Guide to Administering Platelet-Rich Plasma



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The medical significance of our contributions serves as our primary benchmark of success and will continue into the future as the legacy of Arthrex.

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Joint injury and tendinopathy are common causes of pain and loss of performance in horses of all disciplines and ages. Addressing the pathology that is leading to the pain is the cornerstone of a treatment protocol. This could require a combination of injections, conservative therapies such as shock wave, exercise rehabilitation, and surgery. The goal of single or combinational therapy is to modify the disease process and return the horse to pain-free function and not simply to alleviate pain which can further the disease process.

Joint Injury

When considering the use of injectable therapies in the joint, the overall goal is to improve function. This improvement can be from the pain management benefits of injectable products by reducing the inflammatory and pain-sensing molecules present in a painful joint. Some products that are popular in the field may also have potential regenerative effects on the joint tissues that could be the source of joint pain by attempting to return the tissues to native function and structure. The most common treatments for joint pain in an equine patient are corticosteroids, hyaluronic acid (HA), polyacrylamide hydrogels (Arthamid), platelet-rich plasma (PRP), autologous serum rich in interleukin-1 receptor antagonist protein (IRAP), alpha-2-macroglobulin (A2M), and mesenchymal stromal cells (also known as stem cells).

Corticosteroid Overview

Corticosteroid injections provide anti-inflammatory and immunosuppressive effects when administered intra-articularly. The use of corticosteroids in orthopedic applications has fallen under scrutiny due to the concerns of profound pain relief leading to subsequent injury and degeneration of articular tissues, risks of articular infections, and potential serious systemic effects with repeated use.[1] There are two different forms of corticosteroids often used for joint injections: crystalline corticosteroids (eg, triamcinolone) and noncrystalline corticosteroids (eg, methylprednisolone). Several studies have evaluated the intra-articular use of corticosteroids in horses. Overall, these studies indicate that corticosteroids provide short-term pain relief for up to 3 weeks, with benefits diminishing at longer follow-up intervals. Functional improvements were also reduced by the 3-week follow-up. There is increasing evidence regarding the negative systemic effects on the metabolic health of horses[2] with implications that go beyond the potential for corticosteroid-induced laminitis.[1,3] These negative effects on metabolic parameters are not observed with orthobiologics such as autologous protein solution.[4]

HA Overview

Hyaluronic acid (HA) is a native component of synovial fluid that bathes the articular surfaces of the articulating joints. HA is a fluid with viscous properties that has been described as helping with shock absorption and lubrication during daily functions, and HA is an important extracellular matrix component of hyaline cartilage.[5] Studies have also indicated that HA concentration is decreased in the synovial joint with the progression of osteoarthritis (OA) leading to a logical assumption that intra-articular treatment with HA may lead to improved pain, function, and return of joint fluid components vital to cartilage health.

It is important to note that there are different formulations of commercially available HA that vary in molecular weight, concentration, and cross-linking. While there is likely a benefit with low and medium molecular weight formulations, the literature tends to favor the high molecular weight formulations of HA.[1] These high molecular weight formulations have been shown to provide similar properties to naturally occurring HA while retaining the ability to be cleared from the joint through synovial capillaries. Cross-linking HA is another formulation difference of various products on the market. Cross-linking extends the retention time of HA in the joint with presumably longer effective treatment time. The molecular agents used to induce the unnatural crosslinking of HA can promote an inflammatory response and unwanted negative adverse events.[6] HA treatment also potentially increases the endogenous production of HA within a joint, further supporting the potential efficacy of HA injections.

While HA may be a reasonable treatment option under certain conditions, it is not a cure for OA or a regenerative therapy. Studies indicate improvements in kinetic outcomes and limb function following HA treatment with peak clinical benefits between 4 and 8 weeks post-treatment. Other studies have shown no improvements in histologic assessment of cartilage degradation with HA injections in a cranial cruciate transection model. These results indicate that the injection of HA may provide some short-term pain relief through improved joint motion and a washing out of the pathological joint fluid, but does not protect against the progression of cartilage damage. The routine use of intra-articular HA is hindered by the short duration of benefits and the need for multiple injections. Combination therapy of HA and corticosteroids has been suggested as a treatment option due to their different modalities to improve joint function with OA. A study of combination treatment of HA with triamcinolone compared to triamcinolone alone indicated there was no additive benefit of combining the two treatments at the final timepoint of 3 months.[2] In other studies, there were improvements in the subjective measures that were reported by the owners, but this

combination was not difference compared to autologous fluid or stem cell-based treatments. These results indicate that symptom-based treatment modalities may not provide any improved clinical outcomes over autologous treatments such as biologic options. Additionally, they do not promote and may inhibit the cartilage healing that the autologous fluid treatments may provide.

