

**University of Veterinary Medicine
Department of Surgery**

Elmaradt csontegyesüléssel sárcsonttörés kezelése mediális és kaudális csontárgzító lemezek kombinációjával négyéves ivartalanított kankutyánál: Szakirodalmi áttekintés és esettanulmány.

Management of a non-union tibial fracture using a combination of medial and caudal locking bone plates in a four-year-old neutered male dog: A literature review and case report.



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Absztrakt

Kutyáknál a sípcsonttörés viszonylag gyakori esemény, és leggyakrabban trauma következménye. Ezeknek a sérüléseknek a kezelése – a kezelési lehetőségek közötti különbségek mellett – jelentős kihívást jelenthet a kezelő állatorvosok számára az anatómia és gyógyulás komplexitása miatt.

Sor került a szakirodalom szisztematikus áttekintésére annak érdekében, hogy a szerző bővítse a témához kapcsolódó legfrissebb publikációkkal kapcsolatos ismereteit. Ez magában foglalta a sípcsont anatómiájának, a törésgyógyulás fiziológiájának, a sebészeti módszertannak és a lemezek alkalmazásának elemzését. Ez lehetővé tette a szerző számára, hogy azonosítsa a szakirodalom aktuális hiányosságait, és tájékozott, bizonyítékokon alapuló beszámolót hozzon létre egy összetett sebészeti esetről.

Ez az esettanulmány egy négyéves, a sípcsont elmaradt csontegyesüléssel járó törését mutató ivartalanított kankutya esetét írja le. Több korábbi műtét, több csontalagút és a sípcsont gyenge csontsűrűsége miatt újszerű rögzítési technika került alkalmazásra. Ez magában foglalta a sípcsont kaudális és mediális aspektusainak rögzítőlemezzel történő rögzítését.

A jelentés a sípcsont sikeres helyreállításáról és az érintett végtag teljes klinikai funkciójának visszatéréséről számol be. Ez támogatja az új rögzítési technika alkalmazását, és arra ösztönzi az állatorvosokat, hogy fontolják meg annak alkalmazását hasonló esetekben.

Abstract

Tibial fractures in canines are a relatively common event, most frequently as a result of trauma. Management of these injuries can pose a significant challenge for veterinary surgeons due to complexities in anatomy and healing in addition to the variance in treatment options.

A systematic review of the literature was undertaken to enhance the author's understanding of the most up to date publications pertinent to the subject. This involved analysis of tibial anatomy, physiology of fracture healing, surgical methodology, and the use of plating. This enabled the author to identify current gaps in the literature and create an informed, evidence-based narrative of a complex surgical case.

This case study describes a four-year-old neutered male dog presenting with a non-union fracture of the tibia. Due to multiple previous surgeries, several bone tunnels, and poor bone density of the tibia, a novel fixation technique was performed. This involved plating both the caudal and medial aspects of the tibia with a locking plate.

This report describes a successful recovery of the tibia and a return to full clinical function of the affected limb. This supports the use of the novel fixation technique and encourages veterinary surgeons to consider its use in similar cases.

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1. Introduction

A fracture is a complete or incomplete discontinuation in the continuity of bone or cartilage(1). Fractures can result in significant injuries to both soft tissue and vasculature as well as significantly compromising the function and stability of the locomotor system(1). In dogs, a fracture of the tibia is a relatively common event, accounting for 20% of all long bone fractures and 10% of all fractures(2, 3). The majority of these fractures occur as a result of trauma(2, 3). Management and repair of these injuries can pose a significant challenge to veterinary surgeons due to specific anatomical features of the tibia, fracture configuration, open/closed fractures, fracture healing potential, and the variation in treatment options.

Many tibial fractures are diaphyseal fractures involving the shaft or central part of the bone(4) such as that described in this case. In dogs, there is limited soft tissue covering of the medial aspect of the diaphysis which can contribute to complex fracture configurations and complications tibial fracture healing(3, 5). The marginal vascular supply and lack of extraosseous soft tissue have been implicated as having potential detrimental impacts resulting in delayed union and/or non-union fracture healing(5). Comprehensive understanding of the tibial anatomy and the precise application of implants are widely considered crucial to achieve successful treatment outcomes. As a consequence, selecting the most appropriate surgical management for tibial diaphyseal fractures in canines can prove critical.

This thesis describes the case of a four year old neutered male labradoodle with a tibial diaphyseal fracture. Several attempts were made at fixation which ultimately resulted in a non-union fracture. This case report describes a novel repair of a tibial non-union fracture using a combination of medial and caudal Synthes locking bone plates. A Synthetic bone morphogenic protein graft was also necessary to encourage active bone healing of the fracture. It is envisaged that by describing this technique, surgical colleagues will have the opportunity to learn the advantages and disadvantages of this method and better inform future decisions in care.

A Systematic review of the available literature was undertaken to enhance the author's understanding of current data pertinent to the subject. This review aimed to explore the following specific areas:

- The anatomy of the tibia
- Fracture healing
- Surgical principles, perspectives, and techniques in fracture management
- Application of bone plates in the repair of tibial fractures

By delving into these subgroups, the review seeks to identify existing gaps in the current literature. This dissertation further emphasises the significance of presenting this case report, which introduces a fresh perspective on addressing a common challenge in veterinary care for canines, including the placement of a caudal bone plate in the tibia.

2. Literature review

2.1 Anatomy of the Tibia

A comprehensive knowledge of the tibia is essential in understanding management of complex tibial fractures.

2.1.1. Bone anatomy

The tibia is a long bone which lies in the medial region of lower the hindlimb of a dog(4). Tibias vary in size and shape amongst dog breeds(2, 6). Long bones consists of a shaft known as the diaphysis containing the medullary canal and a metaphysis at each extremity of the bone which is below the growth plate (the physis)(7). From a surgical prospective, the tibia can be divided up into three thirds. The proximal third which articulates with the femur, the tibial shaft or middle third and distal third with a cochlea which articulates with the talus of the tarsus joint(8). The tibia also articulates proximally and distally with the fibula on the lateral side. The fibula is a thin bone with little weight bearing in dogs(4). Generally fracture repair of the fibula is generally not indicated(1).

The gross anatomy of the tibia is detailed. The proximal half of the tibia is triangular in cross-section with three surfaces (caudal, medial, lateral) while the distal half is closer to cylindrical in cross section. The proximal articulating surface of the tibia is triangular in cross-section and relatively flat, creating the tibial plateau(9, 10). This surface articulates with the medial and lateral femoral condyles(9, 10).

The very narrow cranial third contains the tibial tuberosity, where the quadriceps tendon structure inserts(9). The medial malleolus is located distally and overlies the talus bone(8). The medial surface of the tibia is broad and relatively straight in shape. Bone plates are typically applied to this surface. The lateral surface is concave in shape to accommodate the cranial tibial muscle(8) and is not used for bone plate application. The caudal tibial surface is also broad and relatively straight.

Long bones in dogs have a wide flared metaphyseal region proximally and distally(4). Cats do not have this anatomical feature in long bones and instead typically have straight bones(11). The tibia of dogs curves medial to lateral in the proximal half and lateral to medial in the distal half, giving rise to the ‘S’ shape of the tibia(10). We have selected the medial and caudal surface in the management of this complex tibial fracture.

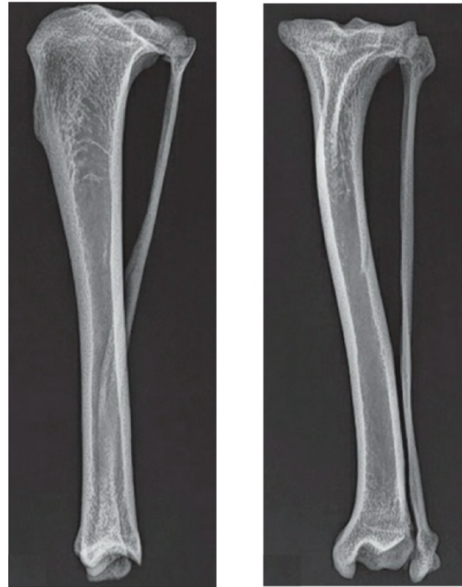


Figure 2.1 Canine anatomy medial and cranial aspect. (9)

2.1.2. Tibial vasculature supply.

In addition to bone structure and ligaments, the blood supplies to the tibia, particularly the diaphysis, inform the surgeon of their options for healing and risks of non-healing.

The canine tibia bone have three sources of arterial blood supply: nutrient artery, periosteal vessels and the proximal and distal metaphyseal arteries(5, 12, 13). The nutrient artery, supplied by the caudal tibial artery. A branch of the popliteal artery, enters the medullary canal via the nutrient foramen found on the caudolateral aspect of the proximal third of the diaphysis(14). Upon reaching the medullary canal, the nutrient artery divides into proximal and distal branches, with terminal branches anastomosing with the metaphyseal arteries at each end(15). This allows the metaphyseal arteries to maintain the medullary arterial blood supply if the nutrient artery has been interrupted due to fracture or surgical procedures(15).

The nutrient artery supplies two-thirds of the blood supply to the tibial bone. Of the nutrient artery's blood supply, 30% supplies the medullary cavity with the remaining 70% supplying the inner two thirds of the inner tibial cortex, via medullary arteries(16).

Periosteal arteries supply the remaining one third of this tibial bone through superficial vessels and soft tissue attachments to the bone(16). Vessels of the medullary canal anastomose with those of the periosteum within the cortex. Following a fracture this periosteal blood supply along with associated extraosseous soft tissue supplies the healing bone and callus formation until the reestablishment of the blood supply via the nutrient artery(5, 16-19). This is a critical detail. It is the surgeons priority to protect these surrounding vessels by splinting the fracture early prior to surgical management and to minimise further iatrogenic damage to these structures during surgical repair of the fracture.

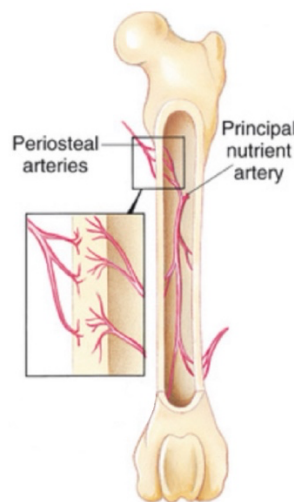


Figure 2.2 Blood supply of a canine long bone: femur. (20)

2.1.3. Tibial soft tissue envelope

Knowledge of the anatomy of the soft tissue surrounding the tibia is crucial for the surgeon to establish a safe approach during fixation of tibial diaphyseal fractures. An intricate knowledge of the tibial muscles allows the surgeon to access the surgical site in the most efficient manner. This reduces unnecessary iatrogenic damage to local musculoskeletal and neurovascular structures. There is limited soft tissue coverage of the tibia in comparison to other long bones such as the femur. This can lead to prolonged healing

times, leading to delayed or non-unions (3, 5). The tibia serves as an attachment for a limited number of small muscles/tendons. This includes the quadriceps femoris muscle (stifle joint extensor), the caudal part of the sartorius muscle (stifle joint flexor), the biceps femoris muscle (as the patella tendon of the stifle) and cranial tibial muscle (tarsal joint flexor)(4).

