:270, 1982
139. Markel M: Bone Structure and the Response of Bone to Stress, in Nixon A (ed): Equine Fracture Repair. Philadelphia, W.B. Saunders, 1996, pp 3-9
140. Richardson GL, Pool RR, Pascoe JR, et al: Autogenous cancellous bone grafts from the sternum in horses: comparison with other donor sites and results of use in orthopedic surgery. Vet Surg 15:9-15, 1986
141. Sullins K, Veit H, McIlwraith C: Healing of subchondral bone and articular cartilage defects in the horse. Vet Surg 16:103, 1987
142. Enneking W, Burchardt H, Puhl J: Physical and biological aspects of repair in dog cortical bone transplants. J . Bone Joint Surg 57A:232, 1975
143. Burchartdt H, Enneking W: Transplantation of Bone. Surg. Clin. North Am 58:403, 1978
144. Hurtig MB: Experimental use of small osteochondral grafts for resurfacing the equine third carpal bone. Equine Vet J Suppl :23-27, 1988

145. Stover S, Pool R, Lloyd K: Repair of surgically created osteochondral defects with autogenous sternal osteochondral grafts in the horse. Vet Surg 18:76, 1989
146. Radin E, Rose R: Role of subchondral bone in the initiation and progression of cartilage damage. Clin Orthop 213:34-39, 1986
147. Gross A, Silverstein E, Falk J, et al: The allotransplantation of partial joints in the treatment of osteoarthritis of the knee. Clin Orthop 108:7-14, 1975
148. Sartoris DJ, Kusnick C, Resnick D: New concepts in bone grafting. Orthop Rev 16:154-164, 1987
149. Urist M, Lietze A, Mizutani H: A bovine low molecular weight bone morphogenetic protein (BMP) fraction. Clin Orthop Rel Res 234:219-232, 1982
150. Glowacki J, Altobelli D, Mulliken J: Tissue response to bone-derived implants., in Habal MB, Reddi AH (eds): Bone grafts & bone substitutes. Philadelphia, Saunders, 1992, pp 84-92
151. Goran A, Marthy K: Fracture dislocation of the cervical spine: Value of anterior approach with bovine bone interbody fusion. Spine 3:95-102, 1978
152. DeBowes RM, Grant BD, Bagby GW, et al: Cervical vertebral interbody fusion in the horse: a comparative study of bovine xenografts and autografts supported by stainless steel baskets. Am J Vet Res 45:191-199, 1984
153. Plenk H, Hollman K, Wilfert K: Experimental bridging of osseous defects in rats by the implantation of kiel bone containing fresh autologous marrow. J Bone Joint Surg 54:402-416, 1972
154. Auer J, Shenk R: The application of Tricalcium Phosphate cylinders in articular and cortical defects in the horse. Vet Surg 18, 1989
155. Hunter W: On the structure and diseases of articulating cartilages. Philos Trans Roy Soc 42:514-521, 1743
156. Wirth CJ, Rudert M: Techniques of cartilage growth enhancement: a review of the literature. Arthroscopy 12:300-308, 1996
157. Riddle W: Healing of articular cartilage in the horse. J Am Vet Med Assoc 157:1471, 1970

EVALUATIONS OF TWO TECHNIQUES OF CANCELLOUS BONE GRAFTING OF EXPERIMENTAL SUBCHONDRAL BONE CYSTS IN THE MEDIAL FEMORAL CONDYLES OF HORSES.

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ABSTRACT

Objective- To develop a grafting technique which would consistently allow primary bone healing in subchondral bone cysts in the medial femoral condyle of horses.

Study Design- Using a paired design within horses subchondral bone cysts were experimentally created in the medial femoral condyle of five horses using either a conventional drill bit or a compaction drill bit.

Animals or Sample Population- 5 young adult horses.

Methods- The experimentally created subchondral bone cysts were grafted with intact compacted autogenous cancellous bone cylinders harvested from the sternum. The graft sites were radiographed at monthly intervals until completion of the study 6 months after surgery. A treble fluorochrome bone labeling technique was used to assess rate of bone healing.

Results- Successful graft incorporation occurred in 3 of 5 compaction drilled and 2 of 5 conventionally drilled defects. Unsuccessful graft sites in the conventionally drilled group were characterized by lateral and deep cystic expansion while defects in the compaction group had bone resorption with out enlargement.

Conclusions- There was no statistical difference in successful incorporation of using either drilling technique. However, compaction drilling prevented lateral and deep expansion of the defects compared with the conventionally drilled defects.

Clinical Relevance- Further investigation with compaction drilling as an adjunct to treatment of large osteochondral defects in weight bearing surfaces is warranted.

