

Artículo original:

EQUINE REPRODUCTIVE EMERGENCIES

Emergencias reproductivas en equinos

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Medical and surgical emergencies involving the reproductive system are a major part of studfarm medicine. Delayed intervention can result in devastating economic loss on the breeding operation. This paper presents a detailed discussion on the major emergencies in stallions and mares.

REPRODUCTIVE EMERGENCIES IN THE STALLION

Stallion reproductive emergencies present usually as an evident traumatic injury to the genital organ or as a sudden onset of a clinical syndrome (i.e. colic, unexplained lameness, fever or depression). Scrotal and testicular involvement is often signaled by a sudden increase in testicular size. Rarely, stallions may present as an emergency situation with seminal vesiculitis. One could also argue that emergency in stallion should include any sudden decrease in semen quality, libido or ejaculatory disorders. In the present review we will discuss primarily those emergencies that can jeopardize the life and fertility the stallion and are accompanied by severe clinical symptoms. Prognosis in these situations depend on prompt identification of the problem and adequate medical and/or surgical management of the case.

Emergencies associated with the penis

Most penile emergencies present as a paraphimosis. This is often the consequence of a traumatic injury, penile hematoma, penile paralysis or priapism.

Priapism

This condition is defined as a persistent erection and often leads to impotence. This is relatively uncommon in stallions but has been described following administration of phenothiazine derivatives and reserpine. However, recent studies (primarily surveys) have shown that the risk of permanent penile dysfunction following sedation with acepromazine is extremely low (1 in 10000 cases) [18,48]. Priapism has been reported following general anesthesia, inflammatory spinal cord lesions, traumatic injuries, castration, purpura hemorrhagica and generalized malignant melanoma [6,18,19,21,37]. Spontaneous erection and priapism.

was reported in a stallion during intensive medical management for hemorrhagic colitis [19]. A case associated with generalized squamous cell carcinoma was seen by one the authors (AT, clinical observation). Persistent erection is due to a failure of sympathetic stimulation necessary for detumescence. Long standing erection is complicated by increased carbon dioxide tension and increased viscosity of the stagnant blood leading to venous occlusion [4,39]. Urinary difficulty or blockage is a major complication which often leads to severe metabolic compromise. Rapid deterioration of the stallion's health ensues due to intermittent colic, depression and uremia. Reduction of penile tumescence is the first objective of medical management during emergencies. This may be attempted by a combination of manual massage and lubrication in conjunction with cold hydrotherapy or ice water baths. Suspension of the penis with a bandage or a sling allows prevention of dependant edema (Figure 1).



Figure 1: Suspension bandage for management of priapism

Slow intravenous administration of the anticholinergic agent benztropine mesylate (8 mg in 1 liter of 0.9% saline) has some efficacy when given early.⁶⁰ Other pharmacological treatments used include systemic diuretics, corticosteroids. Diphenhydramine (a first generation antihistamine with anticholinergic activity) and terbutaline (a β_2 -adrenergic receptor agonist and tocolytic) have been used for emergency treatment of priapism in other species but their use in stallions is not well documented [37].

Surgical treatment is often required and consists of flushing the sluggish blood from the corpus carvenosus penis (CCP) under general anesthesia. This can be accomplished by placing a 14 gauge needle 4 to 6 cm behind the collum glandis and another needle is placed 10 to 15 cm behind the scrotum. The coagulated blood is flushed from the CCP with heparinized saline solution (10,000 IU heparin in 1 L of 0.9% saline). Failure of detumescence after 3 CCP lavages warrants surgical approach to create a shunt between the CCP and the corpus spongiosum penis [39,40,54]

Postoperative care includes local (DMSO) and systemic anti-inflammatory therapy and antimicrobials. Bladder lavage and placement of an indwelling catheter may be necessary for 2 to 3 days. The use of bethanechol (0.05 $\mu\text{g}/\text{kg}$ PO q 6 hours for 3 days) may enhance bladder function. Complications of priapism include erection and ejaculation failure and permanent penile paralysis [54]. Stallions with such a complication may continue to be used for artificial insemination after ex-copula collection of semen with imipramine and xylazine [28]. Partial phallectomy may be required in some cases [34,56]

Traumatic paraphimosis and phimosis

Paraphimosis and less frequently phimosis are often complications of traumatic injury to the external genitalia [14]. This is a relatively common emergency in studfarm medicine. The stallion should be thoroughly evaluated and proper medical management initiated immediately in order to avoid further complications due to tissue damage and secondary infections [48]. The trauma may be directly to the penis or the adjacent tissue. Local swelling and edema develop very rapidly (within hours), often masking the extent of lesions. Penile hematoma due to ruptured venous plexus is the worst case scenario particularly if the tunica albuginea is also involved. These lesions may be difficult to manage and often require surgical intervention after initial medical stabilization.

Examination should be conducted under sedation as recent trauma and severe edema is extremely painful. Stallions are agitated and often display high respiratory and heart rates. It is important to determine if the stallion is able to urinate (Figure 2). Urinary bladder catheterization may be indicated in severe cases. Administration of diuretics such as furosemide should be considered carefully. In cases where the trauma is so severe that urinary bladder catheterization is not possible an emergency subschial urethrostomy may be considered. The prolapsed tissue should be supported by a harness as close to the ventral body wall as possible for transport [48].

The initial step in the management of penile and preputial trauma is to attempt to reduce the swelling and replace the prolapsed penis. In many cases the swelling is so severe at the time of presentation that replacement is not possible. In these cases,

the penis should be bandaged with elastic compressive bandage and held close to the body wall (Figure 3). During this initial management, the stallion should be kept in a quiet area. Stallions should be given a non-steroidal anti-inflammatories and preventive anti-tetanus treatment. The prolapsed tissue should be evaluated for presence of abrasions, hemorrhage or necrosis [48]. Local or systemic antimicrobial therapy may be considered depending on the extent of the lesions and risk of bacterial contamination. Hydrotherapy with cold water is helpful. Once the edema is reduced, the second step is to try to replace the penis within the prepuce and retain it in position until the retractor muscle function is regained. One technique used by the authors is to place the penis in a 500 ml bottomless plastic bottle and retain it with harnesses (Figure 4). The penis may also be retained by placing purse string sutures in the preputial orifice using an umbilical tape. However this technique is only helpful if the preputial tissue is healthy. The last step in the management of the prolapse is provide support of the penis and prepuce for a few days using a harness This treatment is applied and the stallion is able to retract and maintain the penis within the preputial cavity without help. During this treatment, the authors suggest providing protection to the preputial and penile tissues by using copious amounts of protective a cream containing lanoline (500 g), dexamethasone (80 mg) and oxytetracycline (3.88 g) [7,43].



Figure 2: Paraphimosis in stallion following penile trauma



Figure 3: Bandaging of the penis to reduce swelling in a case of paraphimosis

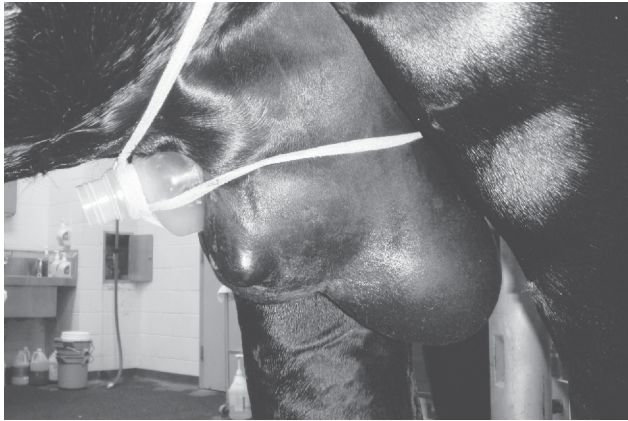


Figure 4: Technique for replacement of paraphimosis using a plastic bottle.

Severe cases with hemorrhage may require surgical drainage initially and removal of the blood clots [31]. Uncomplicated cases usually resolved within 7 to 10 days and the stallion may be returned to activity after a sexual rest of 2 to 3 weeks and re-evaluations. The most common complication of penile and preputial trauma is abnormal erection. In cases of severe loss of function of the penis, manual stimulation or ex-copula ejaculation may be the only alternative to keep the stallion in reproductive activity [19,26]. Severe traumatic phimosis may be managed surgically by reefing [14]. Complications of paraphimosis may require partial or total phallectomy [2].