A medial approach to the tibia is advocated for diaphyseal fractures due to its broad flat bone surface and the minimal muscle attachments allowing for easier exposure of the bone(21). In the case of minimally invasive osteosynthesis, the important landmarks on the proximal medial aspect to be exposed are the patella tendon, the medial collateral ligament and the popliteus muscle(22). This can be achieved by retracting the caudal portion of the sartorius muscle. The limited soft tissue coverage on the distal segment allows for minimal dissection and good visualisation of the medial malleolus(21)

More commonly the medial aspect of the tibia is plated, although in this case report due to multiple previous surgeries resulting in poor bone stock and the occurrence of a non-union fracture, orthogonal plating was applied to the medial and caudal surfaces to increase stability. The caudal surface of the tibia is relatively flat with musculature attachments only present on the proximal half. These being the medial and lateral digital flexor muscles, the caudal tibial muscle and the popliteal muscle(9).

2.2 Fracture Healing

Completing a comprehensive review of the anatomy of the tibia allows us to further understand the process of fracture healing. It gives the reader a more contextual understanding of the underlying physiological mechanisms that contribute to the mending of fractures. This is essential for interpreting the rationale for different surgical techniques.

The process of fracture healing involves the regeneration of tissues, including blood vessels and bone, ideally to its pre-injured state(23). The ideal outcome to any traumatic injury to a long bone is one that culminates in the reconstruction of the structural and functional properties of the bone and the associated limb (24, 25). There are numerous aspects which influence the healing of bone: for example, the presence of excessive movement of the bone fragments, poor surrounding blood supply, the presence of infection, and the age plus overall health of the patient. Most long bone fractures have the potential ability to eventually heal, provided there is an adequate blood supply, adequate biology and less than 2% movement between the fracture ends(4).

2.2.1. Interfragmentary strain theory (IFS)

Movement of less than 2% between the ends of the fracture bone encourages the formation of bone tissue (26). Typically, granulation tissue forms initially at the fracture site as the body attempts to reduce the movement between fragments. This granulation tissue tolerates 100% elongation before it fails(26). Layers of this tissue are formed around the fracture ends. Excessive forces at the fracture site can result in the continuous disruption of granulation tissue, resulting in delayed or non-union of the fracture(26). However, if the granulation avoids disruption, further formation of this tissue will progressively reduce the degree of movement at the fracture site. When there is less than 15% movement at the fracture site, fibrocartilaginous tissue is added to the soft callus to provide further stability and therefore less movement between the bone fragments(27). When movement between the fracture ends is less than 2%, then, and only then will bone tissue form at the fracture site(26). Perren introduced the concept of Interfragmentary Strain (IFS) which defined the amount of relative movement between two fragment ends of a fracture. He quantified the IFS by measuring the size of the fracture gap before and after loading. Indeed, he

developed a formula to measure IFS: the change in the size of the fracture gap during loading, divided by the original size of the fracture gap before(28).

2.2.2. Primary Bone healing (direct healing)

Bone healing occurs by one of two processes referred to as primary and secondary bone healing(1). Primary bone healing, also known as direct bone healing, only occurs when there is absolute stability between the fracture ends (less than 2% interfragmentary strain)(1). This implies perfect anatomical reconstruction of the fracture (distance between the bone ends is <0.01mm) and a constant high interfragmentary compression. These conditions can only be achieved with open reduction and internal fixation (ORIF), typically a short transverse, simple, two-piece diaphyseal long bone fracture repaired with bone plates and screws(29) or between a two piece long oblique fracture that has been anatomically reduced and stabilised with lag screws and a neutralisation plate.

Primary bone healing allows intramembranous ossification to restore the continuity of the cortices at the fracture site(4). Cortical remodelling is achieved by direct remodelling of the lamellar bone, including the haversian canals and blood vessels (30) without the resorption of the fracture surfaces. This form of healing without the formation of periosteal and endosteal callus was originally proposed by Dallas(29, 31). In theory, it is possible to achieve both absolute stability and anatomical reconstruction. However, in practice, complete congruency of the bone surfaces is infrequently achieved. Instead, a combination of contact and gap healing occurs in primary healing(30).

2.2.2.1. Contact healing.

Contact healing occurs when the distance between the bone ends is less than 0.01mm, and the interfragmentary strain is less than 2%.(32). New bone formation occurs under simultaneous resorption and formation, achieved by osteoblasts depositing new bone following the preceding osteoclasts who advance across the fracture removing dead bone(7). This cortical remodelling and union occurs through the internal reconstruction of the haversian system without the resorption of the fracture surface(30).

2.2.2.2. Gap healing.

Gap healing occurs when the distance between the bone ends is less than 1mm and IFS less than 2%. This area is initially filled with a fibrin matrix, and with the aid of angiogenesis is remodelled to collagen tissues and other types of extracellular matrix components (33). Osteoblasts from the periosteum-derived mesenchymal stem cells deposit new bone parallel to the fracture plane, this differs from contact healing which deposit this bone parallel with the long axis of the bone. This process is similar to intramembranous ossification.

2.2.3. Secondary healing (indirect healing).

Secondary bone healing, alternatively referred to as indirect bone, is characterised by tissue differentiation and gradual stiffening of the callus tissue(27). For secondary one healing to take place there must be some form of motion at the fracture site(33). Therefore, this form of healing takes place when fractures naturally heal without surgical intervention or when surgical intervention has taken place but fails to achieve perfect anatomical reconstruction and absolute stability. Instead, relative stability is achieved. Secondary bone healing is characterised by the formation of a callus, characterised by the accumulation of reparative cells and extracellular matrix, extending the bone axially and abaxially beyond the periosteal and endosteal regions(20). Over the course of five stages, secondary bone healing decreases the IFS through two primary mechanisms. One of these mechanisms involves the removal of the dead bone within the fracture gap by osteoclasts, thereby increasing the interfragmentary distance. Consequently, diminishing the IFS to a level which allows for tissue survival. Secondly, an external periosteal callus develops on the abaxial surface of the bone, by enhancing the rigidity of the bone there'll be an increase of stability.

The five stages involved in indirect bone healing are 1. Inflammation, 2. Intramembranous Ossification, 3. Chondrogenesis, 4. Endochondral ossification and 5. Bone remodelling. (25). This entire process is orchestrated and regulated by a multitude of biologically produced mediators, including chemoattractants, along with angiogenesis and growth factors (25, 31).

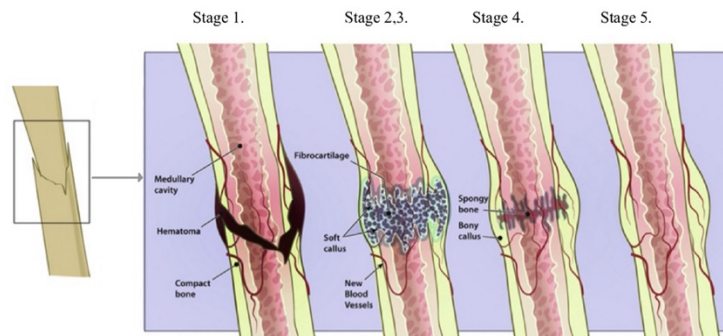


Figure 2.3. Stages of secondary bone healing(34).

Stage 1: Inflammation

This stage begins at the time of fracture until the formation of the soft callus. The goal of this stage is to begin decreasing the strain and motion at the fracture site. This is achieved by the formation of haematoma and granulation tissue which both have a high strain tolerance of 100% (26). At the time of acute bone injury, there will be haemorrhage at the fracture site, inflammatory cells will be activated as a result of the activation for the coagulation cascade. This will result in the rapid and active inflammatory response which floods the site with blood cells, platelets, monocytes and other inflammatory cells (26, 32, 35). Macrophages and platelets release bioactive molecules such as TGF-1 and PDGF into the haematoma(25). These growth factors activate mesenchymal stem cells (MSCs), these stem cells have the ability to differentiate into mature mesenchymal tissue such as cartilage or bone (36, 37). As this inflammatory phase progresses, secondary haemostasis takes place, this results in a haematoma composed of a fibrin matrix containing platelets and growth factors as a framework which will enable the invasion of the migrating cells including local tissue macrophages and polymorphonuclear neutrophils. This fibrin rich framework forms along the cortex, medullary cavity and extends beyond the periosteum into the soft tissue. This matrix will remodel into granulation tissue known as external callus.

Stage 2. Intramembranous ossification

Intramembranous ossification begins towards the end part of the preceding inflammatory stage, it occurs within the periosteum and the outer cortex adjacent to the fracture site(4). This process closely replicates intramembranous ossification observed during skeletal development(4). The resident macrophages present on the endosteal, and periosteal surfaces play a significant role here(38). New bone is synthesised through the

differentiation of the proliferating osteoprogenitor cells into osteoblasts which are present between the periosteum and the outer cortex, this subperiosteal region and the soft tissue surrounding it begin to form the hard callus(39)

Stage 3. Chondrogenesis

Given that secondary healing is characterised by endochondral ossification, a cartilaginous template precedes the formation of bony callus(40). The goal of this cartilaginous template is to bridge the fracture decreasing strain to a level which allows osteoblast survival. This stage begins once the external callus has been established and is dominated cellularly by chondrocytes and fibroblasts. As a result of the persistent chemotactic gradient, fibroblasts primarily originating from the endosteal and periosteal regions, and MSCs, mainly derived from the periosteum, infiltrate the callus (41). These MSCs undergo chondrogenesis differentiating into chondrocytes, followed by their proliferation and the synthesis of a cartilaginous matrix. Any area within this matrix where there is a deficiency of cartilage, fibroblasts produce fibrous tissue. Fibroblast and chondrocyte proliferation is stimulated by growth factors TGF-B2, TGF-1 and IGF (40, 42). The fibrin rich granulation tissue has now transitioned into a fibrocartilage plug which will splint the fracture producing a semi-rigid soft callus which is avascular(43, 44).