INTRODUCTION

Subchondral bone cysts (SBCs) are an important source of lameness in young horses.[1-9] They are most common in the medial femoral condyle but have been reported in most joints of the horse.[4,5,9,10] The cause of subchondral cysts is unknown, but osteochondrosis,[2,5,10-13] articular trauma,[14,15] or both[1] are considered likely.

Conservative therapy has consisted of variable periods of stall rest.[4,5,7] Although lameness in some horses has been reported to resolve permanently,[4,7,16] some discrepancy exists between definitions of a successful outcome based on the intensity of exercise. Conservative therapy has not produced good overall results as lameness often returns when exercise intensity reaches a certain level.[3,5,6,9,17]

Early surgical interventions used a supra condylar approach to access the cyst from outside the joint followed by introduction of a cancellous bone graft;[18] no results were reported. The outcome improved following curettage of the cysts via arthrotomy or arthroscopy.[3,6,9,17] The practice has been to evacuate the cyst contents and its fibrous lining and to expand the cyst opening to the margins of substantial subchondral bone. Repeated drilling into the debrided cyst margins (forage) to expose the cavity to increased osteogenic potential has been associated with cyst enlargement or development of secondary cysts.[3] Synovial fluid hydraulic pressure through cartilage defects has been incriminated in the pathogenesis of subchondral cysts, and may play a role in these secondary postoperative changes.[14,15,19]

Postoperatively, most subchondral cysts do not completely fill with bone and remain radiolucent.[3,4,6,9,15,17] Defects fill with fibrous connective tissue and fibrocartilage [9,20] which is associated with a decrease in synovitis and deeper bone pain. Simple curettage of the lesions in athletic horses is not totally effective.[3,9,17] In a small number of cases,

packing cancellous bone fragments into curetted defects has yielded success rates similar to those reported for debridement alone.[21,22] Because these were clinical cases, there was no control and only radiographic evaluation of healing. Three of five horses destined to race were sound and back in full race training after 1 year.[22]

A logical approach to achieve the best possible healing of debrided defects would be to use a single perfectly fitting autogenous cancellous bone graft. Such a graft would provide osteogenic potential and a stable scaffold for ingrowth of new vessels.[23-27] When previously performed, good results were obtained when the graft healed primarily.[28] When the grafts healed well, morphologically organized hyaline cartilage formed on the surface of the grafted cancellous bone plugs.[28,29] However, in some horses apparent leakage of synovial fluid between the graft and parent bone caused lysis of both surfaces and loss of the graft.[29] Clinical and experimental experience point to the importance of intact subchondral bone for production of a cartilage surface.[30,31]

Assuming the failure of some grafts was due to leakage of synovial fluid around the grafts, methods have been sought to seal the host-graft interface. Compaction drill bits (Compaction Drill Bits, Instrument Makar, Inc, Okemos, MI) are specialized orthopedic drills designed to displace the bone peripherally rather than remove it from the implant site. The displaced bone becomes densely compacted to the sides and floor of the hole. Compaction Pliers (Compaction Pliers, Instrument Makar, Inc, Okemos, MI) shape and compress the graft ensuring a circular perfectly sized graft for the recipient site. The final compaction size is .75 mm smaller than the compaction hole permitting easy insertion of the graft and allows for bone memory expansion to tighten the fit postoperatively. Hypothetically, clinical subchondral cystic lesions would be enlarged into a cylinder using the appropriate drill to receive the fitted autogenous cancellous plugs.

This project was designed to evaluate the healing of compacted cancellous bone

cylinders when placed into experimental medial femoral condylar defects created using conventional or compaction drilling. We hypothesized that compaction drilling would make the recipient subchondral bone more resistant to synovial fluid pressure. The new surface generated on a stable surface should be superior to that on an unfilled defect a centimeter or larger in diameter and depth.

MATERIAL AND METHODS

Five mixed breed horses two-four years old with radiographically normal femorotibial joints were used for this project. All horses were dewormed and vaccinated routinely. Using a paired design within horses, the medial femoral condyles of each horse were randomly assigned to one of two groups (compacted or noncompacted). Twenty four hours prior to surgery, oxytetracycline (25 mg/kg, IV) was administered for fluorochrome bone labeling. After overnight fasting, each horse received phenylbutazone (2.2 mg/kg, PO) and procaine penicillin G (22,000 IU/kg, IM). The horses were sedated with xylazine (0.66 mg/kg, IV) and general anesthesia was induced with guaifenesin (5% IV to effect) and ketamine (2.2 mg/kg, IV) and maintained using halothane in oxygen. The horses were placed in dorsal recumbency and the stifle and ventral sternal areas were aseptically prepared and draped following standard protocol.

