Complications of intravenous catheterization in horses

A. Schoster
Equine Department, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland

Abstract

Intravenous catheterization is a necessity for continuous administration of intravenous fluids and for intermittent intravenous access to avoid discomfort and potential complications of repeated needle insertions into the vein. Intravenous catheterization is commonly performed and well tolerated in horses, but catheter associated complications have been reported. The most commonly reported complication is thrombophlebitis, but others such as venous air embolism, exsanguination and catheter fragmentation may also occur. This article aims to review clinical signs, pathogenesis, diagnosis, therapy, risk factors and prevention of common catheter associated complications.

Keywords: thrombophlebitis, catheter fragmentation, exsanguination, venous air embolism

Introduction

Intravenous access for administration of medication, blood collection and intravenous fluid therapy is an important aspect of therapy in sick horses. Intravenous catheterization is commonly performed and well tolerated in horses. However, it may also lead to complications, which will be reviewed in this article. Information on clinical signs, pathogenesis, diagnosis, therapy, risk factors and prevention will be discussed.

Thrombophlebitis

Several clinical pictures are commonly grouped under thrombophlebitis. Peri- or paraphlebitis is defined as inflammation of the vein and surrounding tissue and can be septic or aseptic. Thrombophlebitis is defined as an inflammation of the vessel wall followed by formation of a thrombus in the lumen of the vein, which can be septic or aseptic. Vein thrombosis refers to the presence of a thrombus without concurrent mural inflammation; it often represents the long-term outcome of thrombophlebitis (Spurlock et al., 1990; Divers, 2003). Occurrence rate of thrombophlebitis has been reported as 1–29%. Horses undergoing elective surgical procedures have the lowest rate of occurrence of thrombophlebitis, while horses with systemic inflammatory disease have the highest rates (Gulick and Meagher, 1981; Spurlock et al., 1990; Traub-Dargatz and Dargatz, 1994; Lankveld et al., 2001; Mair and Smith, 2005; Geraghty et al., 2009).

Pathogenesis

Horses have a coagulation system, which tends towards hypercoagulability in disease states, leading to excessive activation of coagulation, impairment of fibrinolysis and anticoagulant pathways, resulting in thrombosis.
Complications of intravenous catheterization in horses

A. Schoster

(Dunkel et al., 2010; Mendez-Angulo et al., 2011). Virchow described the three factors leading to thrombus formation (Virchow’s triad). These include local trauma, reduced blood flow and altered coagulation status. Catheter insertion leads to minor trauma and inflammation of the vein, followed by activation of the coagulation cascade. Sick horses often spend a lot of time with their head in a lower position than normal contributing to abnormal blood flow in the vein. This increases the risk of coagulopathies (Divers, 2003). Most cases of thrombophlebitis in a hospital setting are non-septic. If bacterial infection of the thrombus occurs, environmental or skin bacteria will colonize the thrombus, rather than hematogenous infection of the thrombus. The catheter insertion site serves as a portal for bacteria and rarely fungi to gain access to the vein (Geraghty et al., 2009). The most common bacteria isolated are \textit{Staphylococcus} spp., \textit{Enterobacter} spp. and \textit{Streptococcus} spp. (Ettinger et al., 1992; Lankveld et al., 2001; Geraghty et al., 2009).

Drug resistant bacteria have also been cultured from septic thrombi of horses, highlighting the need for culture and sensitivity profiles (Gardner et al., 1991). Bacteria can be cultured from 50–60% of removed catheters tips from horses with signs of thrombophlebitis, however bacteremia and sepsis are rare (Gardner et al., 1991; Ettinger et al., 1992; Tan et al., 2003). Bacterial colonization of the catheter tip does not always correlate with clinical signs of thrombophlebitis. Particularly \textit{Acinetobacter baumanii} and other Gram-negatives can frequently be cultured from catheter tips without associated signs of venous disease, highlighting the need of a clinical diagnosis (Ettinger et al., 1992; Vaneechoutte et al., 2000; Geraghty et al., 2009; Milne and Bradbury, 2009).

Secondary complications of thrombophlebitis include bacteremia, sepsis and dislodging of thrombi leading to pulmonary thromboembolism, endocarditis and infective pleuritis (Tan et al., 2003; Ryu et al., 2004; Aksoy et al., 2008).

