Orthopedic procedures, whether performed on an elective or urgent/emergent basis, are common in small animal veterinary patients. In many instances, definitive treatment occurs at a referral center, with postoperative follow-up taking place at the primary care clinic.

During the recovery period, the primary care veterinarian needs to be able to identify complications in order to intervene as soon as possible. This article reviews basic guidelines and provides tools regarding identification of complications that may occur in adult patients recovering from orthopedic procedures.

COMMON ORTHOPEDIC PROCEDURES
Stabilization techniques for rupture of the cranial cruciate ligament are undoubtedly one of the most common orthopedic procedures performed on an elective basis; these procedures include:

- Tibial plateau leveling osteotomy (TPLO)
- Tibial tuberosity advancement (TTA)
- Lateral imbrication suture (lateral femoral fabellotibial suture).

The TPLO and TTA procedures require an osteotomy and normal bone healing for a successful outcome.

Fracture repairs are a group of orthopedic procedures that are particularly challenging because normal healing is dependent on a multitude of factors. For example:

- What was the fracture configuration?
- What repair method was used?
- What is the patient’s signalment?
- Are any comorbidities present and, if so, will they affect fracture healing?

BONE HEALING
In veterinary medicine, the majority of fractures heal via stabilization of fracture fragments by development of a callus, followed by endochondral ossification, which results in formation of new bone. More specifically, healing of bone can occur via direct (primary)—divided into gap or contact healing—or indirect (secondary) bone healing.1,2

Direct Bone Healing
Clinically, direct (primary) healing occurs via a combination of contact and gap healing,1 and requires rigid internal fixation.

Contact direct healing describes situations in which:

- Fracture/osteotomy surfaces are in direct contact
- Interfragmentary motion is not present
- Fragments are usually under compression.

Gap direct bone healing occurs when an interfragmentary gap of < 1 mm is present.

Indirect Bone Healing
Indirect (secondary) bone healing is common in patients with nonreconstructable fracture configurations, in which biologic fixation (biological osteosynthesis) methods are used. These methods minimize the extent to which the fracture site/callus and its blood supply are approached and disturbed (Figure 1). Examples of biologic fixation methods include the use of minimally traumatic surgical approaches (eg, closed alignment using an intramedullary pin), external fixator systems, and cancellous bone grafting.

Under indirect bone-healing conditions, immediately after fracture occurrence, bone union begins by:

- Accumulation of blood from periosteal, endosteal, and marrow sources, which forms a fracture hematoma
- Development of a soft tissue envelope, which surrounds the fracture site and delivers the needed blood supply to the healing bone until the endosteal, periosteal, and marrow blood supply sources are reestablished.

The phase described above is the

Figure 1. Radiograph of comminuted radius and ulna fracture at time of injury (A) and approximately 8 weeks later, after removal of failed external fixator (B). This fracture is an example of a delayed union; note extensive callus formation. While fracture is not healed, it also provides an excellent example of indirect (secondary) bone healing. Courtesy UC–Davis VMTH
reactive phase, which includes the inflammatory and granulation phases, and it is followed by the reparative and remodeling phases. While the initial phases are relatively short lived, the remodeling phase may continue for years.1

Interfragmentary Strain
Bone healing is also influenced by interfragmentary strain—a measure of the deformation that occurs in the area between fracture ends (Figure 2). The smaller the gap between the fragments, the greater the amount of strain that will occur with a given degree of deformation. Bone formation requires low strain levels.

Orthopedic implants (ie, plate and screw, interlocking nail) are designed to decrease the amount of deformation at the fracture ends. Limiting the amount of deformation decreases interfragmentary strain, which is important because, in order for bone formation to occur via gap direct bone healing, the strain in the fracture gap must be less than 2% (Figure 2).1

The clinical manifestation of this concept is somewhat counterintuitive. For example, one method by which the body reduces strain is to increase the original gap length by osteoclastic resorption.1,2 Therefore, during an early (ie, 6–8 weeks after repair) follow-up radiograph, the fracture

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**TABLE 1. Failure to Achieve Clinical Union of Fragments: Definition, Clinical Signs, & Approach**