A2M Overview

Alpha-2-macroglobulin, otherwise known as A2M, is a large plasma bound glycoprotein (180 kDa) produced primarily by the liver. This protein has the ability to form a tetramer where 4 of these proteins form a larger structure (720 kDa). This tetrameric structure has been shown in vitro to have the potential to trap and inhibit proteases within its "Venus flytrap"-like structure.[7] Trapping occurs through the "bait region," which lies between two of the large glycoproteins, enabling each tetrameric structure to trap and inhibit two proteases. However, complete neutralization of degradative activity appears to be limited by the time required to neutralize a trapped protease, and larger proteases may exceed the size capacity of the trapping region.[8] Other researchers ascribe the anti-inflammatory effect of A2M to the presence of binding receptors on the outside of the tetrameric structure, which bind interleukins (ILs). Unfortunately, when pro-inflammatory proteins, like interleukin-1 beta (IL-1β), is bound, it maintains its inflammatory activity, and these receptors are also able to bind to growth factors, which may have a beneficial effect in autologous blood therapies.[9]

While the tetrameric structure potentially has a wide range of effects it imparts on biological systems, practitioners believe it may have some beneficial treatment effects for inflamed or degradative musculoskeletal tissues. It has been theorized that a concentrated autologous form may be collected by filtering out smaller proteins from a patient's own plasma due to the unusually large size of its tetrameric structure. While research on this topic is still in its infancy, a small number of in vitro and animal models have been studied with minimal clinical research currently published. A mouse ACL transection model for post-traumatic osteoarthritis (PTOA) was injected with one of two doses of A2M or saline immediately after PTOA induction, 3 days after, and weekly for 6 weeks, which resulted in histological reduction in OA progression.[10] A similar ACL transection in a rabbit model was conducted where a single concentration was injected for 3 consecutive days following induction with the study ending at day 10, which resulted in decreased ligament stump swelling and degeneration with increased cell presence in the treatment group.[11] The study included three treatment arms consisting of a single injection of PRP, A2M, or a corticosteroid, with 25 patients per group and Kellgren-Lawrence (K-L) grade 2 or 3 osteoarthritis. At the 12-week follow-up, no significant differences in pain outcomes were observed. However, WOMAC function scores were significantly improved in the A2M group compared with the PRP group.[12]

While the peer-reviewed research on the application of A2M is limited, there is potential that it may add another treatment modality for autologous blood products. The current products marketed for A2M make the claim that they concentrate A2M from the patient's own plasma. While this is theoretically possible, there is limited data available on how well the products concentrate the protein over baseline plasma. This is particularly due to the lack of validated, commercially available A2M assays, necessitating a high level of expertise or advanced analytical equipment to analyze samples for this protein. A recent publication took these additional steps and evaluated commercially available A2M kits available in the equine market.[13] The commercially available A2M product produced only a 1.3-fold increase over baseline A2M concentrations, which did not exceed the levels achieved by currently available PRP products. These results underscore the need for further development of autologous A2M products in the equine market before the clinical efficacy of this treatment strategy can be determined.

PRP Overview

PRP is one of the more commonly examined injectable orthobiologics being used in equine OA. PRP is described as an autologous treatment that is isolated from the patient's own blood and provides potential healing effects on orthopedic pathology by providing growth factors and reducing inflammation in an affected joint. The main growth factors that are consistently found in PRP that help improve the healing capability of damaged cartilage are transforming growth factor-beta (TGF-β) and platelet-derived growth factors (PDGFs). To generate PRP, venous blood is collected into a syringe containing anticoagulant and centrifuged to separate the blood into its unique contents.

PRP is often divided into two types based on centrifugation separation techniques: plasma and buffy coat techniques. Plasma systems are often the simplest and consist of a single, relatively slow-speed spin resulting in platelets suspended in the plasma. This processing method typically results in a 2- to 3-fold increase in platelet concentration compared with the original blood sample. The final product is also generally leukocyte-reduced, provided the red blood cell layer is not disrupted during PRP collection. There are two broad types of buffy coat techniques. The first approach uses a single centrifugation step, typically performed at a relatively high speed and for a longer duration than plasma-based processing techniques. This results in the platelets and leukocytes concentrating into a visible buffy coat above of the red blood cell layer. The other buffy coat-based processing method uses two centrifugation steps performed in either a slow-fast or fast-slow sequence. With a slow spin first, platelets remain suspended in the plasma while leukocytes are

removed. The plasma is then collected and placed into a second device where it undergoes a hard spin allowing the platelets to be pelleted for retrieval. With a fast spin first, the platelets and leukocytes settle into the buffy coat. The buffy coat is then isolated and spun at a slow speed, so a high number of platelets and leukocytes are resuspended into a small volume of total plasma. There is currently no consensus on which style of buffy coat-based processing is superior for application in equine musculoskeletal injuries.