With the hind limbs in flexion, the central portion of the medial femoral condyle was located and bilateral 20-mm arthrotomies were created. A 10-mm diameter X 15-mm deep cylindric defect was created on the weight bearing surface of each medial femoral

condyle. The defects were predrilled using a 4.5-mm drill. The sites designated to be conventionally drilled were enlarged using an 8-mm conventional drill and completed using a 10-mm square-ended drill bit (Codman Cervical Drill Tip, Johnson & Johnson, New Brunswick, NJ). The sites designated to be compaction drilled were completed using a 10-mm Compaction Drill Bit. A Stryker reamer (Stryker Reamer, Stryker, Kalamazoo, MI) which developed 300 rpm was used for drilling. Sterile saline lavage cooled the drill bits and debris was carefully removed using forceps and suction and lavage.

A 15-cm midline incision exposed the sternebrae. The soft tissues were retracted, and the thin cortical bone was removed using a Cobb spinal elevator (Cobb Spinal Elevator, Codman, Johnson & Johnson, New Brunswick, NJ). A 12-mm cylindric sternal autogenous cancellous bone graft was harvested using a Cloward-type dowel cutter (Codman Dowel Cutter, Johnson & Johnson, New Brunswick, NJ). The graft was compacted to a 10-mm diameter using Compaction Pliers and cut to a 15-mm length using a scalpel. The grafts were placed into the condylar defects, and the surfaces were shaped to align with the adjacent subchondral bone (Figure 1).

The arthrotomies were closed using a five-layer simple continuous pattern of 00 polyglactin 910 (Vicryl, Ethicon Inc, Somerville, NJ) in the joint capsule, deep and superficial fascia, and subcutaneous tissue. Care was taken to keep the tissue layers separate. The skin edges were apposed using 00 nylon (Ethilon, Ethicon Inc, Somerville, NJ) in a vertical mattress pattern. The sternal incisions were closed using simple continuous patterns of 0 polyglactin 910 in the superficial fascia and subcutaneous tissue.

The skin margins were apposed using stainless steel staples (Stainless Steel Staples, Richard Allen Medical, Richland, MI). Postoperatively procaine penicillin G (22,000 IU/kg, IM, BID) and phenylbutazone (2.2 mg/kg, PO, BID) were administered for five and 10 days respectively. Stall confinement was enforced for 3 weeks followed by small paddock turnout for the remainder of the study. The skin suture and staples were removed after two weeks, and radiographs were taken at 30-day intervals. Calcein (Calcein, (2,7'[Bis] carboxymethyl-amino methyl fluorescein), Sigma, St Louis, Missouri) (20 mg/kg, IV) was administered at three months as a second fluorochrome bone label and oxytetracycline (25 mg/kg, IV) was repeated at six months (24 hours prior to euthanasia). Lameness evaluations were performed at the beginning of the study and just prior to euthanasia using a four grade system.[32]

Horses were humanely euthanatized using pentobarbital sodium (IV), and the stifle joints were examined. Following gross examination *in situ*, the medial femoral condyles were removed. The specimens were photographed and sectioned through the center of the defect using a band saw. The specimens were photographed and placed into 10% neutral buffered formalin and changed to 70% ethanol after 48 hours until processing. Xeroradiographs were taken of representative bone sections. One section was decalcified for 10 days in resin ion exchange medium of 10% formic acid. Sections were then routinely processed into paraffin blocks, sectioned at 7 µm and stained with haematoxylin and eosin. Another section was embedded in methacrylate, sectioned at 7 or 10 µm and stained with toluidine blue or left unstained for observation of fluorochrome

bone labeling. McNemar's test for paired nominal data was used to evaluate the statistical difference of occurrence of successful vs unsuccessful results. Success was defined as primary healing of the graft.

RESULTS

Minimal edema was present around all incisions by the first postoperative day and resolved after 3-4 days. Horse two developed an acute left hind lameness on postoperative day 6. Arthrocentesis demonstrated no sepsis, and a subcutaneous suture tract abscess was drained on the 10^{th} postoperative day. All other horses were grade 2/4 lame during the first 10 days of stall confinement but improved to grade 1/4 or less prior to paddock turnout. No horses demonstrated lameness at a trot prior to euthanasia. The sternal/pectoral region developed dependent edema beginning on the second post-operative day which increased slightly until day 3-4, and did not completely resolve until 10-14 days after paddock turnout began in the fourth postoperative week. The sternal incisions all healed primarily.