<table>
<thead>
<tr>
<th>DEFINITION</th>
<th>CLINICAL SIGNS</th>
<th>APPROACH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DELAYED UNION</strong></td>
<td>Fracture in which healing is prolonged, but is anticipated to eventually occur</td>
<td>8- to 12-weeks postrepair: • No instability at fracture site • Minimal to no pain on palpation of fracture site • Minimal lameness; any lameness present should be improving</td>
</tr>
<tr>
<td><strong>NONUNION</strong></td>
<td>Fracture that fails to heal regardless of time since repair</td>
<td></td>
</tr>
<tr>
<td>Viable</td>
<td>Fracture typically affected by motion and/or lack of adequate mechanical stability; following are present: • Adequate biologic environment • Healing response • Excessive callus formation around fracture site</td>
<td>• Clinical instability may be noted at fracture site; with loose implants, may be gross instability • Significant pain on palpation, or with use of limb • Worsening lameness • Migrating broken implants may protrude through skin • Muscle atrophy and/or stiffness</td>
</tr>
<tr>
<td>Nonviable</td>
<td>Fracture that fails to heal accompanied by cessation of osteogenic activity; fracture site is avascular and biologically inactive</td>
<td>• Instability may be noted at fracture site • Pain on palpation of surgery site and with limb use • Static nonweight-bearing lameness typical • Fistula/draining tract may be present (necrotic cases) • Severe muscle atrophy • Significant loss of joint mobility/ range of motion</td>
</tr>
<tr>
<td><strong>MALUNION</strong></td>
<td>Fracture that heals but normal form and function of limb is not restored</td>
<td>• Mild cases: May be difficult to detect any abnormality clinically • Severe cases: Angular or torsional deformity apparent and originates at fracture site</td>
</tr>
</tbody>
</table>

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a. For example, if converting from external fixator to plate, remove all fixator components
b. Surgery involves (1) opening medullary cavity; (2) en bloc removal of affected bone ends, compression of ends using plate and screw fixation (optional); (3) autologous and/or allogeneic bone graft (essential); (4) if available, consider rhBMP-2

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![Figure 2. Strain is the change in gap length divided by original gap length. This graphical depiction of strain demonstrates how osteoclastic resorption increases the initial fracture gap and, as a result, decreases interfragmentary strain to a point where bone will form.](image-url)
gap may be wider than it was initially, but this is considered normal (Figure 3). However, if such a gap is noted during subsequent radiographic evaluations, it is considered a complication in healing.

As it heals, an unstabilized fracture or a fracture that is being addressed using indirect (secondary) bone healing can decrease strain by both formation of a fracture callus (which stabilizes the fracture and, therefore, decreases deformation) and osteoclastic resorption. \(^1\,^2\)

**COMPLICATIONS OF BONE HEALING**

Despite veterinarians’ best efforts, complications occur in bone healing, resulting in increased morbidity for patients and increased economic burden for clients.\(^3\) Examples of such complications include:

- Failure to achieve functional clinical union of fragments (Tables 1 and 2)
- Osteomyelitis
- Bone-implant construct failure (construct can fail due to problems with the implant or bone, or implant attachment to the bone)
- Fracture disease (eg, atrophy, stiffness, adhesions).

For most orthopedic cases, follow-up clinical evaluations with radiographs are generally recommended 6 to 8 weeks after surgery; further follow-up depends on the specific needs of the patient. However, in situations with precarious fixations or concern regarding client compliance after surgery, radiographs may be required 4 weeks after surgery.

**DELAYED UNION, NONUNION, & MALUNION**

These complications (Tables 1 and 2) tend to occur when the mechanical and biological environment necessary for bone healing is not optimal.

Biologically, it is essential to minimize disruption of the natural bone healing process by:

- Minimizing dissection
- Preserving surrounding soft tissue structures
- Maintaining the fracture hematoma.

From a mechanical point of view, the aim is to provide:

- Proper alignment of fracture fragments
- Adequate stability at the fracture site such that healing (bone formation) can occur (see Interfragmentary Strain).\(^1\)

A mnemonic has been developed to outline fracture assessment (Table 3, page 75).