### Risk factors

Risk factors include catheter-associated factors, patient factors and medication administered (Tab. 1) (Dickson et al., 1990; Spurlock et al., 1990; Meister et al., 1993; Traub-Dargatz and Dargatz, 1994; Lankveld et al., 2001; Dolente et al., 2005; Geraghty et al., 2009). Both catheter dwell time and catheter material have an influence on local trauma to the vein. Rigid materials have a greater potential for causing endothelial damage (Spurlock et al., 1990). Horses with systemic disease, particularly gastrointestinal disease, have an increased risk of developing thrombophlebitis. Horses that had antimicrobials or non-steroidal anti-inflammatory medication administered through the catheter had a lower risk of thrombophlebitis compared to horses that did not have these medications administered (Traub-Dargatz and Dargatz, 1994; Geraghty et al., 2009). Administration of irritating medication and total parenteral nutrition are also common risk factors for thrombophlebitis. Foals have a higher risk of catheter associated problems compared to adults (Ettinger et al., 1992). As common sources of bacterial contamination are the skin of horses or nosocomial pathogens from the hospital environment. Animals which are recumbent or spend a larger than normal time in recumbency are at higher risk of septic thrombophlebitis (foals, sick animals, colics) (Gardner et al., 1991; Ettinger et al., 1992).

### Clinical signs and diagnosis of thrombophlebitis

Diagnosis is made based on clinical signs, ultrasonographic examination and blood work. Clinical signs often appear 24–48h after removal of a catheter, or while the catheter is still in place (Lankveld et al., 2001). Thrombosis usually starts at the insertion site or the tip of the catheter. Clinical signs include pain, heat or swelling of the vein, firmness ‘rope like appearance and feel of the vein’ and lack of fill in the vein distal to the catheter (Fig. 1 A and B). Swelling of the surrounding tissue and head may also occur. Often fever appears as a first clinical sign but can also occur later in the course of the disease. Limb edema and cellulitis are seen in association with cephalic vein thrombosis.

Acute phase proteins such as fibrinogen and serum amyloid A as well as leukocytes and neutrophils are often elevated (Westerman et al., 2016). Patients with any of the clinical signs, fever or unexplained leukocytosis (neutrophilia) or elevated acute phase proteins should have their catheterized vein evaluated immediately by clinical examination and ultrasound.

### Table 1: Risk factors for development of thrombophlebitis after intravenous catheterization in horses.

<table>
<thead>
<tr>
<th>Catheter factors</th>
<th>Improper placement</th>
<th>Diameter</th>
<th>Flexibility</th>
<th>Composition</th>
<th>Dwell time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient Factors</td>
<td>Endotoxemia</td>
<td>Systemic inflammatory response syndrome</td>
<td>Gastrointestinal disease</td>
<td>Foals</td>
<td></td>
</tr>
<tr>
<td>Medication administered</td>
<td>Injection of irritating medication</td>
<td>Anesthetic induction technique</td>
<td>Parenteral nutrition</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Ultrasonography examination is useful to characterize the extent and monitor the progression of the disease. Ultrasound can be used to detect catheter related abnormalities that are not yet clinically evident. Serial examinations are often useful as changes might not be obvious on the first examination but become more pronounced 12–24 hours later (Geraghty et al., 2009). A linear transducer with 7.5 or 10-11 MHz, shallow depth and focal point should be used. For better visualization the horse should be clipped and alcohol or ultrasound gel applied to the area. The vein should be evaluated in transverse and longitudinal planes along its entire length while it is filled and under physiologic conditions. Special attention should be paid to the catheter entrance site as well as tip of the catheter. The normal jugular vein is a circular hypoechoic structure on transverse section, and tubular structure in a longitudinal view, surrounded by a slightly more hyperechoic wall. Thickening of the wall, surrounding tissue edema, fluid accumulation, gas as well as thrombi can be identified in case of thrombophlebitis (Fig. 2 A and B). A thrombus is a homogenous hyperechoic mass attached to the vessel wall on at least one side and protruding into the lumen (Fig. 3 A). Blood flow can be imaged past the thrombus if it is only partially occlusive using pulsed wave or color-coded Doppler (Fig. 3 B). Thrombosis can extend to involve the external maxillary vein and the linguofacial vein cranially or extend down the thoracic inlet caudally (Gardner et al., 1991; Geraghty et al., 2009). A septic thrombus is a cavitated heterogeneous mass, fully or partially obstructing the vein. The anechoic areas represent fluid accumulation secondary to infection. A needle aspirate of the fluid cavity for bacterial culture and gram stain can be performed after aseptically preparing the site.