Stage 4. Endochondral Ossification (Hard callus Formation)

In the process of endochondral ossification, this stage represents the peak of osteogenesis activity, marked by the elevated levels of osteoblast function and development of mineralised bone matrix as its key features (44).

To enhance the rigidity of the soft callus, the chondrocytes undergo hypertrophy. It begins in the area adjacent to the intramembranous ossification mentioned earlier and mirrors the process of endochondral ossification seen at the physis during bone development (25, 45). These enlarged chondrocytes generate collagen type 10, while reduce the expression of collagen type 2 (46). This type 10 serves as a histological indicator for hypertrophic chondrocytes and the process of endochondral ossification(4). These chondrocytes produce matrix degrading proteinases like MMP, which facilitate the matrix breakdown in preparation for calcification. Pericellular mineralisation begins in the cartilage adjacent to the fracture ends. The appearance of collagen type 10 also induces tissue hypoxia, prompting the release of vascular endothelial growth factor by the hypertrophic cells,

thereby promoting vascular invasion through angiogenesis. Some authors suggest that vascularisation precedes the mineralisation in the cortex (47). Additionally, members of the bone morphogenic family play a crucial role as the mediators of this stage. Derived from perivascular MSCs, osteoblasts progress with the advancing vascular networks at the periphery of the fibrocartilage. Osteoclasts eliminate the mineralised fibrocartilage which osteoblasts contribute to depositing osteoid to initiate the woven bone formation(45, 48). This sequence continues until the fracture is fully bridged. The acquired structural strength and rigidity of this robust hard callus prove adequate for the bones to return to normal function.

Stage 5: Bone Remodelling.

The abnormal appearance and diameter of a bone that has healed by secondary healing is not permanent. The final stage of secondary bone healing is the remodelling of woven bone (deposited during secondary healing), to lamellar bone (primary bone), a structurally stronger form of bone(34). This process takes months to years. It involves the co-ordinated resorption of woven bone by osteoclasts and the production of the lamellar bone by osteoblasts (4). One of the primary regulators of bone remodelling is Wolff's Law, which states that bone in a healthy animal will adapt to the degree of mechanical loading (49).

2.3 Delayed unions and non-unions.

The management of canine fractures typically carries a positive prognosis because bone is uniquely able to regain a significant portion of its original properties and therefore resume its function(30). While the majority of fractures of the tibia managed surgically typically heal without complications, occasionally problems may arise. In a study of 195 dogs and cats, non-union occurred in approximately 4% of tibial diaphyseal fractures(3). There are many factors which influence bone healing, these being intrinsic factors for example the individual patient and the local fracture environment, and extrinsic factors like the surgeons chosen repair technique and the post-op management and owner compliance(4). If complications arise, it can result in delayed unions, non-unions and malunions. By definition, a delayed unions occurs when a fracture takes a longer time to heal than anticipated but eventually goes on to healing(1). A non-union is a fracture that has an arrested reparative process with no possibility of healing without surgical intervention(20, 50). The most common causes of delayed or non-unions of fractures are poor local biological or mechanical fracture environments(1). For example, poor selection of implants resulting in inadequate stability of the fragments, inadequate contact of bone fragments because of poor alignment or too large an interfragmentary gap, impairment of blood supply, infection, or loss of bone/bone fragments from open fracture or surgical intervention. Other general factors such as age, corticosteroid treatment, and systemic disease may also influence bone healing(51). Typically, the diagnosis of delayed and/or non-union fractures are diagnosed using clinical signs and sequential radiographs.

2.3.1. Mechanical environment.

The mechanical environment of a fracture is determined by the anatomical reduction and stability of the fracture fragments. An appropriate mechanical environment is key for achieving clinical union(23). Adequate alignment and reduction are essential to ensure fragments are stable and in close proximity to allow the formation of callus across the fracture site. The size of a fracture gap varies and depends on the individual fracture configuration, objective of the surgery team- perfect anatomical reconstruction and absolute stability, the success of the surgical technique to achieve its objectives, the stiffness and fatigue resistance of the selected implants and the post-operative care of the patient. Wider fracture gaps typically take longer to heal, in one particular research study, a non-union occurred when a 21mm section of the diaphysis was removed from a dog's

femur which had an outer diameter of 14mm despite stabilising the fracture gap with a bone plate(52). This approach was also explored in many other studies involving various bones with non-union achieved with varying length of fracture gap(53-57). Based on these studies it is evident that there is no precise threshold for fracture gap leading to non-union, however gaps approaching the width of the bone should be avoided.

Another mechanical factor which influences bone healing are the forces that are experienced at the fracture gap and their resulting motion on the fragments. Motion strongly influences the development of tissues found in direct healing and plays a crucial role in initiating proliferation and differentiation of MSCs(58). Too much motion can delay or stop healing while too little motion, for example, due to an extremely stiff fracture fixation, can eliminate essential movement at the fracture site(58). Direction and magnitude of the forces at the fracture site also have an influence on MSC proliferation and differentiation(59).

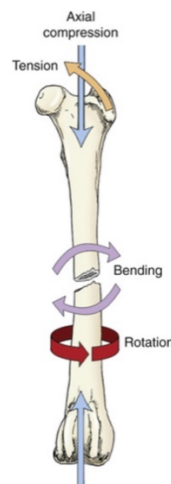


Figure 2.4: Forces experienced in long bones and must be considered by the surgeon when choosing an implant.

During weight bearing mostly axial forces are experienced across the diaphysis of long bones resulting in axial compression and tensile strain about the circumference (*figure 2.4*), therefore the healing tissue of the diaphyseal fractures will undergo compression in the centre, with stem cells favouring chondrogenesis while the abaxial surface of the callus

experiences a low to moderate magnitude of tensile strain and hydrostatic tensile stress which is the optimal environment for bone healing (60, 61). It is crucial that forces and motion remain within the tolerable limit for the healing tissue, or a fibroblastic lineage will form. The latter is unfavourable due to its slow and indirect nature for transformation to bone, leading to delayed unions or non-union(59, 62, 63). In contrast delayed or non-union can also be seen if too little strain is experienced by the fracture callus resulting in a phenomenon called stress protection(26, 64). Ideally some strain must be experienced at very low loads for cells to have adequate mechanical signalling to differentiate into bone(27).

2.3.2. Biological environment.

In the case of a fracture, the biological environment of a fracture is also crucial for optimal healing. Typically, biology can be divided into cells and growth factors. The role of growth factors has been extensively studied and reviewed with Bone Morphogenic Protein 2 (BMP-2) proven to be essential(65, 66). This was utilised in this specific case due to lack of radiographic evidence of callus formation at the fracture site after eight weeks.

Growth factors are initially released from the extracellular matrix followed by haematoma formation and platelet degranulation, which trigger a cascade of numerous factors influencing cellular activity, their concentrations adhering to a specific temporal sequence, increasing and decreasing in a defined order(4). We understand that the fracture haematoma is critical in fracture healing(67-70). We also know that the removal of the haematoma at the time of the fracture repair will prolong the fracture healing process. It is crucial that the surgeon understands the consequences of removing the fracture haematoma at the time of the repair and that they achieve their objectives when performing surgery to facilitate healing and a good functional outcome(71). Since these growth factors are initially released from the local environment around the fracture it is crucial to concentrate on preserving this environment during surgical intervention, Minimally invasive techniques aimed at preserving the soft tissue and local vascular structures are frequently documented and utilised (21, 63, 72-75). In contrast, techniques such as open reduction and internal fixation (ORIF) may remove or compromise this local environment leading to delayed healing(76). Healing time of the tibia is influenced by both negative intrinsic and extrinsic factors. Intrinsic factors, such as diminished or compromised blood supply, its limited soft tissue envelope have been implicated to cause this delayed union or non-

union(3,5). Aging can also have a detrimental effect(75), with advanced age increasing the odds of delayed unions(50). In a study of fracture non-union, 18 domestic cats that developed non-union, had a median age of five years(77).

Extrinsic factors, which are introduced by the surgeon, such as disruption of the local environment, surgical approaches that elevate the soft tissue attachments, haematoma removal through lavage and bone reaming(78, 79), also have adverse effects on healing. The primary cell type during fracture healing are mesenchymal stem cells, these cells exhibit their highest activity in young animals and experience a decline in number as the animal ages(80-82). A crucial origin of these stem cells is the inner layer of the periosteum, known as the cambium layer. MSCs can also present in the bone marrow within the metaphyseal region of bone. Hence, a fracture that occurs in a young patient, within the metaphyseal region, with thick highly vascularised periosteum, coupled with a rich soft tissue envelope and moderate hydrostatic tension have the greatest cell activity. It has been hypothesised that comminuted fractures cause more extensive damage to the periosteal and extra-osseous vasculature consequently leading to increased incidence in delayed and non-unions(77). In the case of a tibial diaphyseal comminuted fracture, with this high impact fracture resulting in excessive damage, in this location where minimal soft tissue coverage and muscle attachments, along with damage to the periosteum and its vasculature, will have an increased incidence in delayed or non-unions(3, 5).

2.3.3. Delayed unions.

Delayed union is defined as a fracture which has not healed in the anticipated time frame but one which does eventually heal(83). The normal time for a fracture to heal varies, depending on the given bone, fracture location and type, age of animal and method of fixation(83). Generally in the case of long bone fractures, radiographic evidence of bone bridging of all fracture lines can be seen at 12-16 weeks in skeletally mature dogs and 3-6 weeks in skeletally immature dogs(84). In the case of the distal half of the tibia, there are limited muscle attachments which results in a slower rate of bone healing compared to the proximal tibia which has greater muscle attachments on the lateral side(85). In a study of 442 dogs with 461 bone fractures, no delayed or non-unions were recorded for ilial fractures, which was speculated to be due to the generous soft tissue envelope which

surrounds the bone increasing the likely hood for the fractures to heal uneventfully, unlike tibia's in this study(50).

The most common reasons for delayed unions commonly stem from mechanical instability, resulting from insufficient or disrupted fixation of fracture fragments(1). Delayed unions can be seen clinically on radiographs showing a persistent fracture line, evidence of minimal or no bridging and presence of osteogenic activity (callus formation) and no significant sclerosis.