**TABLE 2. Failure to Achieve Clinical Union of Fragments: Radiographic Changes**

<table>
<thead>
<tr>
<th>TYPE</th>
<th>RADIOGRAPHIC CHANGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>DELAYED UNION</td>
<td>Evidence of progression toward healing:</td>
</tr>
<tr>
<td></td>
<td>• Fracture lines present without adequate bridging</td>
</tr>
<tr>
<td></td>
<td>• No evidence of implant failure or loosening</td>
</tr>
<tr>
<td>NONUNION</td>
<td></td>
</tr>
<tr>
<td>Nonunion Viable</td>
<td>In general, lucent line remains at fracture site (fibrous tissue and cartilage), with ineffective bridging callus</td>
</tr>
<tr>
<td>Hypertrophic</td>
<td>Exuberant callus extends from cortical margin, but does not bridge fracture; referred to as elephant’s foot</td>
</tr>
<tr>
<td>Moderately</td>
<td>Has less callus than hypertrophic; referred to as horse’s foot</td>
</tr>
<tr>
<td>Hypertrophic</td>
<td></td>
</tr>
<tr>
<td>Oligotrophic</td>
<td>No radiographic evidence of activity; thus, no callus; rounding of fracture edges and bone resorption. Fragment ends maintain a hazy appearance—consistent with vascularity.</td>
</tr>
<tr>
<td>Nonunion Nonviable</td>
<td></td>
</tr>
<tr>
<td>Dystrophic</td>
<td>Nonviable fracture ends that often appear sclerotic (increased mineral opacity of cortical bone), with closure of medullary cavity; bony callus may be evident because vascular supply leaving fracture is intact</td>
</tr>
<tr>
<td>Necrotic</td>
<td>Often has lucency and bone loss associated with implants; may have draining tract/fistula or sequestrum</td>
</tr>
<tr>
<td>Defect</td>
<td>Occurs when gap at fracture site exceeds biologic healing process of bone; gap becomes filled with fibrous tissue and/or muscle, which appears radiographically as a radiolucent gap</td>
</tr>
<tr>
<td>Atrophic</td>
<td>Characterized by loss of bone from fracture site, often by resorption; results in rounded sclerotic bone ends that recede from fracture site</td>
</tr>
<tr>
<td>MALUNION</td>
<td>Fracture may either appear healed or have large bridging callus consistent with progression toward normal healing via secondary bone healing</td>
</tr>
</tbody>
</table>

\[Figure 3. Immediate postoperative radiograph (A) and another radiograph 8 weeks after surgery (B); note that the gap in the ulna (blue arrow) has increased in size, which is normal at this stage of healing.\]
Delayed Union
Diagnosis of a delayed union can be challenging; by definition, it is a fracture that has not healed in the typical time frame for a given fracture in a given animal.\textsuperscript{4,5} Thus, diagnosis is dependent on the knowledge of what is typical for a particular fracture (Table 4).

Causes. Causes of delayed unions can be classified as mechanical, biologic, or both.

• Biologic: For osteotomy procedures, such as TPLO or TTA, a delayed union is most often due to biologic causes, such as periosteal damage, infection, and impairment of local blood supply.\textsuperscript{4,5}

• Mechanical: Mechanical causes relate to excessive fracture gaps when bone is lost during trauma or during surgery, inadequate immobilization or immobilization for an insufficient period of time, or interposition of soft tissue structures between fracture ends (Figures 1 and 4).\textsuperscript{4,5}

Patient comorbidities are also an important consideration: advanced age, concomitant corticosteroid administration, and metabolic disease (eg, hyperadrenocorticism) can play a role in fracture healing.

Evaluation. If a fracture or osteotomy is not healing in the expected amount of time, careful evaluation of implant construct and thorough patient evaluation are necessary.

The following findings are consistent with instability, and intervention is usually advised:

• Broken implants (Figures 5 and 6)
• Radiolucency associated with bone/implant interface (Figure 7)
• Pain on palpation of the fracture site
• Increasing lameness.

A key radiographic finding that differentiates a delayed union from a nonunion is the absence of sclerotic bone at fracture ends in patients with a delayed union.