Management of scrotal enlargement

Scrotal enlargement is a common complaint in breeding stallions and should be considered an emergency. Diagnosis is based on a detailed clinical evaluation and ultrasonographic imaging of the scrotum [42,45,53]. Clinical assessment in the field determines the level of emergency and need for critical care. Increased scrotal size may be sudden with or without acute signs of pain or colic. The increase in scrotal size may also be progressive with intermittent clinical signs of pain or observed during an examination for reduced fertility. Sudden increase of scrotal size is often associated with trauma (testicular hemorrhage, scrotal hernia, testicular torsion) or an acute infectious process (orchitis or epididymitis). Progressive increase in testicular size is often due to testicular neoplasia, hydrocele or local edema [45,53].

Complete blood count and blood biochemistry are indicated if the stallion is showing severe colic or a possible gastrointestinal compromise. Depending on the epidemiological conditions, the clinician may consider diagnostics for equine viral arteritis (EVA), infectious equine anemia (IEA) as well as dourine. Scrotal/inguinal hernias with intestinal incarceration, severe testicular cord torsion, testicular hemorrhage and orchitis are particularly painful. Stallion should be sedated before examination of the scrotum and its content by palpation and ultrasonography (xylazine 0.2 to 1.1 mg/kg or detomidine (10-40 µg/kg) or Butorphanol (0.05-0.075 mg/kg) [45,53].

Testicular trauma

Scrotal or testicular trauma is characterized rapid swelling of the area. The scrotal skin may be too tense preventing adequate palpation of scrotal sac content. Ultrasonography allows differentiation between lesions involving just the scrotum and those involving the testis [45]. Severe scrotal trauma may be complicated by penetrating wounds, testicular hemorrhage and albuginea rupture. Testicular hemorrhage is extremely painful. Initial treatment may include hydrotherapy, anti-inflammatory drugs and diuretics. Unilateral castration is recommended for severe testicular trauma and rupture of the albuginea (Figure 5). Antimicrobial therapy and tetanus preventive measures should be considered in cases of open wounds [53]. Complication of testicular trauma include subfertility or sterility due to severe testicular degeneration [33,35] or development of anti-sperm antibodies [62].

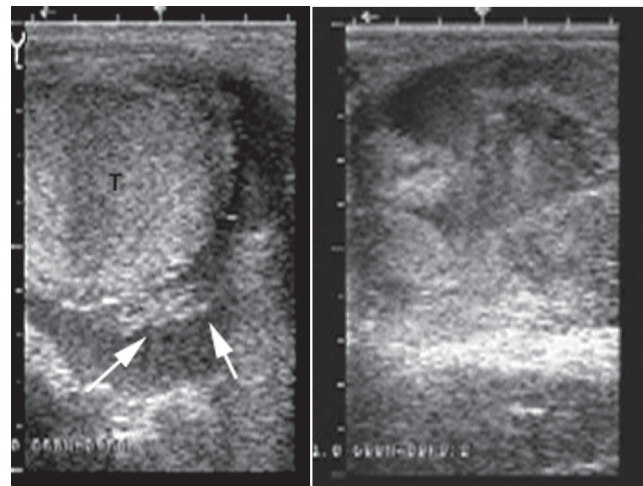


Figure 5: Ultrasonography of testicular hemorrhage with rupture of the albuginea

Scrotal and inguinal hernia

Inguinal hernia is defined as the passage of abdominal viscera (omentum, ileum or distal jejunum) through the vaginal ring into the inguinal canal (between the vaginal ring and the external inguinal ring). The hernia is called scrotal when the viscera are completely within the scrotal sac and lie adjacent to the testis. Although considered an acquired condition in the mature stallion, congenital large vaginal ring may be a predisposing factor. Scrotal or inguinal hernias usually occur after breeding, exercise or transport but they may also occur in resting stallions or after rolling. They are often unilateral, although bilateral herniation has been described in a show-jumping stallion [1]. Most hernias are indirect (passing directly into the vaginal cavity through the vaginal ring). However, direct scrotal/inguinal hernias (through a rent in the body wall) have been described [1,57]. Mortality rate varies between 26 and 62.5% if no prompt diagnosis and treatment [45].

Clinical signs are variable and depend primarily on the nature of herniated organs and the degree of their compromise. Stallions may initially show just an abnormal gait or some

depression slowly progressing to a colicky syndrome of variable intensity. Inguinal hernia should always be a differential in the work up of mild to severe colic in a stallion. Incarceration of intestinal loops causes a severe colicky syndrome and may require immediate surgery [30]. Intestinal motility and reduction or even absence of gut sounds may be noted. Gastric reflux may be present particularly in cases of strangulating hernias [45,48]. Heart (>60 bpm/min) and respiratory rates (>30/min) are often elevated. In severe cases, the stallion may show depression and dehydration. Rectal temperature may be increased in some cases ($\geq 39.4^{\circ}\text{C}$). Mucous membranes may be injected and the capillary refill time increased (> 3 sec). Absence of borborygmus is common in strangulated hernias. The hematocrit (PCV) ranges from 38 to 71% and total plasma protein may reach 8.4 g/dl. Blood pH may be normal to acidic (lactate >22 mg/dl). CBC may normal or show a stress leukogram (>14 000 BC/mm³) and neutrophilia. Abdominocentesis may reveal increased protein and leukocyte (modified transudate or peritonitis) [48].

Diagnosis of scrotal or inguinal hernia requires a thorough ultrasonography of the scrotum and its content (Figure 6). Transrectal palpation may reveal edematous intestinal loops within the vaginal ring or distended intestines in the caudal abdomen. Loss of intestinal motility along with severe colic signs usually suggest small intestine incarceration and vascular lesions that require immediate surgery [8,20,29,48,59]. Unilateral castration and laparoscopic repair remain the best course of action in uncomplicated cases [48,59]. Complicated cases may require castration as well as celiotomy to better evaluate the degree of intestinal compromise [53]. Laparoscopic closure of the vaginal ring prevents further complications [36].

Testicular cord torsion

Testicular cord torsion is a frequent cause of increased scrotal size. Torsions of 180° or less have usually very little clinical consequence on the stallion. Clinical symptoms are present when the torsion is greater than 270° . At first the stallion may show just a mild colic that is responsive to analgesia, a hindlimb lameness or stiff gait. Scrotal and preputial edema increases slowly and becomes severe with development of ischemic damage and gangrene. Severe torsion results in vascular compromise (spermatic artery thrombosis), edematous and tense scrotum and hemorrhagic necrosis.15 Diagnosis of torsion may be difficult because of the vascular complications.

The condition should be suspected in all stallions with sudden painful increase of scrotal size with absence of evidence of orchitis or hernia. Vascular compromise may be revealed upon evaluation with Doppler ultrasonography [45,48]. Although orchiopexy has been proposed in some cases, unilateral castration is usually the treatment of choice in breeding stallions with severe testicular cord torsion [41]. Urgent intervention and proper post-operative care reduces effects on the contralateral testicle and preserves fertility. Endocarditis has been reported as a potential complication [24].