2.3.4. Non-unions.

A non-union is typically diagnosed clinically and radiographically. These fractures show the persistence of a fracture line radiographically and a definite lack of progressive fracture healing in sequential radiographs. Non-unions are widely classified according to Weber-Czech classification(86), according to their biologically active or inactive form, i.e. depending on the presence of osteogenic potential and viability which in turn is dependent on adequate blood supply(64, 86). The accuracy of this classification system has been questioned as it was developed in a time when fractures were managed non-surgically. Non-unions fractures are classified as viable or non-viable non-unions.

2.3.4.1. Viable non-union.

Viable non-unions are characterised by the presence of radiographic callus formation at the fracture ends. This is a critical finding and indicates that there is active biology at the fracture site. That is adequate blood supply and proliferating new bone. They are further subdivided by their variable degrees of callus present, into hypertrophic, moderately hypertrophic, and oligotrophic.

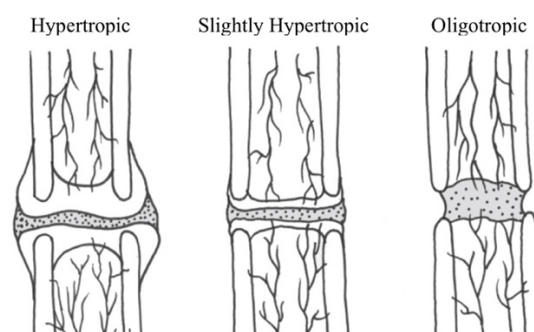


Figure 2.5: Viable non-unions showing callus formation(87).

Hypertrophic non-union have a characteristic “elephant foot” callus formation seen at both sides of the fracture line on radiographs, showing the presence of an abundant callus which remains unossified due excessive motion exceeding the tolerable limit of strain bone or cartilage can withstand, but good biological potential for healing(88), resulting in the formation of fibrous tissue instead. Moderately hypertrophic non-union is characterised by a lesser callus formation or “horse foot” callus at fragment ends. Oligotrophic non-union is a viable non-union although no radiographic evidence of callus formation due to fracture being bridged by fibrous tissue. Viable non-union usually have adequate biological activity but inadequate mechanical environment, therefore the underlying problem to the non-union is motion. This is important as bone production during fracture healing is influenced by stress and strain(89, 90). All aspects of the mechanical environment need to be investigated, whether it be alignment and apposition or the forces and motion of the fragments.

2.3.4.2. Nonviable non-union.

Nonviable non-unions, in significant contrast, are not biologically active. According to Weber & Cech they are also avascular although this has been disproven in recent years by Nicholas et al (88). In the case of non-unions, even with adequate fixation because they are biologically inactive, clinical union may not occur. These can be subdivided into dystrophic, necrotic, defect and atrophic.

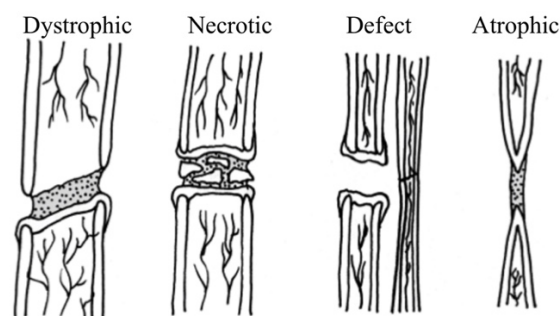


Figure 2.6: Non-viable non-union classification(87).

Dystrophic non-union is when non-viable bone is present on one or both sides of the fracture line due to poor vascularisation, this can be seen in a distal diaphyseal radius and ulna fracture. Necrotic nonviable non-union is the result of fragments not captured by the callus because of excessive motion and infection. These fragments remain in the fracture

gap and never become vascularised. They are known as sequestrum (dead piece of infected bone separated from the surrounding tissue). These biologically dead bone fragments prevent healing. A non-union is classified as a defect when the fracture gap is too large for osteosynthesis to occur. Instead, the fracture gap fills with fibrous tissue or muscle. Atrophic non-union are typically completely absent of any radiographic callus formation of the fracture gap and the resorption of the fracture ends.

2.4 Principles of fracture healing from a surgeon's perspective.

The technique of the surgeon plays a huge role in the successful planning and management of any long bone fracture(91). Indeed, a successful surgery doesn't just start at the first incision but also is influenced careful surgical planning, an appropriate surgical approach used and suitable implants(92). The surgical principles proposed by Halstead in 1890 should always be followed.

His first principal is gentle tissue handling. This includes minimal handling of the tissue, using appropriate instrumentation while sometimes the use of surgeons hands is preferred to minimise crush injuries(93) and cautious and deliberate tissue dissection(94). In the case of long procedures tissues must remain moist(84). His second principal looks at haemorrhage control of the surgical site, it is important to minimise blood loss to prevent hypovolaemia and reduced blood pressure, but also to allow for vision of the surgical site. Strict aseptic technique is a crucial, especially in orthopaedics because contaminated implants can result in infection, delayed healing and increase the risk of implant failure(95).

The fourth principle of Halsted requires the preservation of blood supply to the surrounding tissues, as previously mentioned, the surrounding blood supply dramatically influences fracture healing(76). His last two principals look at the closure of the surgical site by ensuring elimination of dead space and opposing the tissues accurately with minimal tension. Elimination of dead space is crucial to prevent the formation of a seroma or haematoma. Excessive tension on the suture influences wound healing (96), and potentially can result in wound breakdown increasing the risk of surgical site infection. Therefore, for optimal healing, the surgeon must practice gentle tissue handling and accurate suture placement with minimal tension (91).

One of the goals of fracture repair is the return of function of the limb and early ambulation. Based on this idea, a surgical technique known as Open reduction and internal fixation (ORIF) was developed by Lambotte and Danis. Reduction is the process of replacing fracture fragments to their original anatomical position, described as apposition of the fracture fragments(1). There are three methods of reduction: closed, open and indirect reduction.

Closed reduction is the traction and manipulation of the fracture fragments into their original position without the use of an open surgical site. Open reduction is the reduction of fragments by direct observation and manipulation through a surgical approach directly to the fracture site. Indirect reduction is the manipulation of fragments through a surgical site some distance from the fracture. The AO foundation created several principals for fracture treatment originally with the idea of ORIF(97), these being:

1. Restoration of fracture fragments to anatomical reduction.
2. A stable fixation that is appropriate to the clinical and biomechanical case.
3. Maintaining blood circulation to the bone fragments and surrounding soft tissue envelope by practising gentle surgical techniques and reduction.
4. Early mobilisation of the limb allowing for early ambulation.

ORIF is the reduction of fractures under direct vision and placement of internal fixation(1). ORIF should encourage anatomical reduction and stabilisation of the bone through interfragmentary compression and rigid fixation, resulting in absolute mechanical stability which allows for early weight bearing and locomotion(87). Accurate anatomical reduction can result in load sharing between the bone and the plate. The level of fracture stability achieved by the surgeon dictates the mode of healing as bone responds to stress and strain(27). In ORIF, the goal is to achieve perfect anatomical reconstruction of the fracture and absolute stability between the fragments. This can be achieved in simple two-piece fractures using bone plates as a compression plate or using a combination of lag screws and a neutralisation plate. This allows direct haversian bridging via primary healing of the fracture gap. Due to primary healing and the resultant absence of callus formation, it may take longer for bone to reach normal functional strength although with this stable repair complications are less likely to occur. It is an invasive surgical approach to bone which can lead to increased healing time due to increased tissue damage and fragment manipulation. Therefore, ORIF is typically reserved for simple two-piece fractures where internal fixation results in the perfect anatomical reconstruction and absolute stability, load sharing implants which reduce the risk of implant failure and a high likelihood of a successful outcome.

Although there are mechanical benefits, anatomical reduction of the fracture incurs a significant biological cost, via disturbance of the fracture haematoma, periosteal devascularisation and iatrogenic surgical trauma leading to delayed unions. Major

complications from the application of ORIF are for example implant failure, osteomyelitis and delayed or non-unions(98). In one particular dog study of tibial fractures fixed with ORIF, 18% of cases need revision surgery due to complications (99). It has been proposed that many of these complications are largely associated with disruption of the fracture haematoma and adjacent soft tissue which subsequently causing both delayed healing and impairment(100). Therefore, anatomical reconstruction may not be advantageous for fractures of the diaphysis and approaches preserving the biological environment often preferred(26, 76, 101-103).

In recent years, AO foundation surgical principals have undergone a gradual shift to the idea of preserving this biological environment, referred to as biological osteosynthesis (76, 102, 104). The concept of biological osteosynthesis is based on less precise reconstruction of the fracture fragments and more rigid fixation would minimise iatrogenic trauma to the fracture site, promote early callus formation and rapid secondary healing. In complex fractures (three or more fragments) the current focus is on limb alignment rather than anatomical reduction and on achieving the best possible construct stability rather than rigid interfragmentary stability(105). Nowadays, anatomical reduction is indicated for simple two-piece fractures and articular fractures.

Complex fractures are managed by leaving the fracture site and bridging the fracture site with longer bone plates(26). The use of these bridging plates allow plates to bypass the fracture site altogether (28). These complex fracture configurations are not reconstructed, the bone plate implants experience significantly higher forces as there is no load sharing with bone, so it is important to select an appropriately stiff implant. In a human study, less invasive plating was found to decrease healing times and complication rates associated with femoral fracture(106).

Surgical principals for biological osteosynthesis are as follows:

1. Indirect reduction of the fracture through the utilisation of limited surgical approaches with the aim of minimal to no disturbance of the haematoma.
2. Use of bridging implants for fracture stabilisation rather than anatomical reduction with neutralisation plates.
3. Reduced dependence on additional implants e.g. Cerclage wire, K-wires, interfragmentary screw etc.

These principals can be implemented by two techniques, known as 'Open But Do Not Touch' (OBDNT) or Minimally Invasive Osteosynthesis (MIO).

OBDNT allows the surgeon to incise the skin over the fracture site to access the bone. However, the fragments and the fracture haematoma are not touched. Alignment of fracture fragments is re-established by the manipulation of major bone fragments. This technique significantly reduced iatrogenic damage to the surrounding soft tissue envelope and the fracture haematoma. Attention must be given to the use of stiffer implants in these cases.

MIO is technically more difficult procedure which results in significantly less damage to the surrounding soft tissue envelope and potentially, faster fracture healing times. Rather than the traditional long skin surgical incision to access as fracture site, the MIO technique encourages small stab incisions at either extremity of the long bone through which the surgeon can carefully place the implant. In minimally invasive plating osteosyntheses, the bone plate is placed epiperiosteally to preserve the blood supply of the periosteum. This allows for the preservation of fracture haematoma and blood supply to the fragments and surrounding soft tissue(107), which in turn has been proven to improve callus formation, maturation and remodelling(100).