In addition to platelets, it is important to consider leukocytes and red blood concentrations in a PRP preparation. The common naming conventions for PRP are based on the number of leukocytes present. A pure PRP is a formulation that contains almost complete depletion of white blood cells. A leukocyte-poor formulation indicates significant decrease in leukocytes from baseline whole blood, but not complete depletion. A leukocyte neutral formulation is when the final white blood cell concentration is comparable to the starting whole blood concentration. Finally, a leukocyte-rich formulation indicates the concentration of leukocytes is increased over the concentration in the starting whole blood. Leukocytes-rich PRP also contain the highest volume of red blood cells of all PRP products. Within leuko-reduced PRP, the type of leukocytes that are present should also be considered. Neutrophils secrete proinflammatory proteins and matrix metalloproteinases, which are undesirable in an already inflamed tissue. In a chronic soft-tissue injury like a tendinopathy where scar tissue is present, neutrophils might be desirable in a PRP to reset the healing cascade. It is important to understand if a PRP is leuko-reduced, and also if it is neutrophil-rich or neutrophil-poor.

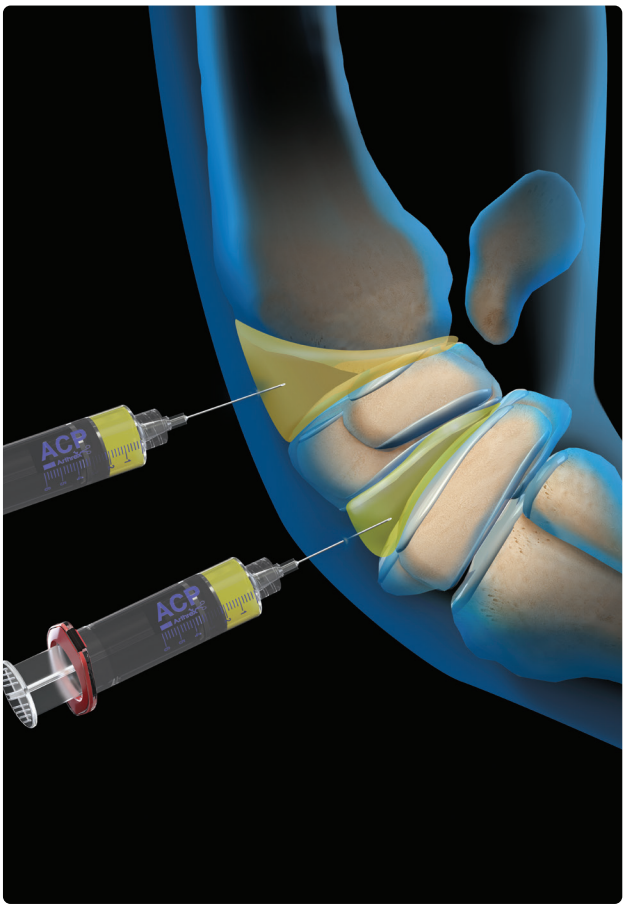
IRAP Overview

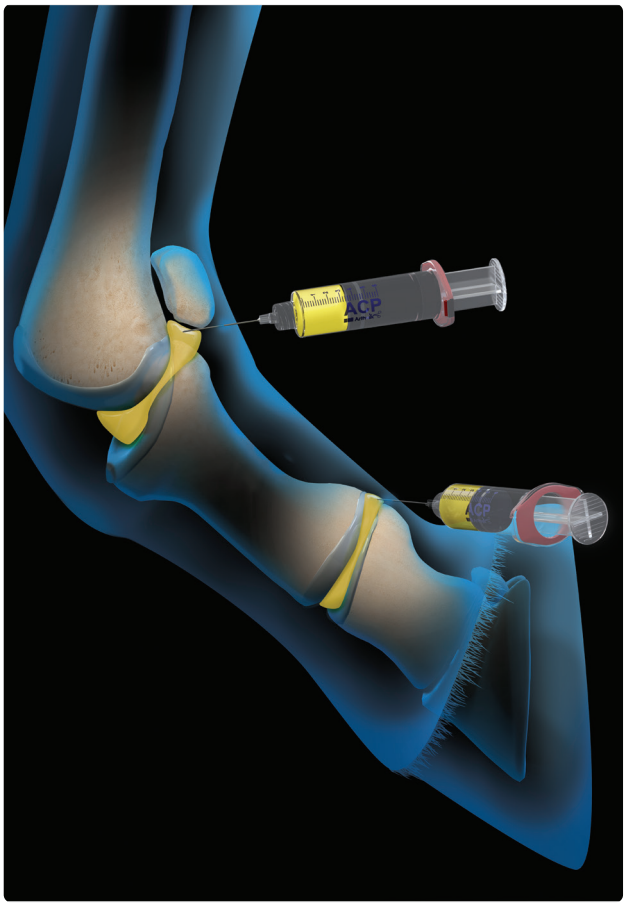
Autologous interleukin-1 receptor antagonist (IL-1RA) protein, or IRAP for short, is another autologous orthobiologic commonly used in equine veterinary medicine. Like all biologics, the protein content of IRAP is complex and contains a milieu of growth factors, chemokines, and cytokines. IRAP products are unique in their processing of autologous blood with glass beads to increase production and concentration of IL-1RA protein. Monocytes adhere to the glass beads and become stimulated to secreteIL-1RA protein. There are two other factors necessary for this production to occur. First, the blood with glass beads needs to be maintained around body temperature. The second factor is time. Current industry guidelines suggest that 16 to 24 hours is required to optimize IL-1RA production relative to other proteins produced by blood cells. This timeline fits well in clinical practice where blood can be drawn and incubated with the glass beads overnight until the final processing is performed. The final processing step consists of high-speed centrifugation to remove cellular debris, resulting in an IL-1RA-enriched serum. The serum is then filtered through a syringe Millipore filter and injected into the patient. Any remaining IRAP can be frozen for repeat injections if indicated which is then filtered through a syringe Millipore filter, injected into the patient with any remaining IRAP frozen for repeat injection if indicated.