The radiographic appearance of the graft sites immediately after surgery was a uniform but slightly decreased density compared to the adjacent parent bone and was similar regardless of graft type (Figure 2). One month later the appearance was unchanged except in horse two which demonstrated dome-shaped lytic enlargements of both defects. At 60 days, similar lytic enlargement was present in the remaining three sites

(Horses 4N, 5C and 5N) which ultimately went on to failure (Table 1). At 90 days these lytic defects had reached maximum size, and no further radiographic changes occurred. Defects in horses 1C, 1N, 3C, 3N and 4C were more difficult to distinguish from parent bone at 90 days and were indistinguishable deeper than 3 mm from the surface by 120 days.

Gross Necropsy Observations

The incisions healed completely in all horses, and there was no gross evidence of soft tissue inflammation. The appearance of the joint capsule and synovial membrane was unremarkably similar between groups regardless of graft outcome. The medial menisci and proximal tibial articular surfaces were grossly normal in all joints.

The condylar defects were easily identified. In four of the five horses, both defects were covered with an irregular off-white dense tissue and thin dull grey tissue (Figure3). The articular cartilage adjacent to the grafts was intact, and the defects remained 10 mm in diameter. In the remaining horse (# 1) there was no gross evidence of resurfacing on either graft site. Graft incorporation could not always be determined based on the surface appearance of any defects due to consistent irregular graft surfacing.

On cut sections through the graft sites there was successful graft incorporation (the margins cancellous graft had healed well to parent bone) in 3/5 of the compacted joints and 2/5 of the conventionally-drilled joints (Figure 4). The remaining 2/5

compacted and 3/5 conventionally-drilled graft sites demonstrated variable graft resorption or resorption of parent bone adjacent to the graft. The unsuccessful grafts in the compacted group were characterized by graft resorption with minimal or no lateral or deep expansion of the parent bone defect (Figure 5). In the conventionally-drilled group, all unsuccessful graft sites expanded laterally and deeply into the subchondral bone as either solitary or multiple cystic lesions (Figure 6). The largest defect measured 2.4 cm deep X 1.6 cm wide. Utilizing the McNemar's test for paired nominal data, no statistical difference was found between success and failure of the graft groups.

Histology

The histologic appearance of the graft sites varied within individual horses and within the graft types (Table 1). Successful sites had active bone formation characterized by wide osteoid seams lined by plump osteoblasts. Areas of bone formation alternated with areas of resorption which were characterized by Howship's lacunae with or without osteoclasts. These areas of remodeling within the graft extended a maximum of 5 mm into the subchondral bone and were less than 5 mm wide.

Unsuccessful sites were lined by a poorly staining fibrous tissue and were devoid of bone and cartilage or consisted of a combination of fibrocartilage and fibrous tissue. The detritus within the defect consisted of fibrous tissue and spicules of necrotic bone. The periphery of the defects had extensive scalloping of bone by osteoclasts and abundant

reactive bone with wide osteoid seams. The outer edges had formation of new lamellar bone on the existing trabeculae.

The newly formed surface cartilage consisted of a combination of fibrocartilage, fibrous tissue and hyaline-type cartilage with irregular areas of mineralization. There were irregular clefts extending variable distances into the defects. The hyaline cartilage had an abnormal arrangement of the superficial, intermediate and radiate zones. Fibrocartilage and fibrous tissue dominated the surfaces in most defects. The tissue type of the surface cartilage appeared to vary independently of whether the graft site was successful or not.

Fluorochrome Labeling

There was mild, nonspecific diffuse staining of both the parent and graft bone throughout, and frequently single bright green (calcein) bands lined trabecular bone, corresponding to areas of active bone remodeling. Only rarely were a few residual single yellow (tetracycline) bands lining the trabecular bone. No distinction could be made between the graft and parent bone using fluorescent microscopy.

DISCUSSION

Successful incorporation of intact cancellous bone cylinder grafts in the medial femoral condyle can result in uniform cartilage formation on the articular surface.[28]

However, in previous work graft site incorporation was successful in only 50% of cases and unsuccessful cases resulted in cystic enlargement.[29] This project was designed to address problems associated with the previous grafting technique. The hypothesis was that cystic enlargement and in previous work was associated with synovial fluid influx along the host graft interface. Drilling the cystic defect with a compaction drill bit forms lateral walls of compacted cancellous bone that should be more resistant to synovial fluid pressure. Additionally, the cancellous cylinders in this project were compacted and allowed to expand within the defect forming a tight seal.

The overall results in this study closely paralleled previous similar work in that approximately half of the grafted cancellous cylinders healed primarily to the surrounding parent bone.[28, 29] However, based on gross and histologic results drilling with the compaction drill bit did prevent lateral expansion of all defects. Lateral expansion as well as secondary cyst formation developed in all unsuccessful conventionally drilled graft sites. Graft disruption and resorption occurred in 2 horses in the compaction group. Failure may have been associated with either an insufficient seal and adhesion from graft to host bone or inhibition of revascularization due to the increased density of the graft associated with compaction. Since successful grafts also occurred in the compaction-drilled sites, it is unknown whether the denser walls affected graft healing. Both horses (2 and 5) with failure in the compaction group had bilateral failure raising a question about an individual horse factor in success or failure. It was noted at the time of surgery in both of these horses that the sternal grafts formed very compacted cylinders that did not expand in the

defect, thus forming a weak seal at the interface. Sufficient numbers were unavailable to statistically examine the effect of individual horses on the results.