A sliding plate technique is used in MIPO via 'keyhole' holes remote from the fracture(4). The use of longer bone plates allows for the placement of screws at each extremity (108). It is important to note, to classify a surgery as MIO, it is not dependant on the size of the cutaneous incision but rather the avoidance of tissue handling to the fracture site. In the case of MIPO the fracture site cannot be directly observed, this can be difficult for the surgeon to realign the bone fragments and place fixation, therefore good anatomical knowledge of the unseen bone and adjacent structures is essential. Intra-operative imaging such as fluoroscopy is used to achieve proper bone alignment and implant placement.

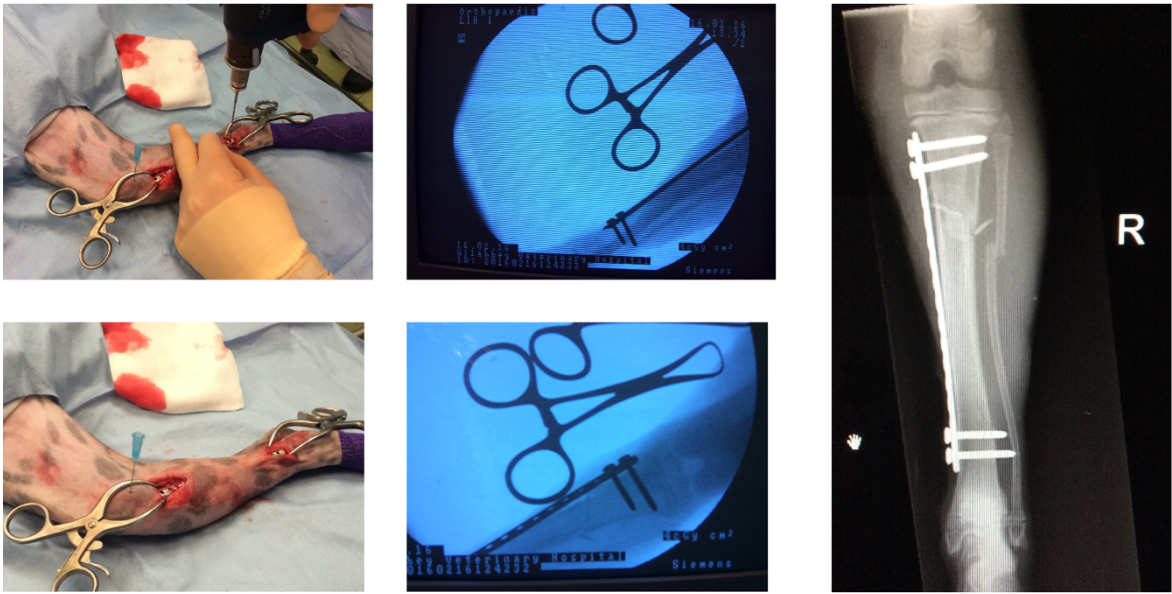


Figure 2.7: Minimally invasive osteosynthesis applied to a canine tibia with the use of a sliding bone plate and fluoroscopy (images courtesy of S. Guerin).

2.5 Bone plates

A primary goal in fracture treatment is the early return to function of the injured limb. Any fracture repair requires adequate stability with less than 2% movement at the fracture site and appropriate biology to promote the formation of bone. The repair of fractures using open reduction and internal fixation with bone plates and screws is considered a very successful option in veterinary orthopaedics (application of internal fixation using bone plates is ideal for accomplishing this). This is due to the ability of bone plates to restore rigid stability to the reconstructed fracture when properly applied(109).

Bone plates are adaptable to many situations whether it be a toy or giant breed dog, and whether it be a simple or complex fracture. Similarly, it can be used appropriately in most long bone fractures. The use of bone plates was originally restricted to diaphyseal fractures of the long bones because of the principle of application insisted on a minimum of three bicortical screws on either side of the fracture site. Metaphyseal and articular fractures often had inadequate bone stock on one side for the classical application of bone plates. More recent designs and a broadened application of bone plates using concurrent intramedullary pin or second bone plate dramatically increased the potential application of bone plates.

Plates can be used on most fractures of the tibial diaphysis, including non-unions(1). Many different designs and sizes of plates are available in the veterinary industry depending on their intended site of application and strength required. For the surgeon to achieve the optimal results from bone plates it is crucial to have an extensive knowledge of the anatomy, physiology, and possible forces acting on the fracture healing process. Many factors must be considered by the surgeon before the application of a bone plate to stabilise long bone fractures. This includes the age, the weight and behaviour of the patient, the dimension of the bone, the number and size of bone fragments, the expected biomechanical loads experienced at the fracture site, the damage to the surrounding tissue and the type and size of the plate(110).

Bone plates are made from stainless steel and titanium. Stainless steel implants are significantly stiffer than titanium and bone. Indeed, stainless steel bone plates and screws are commonly used in the management of dog and cat long bone fracture management (111). In addition, stainless steel implants are biologically well tolerated and relatively inexpensive (90). Stainless steel also has the advantage of being ductile allowing contouring to the surface of the fractured bone without breaking (112). In contrast, it is not as stiff as stainless steel and instead more closely matches the elasticity of bone, which may be more suitable to a fracture healing in an area where more strain is required for fracture healing response to develop (112). The use of titanium bone plates has been limited by surgeon preference and financial restriction compared to stainless steel (110). In the context of this case study, we will specifically address plates constructed from stainless steel for the fixation of diaphyseal fractures.

Traditionally long bone fractures were repaired with straight bone plates. Johnston and Tobias, in their textbook *'Veterinary surgery'* describe the sizes of standard bone plates used in orthopaedic surgery are named on the basis of the diameter of the screws used to secure them: 1.5, 2.0, 2.4, 2.7, 3.5 and 4.5mm. These standard bone plates typically come in a variety of lengths, typically starting with four holes and finishing with 12/14 hole plates. Custom-made plate lengths are now available giving the surgeon access to bone plates of any length.

Unstable fracture configurations, and fractures in very active patients, often require stiffer bone plate implants. The surgeon must plan these fracture repairs using a different approach when compared with simple fracture configurations. Bigger implants are selected these may include bigger sized plate/screw selection (based on the width of the bone), thicker bone plates, wider bone plates or longer bone plates (110). Stiffer constructs can also be achieved by a shorter working length of the plate over a complex fracture, locking bone plates or augmenting the bone plate implant with an intramedullary pin or second bone plate. This led to the development of broad plate, this allows more screws per unit length of the plate and also come with screws in a staggered formation to improve the holding strength of the plate (110). Cutable plates are also available in the smaller screw sizes. These plates are available in long lengths such as 50 screw holes and can be cut to the desired length (4, 110)



Figure 2.8: Broad and standard LCP plates (photo courtesy of S. Guerin)

The standard straight bone plate design has been expanded dramatically to now offer surgeons the options of various plate designs- including T-plates, acetabular plates, supracondylar plates etc.

The surgeon has direct control over the amount of additional soft tissue injury caused by the fracture repair technique selected. Careful tissue handling in all cases and Halsted's principals are still applicable. The technique used by the surgeon to apply the plate is dependent on the specific fracture involved. In the case of a simple two piece transverse/oblique fracture, concurrent damage to the surrounding soft tissues is typically low, indicating a good soft tissue envelope and biological environment. In addition, perfect anatomical reconstruction of the bone can be achieved resulting in load sharing between the bone and plate. In this instance, the plate functions as either a compression or neutralisation plate. In complex fractures, where anatomical reconstruction of the fracture is not achieved, the standard bone plate functions in a bridging manner(110). Load-sharing is not present, and the applied bone plate experiences greater forces, with a greater risk of failure. Here the load transfer occurs entirely through the bone plate and screws. The plate is secured to the bone using screws in the major proximal and distal fragments. This means the plate bears the entire load experienced by the bone until callus bridges the gap. Due to the complex fracture configuration, the surgeon typically elects not to fill the screw holes over the multiple bone fragments as to not disturb the fracture site resulting in empty screw holes and weakening of the implant. This encouraged the development of the lengthening plate. The lengthening plate has a section of solid metal in the mid-portion of the plate which is positioned over the comminuted segment of the fracture(4). This solid piece of metal significantly increases the stiffness of the bone plate and spans the fracture

site. However, these lengthening plates are infrequently used in veterinary orthopaedics because they are a significant additional expense to the surgeon.

2.5.1. Plate-rod technique

An alternative option to manage complex fractures that are not anatomically reconstructed is to combine the standard bone plate with an intramedullary pin (IM pin) of appropriate size(78). This technique is known as the plate-rod technique.

The plate-rod technique is a practical and effective approach of fracture fixation for tibial comminuted fractures when the objective is biological osteosynthesis(4). This technique has also been shown to be superior in providing greater stability in a study of canine tibial diaphyseal fractures compared to interlocking nails(113). In the tibia, the IM pin is placed using a normograde technique to alignment the major fragments and restore the bone to its original length. A standard bone plate of appropriate size and length is then applied typically with three/four bicortical bone screws in the major bone fragments(104) . In the tibia, IM pins are never placed in a retrograde fashion due to the risk of damage to the stifle joint. In a complex fracture, the attachment of the plate to the major proximal and distal fragments prevents axial collapse and rotation, while the IM pin significantly increases the bending strength of the repair in all directions(114). This plate-rod combination reduces plate strain and improves overall stiffness of the construct. This enables the fatigue life of the plate to be increased while decreasing the risk of plastic deformation(114). Plastic deformation is when a material gets permanently distorted, such as that described in this case.

In a single in-vitro study, bone plates measuring 3.5mm were used to bridge a 20mm fracture gap along with the use of intramedullary pins which occupied 30%, 40% and 50% of the medullary canal. It was found that the incremental increase of 10% in canal filling, plate strain was reduced by approximately 20%(115). In the same paper, IM pins filling 30%, 40% and 50% of the medullary canal had an overall increase in the constructs stiffness by 6%, 40% and 78% respectively(114). Clinically, it was found that the plate-rod technique using rods filling 50% of the medullary canal may be too rigid(114), which in turn decreases the beneficial effect of micromotion and its role in the formation of callus in

secondary bone healing(116). In drawing conclusions from this study, it is advisable to choose an IM pin that is roughly 30-40% of the diameter of the medullary canal of the tibia in question(72).