PRP Joint Injection Reference Guidelines

Carpus

- Maintain the limb in flexion.
- Enter the radiocarpal joint dorsally between the distal radius and the proximal aspect of the radial carpal bone, medial or lateral to the extensor carpi radialis tendon within the palpable joint space. Insert the needle perpendicular to the skin.
- Enter the intercarpal joint dorsally between the distal edge of the radial carpal bone and the proximal edge of the third carpal bone, medial or lateral to the extensor carpi radialis tendon within the palpable joint space. Insert the needle perpendicular to the skin. Note that the intercarpal joint communicates with the carpometacarpal joint.





Fetlock

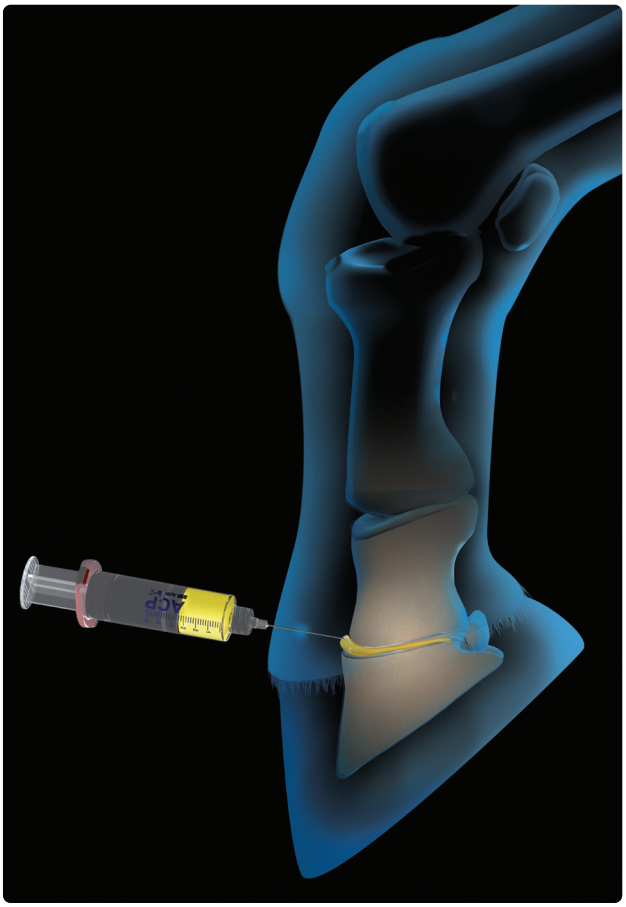
- Position the horse weight-bearing or hold the limb in flexion.
- Enter the joint laterally between the palmarodistal aspect of the third metacarpal bone, the dorsal edge of the suspensory ligament branch, the distal end of metacarpal IV, and proximal to the collateral sesamoidean ligament. Angle the needle tip slightly downward toward the center of the pouch.