Fluorochrome bone labeling confirmed complete revascularization of successful graft sites in both groups at 3 months after surgery with indications of calcein labeling present in the graft sites. No distinction could be made grossly, with fluorochrome labeling or on histology between successful graft sites in the two groups.

In previous work, successful grafts became covered with a uniform cartilage surface.[28] Surfacing of successful grafts in this study was not as uniform as in previous work. Successful grafts were resurfaced with similar tissue regardless of the drilling technique used. The joint surfaces were irregular and were covered with a combination of fibrous tissue, hyaline cartilage, and fibrocartilage, but primarily fibrocartilage. Joint resurfacing on a successful graft would seem to seal the defect and limit synovitis and subsequent degenerative changes.

In summary, a method of filling defects of subchondral cysts that yields consistent results remains to be identified. Although the specific reason for some horses continuing to be lame after cyst debridement is unknown, logically, a procedure yielding a complete healing would seem to be beneficial. This procedure is not consistent enough for clinical application.

REFERENCE

1. Baxter G: Subchondral Cystic Lesions in Horses, in McIlwraith W, Trotter G (eds): Joint Disease in the Horse. Philadelphia, W.B. Saunders, 1996, pp 384-397
2. Bramlage L: Osteochondrosis Related Bone Cysts. Proc American Association of Equine Practitioners. 39, pp 83-85, 1993
3. Howard RD, McIlwraith CW, Trotter GW: Arthroscopic surgery for subchondral cystic lesions of the medial femoral condyle in horses: 41 cases (1988-1991). J Am Vet Med Assoc 206:842-850, 1995
4. Jeffcott LB, Kold SE: Clinical and radiological aspects of stifle bone cysts in the horse. Equine Vet J 14:40-46, 1982
5. McIlwraith CW: Subchondral cystic lesions (osteochondrosis) in the horse. Comp Cont Educ Pract Vet 4:S394-S404, 1982
6. McIlwraith W: Subchondral cystic lesion in the horse- the indications, methods, and results of surgery. Equine Vet Educ 2:75-80, 1990
7. Stewart B, Reid CF: Osseous cyst-like lesions of the medial femoral condyle in the horse. J Am Vet Med Assoc 180:254-257, 1982
8. Verschooten F, De Moor A: Subchondral cystic and related lesions affecting the equine pedal bone and stifle. Equine Vet J 14:47-54, 1982
9. White NA, McIlwraith CW, Allen D: Curettage of subchondral bone cysts in medial femoral condyles of the horse. Equine Vet J Suppl 6:120-124, 1988
10. Trotter GW, McIlwraith CW: Osteochondritis dissecans and subchondral cystic lesions and their relationship to osteochondrosis in the horse. J Equine Vet Sci 1:157-162, 1981
11. Rooney J: Osteochondrosis in the horse. Mod Vet Pract 56:41-43, 113-116, 1975
12. Watkins J: Osteochondrosis, in Auer J (ed): Equine Surgery. Philadelphia, W.B. Saunders, 1992, pp 971-984
13. Nixon AJ: Osteochondrosis and osteochondritis dissecans of the equine fetlock. Compend Contin Educ Pract Vet 12:1463-1475, 1990
14. Ray C, Baxter G, McIlwraith C: Development of subchondral cystic lesions following articular cartilage and subchondral bone damage in young horses. Equine Vet J 28:225-232, 1996
15. Kold SE, Hickman J, Melsen F: An experimental study of the healing process of equine chondral and osteochondral defects. Equine Vet J 18:18-24, 1986
16. Rose JA, Sande RD, Rose EM: Results of conservative management of osteochondrosis in the horse. Proc Am Assoc Equine Pract 31:617-626, 1985
17. Lewis R: A retrospective study of diagnostic and surgical arthroscopy of the equine femorotibial joint. Proc Assoc Am Equine Pract. 33, pp 887-893, 1987