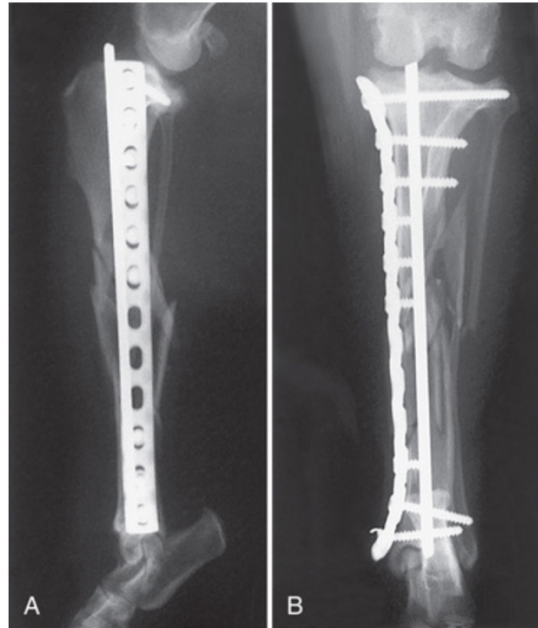


Figure 2.9: Plate-rod technique employed for the management of a comminuted fracture of the tibia(15).

2.5.2. Dual plating

The utilisation of dual plate fixation for the treatment of fractures has been described both clinically and biomechanically in veterinary and human literature(117-121). Orthogonal plating (placement of plates at a 90 degree angle to each other) of the tibia has been shown to enhance construct stiffness during mediolateral bending and has increased failure load in axial compression when compared to single plating and plate-rod constructs(122).

Locked orthogonal plates may be indicated in non-reducible tibial diaphyseal fractures where the risk of implant failure is considerable(122).

The placement of orthogonal plating allows the surgeon to maximise cortices purchased as screws can be placed on different planes at almost the same level(11). In this case report it allowed the engagement of 12 cortices above and below the fracture line to be achieved through the application of 3 bicortical screws in the proximal and distal fragments in both plate constructs respectively.

With the application of orthogonal plating to a fracture repair, a concern that may arise is the construct may be too stiff(120), resulting in ‘stress protection’ of the fracture repair. However, this has been disputed in a human biomechanical study, which revealed the application of orthogonal bone plating to a 1cm fracture gap did not exceed the stiffness of the intact model(119). This was further reinforced in a feline study, where no non-unions were observed in 11 tibial fractures repaired by orthogonal plating with all tibias achieving clinical union(11).

Locked orthogonal plates were applied to the tibial fracture in this case study.

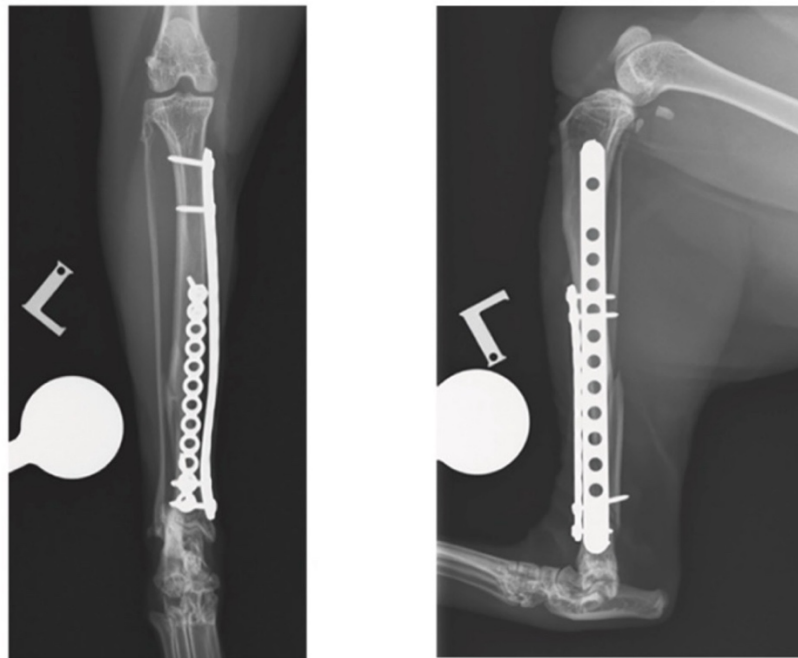


Figure 2.10: The application of orthogonal plating for a feline tibial fracture. Plates placed on the medial and cranial aspects(11).

2.6 Locking plates

Locking plates, were developed to overcome the limitations of conventional plates(123). Locking plate systems have significant benefits in that they require significantly less contouring to the bone which compared to the traditional friction bone plate systems(124). This saves the surgeon considerable time, but also reduces the risk of fracture displacement when tightening the bone screws. When the locking screws engage the plate, no further tightening is possible, therefore, the bone segment is locked into its relative position(125). The locking plates are allowed to ‘stand off’ the underlying bone surface typically by 2mm and this reduced ‘foot print’ resulted in preservation of the underlying periosteum, resulting in an biological advantage and potentially faster healing tissue(126). Locking plates are also less reliant on the underlying bone density for purchase and stability. This is especially relevant in the case of osteoporotic bones(127, 128), as described in this case report. There is also a significant shift towards biological osteosynthesis which encompasses functional fracture alignment, relative fracture stability, and the promotion of optimal biological environment by minimal disturbance of the periosteum and surrounding soft tissue envelope of the fracture.

The effectiveness of bridging osteosynthesis prompted an interest in the development of an internal fixator(127). It was this development that led to the creation of the locking plate. By securely attaching the screw to the plate, the screw-plate mechanism acts as a fixed angle construct similar to the external fixator(129). The conventional plate relies upon friction between the bone and the plate for the maintenance of stability, whilst a locking plate does not rely on bone-plate contact for strength. This eliminated the need for anatomical contouring of the plate, saving surgical time and minimises fracture displacement which is extremely beneficial for minimally invasive osteosynthesis(127). Both clinical and laboratory examinations have proven, for certain fractures, that locking plate mechanisms offered enhanced structural strength (26, 128-135). Their ability to function when off-set from the bone, allows prevention of tight bone-plate contact, minimising the periosteal damage and maintaining extraosseous vascular supply thereby enhancing healing(126).

As with the idea of biological osteosynthesis, the implant spans a longer segment of the bone which allows for minimal disturbance to the fracture(136). The stability of conventional screw and plate constructs rely on frictional forces created between the plate and the bone during axial loading (compression) or pure tension (137). These axial forces during ambulation are converted to shear forces at the bone-plate interface, with the tightest screw experiencing the largest loads. Up to a certain point, increasing screw torque can increase the strength of the construct(138). The strength of the construct depends on the ability of the screw to resist shear force or the ability of bone to resist compression(137).

Therefore, the resistance of screw pull-out is determined by the thickness and quality of bone and also the diameter of the screw. Typically, the weakest component of the conventional plate-screw construct is the sheer strength of the interface between the bone and screw(138). In locking implants, due to the construct's angular stability, the sheer stress generated during axial loading or bending is converted to compressive stress at the screw bone interface(136) and distributed between all screws.

Cortical bone, as seen in the diaphysis of the tibia, is more resistant to compressive forces than sheer(139). Therefore, the failure of locking screws requires failure of a large area of bone due to compression rather than stress concentration at a single screw-bone interface leading to screw pull-out as seen in compression plates(137, 138). This increased strength of the angle-stable locking screws against pull-out provides sufficient stability with fewer screws. This decreased dependence on screw pull-out for stability, is extremely beneficial for poor quality bone as seen in older patients or patients with previous surgeries such as the dog in this case report.

Locking bone plates appear to be replacing the use friction bone plates human surgery. This is also starting to be seen in veterinary orthopaedics. The classical indications for locking bone plates are complex fractures, weak/osteoporotic bone, and periarticular fractures(124, 129, 130, 140).

The current principals of locking plate systems in small animal orthopaedic surgery are:

- In general long bone plates should be used, spanning 80-90% of the bone(104, 141, 142).

- ‘Far, far, near, near’ technique for placement of screws, placing screws at end of plate and near the fracture to maximise construct stiffness(4)
- A minimum of 3 screws per fracture segment(104). When it comes to axial stiffness, there is little mechanical advantage in using more than 3 screws per fracture segment, and placing the 3rd screw closer to the fracture gap will increase construct stiffness(142).
- Placement of screw proximity to fracture gap depends on size of the fracture gap. In a small fracture gap (1mm), placement of screw closest to fracture should be one to two holes afar. In a larger fracture gap (greater than or equal to 6mm) placement of the innermost screw as close to the fracture line as practical is advised(142).
- Use of monocortical or biocortical locking screws.

In our case report, the use of locking bone plates was shown to be appropriate. This suitability stemmed from the significantly compromised bone quality attributed to osteoporosis resulting from numerous prior surgeries and the presence of multiple bone tunnels from previous implants. The depletion of the biological environment due to multiple surgical interventions also served as an additional indication for the use of locking bone plates in this case.

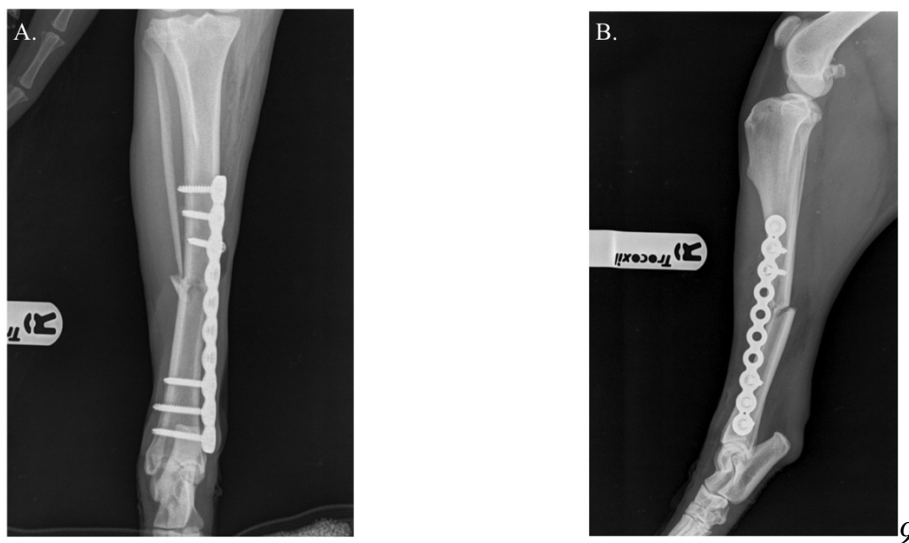
3. Case report

Management of a non-union tibial fracture using a combination of medial and caudal locking bone plates in a four-year-old male entire dog.