Infection. The following can be noted if infection is present:
• Excessive periosteal reaction (Figure 7), radiolucency associated with implant/bone interface, draining tracts (Figure 8, page 76), sudden onset lameness, and pain associated with implant or fracture site.

If the construct is otherwise stable, a fracture will heal despite infection. While deep percutaneous aspirates can be valuable in identifying the microbial organism and susceptibility pattern, they must be interpreted carefully because contamination with skin organisms during sampling is common.

While the definitive diagnosis of a delayed union can be difficult, suspicion of an inadequate biologic or mechanical environment warrants prompt intervention.

Nonunion
A nonunion is characterized by failure of bone healing, cessation of osteogenic activity at the fracture site, and required surgical intervention to achieve a functional outcome.

Classification. Nonunions are further divided into:
• Viable: Hypertrophic, moderately hypertrophic (Figure 9, page 76), and oligotrophic

TABLE 3. The Four As of Fracture Assessment

<table>
<thead>
<tr>
<th>Alignment</th>
<th>• Assess entire bone and, in particular, joint above and joint below. • Evaluate limb in terms of angular and torsional alignment relative to normal. • Goal is to return and maintain limb in normal alignment.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apposition</td>
<td>• Evaluate realignment of fracture fragments for apposition. • Desired amount of apposition is dependent upon method of fixation. • Maintenance of apposition is important during follow-up evaluations.</td>
</tr>
<tr>
<td>Apparatus</td>
<td>• Assess appropriateness of implants chosen and state of implants. • Evaluate each individual implant for evidence of current or impending failure.</td>
</tr>
<tr>
<td>Activity</td>
<td>• Assess biological activity of bone in response to fixation (ie, callus formation). • Evaluate fracture site for evidence of lysis and periosteal new bone formation. • Evaluation requires knowledge of patient’s age, time since repair, and consideration of factors, such as infection or other wounds/injuries.</td>
</tr>
</tbody>
</table>


TABLE 4. Typical Fracture Healing Times Based on Age of Dog

<table>
<thead>
<tr>
<th>AGE</th>
<th>TYPE I (SOME II) FIXATOR</th>
<th>PLATE or TYPE III (SOME II) FIXATOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 Months</td>
<td>2–3 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>3 to 6 Months</td>
<td>4–6 weeks</td>
<td>6–12 weeks</td>
</tr>
<tr>
<td>7 to 12 Months</td>
<td>5–8 weeks</td>
<td>12–16 weeks</td>
</tr>
<tr>
<td>&gt; 12 Months</td>
<td>7–12 weeks</td>
<td>16–30 weeks</td>
</tr>
</tbody>
</table>

a. Clinical union will take longer with increasing age and/or increasing complexity of fracture.

Figure 7. Lateral and craniocaudal radiographs of right humerus of a dog that sustained a humeral fracture 4 months previously. The fracture was repaired with an external fixator, threaded intramedullary pin, and single cerclage wire. The cerclage wire appears to have untwisted and is in the fracture site (A and B). Particularly in A, an area/ring of lucency (arrowhead) is apparent around the transcondylar pin, which is consistent with a loose implant. The significant periosteal reaction on the medial aspect of humeral condyle (*) is likely a result of motion but could also result from infection.

Figure 6. A dog in which a carpal arthrodesis was performed 8 months previously; note the broken screws (*), likely related to excessive mechanical stress associated with this procedure. Courtesy UC–Davis VMTH
• Nonviable: Dystrophic, necrotic (Figure 10), defect, and atrophic (Figure 11).

A viable nonunion often has an adequate blood supply and biologic environment but lacks sufficient mechanical stability, while a nonviable union is characterized by its avascular and biologically inactive environment.

Additional Causes. In addition to impaired blood supply, a nonunion can also occur secondary to:
• Technical failures during the repair (Figure 7)
• Bone loss as a result of injury or surgery
• Devascularization of fragments during surgical approach and dissection
• Infection (Figure 10)
• Instability (eg, mismatch of implant to bone stiffness)
• Poor fracture reduction (eg, inappropriate choice of implants) (Figure 12)
• Neoplasia.