18. Evans H, Jenny J: Surgical and clinical management of subchondral "bone cysts". Proc Am Assoc Equine Pract. 16, pp 195-197, 1970
19. Landells J: The bone cysts of osteoarthritis. J. Bone Joint Surg 35-B:643-649, 1953
20. Jeffcott LB, Kold SE, Melsen F: Aspects of the pathology of stifle bone cysts in the horse. Equine Vet J 15:304-311, 1983
21. Kold SE, Hickman J: Use of an autogenous cancellous bone graft in the treatment of subchondral bone cysts in the medial femoral condyle of the horse. Equine Vet J 15:312-316, 1983
22. Kold SE, Hickman J: Results of treatment of subchondral bone cysts in the medial condyle of the equine femur with an autogenous cancellous bone graft. Equine Vet J 16:414-418, 1984
23. Gross TP, Jinnah RH, Clarke HJ, et al: The biology of bone grafting. Orthopedics 14:563-568, 1991
24. Johnson AL: Principles of bone grafting. Semin Vet Med Surg (Small Anim) 6:90-99, 1991
25. Kold SE, Hickman J, Melsen F: Qualitative aspects of the incorporation of equine cancellous bone grafts. Equine Vet J 19:111-119, 1987
26. Markel M: Bone Grafts and Bone Substitutes, in Nixon A (ed): Equine Fracture Repair. Philadelphia, PA, W.B. Saunders, 1996, pp 87-92
27. Stevenson S: Bone Grafting, in Slatter DB (ed): Small Animal Surgery. Philadelphia, PA, W.B. Saunders, 1985, pp 2035-2048
28. Sullins K, Veit H, McIlwraith C: Healing of subchondral bone and articular cartilage defects in the horse. Vet Surg 16:103, 1987
29. Sullins K, Veit H, McIlwraith C: Osteochondral grafts to fill large articular defects in horses. Vet Surg 18, 1989
30. Sullins K, McIlwraith C, Powers B, et al: Evaluation of periosteal grafts in articular cartilage repair in horses. Vet Surg 14:66, 1985
31. Vachon AM, McIlwraith CW, Powers BE, et al: Morphologic and biochemical study of sternal cartilage autografts for resurfacing induced osteochondral defects in horses. Am J Vet Res 53:1038-1047, 1992
32. Stashak TS: Diagnosis of Lameness, in Stashak TS (ed): Adams' Lameness in Horses. Philadelphia, PA, Lea & Febiger, 1987, pp 100-156

LIST OF FIGURES

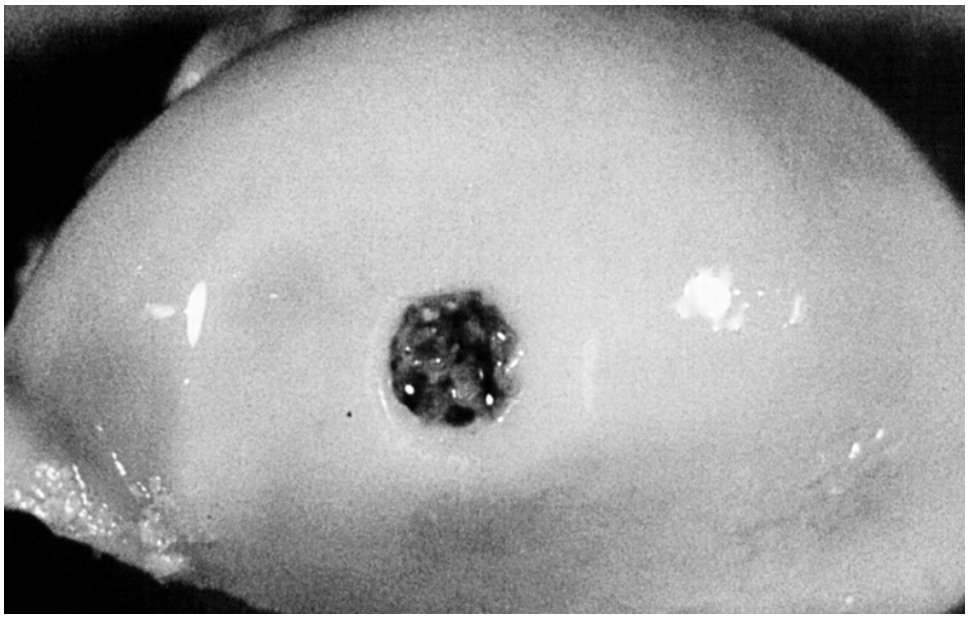

Figure 1. Surface of the condyle with the graft in situ.