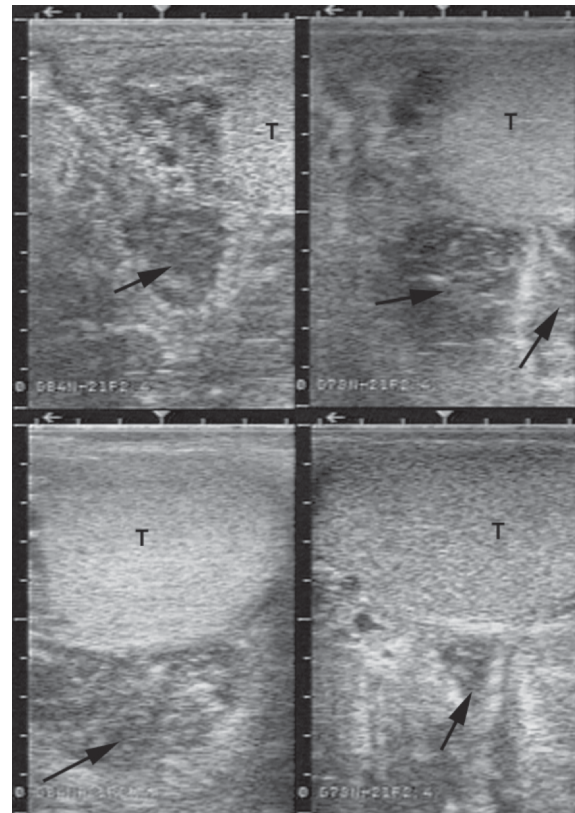


Figure 6: Ultrasonography of scrotal hernia (T= testicular parenchyma, arrows indicate intestinal loops).

Orchitis and epididymitis

Although uncommon, orchitis, periorchitis and epididymitis should be considered as differentials in the diagnosis of causes of scrotal enlargement. Orchitis may be due to viruses (EIA, EVA, Influenza), parasites (Larva migrans of *Strongylus edentatus*) and bacteria (*Strep. equi zooepidemicus*, *Pseudomonas spp.*, *Actinobacillus equuli*, *Corynebacterium pseudotuberculosis*) [22,27,48]. Ultrasonography may be helpful in identifying the lesions (Figure 7). Neutrophilia, fever and hyperfibrinogenemia without cardiovascular signs are often suggestive of orchitis [22,61]. Cytological evaluation of a fine needle aspirate may confirm the diagnosis. Although antimicrobial may help in some cases, unilateral castration is often the best approach as most affected testis will undergo severe atrophy [48,53].

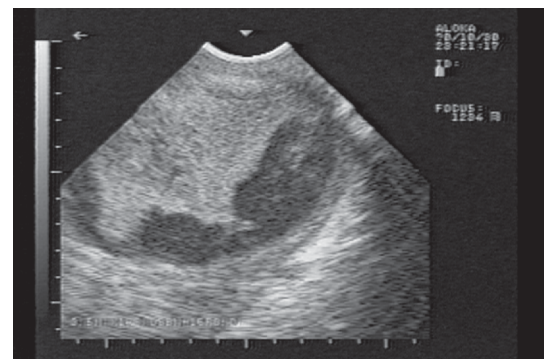


Figure 7: Ultrasonography of a testicular abscess due to *C. pseudotuberculosis*

Neoplasia

Scrotal enlargement may be observed in cases of testicular neoplasia. However, these are often insidious lesions and do not present as an emergency unless there are other complicating factors. Several types of testicular neoplasia have been reported and include non-germinal (interstitial cell, sertoli cell, leiomyoma, lipoma, mast cell) and germinal neoplasms (seminomas, undifferentiated germ cell tumors). Tumors of the tunica vaginalis (mesothelioma) have also been reported. Seminomas are the most commonly reported testicular tumors. They are usually benign but metastatic seminomas have been reported. Diagnosis may be confirmed by testicular biopsy. Unilateral castration and complete evaluation of the testicular cord and internal organs by ultrasonography is highly recommended [45,48,58].

Other testicular disorders

Scrotal enlargement may also be the result of abnormal blood and lymphatic flow due to anomalies of the vessels (varicocele), increased ambient temperature (hydrocele) or as a consequence of other diseases resulting in generalized edema (enteropathy, hypoproteinemia, liver or kidney disease) or infectious diseases (EVA, IEA, Dourine) [45,48]. Hydrocele, accumulation of various quantities of fluid within the vaginal cavity, is very common in hot and humid environment and may reach level that could result in reduced fertility. Diagnosis of hydrocele is confirmed by ultrasonography (Figure 8). The fluid may be anechoic or present some proteinaceous material (Figure 9) [45]. Hydrocele can be managed by changing the stallion's environment (air conditioning to reduce heat and humidity) and regular exercise. Accumulation of large quantities of fluid may require aseptic aspiration. In most instances, systemic anti-inflammatory drugs and cold water hydrotherapy of the scrotum are included as part of the therapeutic plan. Scrotal enlargement may be secondary to abscessation or scrotal skin diseases (cutaneous lymphosarcoma) (Figure 10) [45,53].



Figure 8: Severe hydrocele in a stallion

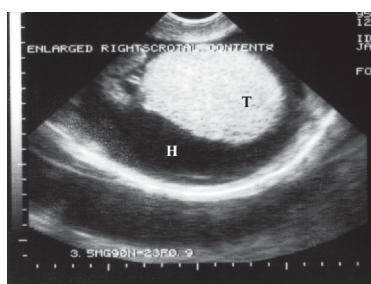


Figure 9: Ultrasonographic appearance of hydrocele



Figure 10: Scrotal abscess and cutaneous lymphosarcoma

REPRODUCTIVE EMERGENCIES IN THE MARE

Emergencies involving the reproductive system in the mare represent a large portion of equine emergencies in studfarm medicine practice. Obstetrical emergencies are without doubt the most common reproductive emergency and will not be discussed here. The reader is referred to a recent discussion of our approach to obstetrical intervention in the mares for review.⁴⁷ In the present review we focus primarily on reproductive emergencies in the open mare, during pregnancy and in the immediate postpartum period.

Reproductive emergencies in the open mare

Most emergencies involving the reproductive tract in open mares are due to injuries during breeding or pasture accidents (kicks from other mares). Breeding trauma occurs when breeding is unattended, with an overaggressive stallion, or when there is a large disproportion between the stallion penis size and the mare's caudal genitalia. In well managed breeding operation accidental injury to the vagina and cervix is usually prevented by the use of breeding rolls. Vaginal and rectal trauma has been observed by the authors in pasture bred mares. Clinical signs include straining, colic or post-mating hemorrhage, rectal prolapse, and anal or perineal masses. The extent of the lesions should be assessed by endoscopy after sedation. Genital trauma due to breeding is mostly located in the vagina. The cervix is rarely involved. Most injuries are superficial but full thickness tears may occur and can become rapidly complicated by peritonitis and severe colic. Vaginal or perivaginal adhesions may also be a consequence of genital trauma during mating [16].

Severe traumatic injuries to the vulva and clitoris can be sustained by mares kicking each other. Clitoral injuries are very painful and can bleed intensely (Figure 11). These injuries often require immediate clitoridectomy, pain management and preventive anti-infectious and anti-tetanus measures.



Figure 11: Severe hemorrhage following traumatic injury to the clitoris

Reproductive emergencies in the pregnant mare

Reproductive emergencies in the pregnant mare have been reviewed recently by the authors [51]. The emergency is not related only to the health and welfare of the mare but also to the risk of compromise of fetal viability and abortion. Clinical situations that need to be considered as emergencies in the pregnant mare include severe respiratory distress, colic, anorexia, unexplained fever, presence of abnormal vaginal discharge, premature mammary gland enlargement or lactation, abnormal abdominal girth or shape.

Evaluation and management of the colicky pregnant mare

Colic in the pregnant mare may be of gastrointestinal or genital origin. Colic causes of genital origin include first stage of labor or abortion, uterine torsion, uterine rupture, hemorrhage, and fetal membrane hydrops. Ventral wall defects and herniation through a ruptured prepubic tendon may also be considered genital because they are induced by the heavy gravid uterus. Approach to diagnosis is not different from other cases of colic except that the examination should also include the health and well-being of the fetus. Diagnosis of the cause of colic can be challenging particularly in late pregnancy because per rectum evaluation and abdominocentesis are severely restricted by the bulk of the gravid uterus.

Historical data that are important include the mare's age, parity, stage of pregnancy (breeding date), previous colic or surgery,

response to analgesia, and degree and duration of pain. Type of colic should be defined as to degree of severity; timing (before or after feeding) and frequency (persistent vs. intermittent). Mares in poor body condition or severely undernourished may develop postprandial mild colic because of gastric ulcers. Some mares may be just quiet, dull and reluctant to move others will go off feed intermittently. Mares with violent colic should be immediately referred to a surgical facility [49].