Clinical Signs. Clinical signs of a nonunion can be variable, but common signs include:
• New, persistent, or worsening lameness
• Muscle atrophy and stiffness
• Palpable instability
• Pain on palpation or with use of the limb.

Prevention. Prevention of a nonunion is key because treatment can be difficult and nonviable nonunions, in particular, can have a poor to guarded prognosis.

Treatment. Successful treatment of a viable nonunion centers on removal of fibrous tissue in the fracture gap, addition of a graft, and rigid fixation (Figure 9), while a nonviable nonunion must be approached with the focus on preservation of soft tissue structures; as in all other fractures, rigid fixation is of paramount importance.4,5

1. Remove, reposition, or replace implants.
2. Open medullary cavity and remove sclerotic/atrophic bone ends.
3. Lavage area to remove any infection/contamination.
4. Place a suitable autologous, autogenous, or synthetic graft.

Although potentially cost prohibitive, use of recombinant human bone morphogenetic protein 2 (rhBMP-2) can contribute to a successful outcome in cases of nonviable nonunions, and its use has been documented in veterinary medicine.9

OSTEOMYELITIS AND SOFT TISSUE INFECTION

Posttraumatic osteomyelitis is not very common in elective orthopedic procedures and fracture repairs.10 However, open fractures (Figures 4 and 5) are particularly prone to infection, with the risk increasing with severity of injury.11

In the Literature

Despite our best efforts, infection can occur during elective procedures and, therefore, must be a consideration in postoperative monitoring. The infection rate associated with the TPLO procedure (Figure 10) has been reported, at the highest, as 8.4%, but a more recent study identified a rate of 3.8% for superficial or deep surgical site infections (SSI).12 While this information does not apply to orthopedic surgical procedures as a whole, it does provide a current and relevant idea of the impact of posttraumatic osteomyelitis.
Orthopedic Follow-Up Evaluations: Identifying Complications

Clinical Signs
Clinical signs associated with an infection can be variable and will depend on time since surgery. Signs often include:
• Inflammation and swelling at the surgery site
• Pain on palpation over the implant or fracture site
• Draining tracts (Figure 8)
• New/worsening or sudden onset lameness.

Diagnosis
The following can be noted if infection is present:
• Excessive periosteal reaction (Figure 7)
• Radiolucency associated with implant/bone interface.

While deep percutaneous aspirates of the infected area can be valuable in identifying the microbial organism and its susceptibility pattern, they must be interpreted carefully because contamination with skin organisms during sampling is common.

Staphylococcus species are the most common causative organism. That being said, it is crucial that samples of bone, deep tissue, and representative implants are submitted for culture and susceptibility analysis.

Antimicrobial Therapy
Infections associated with the incision or surrounding soft tissue can often be treated with antimicrobial drugs and have minimal impact on bone healing, but those associated with an implant or the bone itself are more problematic. However, if the construct is otherwise stable, a fracture will heal despite infection.

Microbial infections involving orthopedic implants often develop a bacterial biofilm, which confers resistance to systemic antimicrobial drugs. Thus, eradication of the infection necessitates removal of the implant once healing is complete. Of similar importance is the.

Case Example: Delayed/Nonunion Complications
Delayed/nonunion complications commonly occur in small/toy breed dogs with fractures in the distal ½ to ¼ of the radial diaphysis (Figure 11).

Morphometric studies have demonstrated a propensity for radial fractures in toy breeds compared with large breed dogs; the radius of toy breed dogs also has a decreased vascular supply compared with that of larger breeds. These unique mechanical and biologic properties likely contribute to the high rate (83%) of malalignment or nonunion complications when these fractures are treated with external coaptation alone. This emphasizes the need for adequate apposition and rigid fixation (eg, bone plate or external skeletal fixator) with preservation of the blood supply during fracture repair. In essence, a biological approach to the repair is advocated.
removal of any avascular bone and/or sequestra (Figure 13) that may be present.

Antimicrobial therapy should be guided by the results of culture and susceptibility analysis, and should be continued for a minimum of 6 to 8 weeks.