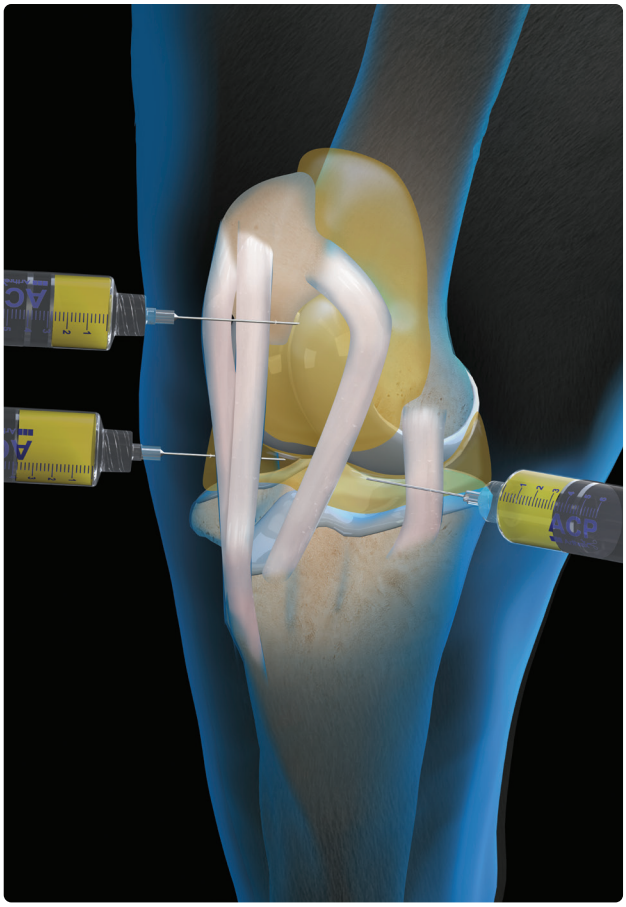
Pastern

- Maintain the limb in flexion for the palmar approach. Palpate the bony prominence of the proximopalmar middle phalanx, then insert the needle just proximal to this prominence and perpendicular to the sagittal plane of the proximal phalanx.
- Ensure the horse is weight-bearing for the dorsal approach. Locate the dorsal aspect of the distolateral proximal phalanx at the origin of the lateral collateral ligament. Insert the needle just distal to this eminence, beneath the common digital extensor tendon in the forelimb or the long digital extensor tendon in the hindlimb, and parallel to the ground.

Coffin

- Ensure the horse is weight-bearing.
- The dorsal inclined approach is performed by inserting the needle on the dorsal midline 1 cm proximal to the coronary band and perpendicular to the slope of the middle phalanx until the middle phalanx is encountered.



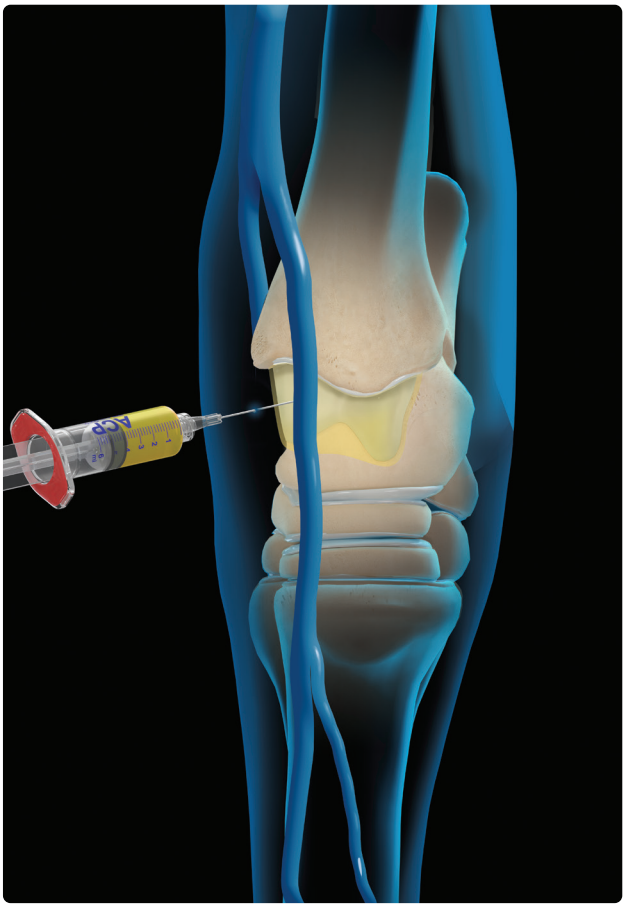


Stifle

- Although local anesthetic may diffuse between compartments, it is unclear whether biologics or other medications diffuse between compartments. Therefore, it is recommended to treat each affected compartment separately.
- All injections can be performed with the horse in a weight-bearing position. Direct all needles to the ground.
- Inject the femoropatellar joint between the middle and medial patellar ligaments, 3 cm proximal to the proximal tibial tuberosity.
- Enter the medial femorotibial compartment between the medial patellar ligament and the medial femorotibial ligament just proximal to the proximal tibia.
- Enter the lateral femorotibial compartment at the caudal aspect of lateral patellar ligament just proximal to the tibia