The extent of the emergency can be appreciated by evaluation of heart rate, respiratory rate and effort, capillary refill time, and gut sounds. Severe hyperemia, icterus or paleness suggest cardiovascular compromise or endotoxemia. Degree of dehydration should be estimated. Nasogastric reflux is always significant and should be investigated. The overall appearance of the mare (girth) and any abdominal distension should be noted.

Primiparous or old mares may experience mild discomfort or low grade colic due to fetal movements and position or slight displacement of the large colon by the gravid uterus. Signs that may be observed include yawning, pawing, dullness, and occasional rolling or grunting while in lateral recumbency. Physical examination parameters are often within normal limits but fluctuation of heart rate may be noticed. Gut sounds should still be present. Transrectal palpation in low grade colic allows the clinician to rule out slight large colon impaction and uterine torsion. In the last trimester of pregnancy the fetus should be in the anterior dorsopubic presentation. Deviations from this normal presentation may cause discomfort to the mare. Administration of analgesics (low dose of flunixin meglumine) and laxatives (mineral oil or magnesium sulfate by stomach tube) should alleviate some of these episodes of colic. Mares that are chronically showing discomfort or mild colic without evidence of genital or gastrointestinal abnormalities should be on diet favoring soft feces (such as a bran mash) [49].

Mild to moderate colic signs may also be seen during the first stage of labor or impending abortion. Transrectal palpation and vaginoscopy may reveal an open cervix with a fetus engaged in the birth canal.

Per rectum palpation should be conducted carefully as painful mares may fall suddenly to the ground. Lack of abnormalities on transrectal palpation does not rule out gastrointestinal involvement. Transcutaneous abdominal ultrasonography should be performed on all mares to re-evaluate the gastrointestinal tract as well as the fetus and placenta.

If the mare needs to be sedated, the drugs of choice are xylazine or romifidine because of their rapid action and clearance. Caudal epidural analgesia may be required in mares that strain. Care should be taken to avoid rectal tears particularly in mare with scant fecal output, dry or edematous rectal mucosa. Inability to easily advance the arm into the rectum suggests severe hydrops or large intestine distension. Access to the fetus and position of the broad ligaments allows diagnosis of uterine torsion. Palpation of distended small intestine should warrant further evaluation for a non-

strangulating or strangulating/ischemic small intestinal lesion [49].

Palpation of the large colon may reveal displacement or gas distention. Palpation of tight tenia (bands) across the pelvic inlet is a significant finding and should be taken seriously. Distention of the large colon, cecum, and small intestine may be a consequence of external compression of those structures by the fetus. Cecal rupture should be considered in mares with signs of endotoxic shock and contaminated peritoneal fluid. The small colon should be palpable with the characteristic fecal balls present within. Impaction or gas distention may be the result of compression or impingement against the pelvic brim by the fetus.

Abdominocentesis is often difficult in late pregnancy and is best performed with a teat cannula after identification of a ventral fluid pocket by ultrasonography as close to the xyphoid as possible. Complete blood count, serum biochemistry, and blood gas determination should be considered on a case by case basis.

The fetus and placenta should be evaluated by transrectal and transabdominal ultrasonography (Figure 12). The minimum database should include determination of the combined uteroplacental thickness (CUPT) and a baseline biophysical profile of the fetus (heart rate, breathing movements, fetal activity and tone). Increased CUPT and evidence of fetal stress (sustained bradycardia or tachycardia) may indicate impending delivery. Increased CUPT (>8 mm at 270-300 days, >10 mm at 300-330 days or > 12mm at more than 330 days of gestation) at the cervical star in mares with mucopurulent discharges suggest an ascendant placentitis [49]



Figure 12: Ultrasonographic measurement of the combined uteroplacental thickness (CUPT) at the level of the cervical star (arrows indicate middle uterine artery)

Studies have shown an increased risk for abortion, stillbirths or birth of compromised foals in mares with severe gastrointestinal colic particularly if they require surgery. These complications are primarily due to the effect of endotoxemia and intra-operative hypoxemia and hypotension. Preventative treatment against endotoxemia, placentitis, and uterine contractions should

be part of any treatment protocol for post-colic management in the pregnant mare (Table 1). Altrenogest is commonly used at double dose to maintain myometrial quiescence in mares following severe colic episodes or after colic surgery. Clenbuterol is also used for the same purpose in colicky pregnant mares undergoing medical or surgical treatment [3]. Recent reports suggest that altrenogest treatment in the last 2 months of pregnancy induces an earlier parturition and prolongs the second stage of labor causing increased problems in newborn foals. Also, foals born to altrenogest-treated mares have a lower neutrophil to lymphocyte ratio. However, review of medical and colic cases did not show any effect of altrenogest on pregnancy outcome. Episiotomy is recommended for advanced pregnant mares undergoing abdominal surgery in order to avoid cervical/placental contamination during the long period of dorsal recumbency. Mares can deliver normally after ventral abdominal surgery but foaling should be monitored closely [49].

Table 1: Treatments proposed for management of placentitis and prevention of complications following abdominal surgery in the pregnant mare

Drugs	Dose
Antimicrobials*	
Ceftiofur	20 mg/kg IV or IM q 12 hours
Trimethoprim sulfadiazine**	25 mg/kg PO q 12 hours
Gentamicin**	6.6 mg/kg IV q 24 hours
Cefazolin	20 mg/kg IV q 6 hours
Procaine Penicillin **	22000 UI/kg IM q 12 hours
Potassium Penicillin G**	22000 UI/kg, IV q 6 hours
Anti-inflammatories***	
Flunixin meglumine	0.25 mg/kg PO or IV q 8 hours
Phenylbutazone	2.2- 4.4 mg/kg PO q 12 hours
Pentoxifylline	1.1 mg/kg IV q 24 hours, or 8.5 g/kg PO q 12 hours
Acetylsalicylic acid	50 mg/kg PO q 12 hours
Tocolytics	
Altrenogest	0.088 mg/kg PO q 24 hours
Clenbuterol	1 µg/kg q 12 hours
Other supportive treatments	
Isosuprine	0.4-0.6 mg/kg PO q 24 hours
Oxygen	10-15 liters/minute intranasal insufflation
Vitamin E	1000-10,000 UI PO q 24 hours
Dextrose	2.5-5%, 1 to 2 mg/kg/minute

* Antibiotic choice is indicated by culture. It is important to note that some cases of placentitis may be due to fungal infection. Cytological evaluation of discharge may help identify these infections. Antibiotic of choice for systemic treatment of placentitis

***Anti-ulcer therapy (omeprazole) is recommended

Uterine torsion

Uterine torsion occurs generally after the 8th month of pregnancy. However, cases have been reported as early as 110 days. Suspected predisposing factors to uterine torsion include increased fetal activity and sudden drop of the mare before rolling. History may reveal excess activity of the fetus observed in the flank, recurrent colic syndrome, straining, and premature lactation [10,49,51].

Degree of pain is variable and depends on degree of torsion. Complaints may include restlessness, sweating, anorexia or poor appetite, frequent urination, wide-legged stance in the hind limbs, stretching, self-auscultation, rolling, and kicking at the abdomen. Some mares may show mild signs of colic on and off for a few weeks. The most common historical fact is non-responsiveness to analgesic treatment for mild colic [10,11].

Diagnosis is made by transrectal palpation of the broad ligaments. The ligament on the side of the torsion tends to be more caudal and is palpable as a tight vertical band while the opposite ligament is pulled horizontally across the top of the uterus (Figure 13). The degree of torsion varies from 180° to 360° (rarely greater than 360°) and is sometimes difficult to ascertain both the degree and direction of the torsion. Although earlier reports suggest higher frequency of counterclockwise torsions, this is not supported by recent observations.

Fetal and placental evaluation should be performed by transabdominal ultrasonography. Long-standing uterine torsion may show changes such as uterine and placental edema and placental separation (Figures 14). Abdominocentesis is indicated if the mare is severely affected, as uterine rupture may be a complication of uterine torsion [49].