Follow-Up & Prognosis
Serial radiographs every 4 to 6 weeks until complete healing and resolution of radiographic signs of infection are advised.10

Prognosis is generally good for normal function unless there is significant soft tissue loss/involvement or infection is associated with a total joint implant that needs to be removed.10,11

BONE-IMPLANT CONSTRUCT FAILURE
Bone-implant construct failure can occur:
• With failure at the implant level, such as a broken screw (Figure 6) or bent plate
• In association with the bone, such as a tibial tuberosity fracture after TPLO or nonunion (Figure 14).

A very small number of these complications are attributed to the implant alone, with the major cause identified as technical errors, including:
• Inappropriately sized implants
• Inappropriate implant placements
• Use of cerclage and intramedullary pin for repair of a transverse long bone fracture (Figure 12)13
• Poor owner compliance.

Radiographic Evaluation
When evaluating serial follow-up radiographs after fracture fixation, it is generally important to evaluate several criteria:
• Evidence of implant loosening or breakage (Figures 4, 5, and 7): Breakage may not be obvious and can be obscured on a single radiographic view; thus, orthogonal views are essential. If there is evidence of loosening, assess the position of the implant relative to previous radiographs (Figure 6 and 14).
• Loss of cortical bone adjacent to the implant(s) or radiolucency: May occur with loosening or infection (Figures 7 and 10)
• Loss of reduction at fracture site or loss of alignment (Figure 7)
• Evidence of progression toward normal healing at fracture site: Is there a bridging callus? Do the fracture ends appear more rounded and less distinct?

Management
While some failures require surgical intervention, others can be managed nonsurgically. In general, the greater time since surgery, the less likely bone-implant construct failure will occur. For elective osteotomies, such as TPLO and TTA, once the 6- to 8-week follow-up evaluation is reached, the chance for bone-implant construct failure is quite low.

FRACTURE DISEASE
Fracture disease describes any other postoperative complication associated with the initial injury, fracture, or repair. Some of the more common issues are:
• Muscle atrophy
• Joint stiffness
• Fracture distal to the implant
• Articular cartilage degeneration
• Adhesion of muscle to bone/muscle scarring.

Disuse of the affected limb contributes significantly to muscle atrophy, joint stiffness, and osteopenia, which helps emphasize the importance of postoperative physical therapy.

Quadriceps contracture (Figure 15) is the most common and severe form of fracture disease in small animal patients. It is of greatest concern in:
• Young dogs and cats with femoral fractures
• Animals managed with prolonged coaptation with the limb in extension.

Quadriceps Contracture Prevention

During follow-up evaluations, the quadriceps muscles should be palpated for evidence of persistent firmness (permanent contraction); in addition, stifle range of motion should be assessed. Use of a goniometer to measure and record maximal angle of flexion and extension is important for ongoing comparisons.

For the stifle:16
• Normal flexion angle is < 45 degrees
• Normal extension angle is approximately 162 degrees.

In one reported case of quadriceps contracture, a loss of stiffe flexion, nonweight-bearing lameness, knuckling, and internal rotation were present 22 days after a second attempt to repair a femur fracture.16 Therefore, young dogs (< 12 months) should be evaluated for these signs at 10 to 14 days after surgery; then at 4- and 8-weeks postoperatively.

Quadriceps Contracture Evaluation

During follow-up evaluations, the quadriceps muscles are often used to assess muscle strength. During follow-up evaluations, the quadriceps muscles should be palpated for evidence of persistent firmness (permanent contraction); in addition, stifle range of motion should be assessed. Use of a goniometer to measure and record maximal angle of flexion and extension is important for ongoing comparisons.

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In Summary

Orthopedic procedures are commonly performed in small animal patients, and whether it is an elective procedure or urgent/emergent fracture repair, follow-up evaluations are critical in reaching a desirable outcome.

Due to the increasing frequency of these procedures, primary care veterinarians are often responsible for follow-up visits. In some cases, the primary veterinarian may be comfortable performing the evaluation, taking radiographs, and interpreting progress, while others prefer to perform the evaluation; then consult the surgeon.

In either case, a team approach between the referral surgeon and primary veterinarian is optimal in order to achieve success in managing patients after an orthopedic procedure has been performed.

References


Suggested Reading


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September/October 2014 Today’s Veterinary Practice 79