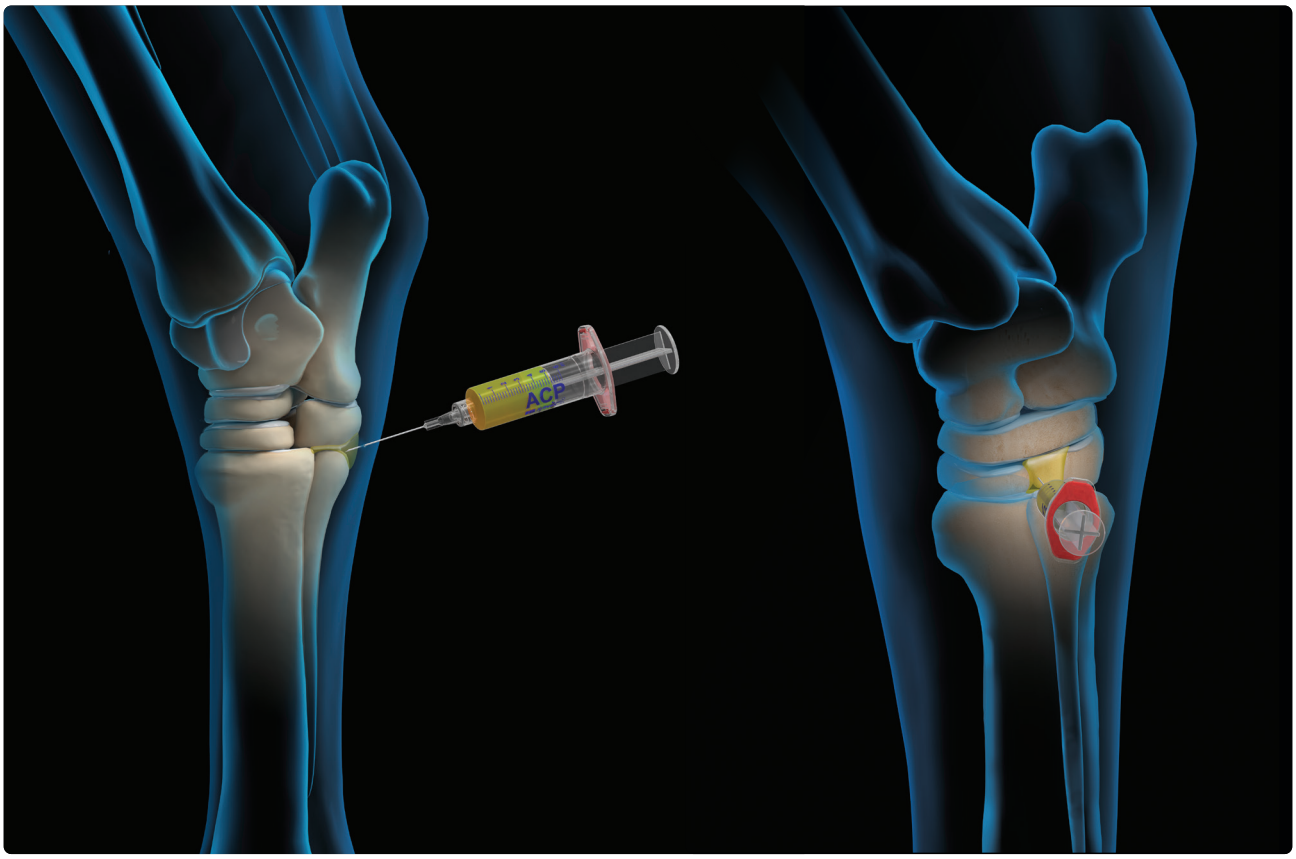
Tibiotarsal

- This joint is easily injected without ultrasound guidance.
- Position the horse weight-bearing.
- Insert a 20-guage, 1.5-inch needle at the distal end of the medial malleolus, medial or lateral to the saphenous vein. The area medial to the saphenous vein provides a larger access site and is therefore preferred to the lateral area.



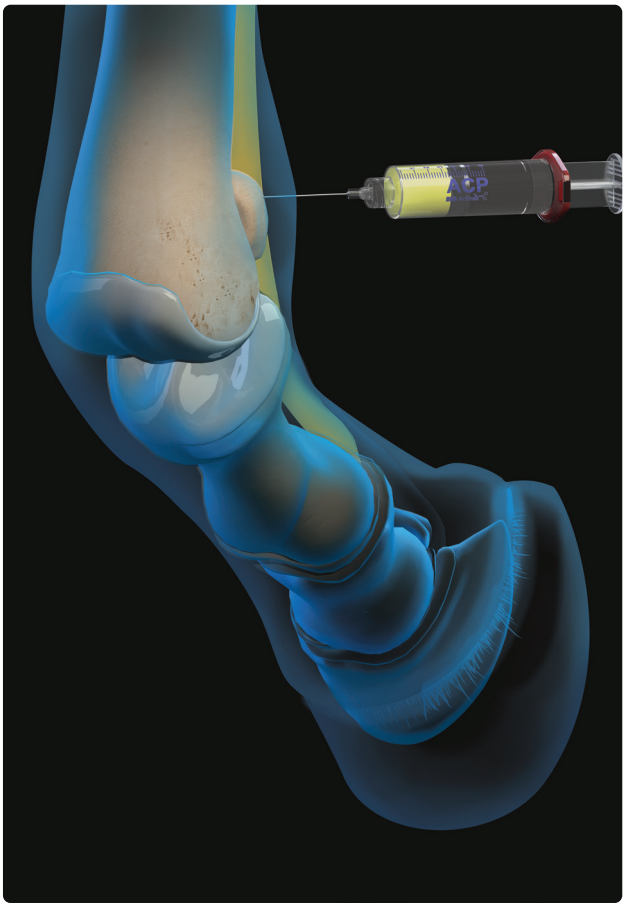
Lower Hock Joints

- Medicate both the distal intertarsal and tarsometatarsal joints individually, as these joints do not always communicate.
- Position the horse weight-bearing. Identify the distal intertarsal joint between the fused first and second tarsal bones, the third tarsal bone, and the central tarsal bone. Locate the injection site medial to the distal edge of the cunean tendon and approximately level with the distal aspect of the chestnut. Insert the needle parallel to the ground until bone is encountered.
- Position the horse weight-bearing, or hold the limb if restraint is needed. Enter the tarsometatarsal joint approximately 0.5 cm above the head of metatarsal IV and 1.5 cm lateral to the edge of the superficial digital flexor tendon. Insert the needle in a dorsomedial direction, parallel to the ground, until bone is encountered.