**Treatment**

If the catheter is still in place when the diagnosis is made, it should be removed. The catheter site should be aseptically prepared and the catheter removed in a sterile procedure. The tip of the catheter should be cut off with sterile scissors and a culture (aerobic and anaerobic) and sensitivity profile performed.

Thrombus development occurs mostly in the first 24h of injury, therefore treatment should be instituted as early as possible (Dias and Neto, 2013). The choice of treatment depends on the severity of the disease and centers on decreasing the present inflammation, preventing formation of or dissolving an existing thrombus and preventing or treating secondary bacterial infection. Independent of the severity of the disease the affected vein should no longer be used for venipuncture.

Local therapy includes hydrotherapy and hot packs, application of anti-inflammatory ointments and DMSO.

If the thrombophlebitis is extensive and clinical or ultrasonographic signs of a septic thrombus are present, systemic treatment should be instituted. Anti-inflammatory medication should be used to reduce signs of inflammation (flunixin meglumin 1.1mg/kg IV q12h, meloxicam 0.6mg/kg PO or IV q24h). Broad-spectrum antimicrobials such as penicillin (Na-Penicillin 30.000IU/kg IV q6h) combined with aminoglycoside (gentamicin 6.6mg/kg IV q24h), or trimethoprim sulfamethoxazole (24mg/kg PO q12h) are indicated to prevent or treat bacterial infection. If a culture and sensitivity profile were performed, antimicrobial therapy should be adjusted accordingly. Anaerobic bacteria were
Complications of intravenous catheterization in horses

A. Schoster

Cultured from 8% of septic thrombi in one study (Gardner et al., 1991). If anaerobic infection is suspected based on the presence of gas on ultrasound, antimicrobials with anaerobic action need to be chosen. Metronidazol (25mg/kg PO, q12h) is effective, but its use is only permitted in non-food producing animals in Switzerland. Penicillin (Na-Penicillin 30.000IU/kg IV q6h) or oxytetracycline (7mg/kg IV q 12h) can be used alternatively. Systemic treatment should be continued for several days until the clinical signs of inflammation have subsided and inflammatory changes on blood work have normalized. On ultrasound the thrombus should appear unchanged over several days and gas should be absent before treatment is discontinued.

To prevent formation of a thrombus or attempt to resolve an existing thrombus local and systemic therapy can be attempted. Local treatment with heparinized creams can be attempted, however high concentrations of heparin are needed to achieve similar levels in the corium compared to intravenous application of heparin. Low molecular weight heparin (50-100IE/kg, SQ, q24h) had a better clinical effect than heparin (40-100IE/Kg, IV or SQ q6h) for prevention of thrombophlebitis and was associated with fewer adverse effects (Schwarzwald et al., 2002; Feige et al., 2003). Acetylsalicylic acid, a prostaglandin inhibitor, reduces thrombocyte aggregation and can be used at a dose of 20mg/kg PO q48h. Treatment duration depends on the severity of the thrombus and financial means of the owner. Treatment should be discontinued once the thrombus does not appear to grow and clinical signs of inflammation have subsided.

Other anticoagulants (phenprocoumon, warfarin, clopidogrel) have been studied but are difficult to administer in a practice setting due to monitoring requirements or they have only been evaluated in a research setting (Scott et al., 1980; Bubeck et al., 2005; Brainard et al., 2011; Brainard et al., 2012). Recombinant tissue plasminogen activator has recently been evaluated in vivo and in vitro for its thrombolytic effects in healthy horses. While initial results appear promising more studies are needed to evaluate the safety and effectiveness (Baumer et al., 2013).

In case of fluid accumulation around the vein, drainage should be established. Often a small skin nick is sufficient (Fig. 4). In severe cases, surgical treatment is necessary to remove accumulation of necrotic tissue and infected material from the vein and surrounding area. Phlebotomy, thrombectomy or venous grafts can be performed to remove infected thrombi and restore blood flow in selected cases (Cannon, 1983; Rijkenhuizen and van Swieten, 1998; Wiemer et al., 2005; Russell et al., 2010).

**Prognosis**

The outcome of thrombophlebitis is variable. The thrombus may undergo recanalization and blood flow may be partially restored. If the vein becomes fully occluded, collateral vessels will form after some time. The affected vein should not be used in the future for injections or catheterizations. Thrombophlebitis did not affect the horses’ performance on a pleasure riding and show horse level, even when occlusion was bilateral. However, in racing Standardbreds thrombophlebitis was associated with a decreased chance of return to racing (Moreau and Lavoie, 2009).