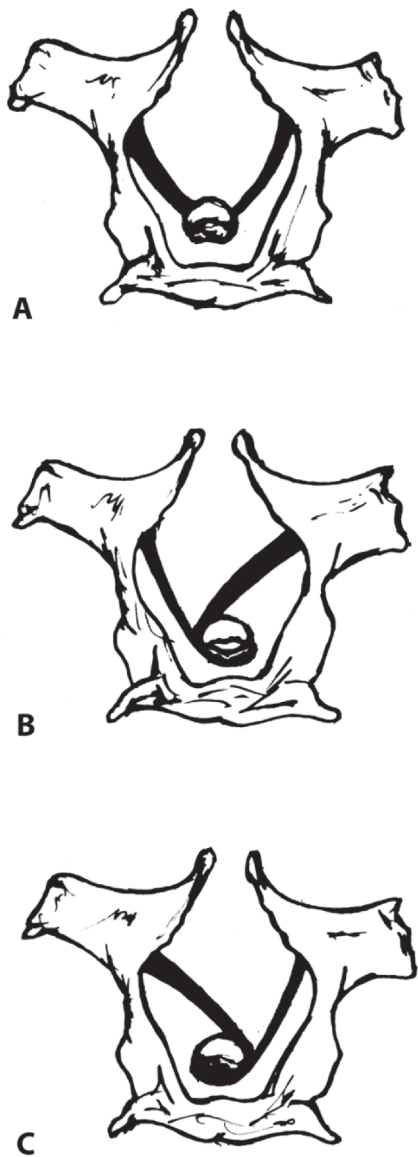


Figure 13: Position of broad ligament. A) normal, B) counterclockwise (left) torsion, C) clockwise (right) torsion

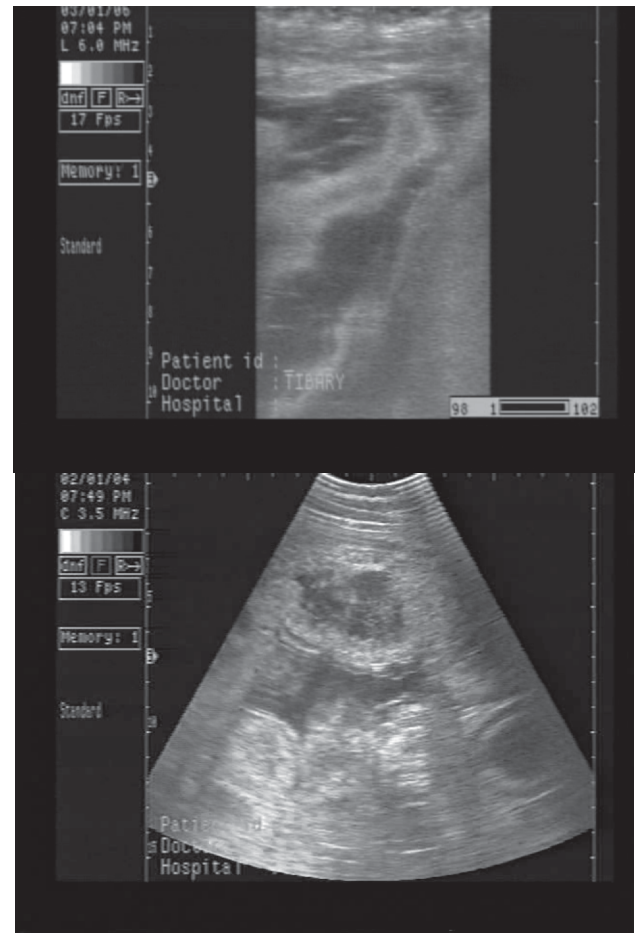


Figure 14: Ultrasonography of the uterus in mare with a severe uterine torsion. A) uterine edema and congestion, B) Placental separation

Three approaches are described for the correction of uterine torsion: rolling under general anesthesia, standing flank laparotomy and ventral midline laparotomy. The choice of correction technique depends on the stage of pregnancy, mare breed, and degree of dam and fetal compromise. At term, the fetus may be manipulated vaginally to correct the uterine torsion. Manipulations are performed in the standing mare after epidural anesthesia. The mare should be placed on a slope so that the hindquarters are slightly elevated in order to facilitate the manipulation [10].

Rolling under general anesthesia may be performed at any stage. The mare should be positioned on the side of the direction of the torsion and rolled while an aid is maintaining the uterus by exerting pressure using a plank. It may be necessary to roll the mare several times before the torsion is corrected. The mare is placed in a sternal position and checked per rectum after each roll. This technique has been associated with low incidence of uterine rupture and hemorrhage, however, these complications are relatively rare when the torsion is diagnosed and treated early [10,49].

Surgical correction of uterine torsion may be accomplished by laparotomy using a standing flank approach on the side of the torsion or under general anesthesia using a ventral midline approach. Standing flank laparotomy offers the advantage of

avoiding anesthetic complications. The incision is made on the side of the uterine torsion. A fetal hock is grasped gently and the whole feto-uterine unit is rocked back and forth until it completely flips over. Large (near term) or dead fetuses may not respond well to this technique and it may be necessary for another person to assist with the pendulum swing of the uterus via the other flank [2]. In large draft horses, correction of torsion via flank laparotomy is the preferred method in the authors' experience. In these breeds, in the last month of pregnancy a bilateral flank approach is often needed. Detorsion of the uterus may be facilitated by flooding the peritoneal cavity with large amounts of sterile fluid (LRS) [51].

Midline celiotomy is generally the best choice when the fetus is large or when there is a high risk for uterine wall compromise. In cases of severe uterine damage, delivery of the fetus may be the only choice to preserve the life of the mare. This approach is also recommended for chronic uterine torsion [10,25].

Medical management after correction of the uterine torsion is similar to that described for placentitis or post-surgical colic except that antimicrobials are used only when surgery was the method of correction. Uterine thickness and vascular integrity should be monitored daily for 3 to 5 days. Endocrinological evaluation (progesterone and estrone sulfate) may be indicated in cases which show changes in the utero-placental unit. Prognosis for fetal viability and normal foaling is excellent if the condition is diagnosed and treated before the uterus is compromised or peritoneal fluid changes occur. Outcome of uterine torsion has been summarized recently in a retrospective study of 63 cases. Mare and foal survival rates were significantly lower (65% and 32%) when mares were at more than 320 days of gestation compared to mares less than 320 days of pregnancy (97% and 72%) [10]. The poor survival of foals in advanced pregnancy is attributed to fetal hypoxemia due to disruption of uterine blood flow. Complications of uterine torsion include uterine rupture, irreversible thrombosis and uterine ischemia, hematoma, and extreme venous congestion. Gastrointestinal complications of the uterine torsion include constriction of the small intestine [10,49].



Figure 15: Vaginal varicose veins

Hemorrhagic vaginal discharge

The most common cause of hemorrhagic vaginal discharge without overt signs of distress in the mare is bleeding from varicose veins. This is mostly seen in older mares particularly of large breeds. The amount of blood varies from slight spotting to large pool of clotted blood seen when the mare is laying down. Diagnosis is confirmed by vaginoscopy using a Polanski speculum. Large ulcerated vaginal varicose veins may be visualized laterally on the cranial aspect of the vestibulovaginal fold or on the dorsal vaginal wall (Figure 15). Astringent cream (Preparation H®) may help reduce the bleeding. Alternately, the veins can be cauterized by laser .51 Other causes of bloody vaginal discharge include impending abortion or foaling and urinary bladder disease which can be directly investigated by cystoscopy and urine analysis.

Mucopurulent vaginal discharge

Mucopurulent vaginal discharge is primarily due to ascending placentitis, particularly if there are other predisposing factors (i.e. advanced age, loss of body condition, abnormal perineal conformation, etc.). Mares with placentitis will often show premature mammary development and lactation. Copious thick mucopurulent vaginal discharge may suggest fungal placentitis. Vaginoscopy allows determination of the origin and amount of discharge and sampling for cytological and microbiological evaluation. Transrectal evaluation usually shows increased placental thickness at the cervical start and placental separation with pockets of purulent content (Figure 16). Placentitis can be managed medically with anti-inflammatories, antimicrobial therapy, tocolytics, scavengers of inflammatory products, and improvement of blood flow and fetal oxygenation (Table 1) [49].