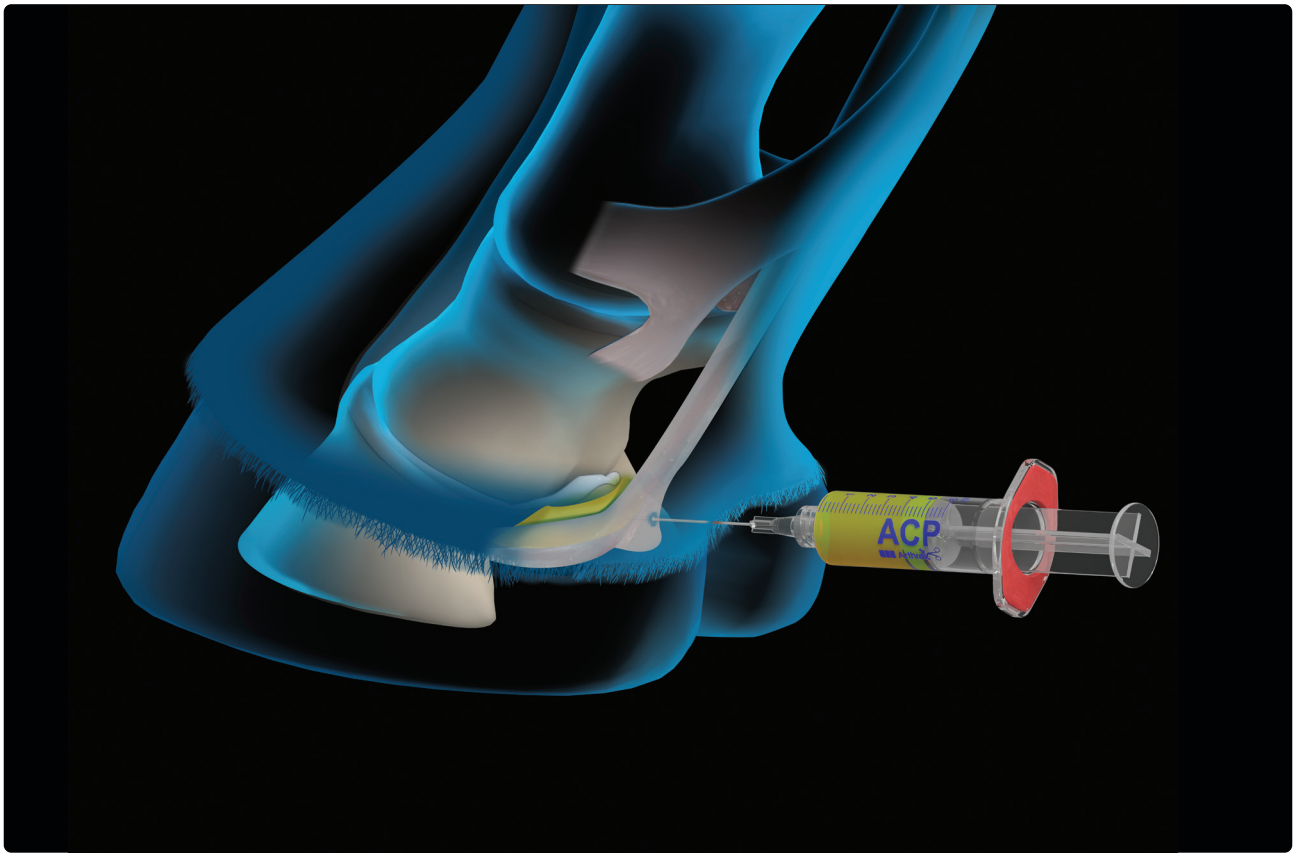
Proximal digital tendon sheath

- Maintain the limb in flexion for the axial sesamoidean approach. Insert the needle in a transverse plane at the level of the midbody of the palpable axial edge of the lateral or medial proximal sesamoid bone. Advance the needle through the palmar annular ligament, axial to the superficial flexor tendon, toward the intersesamoidean region.
- If the sheath is distended, the proximolateral approach is relatively simple. Position the horse weight-bearing or maintain the limb in flexion. Enter the sheath 1 cm proximal to the palmar annular ligament between the deep digital flexor tendon and the lateral branch of the suspensory ligament. Insert the needle parallel to the ground. Note that the proximolateral approach often results in blood contamination and is therefore not recommended for synoviocentesis.