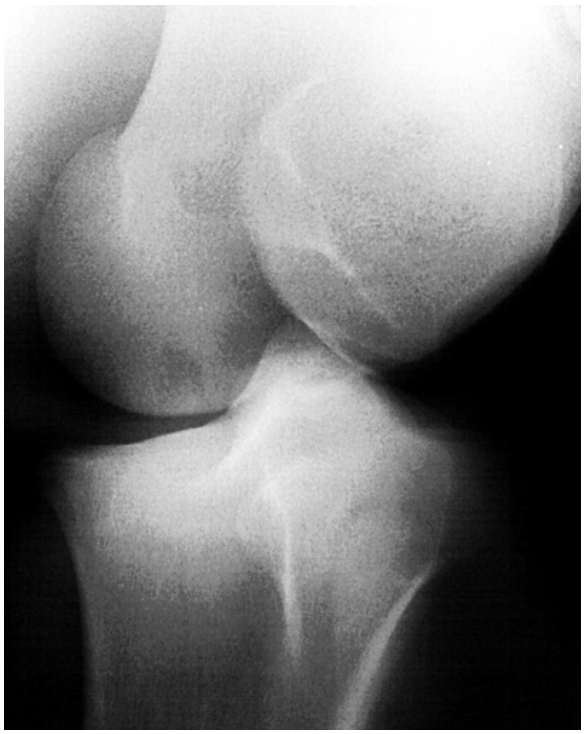

Figure 2. A post-surgical radiograph (PALM) of the graft site. Note the decreased density of the graft in the weight-bearing surface of the medial femoral condyle.

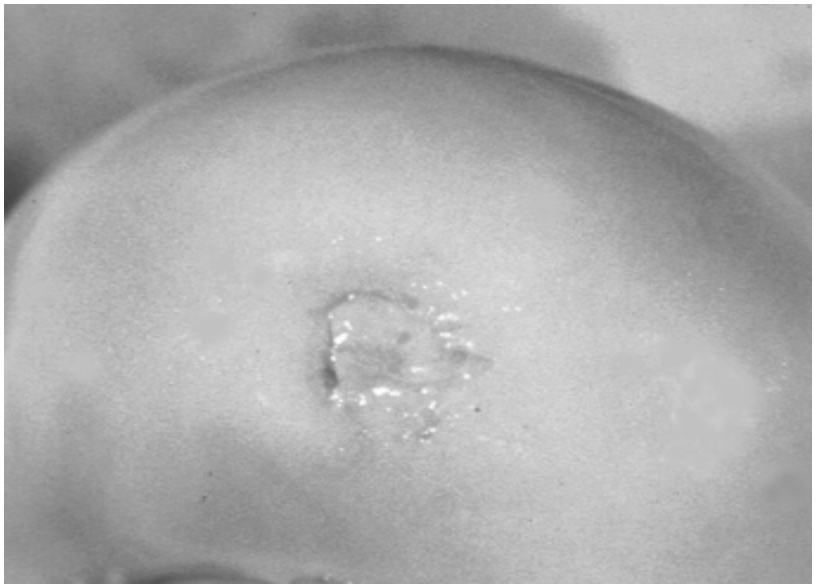

Figure 3 Surface of successful graft. Note the irregular surface with notable solid areas. It was not possible to determine the outcome of the underlying cancellous graft from looking at the surface.

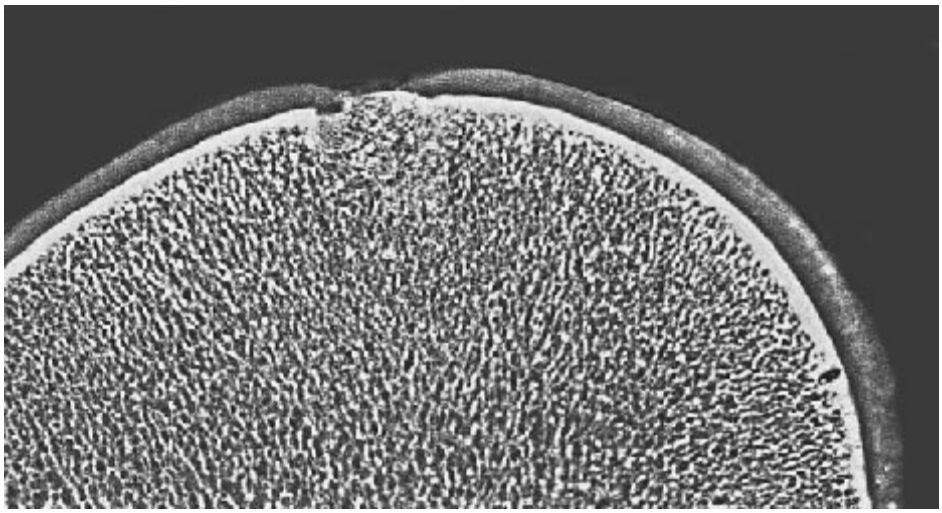

Figure 4 Xeroradiograph of a cut section of a successful graft. The margins of the cancellous graft have healed well to parent bone, but the surface bone has become slightly elevated.