**Prevention**

Careful selection of an appropriate vein and catheter type is imperative. In horses the jugular veins are most commonly used for peripheral venous access. Both jugular veins should be patent and without signs of inflammation. If one of the jugular veins shows signs of pre-existing disease, an alternative to the jugular veins should be considered for catheterization to avoid the potential life threatening complication of bilateral thrombophlebitis. The lateral thoracic vein, cephalic vein or saphenous vein are alternatives for placement of an indwelling intravenous catheter. If parenteral nutrition is planned, a large vein (lateral thoracic or jugular) should be used to minimize endothelial irritation. Polyurethane catheters should be used if saphenous veins, cephalic veins or the lateral thoracic veins are used (Divers, 2003).

There are no guidelines regarding optimal site preparation for catheter placement. Independent of the product used, disinfection results in decreased bacterial contam-
Complications of intra-venous catheterization in horses
A. Schoster

The following ranking order of decreasing thrombogenicity of catheter material commonly used has been established: polyethylene (including teflon), polyurethane and silastic. (Spurlock et al., 1990; Aksoy et al., 2008). Catheter type has less influence on development of thrombophlebitis in horses compared to humans, and patient factors as well as dwell times play a bigger role (Dickson et al., 1990; Lankveld et al., 2001; Milne and Bradbury, 2009). The rigidity of Teflon (polytetrafluoroethylene) catheters causes more vessel wall irritation and increases the risk of thrombophlebitis. They can be introduced over a needle or stylet and usually lie against the vessel wall. Polyurethane or silicone catheters are less rigid, and cause fewer traumas to the vessel wall. Due to their flexibility they have to be introduced with an over the wire or peel away technique, which require fastidious aseptic technique and skill. They usually lie within the vessel lumen, further decreasing the risk of wall trauma (Spurlock et al., 1990). To further reduce thrombogenicity catheters should be of adequate length (20–30 cm). Shorter catheters are prone to more movement and inducing trauma to the vessel wall (Spurlock et al., 1990). While there is not enough evidence in the horse to prove that catheter diameter is associated with thrombophlebitis, this is a known risk factor in humans. The size of the lumen should be adequate for the desired fluid rate but not too large (Traub-Dargatz and Dargatz, 1994). For adult middle and large sized horses 14G catheters are recommended, if large volumes of fluid have to be administered 8G–12G can be used. For foals and smaller horses 14G and 16G can be used. In foals, small horses and systemically compromised patients long-term over the wire polyurethane catheters should be used to avoid dislodging, kinking and other complications. Double lumen catheters should be restricted to horses receiving parenteral nutrition. The correct procedure of catheter insertion and securing depends on the type of catheter used and is beyond the scope of this review. An excellent summary is given by Barakzai and Chandler (2003).

Dwell times of catheters are much longer in human medicine compared to horses. Short-term catheters made of teflon should remain in the vein for a maximum of 2–3 days (Lankveld et al., 2001). Long-term catheters made of polyurethane or silicon can remain in the vein for up to 1–2 months if no signs of inflammation are present. Indwelling catheters should be assessed twice daily for signs of developing inflammation (Divers, 2003). In addition to clinical examination, ultrasound should be used periodically to detect early signs of disease, particularly in high-risk patients (Geraghty et al., 2009). Catheters should be flushed every 4–6h with saline if not used. Whether or not the addition of heparin (1–10IE Heparin/mL) is needed is debated but it is commonly added. An extension should be connected to the catheter permanently to avoid constant manipulation of the catheter. A bandage should not be placed over the catheter to easily identify problems and prevent problems with blood circulation. Foals might need a bandage placed around the catheter to avoid them tempering with the catheter. When injecting medication through the medication cap, the catheter cap should be wiped with alcohol prior to insertion of the needle. The injection cap should be changed daily. The catheterized vein should not be used for venipuncture. Catheters placed in saphenous, cephalic or lateral thoracic veins should be flushed every 4 hours and protected by bandages to prevent trauma to the catheters (Barakzai and Chandler, 2003).