Signalment: Chewie, a four-year-old male labradoodle dog, weighing 21kg presented with a non-union fracture repair of his right tibia.

History: The dog suffered a mid-diaphyseal multiple fracture of the right tibia three months earlier while playing “off-lead” in a wooded area with another dog. The dog presented to his primary care veterinarian and radiographs taken. These orthogonal radiographs identified a tibial fracture with two large bone fragments and a number of small fragments present at the fracture site (unfortunately, we do not have access to these images).

The primary care veterinarian performed the initial surgeries. The first surgery took place two days after the injury. Open reduction and internal fixation was attempted with a 10 hole 3.5mm PAX plate placed on the medial aspect of the tibia. Three bone screws were placed in the proximal and distal fragments respectively (*Figure 3.1*). Four holes were left vacant over the fracture site.



*Figure 3.1: Post operative radiographs by primary care veterinarian.
(A.) Post-op caudo-cranial radiograph. (B.) Post-op mediolateral view radiograph*

Twenty-four hours later, mal-alignment of the right tibia was observed and bending of the bone plate confirmed. A second surgery was performed, allowing removal of the bone plate and the application of a 3,3 Type 1a external fixation frame to the medial aspect of the tibia. (*Figure 3.2 (A)*)

An additional connecting bar was added to the external fixation frame at a later date. (*Figure 3.2 (B)*)

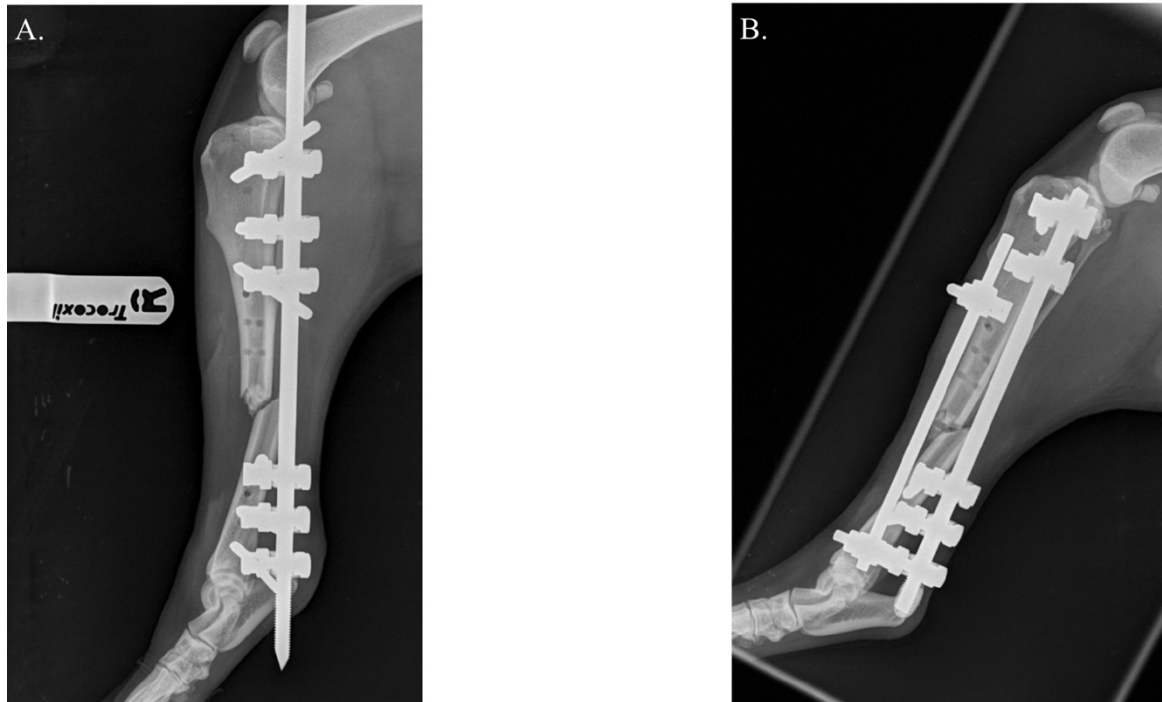


Figure 3.2: (A) Mediolateral view post 2nd surgery of external fixation. (B) Mediolateral view after 3rd surgery of additional connecting bar.

No significant callus formation was detected radiographically. Accordingly, a fourth surgery was performed three weeks later. A 10 hole 3.5mm Synthes LCP plate was applied to the cranial aspect of the tibia with three screws proximally and distally respectively. The external fixation frame remained in place.

The owner reported intermittent weight bearing during this period and malalignment of the operated limb. The dog was referred to Dr. Shane Guerin of Veterinary Specialists Ireland at this stage for further evaluation and treatment.

Physical examination: On presentation the dog was in good physical condition. He was intermittently weight bearing on the right hindlimb and mal-alignment was noted with external rotation of the distal foot. A Type 1a external fixation frame was present on the medial aspect of the tibia with purulent discharge from the proximal pins. Significant muscle atrophy was recorded in the proximal right hindlimb when compared with the contralateral limb.

Diagnostic imaging: Radiographs of the right tibia (*Figure 3.3 (A)*), confirmed the presence of the 3,3 Type 1a external fixation frame with a cranially placed bone plate and screws. A non-union of the tibial fracture was diagnosed with significant mal-alignment. No callus formation was detected on the radiographs. Radiographs of the contralateral tibia were taken to facilitate fracture planning.

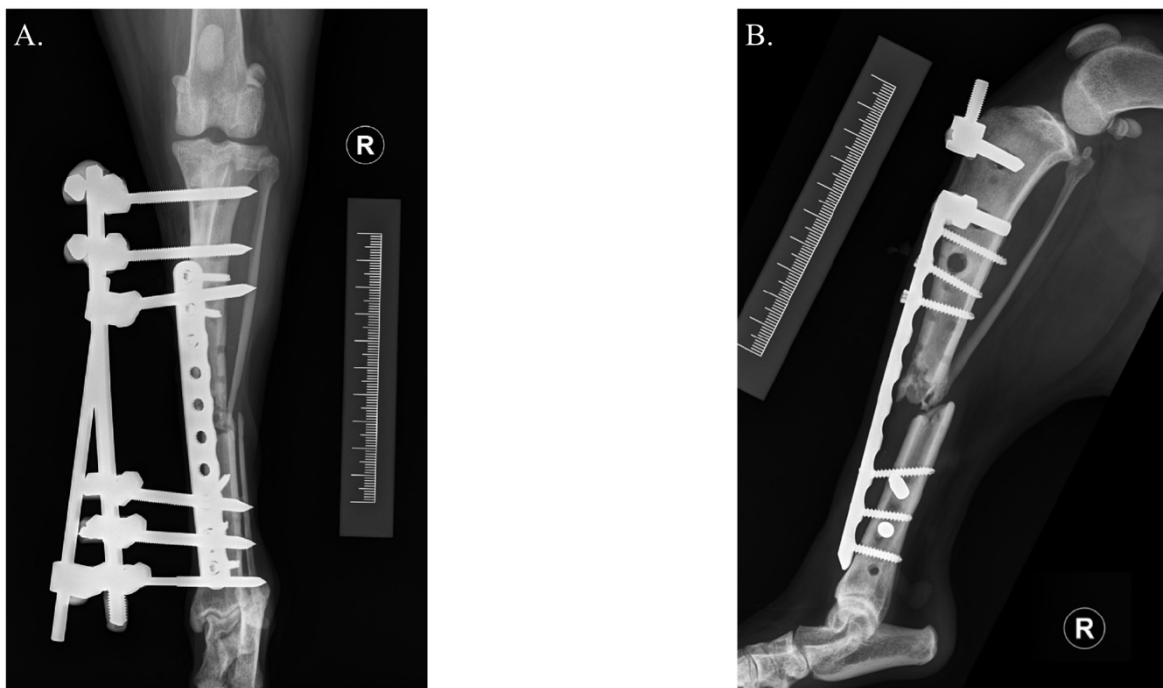


Figure 3.3: (A) Caudocranial radiographic view of tibia of dog upon presentation. (B.) Mediolateral view after removal of external fixation frame, two of the pins were loose and were removed digitally.

Procedure: A sedation and general anaesthetic was administered to the patient according to standard protocols used in the hospital.

After routine preparation, the patient was placed in dorsal recumbency with a hanging limb preparation of the right hindlimb. The remaining external fixation pins were removed. A

craniomedial approach to the right tibia was performed. The cranially located bone plate and screws were removed. Two bone screws were submitted for bacterial culture and antibiotic sensitivity. The fracture site was aggressively debrided and flushed copiously with warm saline. An osteotomy of both fracture ends was carried out to expose bleeding medullary canals.

Accurate anatomical reconstruction was achieved of the tibia with the aid of a temporary k-wire and the fracture stabilised with two orthogonal locking bone plates.

Multiple bone tunnels and poor bone density as a result of previous implant placement limited the fracture repair planning. A long 14 hole 3.5mm Synthes LCP was placed on the medial surface of the tibia with 3 bicortical locking screws placed in the proximal and distal fragments respectively. A 10 hole 2.7mm Synthes LCP was then placed on the caudal tibial surface with two locking bicortical bone screws in the proximal and distal fragments respectively. The surgical site was flushed copiously with warm saline. A synthetic bone graft was placed at the fracture site. The subcutaneous tissues closed with 3/0 polyglycolic acid and the skin with 3/0 nylon.