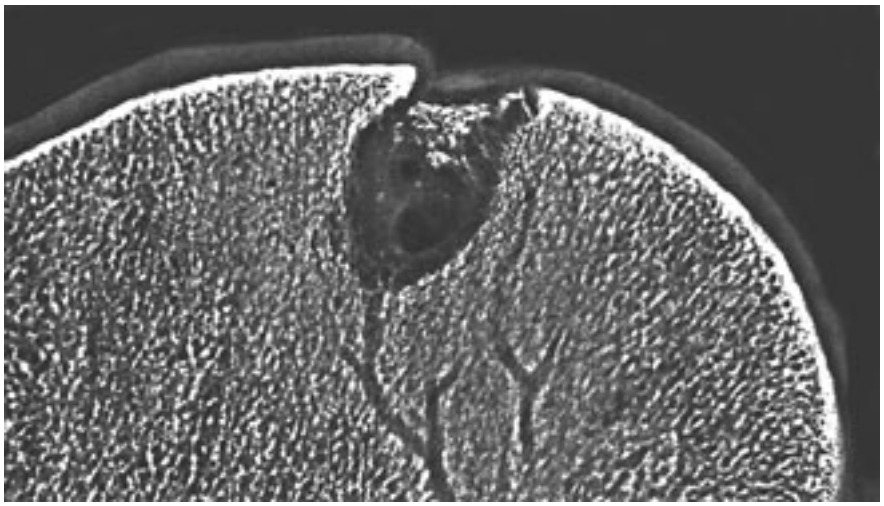

Figure 5 Unsuccessful graft from a compaction drilled graft site. Note the limited expansion of the graft site, but there is almost complete resorption of the graft. Lucent lines in the parent bone indicate further lysis is possible.

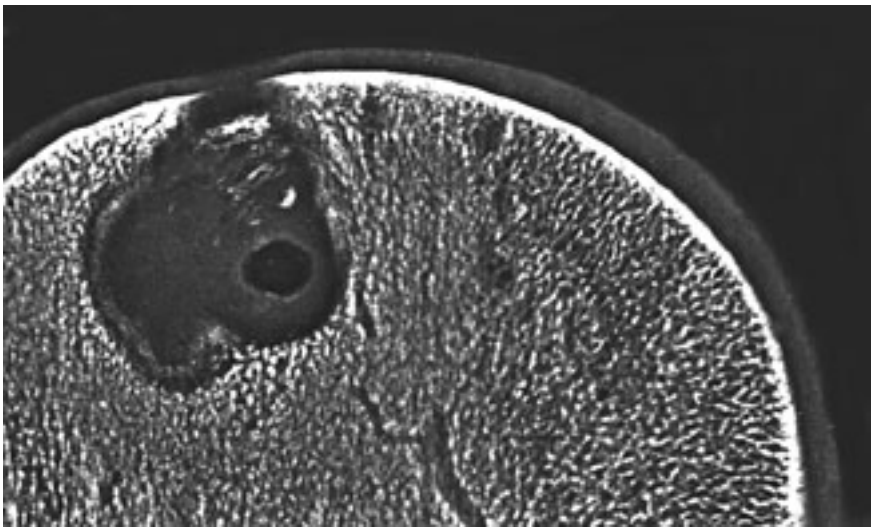

Figure 6 Unsuccessful graft from a conventionally-drilled defect. Note the expansion of the defect into the surrounding parent bone. Lucent lines in the parent bone indicate further expansion is possible.

Table 1. Summary of Results

Horse	Graft Result	Joint Surface
1C (compacted)	Successful graft incorporation	Very thin fibrous tissue surface with minimal amounts of fibrocartilage
1N (noncompacted)	Successful graft incorporation	Very thin fibrous tissue surface with minimal amounts of fibrocartilage
2C	Failure of graft incorporation with joint surface collapse No expansion of defect	Thickened hyaline cartilage combined with increased fibrocartilage
2N	Large cystic formation with lateral and deep expansion	Thickened hyaline cartilage combined with increased fibrocartilage
3C	Successful graft incorporation	Irregular surface with multiple clefts and cracks a combination of hyaline and fibrocartilage
3N	Deep graft incorporation with joint surface collapse	Multiple clefts and cracks with a combination of hyaline and fibrocartilage
4C	Successful graft incorporation	Disorganized fibrocartilage with multiple clefts and cracks
4N	Lateral and deep expansion with diffuse lysis and resorption	A combination of fibrous tissue and fibrocartilage
5C	Complete graft resorption without expansion. Lesion lined with fibrous tissue	Primarily fibrous tissue with some fibrocartilage
5N	Large cystic formation with lateral and deep expansion	Cartilage mostly absent with a thin fibrocartilage surface

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