Navicular Bursa

- The radiographically guided, tendon-sparing lateral approach is described.
- Desensitize the region by administering local anesthetic around the lateral and medial palmar digital nerves at the level of the proximal sesamoid bones. Elevate the horse on blocks to facilitate radiographic positioning.
- Insert the needle at the proximal extent of the collateral cartilage, dorsal to the neurovascular bundle, and dorsal to the dorsoabaxial margin of the deep digital flexor tendon.
- Direct the needle 30° to 40° relative to the horizontal plane for 1 to 2 cm, then obtain a lateromedial radiograph. Adjust and advance the needle as needed until it is positioned within the presumed proximal recess of the navicular bursa, as confirmed by sequential radiographs. Confirm entry into the navicular bursa by injecting 0.25 mL of contrast material diluted 1:[2] with saline and obtaining a radiograph. Once proper needle placement is confirmed, administer the biologic treatment.



Injection guide for average 450 kg horse. If the veterinarian chooses to include antimicrobials, local anesthetic, or other substances, the volume of PRP should be decreased respectively.

Structure	Needle size	PRP volume to inject (mL)
Carpal joint	20-22 gauge, 1.0-1.5 in	3-4 per joint
Fetlock joint	20-22 gauge, 1.0-1.5 in	4-5
Pastern joint	20-22 gauge, 1.0-1.5 in	1-3
Coffin joint	20-22 gauge, 1.5 in	4
Stifle joint	20-21 gauge, 1.5 in	5-8 per compartment
Distal intertarsal and tarsometatarsal joints	20-22 gauge, 1.0 in	1-3 per joint
Proximal digital tendon sheath	20-22 gauge, 1.0 in	6-8
Navicular bursa	18-20 gauge 3.0 in spinal needle	1-3

Ultrasound-Guided Injections

Cervical Facet
The craniodorsal technique is described as it allows joint margins to be clearly identified and facilitates visualization of the needle as it enters the joint. The neck is retained in neutral position after sedation. The affected joint space(s) are identified using radiographic markings such as a metallic bead, or with the transducer-oriented perpendicular to the long axis of the cervical spine localizing the C2-C3 joint space and moving caudally along the neck until the affected joint space is identified. Local anesthetic is injected to desensitize the skin at the injection site. The skin is routinely prepared for joint injections and to optimize ultrasonographic imaging. A range of 18 to 21 gauge, 3.5-inch spinal needles can be used. Smaller-gauge needles are less traumatic to soft tissue, but large-gauge are easier to visualize on ultrasound, and stiffer allowing advancement through deeper tissue planes easier. The ultrasonographic image is manipulated until the joint space is at its widest. The spinal needle is introduced approximately 2 cm craniodorsal to the transducer and directed approximately 20 degrees ventrally using ultrasound guidance to enter the joint space. The stylet is removed, and the joint is injected with no resistance. Synovial fluid is not always obtained, but the injectate can be visualized entering the joint space.

Shoulder
The landmarks for needle placement are easily palpable; however, the joint is quite deep relative to the skin surface so sedation and local anesthetic at the site of needle insertion are recommended. 18- to 21-gauge, 3.5-inch spinal needle is inserted in the notch between the cranial and caudal parts of the greater tubercle of the humerus. The needle is directed parallel to the ground and toward the opposite elbow. Aspiration of synovial fluid confirms accurate needle placement.

Biceps Bursa
The bicipital bursa lies in the bicipital groove, which is located medial to the cranial edge of the greater tubercle of the humerus and under the origin of the biceps brachii. It is injected with the horse sedated and in standing position. At the cranial edge of the greater tubercle of the humerus, a 3.5-inch, 20-gauge needle is inserted parallel to the ground, 45° caudally, and slightly dorsally until the needle tip encounters bone. Synovial fluid can usually be aspirated.

Elbow
The large caudal pouch of the elbow joint is located in the olecranon fossa and is the target site for injection with the horse in standing position. Because the musculature of the triceps is highly variable between horses, ultrasonographic identification of the caudal pouch can facilitate injection. The needle is inserted 0.5 inches ventral and 3 cm cranial to the most proximal palpable point of the ulna. The needle is directed at 45° distomedial for approximately 2 inches. Synovial fluid confirms accurate needle placement.

Hip
The coxofemoral joint can be difficult to inject due to the overlying musculature and the distance from the skin surface to the joint capsule, making identification of landmarks difficult. Ultrasound guidance is strongly recommended. The cranioventral approach is described. The injection is performed on the standing, sedated horse with the skin prepped to optimize ultrasound imaging. The cranial joint margin is identified, and the transducer is moved ventrally until the cranioventral aspect of the joint is identified. A skin bleb of local anesthetic is placed at the site of needle insertion. A 16-gauge, 6-inch needle is introduced cranial to the transducer and advanced under ultrasound guidance until the cranioventral recess of the joint is entered. Synovial fluid is reliably obtained from this location to confirm accurate needle placement.

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Notes

Notes

This is not veterinary advice and Arthrex recommends that veterinarians be trained in the use of any particular product before using it in surgery. A veterinarian must always rely on his or her own professional clinical judgment when deciding whether to use a particular product. A veterinarian must always refer to the package insert, product label and/or instructions for use before using any Arthrex product. Products may not be available in all markets because product availability is subject to the regulatory and/or veterinary practices in individual markets. Please contact your Arthrex representative if you have questions about availability of products in your area.

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