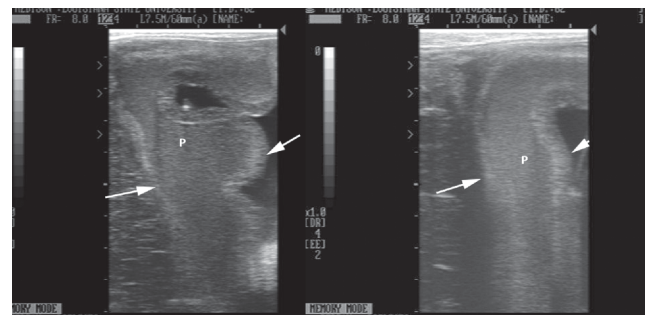


Figure 16: Ultrasonography of experimentally induced ascendant placentitis. Note the increased CUPT (arrows) and accumulation of pus (P) (Courtesy Dr. M LeBlanc).

Premature mammary gland development

Premature lactation is a feature of placentitis, impending abortion, or premature delivery of twins. It is important to verify the breeding date(s) before examination. Increased mammary size may also be observed in cases of mastitis or severe mammary gland edema.

Abnormal ventral wall or abdominal girth

Abnormal ventral wall or abdominal development may be due to prepubic tendon rupture, rupture of the mammary suspensor ligament, body wall hernia or hydrops. The primary complaint in these cases may be obvious excessive ventral edema, abdominal pain, colic, lameness or reluctance to walk, recumbency in the field, or respiratory distress.

Prepubic tendon rupture tends to be acute in onset. Advanced age of the mare, breed (draft) and fetal membranes hydrops are the most important predisposing factors. The mare may present for rapidly developing ventral edema which progresses to the udder and becomes very tense and painful. Palpation and ultrasonography may allow differentiation from rupture of mammary suspensory ligament, severe mastitis and body wall herniation [49].

Fetal membrane hydrops is a rapidly progressing condition (Figure 17). The uterus is generally extremely distended making it very difficult to ballot the fetus by transrectal palpation. In severe cases examination per rectum requires heavy sedation or epidural analgesia. High content of potassium and creatinine in the fluid obtained from the larger cavity indicates hydrops allantois rather than hydrops amnios. Mares with hydrops amnios may be managed until delivery but most cases are terminated to avoid complications such as prepubic tendon rupture, inguinal herniation, and uterine rupture. Termination of pregnancy should be controlled carefully to avoid hypovolemic shock. Fluid should be drained slowly and the mare should receive intravenous hypertonic and isotonic fluid. A plan should be in place to handle complications such as dystocia due to fetal malposition and uterine inertia. Retention of the fetal membranes is a common complication [51].



Fig

Management of mares with body wall defects depends on the severity of the condition. Mares should be on stall rest and provided abdominal support. Administration of analgesics (flunixin meglumine or phenylbutazone) should be part of the management as the condition can be extremely painful. Additional therapy including IV fluids, antimicrobials, and supportive therapy for placental function and fetal well-being (altrenogest, pentoxifylline and vitamin E) may be considered in some cases. Induction of parturition may help salvage the mare

but carries risks of delivery of premature foals. Emergency laparotomy is indicated if an intestinal incarceration is suspected. Elective cesarean section may be considered as a terminal procedure in severely debilitated mares; however, foal survival depends greatly on its readiness for birth. Dexamethasone given intramuscularly at a large dose (100 mg) on 3 consecutive days induced parturition within 1 to 8 days from the last injection and may improve maturation and foal survival outcome [51].

Other complications during late gestation

Several pre-existing conditions or disorders may be exacerbated by advancing pregnancy. The metabolic demands of pregnancy may put mares that already have a compromised liver or kidney function in jeopardy. Similarly, the increased size of the fetus and placental function can increase demands on cardiovascular and respiratory functions. Mares with ruptured suspensory ligament may become incapacitated in late pregnancy [49].

There is a potential association between conditions such as pituitary pars intermedia dysfunction (PPID), equine metabolic syndrome, pregnancy associated laminitis and disturbances of fetal and placental function. Mares with these complications may experience abortion or prolonged pregnancy due to decreased placental function and intrauterine growth retardation [51].

REPRODUCTIVE EMERGENCIES IN THE POSTPARTUM MARE

Postpartum emergencies are defined as any condition that may jeopardize the life or future reproductive ability of the mare and that have a relationship with the process of foaling. Postpartum emergencies are prevented by adoption of strict protocols of pre and post-foaling management (feeding practice, exercise etc.) and evaluation of every foaling mare early in the postpartum. Although this evaluation does not eliminate all emergencies, particularly those of gastrointestinal origin, it does give the practitioner a head start on most of the life-threatening complication such as toxic metritis as reviewed recently by the authors [50,52].

Clinical Assessment of the post-parturient Mare

Mares should be examined within 8 to 12 hours of foaling even when everything seems to be normal. Another evaluation is generally required at 24 to 36 hours which will also be used to insure that the foal had adequate passive transfer of immunoglobulins. Immediate examination of the postparturient mare is warranted anytime the foaling has not been observed of if the foal or the mare does not appear to have a normal postpartum behavior [46,50]. Other factors such as age of the mare, pregnancy length (too short or too long), dystocia, abnormal vaginal discharge, previous complications or metabolic disorders warrant immediate examination.

In addition to physical examination parameters a special attention should be given to assessment of appetite, defecation and micturition. Excessive swelling of the perineal area should be noted and investigated. Evaluation of the vaginal cavity and cervix should be completed by speculum examination and digital palpation. The placenta should be weighed and laid out in letter "F" and examined on both surfaces for completeness and any abnormalities (Figure 18) [12,38,52].

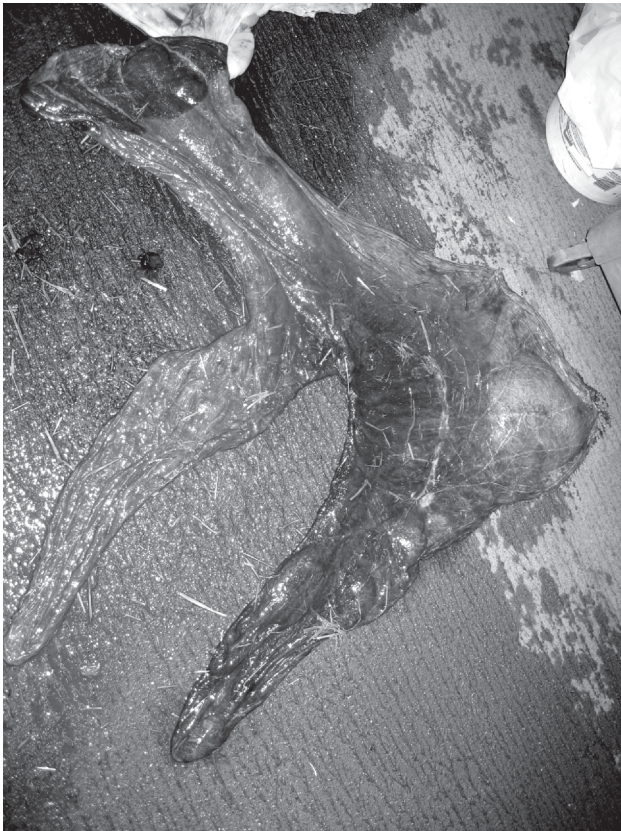


Figure 18: Examination of the placenta

Complaints in the postpartum mare that should be considered an emergency are restlessness or overt agitation, persistent and increasing intensity colic symptoms, foal rejection, absence of defecation or urination, depression, persistent straining and fever. Cases that are not responsive to emergency treatment should be referred for advanced evaluation. Complete blood count (CBC) and serum biochemistry should be obtained before initiation of therapy. Digital pulse should be evaluated frequently. Frequent analysis of peritoneal fluid should be part of the evaluation when the mare is severely depressed, colicky or non-responsive to usually therapy. Immediate action is required if total protein is greater than 30 g/L, leucocytes are more than 15 billion/L or the proportion of neutrophils is greater than 80%. Non-coordination or poor ambulation of the mare and lack of urination warrants further examination for compressive lesions or nerve damage due to trauma. Mares suspected to have a hemorrhage should be confined to a calm area. Sedation should be considered carefully because of the depressive effect it may have on an already compromised mare [50,52].