Venous air embolism

Iatrogenic air embolism following inadvertently left open jugular venous lines have been reported (Bradbury et al., 2005; Holbrook et al., 2007; Sams and Hofmeister, 2008). Air enters into the venous circulation due to the negative pressure compared to the atmospheric pressure. Small amounts of air likely do not result in clinical signs in horses. If large amounts of air enter the circulation, air emboli travel through the right heart into the lung circulation causing cardiac and pulmonary signs. Pulmonary edema develops due to hypoxemia, hypotension, increased vascular permeability and acute inflammation. Cardiac output can be reduced due to embolism. The only scientific report on the amount of intravenous air tolerated in horses is a volume of up to 0.25 mL/kg before clinical symptoms occur (Muir, 1991). Microbubbles diffuse through the alveolar membrane and are exhaled. If the amount of air exceeds the filter capacity of the lungs the air gains access to the systemic arterial circulation and can then be distributed to all organs. Air emboli in the coronary and cerebral circulation can be associated with severe clinical signs (Pellegrini-Masini et al., 2009).

Clinical signs

Clinical signs vary depending on the organ system affected by the air embolus. Horses show signs of cardiovascular collapse, cardiac dysrhythmias, cardiac mur-
Complications of intravenous catheterization in horses
A. Schoster

Diagnosis
Diagnosis can be made based on history (open venous line), and clinical signs. Air embolism can be confirmed on echocardiography or Doppler technologies. During anesthesia volumetric capnography can be used to diagnose venous air embolism (Pellegrini-Masini et al., 2009).

Treatment
Treatment is mostly symptomatic. Intranasal oxygen should be provided to counteract signs of organ hypoperfusion. Intranasal oxygen therapy (8L/min intranasal) also favors clearance of air bubbles. Intravenous fluid therapy should be considered to support cardiovascular function. The use of corticosteroids has been questioned since these drugs aggravate ischemic injury and are not effective to treat cerebral edema (Muth and Shank, 2000). If seizures occur they should be controlled using diazepam (0.05mg/kg IV). If a venous air embolus occurs during anesthesia, treatment should include instituting intermittent positive-pressure ventilation, administering intravenous fluids and increasing the inspired oxygen percentage (Sams and Hofmeister, 2008). In human medicine additional measures such as aspiration of air using a right atrium catheter, hemodynamic support with dobutamine or norepinephrine, mannitol and hyperbaric oxygen therapy have been reported to have favorable outcomes. There is increasing evidence for the use of lidocaine due to its neuroprotective effects (1.3ug/kg/min IV continuous rate infusion) (Muth and Shank, 2000).

Prognosis
In the available case reports, all horses recovered from venous air embolism and survived without long-term complications (Pellegrini-Masini et al., 2009). A one-way valve can be used to prevent air entering the catheter if the catheter extension or injection cap is lost.

Catheter fragmentation
Fragmentation of intravenous catheters made of teflon has been reported (Spurlock et al., 1990; Scarratt et al., 1997). When an over the wire technique is used for catheter placement, the wire can be lost if the technique is faulty. While there is only one case report (Nannarone et al., 2013) describing this complication in horses, in the authors’ opinion this complication occurs more often than reported. Catheter fragments or the guidewire can travel through the heart and become lodged in the pulmonary vasculature. In smaller animals such as ponies and foals the fragments can become lodged in the right heart.

Clinical signs
Catheter fragments or wires often do not cause signs of clinical disease. Potential complications include pulmonary embolism, cardiac arrhythmias, endocarditis, septic thrombosis of the wire, and cardiac tamponade if it penetrated the heart.

Treatment
If the fragment or wire is not associated with signs of disease it can be left in the horse. The location can be assessed using ultrasonography and radiography (Lees et al., 1989; Ames et al., 1991; Scarratt et al., 1997; Little et al., 2002; Culp et al., 2008; Nannarone et al., 2013). Fragments and wires have been retrieved successfully in some instances using surgical and non-surgical approaches in adults and foals (Ames et al., 1991; Hoskinson et al., 1991; Scarratt et al., 1997; Culp et al., 2008; Nannarone et al., 2013). For intravascular retrieval techniques, fluoroscopic guidance is advised, but ultrasound guidance can be attempted if the fragment or wire is located in the heart. In the described non-surgical approaches the fragments or wire were retrieved using a basket or four pronged retrieval catheter or retrieval snare inserted through the jugular vein and a small phlebotomy to remove the fragments or wire (Ames et al., 1991; Hoskinson et al., 1991; Culp et al., 2008; Nannarone et al., 2013). Surgical techniques describing the retrieval of catheter fragments have also been reported (Lees et al., 1989). Complications include further fragmentation of the catheter, embolization and cardiac arrhythmias (Hoskinson et al., 1991).