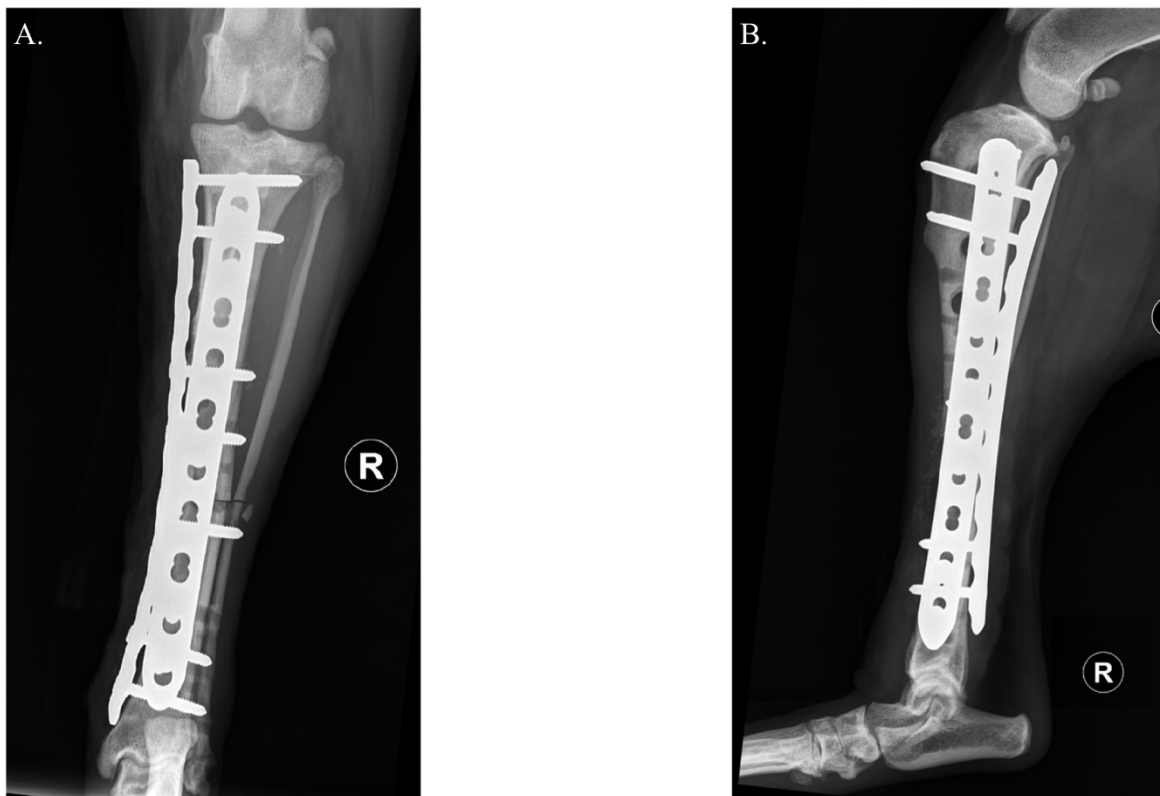


Figure 3.4: Post-operative views of orthogonal plating of the tibia.

Recovery: Chewie recovered well after surgery and quickly started weight bearing on the operated limb. Good limb alignment was achieved. Orthogonal radiographs were taken at four weeks and 8 weeks post operative. These confirmed good alignment of the fracture and stable implants. However, the lack of new callus formation at the fracture site in these sequential radiographs supported the diagnosis of delayed fracture healing.

A further operation at 8 week post-op was undertaken. Identification of the fracture site, on the medial aspect of the tibia was achieved using a small gauge hypodermic needle and fluoroscopy. A 4cm incision was made on the medial aspect directly over the fracture site. A rhBMP-2 (recombinant human bone morphogenic protein) soaked absorbable swab was careful placed around the medial and cranial aspects of the fracture site. The subcutaneous tissues were closed with 3/0 polyglycolic acid sutures and cutaneous with 3/0 nylon.

Further orthogonal radiographs taken 12 weeks post-operation (*Figure 3.5*), identifying new callus formation (*red arrow*) on the cranial aspect of the fracture site confirming progressive fracture healing.

The owners reported the dog had returned to full clinical function with 40 minute walks daily, some of which is “off-lead” exercise.

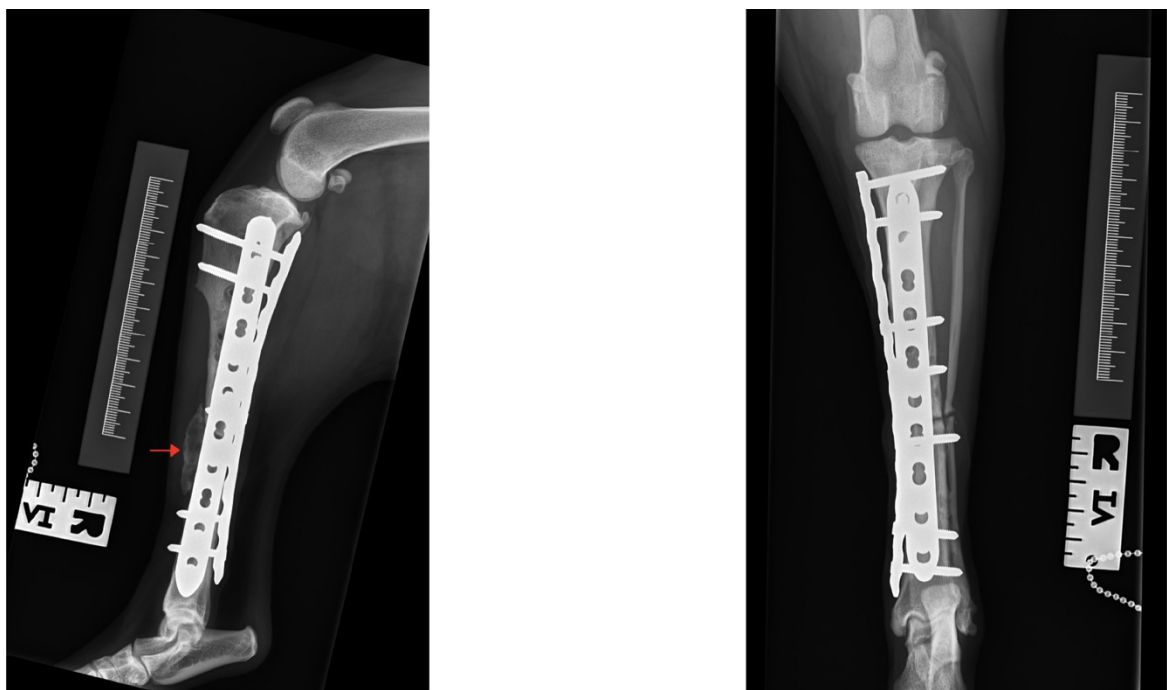


Figure 3.5: 12 week post operative orthogonal radiographs. Red arrow shows callus formation on the lateral aspect.

4. Discussion

This case report demonstrates the potential complications associated with fracture repair of the tibia in the dog. Our case emphasises the importance of an in-depth knowledge of tibial anatomy, fracture healing, surgical methodology when dealing with implants, and the application of bone plates. The current literature has a limited amount of information detailing the management of tibial non-union fractures. In this specific case, the surgeon had to address significant biological damage and poor bone stock. I believe this case study contributes valuable information to this knowledge gap.

The novel surgical technique discussed in this thesis was necessary to overcome the challenges of the presence of a non-union fracture repair of the tibia in a male labradoodle dog with poor bone stock. Numerous previous surgeries were performed at the dog's primary care veterinary clinic. Owing to a series of unsuccessful surgical procedures, including multiple implant placements and removals, several bone tunnels were present. This resulted in poor bone density of the tibia and limited bone stock for further implant placement. In this case, we were able to place two long locking bone plates by using the medial and broad caudal bone surfaces. The latter was used instead of the more typically recommended narrow cranial surface of the tibia. The poor biological environment was addressed using standard techniques, osteotomy of both fracture ends and accurate anatomical reconstruction and absolute stability. A synthetic bone graft was used during this surgery. However, an additional graft of rhBMP2 was considered necessary at eight weeks to incite a radiographic callus formation. This also proved clinically successful and should be considered in such case.

The osteoporotic bone posed a significant dilemma to the surgical team when planning this case. The limited, weak bone stock limited implant placement. A prolonged healing time was also of clinical concern. Bridging bone plates were elected to manage this dog. The surgery team selected the application of orthogonal plating to the tibia. This dual plate construct dramatically increases the construct stability, and is superior when compared to plate-rod technique. This technique offers sufficient mechanical stability at the fracture promoting fracture repair.

The patient in this case study returned to full clinical function, with early ambulation supporting the use of this novel technique. There are limitations to this technique, however, including increased financial burden for the owner with the use of double plates and screws. This surgery was performed by a highly skilled board certified surgeon, in a state of the art small animal hospital. This level of expertise and the facilities are not accessible to most practitioners.

While no two patients are the same, the author encourages veterinary surgeons to consider the use of the caudal surface of the tibial bone in future complex tibial diaphyseal fracture repair when anatomical reconstruction cannot take place. Our contribution to the literature aims to further improve outcomes for dogs like Chewie and increase their chances of returning to their favourite activities like playing “off-lead” with their canine friends. We hope that this technique can augment the quality of life of both dogs and their owners in the future.

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Thesis progress report for veterinary students

Name of student: Claire Synnott
 Neptun code of the student: FAC351
 Name and title of the supervisor: Dr. Shane Guerin & Prof. Németh Tibor.
 Department: Department of Surgery.
 Thesis title: Management of a non-union tibial fracture using a combination of medial and caudal locking bone plates in a four-year-old neutered male dog: A literature review & case report.

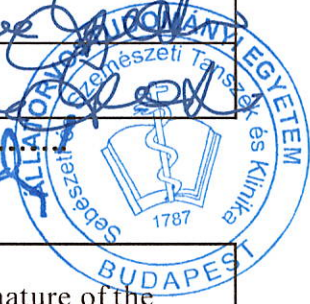
Consultation – 1st semester

Timing				Topic / Remarks of the supervisor	Signature of the supervisor
	year	month h	day		
1.	2022	09	10	overview of topic	<i>[Signature]</i>
2.	2023	1	30	Discussion of thesis title	<i>[Signature]</i>
3.	2023	02	2	Literature review discussion	<i>[Signature]</i>
4.	2023	04	4	lit review checkin	<i>[Signature]</i>
5.	2023	06	20	Discussion	<i>[Signature]</i>

Grade achieved at the end of the first semester: 52

Consultation – 2nd semester

Timing				Topic / Remarks of the supervisor	Signature of the supervisor
	year	month h	day		
1.	2023	7	11	Collect info for case report	<i>[Signature]</i>
2.	2023	7	14	Discuss findings	<i>[Signature]</i>
3.	2023	10	9	Case report	<i>[Signature]</i>





4.	2023	10	16	Evaluation of case report	<i>[Signature]</i>
5.	2023	11	1	Overview of thesis	<i>[Signature]</i>

Grade achieved at the end of the second semester:

5



The thesis meets the requirements of the Study and Examination Rules of the University and the Guide to Thesis Writing.

I accept the thesis and found suitable to defence,

[Signature]

signature of the supervisor

[Signature]

Signature of the student: *Clare Synoth*

Signature of the secretary of the department: *[Signature]*

Date of handing the thesis in: *2023.11.15.*