Retained placenta

The placenta is considered retained if not passed entirely by 3 hours postpartum. This definition may be too conservative as the author has seen mares deliver placenta up to 10 hours postpartum without further complication. However for better management the guidelines of 3 hours ensure safety for the mare. The incidence of retained placenta (RP) in mares varies from 2 to 10% in light breeds. Risk factors include breed, dystocia, abortion or still born, fetal membrane hydrops, placentitis, hypocalcemia, uterine atony and metabolic disorders. Higher incidence of RP (up to 45%) is seen in Friesian and draught breeds.⁵ The tip of the non-pregnant horn seems to be the area most responsible in trapping the placenta. Both total and partial retention can engender serious complications. The objectives of early treatment are to remove the placenta and to reduce bacterial multiplication (prevention of septicemia) and absorption of toxin (prevention of endotoxemia) by the inflamed uterine wall [12,25,44].

Forced extraction of the placenta is contraindicated however some authors have shown some benefit of careful manual removal [13]. Aggressive attempt to removed (pull out) the uterus may result in tears, partial retention, uterine invagination/eversion or prolapse. Necrotic tissue may remain within the endometrial crypts causing further complications with metritis and toxemia [50].

Oxytocin therapy can be administered as low dose bolus (10-20 IU) every 2 hours or preferably as a continuous rate drip (1 IU/minute total dose 60 IU) in LRS or saline. Some mares may experience severe colic with large doses of oxytocin and cause injury to the foal [50].

Chorioallantoic distension associated to low dose oxytocin is a highly effective technique early when the placenta is intact. The chorioallantoic sac is distended by 12 to 20 l (Sterile saline, water with salt 9g/L, water with povidone iodine 1%) using a nasogastric tube firmed held within the placental cavity (Figure 19). The results are usually seen within 5 to 40 minutes. It is important to evaluate calcium levels in non responsive mares and spike fluids with calcium gluconate [50].

If the placental is retained for more than 6 to 8 hours, medical management should include broad spectrum systemic antibiotics (ampicillin, gentamicin, kanamycin, pencillin, ticarcillin, ceftiofure, trimethoprim sulphamethazole). The mare should be monitored for signs of complications (anorexia, fever, depression, laminitis, warm feet, and increased digital pulse). Transabdominal ultrasound during the flushing process may reveal remnant. Supportive therapy in mares showing signs or at risk for toxemia should include fluid therapy and non-steroidal anti-inflammatory drugs (NSAID's) and tetanus prophylaxis [50].

Uterine lavage every 12 hours is highly recommended to eliminate bacteria and debris but care should be taken to first evaluate the uterus presence of tears or rupture (peritoneal fluid analysis). The use of intrauterine antibiotics is controversial (efficacy reduced by the presence of inflammatory products and autolytic tissue). The use of povidone-based oxytetracyclin for intrauterine infusion has been advocated by some authors. The addition of Polymixin B to the systemic treatment may be beneficial for its endotoxin binding properties [50].

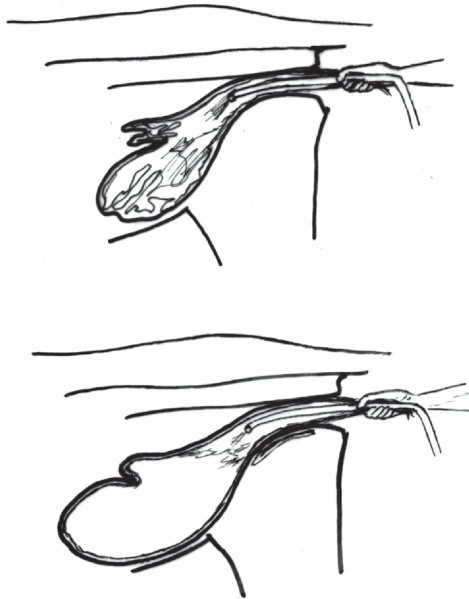


Figure 19: Technique of chorioallantoic distension for the treatment of a retained placenta in the mare.

Septic Metritis

Septic metritis accounts for 8% of postpartum emergencies and may be seen in mares as a result of dystocia, partial or total retained placenta, excessive non-judicious obstetrical manipulations. [17]. The syndrome can be very acute or develop over 2 to 3 days postpartum. Clinical signs are variable and include fever, depression, increased heart rate, injected mucous membrane, toxic line, bounding digital pulse. Palpation per rectum may reveal a thin-walled distended uterus. Vaginal examination may reveal other cervical and vaginal lesions and a foul-smelling discharge. Ultrasonography often shows large volume of intrauterine fluid with high cellularity. Placental tags may be visualized in some mares with a history of retained placenta or unobserved foaling. The uterine wall may show several edema and separation of the endometrium. CBC show severe toxic changes or leukopenia. In

severe cases, peritoneal fluid may show increased number of nucleated cells and total protein concentration. Some mares may be severely dehydrated and have grayish tacky mucous membranes. For unknown reasons, gastric reflux is not uncommon [50].

Postpartum septic metritis is often associated with gram negative bacteria such as *E. coli* and *Klebsiella pneumoniae*. The key in managing these mares is aggressive fluid therapy associated to antibiotics (potassium penicillin 22,000 IU/kg, q 6 h, IV, and gentamicin sulfate 6.6 mg/kg q 24 h, IV) and prevention or management of laminitis. Large volume (10 to 20 liters) lavage of the uterus should be performed twice a day initially to eliminate debris. Fluids should be warm (40-45°C). Initially the lavage should be controlled with transabdominal ultrasound. This would allow the clinician to detect any placental tags. Care should be taken not to damage the uterine wall. Treatment with NSAID's (flunixin meglumin 0.25 mg/kg t.i.d, followed by phenylbutazone 2-4 mg/kg) and soft bedding are highly recommended. Severe cases may require additional therapy with equine plasma polymixin B (1.5 million IU, IV) and pentoxifylline (7.5 mg/kg q 24 h, PO). Management of laminitis includes frog support, hoof icing and heparin therapy. The use of vasodilators (acetylpromazine maleate, 0.02-0.04 mg/kg q 4-6 hours) helps in some cases but mares should be carefully monitored for hypotension. Euthanasia should be considered if the mare is severely debilitated and severe rotation of the third phalanx has occurred [50,52].

Postpartum Hemorrhage

Postpartum hemorrhage has been incriminated in about 40% of postpartum mare deaths and constitutes a high level emergency [17]. Although some of these hemorrhages may occur prior to foaling most are reported in the immediate post-foaling period. In one study urogenital hemorrhage accounted for 16.6% of 163 cases of postpartum emergency in the mare [17].

Predisposing factors include age and dystocia. The mean age of mares with uterine hemorrhage seen by the author was 17.5 (range 8 to 21, n=18). Older mares experience histological changes that modify the elasticity of the arteries. Disruptions of the internal elastic lamina, adventitial elastosis, fibrosis, calcification of intima, degenerative vascular changes have been described in old multiparous mares. These changes are generally grouped under the term of "pregnancy sclerosis". Other predisposing factors cited in the literature include previous episode. The right side seems to be more prone to this injury probably due to displacement of the cecum.

The hemorrhage may be from the middle uterine artery, utero-ovarian arteries, external iliac artery or uterine wall arteries. Hemorrhage may be obvious (expulsion of large volume of blood from the vagina) or may be contained within the uterus (intrauterine hemorrhage), the abdominal cavity (hemoperitoneum) or the broad ligament (Figures 20,21). Broad ligament hemorrhage is very painful and generally occurs from rupture of the proximal uterine artery. Hemorrhage from the external iliac artery or caudal arterial vessels may cause swelling of the perineum on the same side.