Prognosis
In humans there is a fatal outcome in 20% of cases (Heberer et al., 1984). It is likely that the situation in horses is different, as no deaths due to loss of guidewires have been reported.

Prevention
To minimize the risk of catheter breakage the catheter insertion site should be monitored daily for signs of catheter kinking or patient induced trauma to the catheter. The stylet or needle should never be reintroduced into the catheter to avoid cutting of the catheter with the sharp end of the stylet. If a catheter does break inadvertently during removal or introduction the vein should immediately be held off to prevent fragments from travelling caudally.

Perivascular leakage
If the catheter is not introduced correctly, dislodges or cracks at the insertion site administered medication can leak into the surrounding tissue. Shorter catheters are more likely to become dislodged (Scarratt et al., 1997).
This is not a significant problem when isotonic fluids are used. Mild to severe cellulitis can occur when irritating medication is used. Clinical signs consistent with cellulitis, such as pain, swelling and heat develop. Horner syndrome can occur if the inflammation affects the sympathetic branches and ganglia. Some horses become unable to swallow due to inflammation or develop laryngeal hemiplegia due to damage to the vagus nerve. Infusions should be stopped and the catheter removed. Isotonic fluids will be absorbed from the subcutaneous area. Subcutaneous infiltration with isotonic fluids such as saline can be performed to dilute irritating medications. Warm compresses and local anti-inflammatory treatment should be instituted. Systemic antimicrobial and anti-inflammatory therapy and ventral drainage and flushing may become necessary in severe cases (see treatment of thrombophlebitis). Prognosis depends on the severity of the disease.

Exsanguination

Exsanguination from a disconnected infusion port or catheter cap is a rare occurrence and more likely if there is an underlying coagulopathy (Tan et al., 2003). Smaller patients with less total amount of circulating blood volume are at higher risk of exsanguination, e.g. ponies or foals. Horses can generally lose 20–30% of their blood volume (2 ml/kg, 6L in a 500kg horse) without hypotension and corresponding clinical signs (Subcommittee et al., 2013). Clinical signs of hypotension include pale mucous membranes, tachycardia, altered mental state and if severe collapse. If a catheter is dislodged and the horse is bleeding from the insertion site, the area should be held off until blood clotting occurs. A lost catheter cap or infusion port should be replaced and adequate amounts of fluid or whole blood transfusions administered as necessary. One-way valves do not allow blood to exit the catheter without a syringe or infusion port attached and can be attached to catheters to prevent this risk in high-risk animals.

Inadvertent intra-arterial catheterization

If a proper technique is not followed or the horse moves during catheter placement inadvertent intra-arterial catheterization can occur. In this case, arterial blood is seen spurting from the catheter in a pulsatile fashion. Arterial blood is lighter in color than venous blood. If it is unclear whether the vein or the artery has been catheterized a blood gas can be performed to ascertain placement of the catheter in the vein. Arterial catheterization is not associated with any problems as long as no medication is administered. The catheter should be removed, the artery held off for at least 5 minutes to prevent formation of hematoma and another catheter placed intravenously. If medication is administered through an intra-arterial catheter the horse will react with violent behavior, collapse and seizures. The catheter should be removed immediately and venous access established for treatment. To prevent inadvertent arterial catheterization the horse should be restrained adequately and prevented from moving its head and neck during catheter placement, inexperienced people need to be guided by experienced people and the correct location for catheter placed should be chosen. The vein and artery are further apart in the junction of the upper and middle third of the neck, which is the correct site for catheter placement.

Conflict of interest

The authors have no conflict of interest to declare.
Complications lors de cathétérisme veineux chez le cheval

A. Schoster

Le cathétérisme veineux est une nécessité pour l’administration continue de fluides par voie intraveineuse et pour garantir un accès veineux intermittent afin d’éviter l’inconfort et les complications potentielles liées à la pénétration répétée d’une aiguille dans la veine. Le cathétérisme veineux est usuellement pratiqué chez le cheval et il est bien toléré mais des complications associées sont rapportées. La plus commune d’entre elle est la thrombophlébite mais d’autre, telles l’embolie gazeuse, l’exsanguination ou la fragmentation du cathéter peuvent aussi survenir. Cet article vise à résumer les signes cliniques, la pathogènese, le diagnostic, le traitement des facteurs de risque et la prévention des complications communément associées avec le cathétérisme.

References


Complications of intra-venous catheterization in horses

A. Schoster