Clinical signs are general visible immediately or within 24 hours of foaling. The owner may report that the mare is acting painful or showing moderate to severe colicky syndrome, flehmen response, excessive vocalization, depression, sweating, or muscle fasciculation. Mares often show tachycardia (up to 140 bpm). Some mares may be in shock (cold sweat, thready pulse). The mucous membranes are normal initially and become pale or blanched later. Transrectal palpation is extremely painful and should be limited. Transrectal and transabdominal ultrasonography may reveal large hematomas (Figure 20). Transabdominal ultrasonography may show a hemoperitoneum which can be confirmed by abdominocentesis. However, absence of hemoperitoneum does not exclude hemorrhage as it may be contained within the broad ligament and associated structure. Peritoneal fluid shows increased total protein (up to 50g/ml) and normal WBC. The PCV and RBC numbers may be normal earlier due to splenic contraction and fall quickly later on (PCV <18). Severe blood loss leads to stagger due to cerebral anemia. Common complication includes fever, leucopenia, cardiac arrhythmia and thrombophlebitis. Abscess formation within the hematoma has been observed by the authors in 2 mares [50].

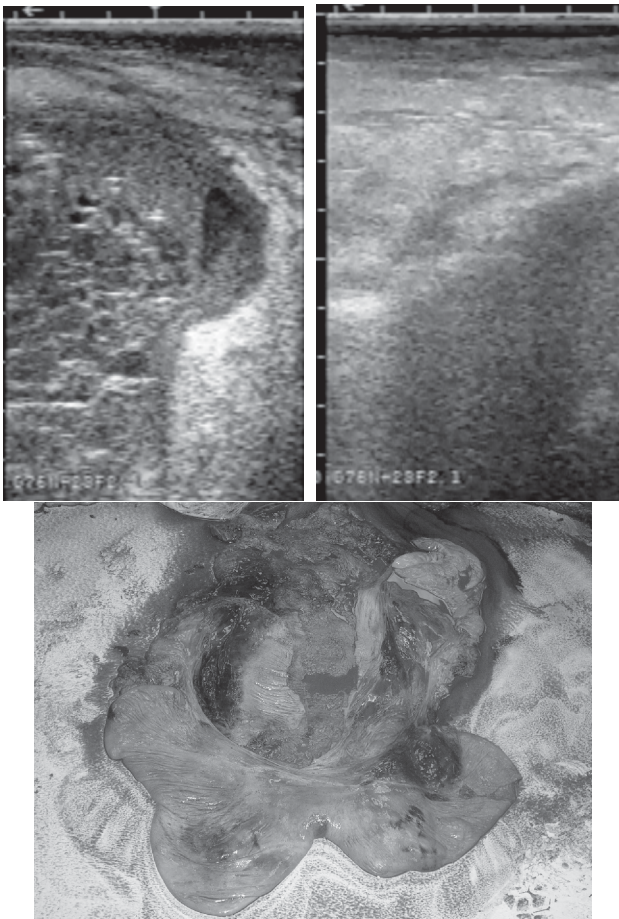


Figure 20: Broad ligament hemorrhage in a mare. A-B transrectal ultrasonography. C) postmortem

Differential diagnosis includes normal 3rd stage pain, cramping which generally responds to spasmolytics (Buscopan® Hyocine-NB-butylbromide) treatment, intraluminal hemorrhage, uterine wall tears (peritonitis). Abdominocentesis in case of uterine tear and peritonitis show increased WBC (> 5x 10⁹/ml),

presence of squam or cell from amiotic and allantoic fluid. Other conditions that should be differentiated include cecal rupture, colonic torsion, incarceration of small intestine or small colon, uterine prolapse (obvious), fractures (pelvis, femur or tibia) and bladder rupture. Precise diagnosis can be reached by careful examination of peritoneal fluid and ultrasonographic imaging of the abdomen [50].



Figure 21: Intrauterine hemorrhage

Client should be cautioned that treatment may be very costly. On initial call the client should be advised to place the mare in a dark quiet stall. Treatment will be based on supportive care to support cardiac output and ensure oxygen delivery (nasal insufflation at a flow rate of 5 to 10 L/min). Fluid therapy should be initiated as soon as possible with a bolus of 2 to 3 L hypertonic saline followed by 10 or 20 L of LRS or Hartman's over 2 to 4 hours. Use of high oncotic pressure colloids (Hetastarch, 3 L) or synthetic oxygen carriers may be useful but expensive. Whole blood transfusion (5 to 8 liters over several hours) is indicated if the PCV falls below 15%. Tranquilizers should be carefully used as they may cause hypotension. Sedation with butorphenol tartrate and xylazine is preferred by most practitioners. Corticosteroids may be beneficial in case of shock. Additional supportive therapy should include antimicrobials, NSAID's and antioxidants (pentoxifylline). The antifibrinolytic drugs, aminocaproic acid, is given as a bolus of 20 g in a liter of saline IV over 20 minutes followed by 20 to 40 g in 10 liters of isotonic fluids every 6 hours. Yunnan baiyao, a hemostatic chinese herb has gained some popularity amongst practitioners and is used per Os, at a dose of 4 g/550 kg horse mixed in 15 to 20 mL water [50].

Prognosis for survival and fertility depends on the extent of the lesions, containment of the hemorrhage and prompt treatment. Rebreeding of mares that have experience postpartum hemorrhage is a common question owners have. The site of hemorrhage (hematoma) may be palpable within the broad ligament for several weeks to months. Conception rate following recovery is often normal if there are no other complications however there is a debate on to whether or not mares are a high risk for another hemorrhage in subsequent foaling [50].

Uterine tears or rupture

Uterine rupture is often a complication of dystocia but may be seen in an uneventful foaling. Prepartum tear may occur following severe uterus torsion or fetal hydrops [52]. Sudden death may result if major blood vessels are involved. In one study, uterine tear was the third most common cause of death in the postpartum mare after uterine artery hemorrhage and gastrointestinal rupture. Uterine tears accounted for 5.5% of 163 mares admitted for postpartum emergencies. Clinical symptoms are variable and depend on the extent of the tear and rapidity of contamination (peritonitis) and include depression, anorexia, colic and fever. In the postpartum mare, the tear is often caused by fetal hoof penetration through the dorsal wall of the uterine body just cranial to the cervix or at the tip of the gravid horn (fetal movements). However, tears may also occur on both the ventral and dorsal aspects of either uterine horns or uterine body. Some studies have reported significantly more right uterine horn tears than left uterine horn tears. Diagnosis may be reached by simultaneous vaginal and transrectal palpation, abdominocentesis (serosanguinous to sanguinous fluid, fetal fluid and bacteria), ultrasound (hemoperitoneum) and celioscopy [52].

Peritoneal fluid often shows increased total protein, WBC and neutrophils. Palpation per vaginam is very sensitive in the diagnosis of uterine body tears but the majority (up to ¾) of uterine horn tears are missed. Uterine tears should be suspected in all mares with evidence of peritonitis on transabdominal ultrasonography of abdominocentesis. The most common isolates are *Escherichia coli*, *Staphylococcus spp.*, *Streptococcus equi subsp. zooepidemicus*, *Streptococcus dysgalactiae subsp. equisimilis*, *Bacillus spp.*, *α-hemolytic streptococci*, *Klebsiella pneumoniae*, *Enterobacter cloacae*, and *Proteus spp* [52].

Uterine tears may be treated medically or surgically. Both types of treatment carry similar prognosis as far as survival and breeding future of the mare. In the surgical approaches, the uterine wall is sutured through a midline or flank laparotomy, laparoscopy and vaginal approach in a Trendelenburg position. Suture after prolapsing the uterus has been successful in some cases in the immediate postpartum period. Medical management of uterine tears aims at promoting placental detachment and uterine involution (oxytocin therapy). Prevention of complication and supportive treatment includes fluid therapy, antimicrobial, non-steroidal anti-inflammatory and anti-toxin therapy. Some cases may require abdominal lavage and use of hemostatic agents (aminocaproic acid, yunan paiyo). Preventive therapeutic measures should be taken to reduce the risk for laminitis. Immediate complications of uterine tears include evisceration.

The authors have seen an entrapment of the spleen within the uterine tear in one case. Mares that present with severe leukopenia, very high heart rate and gastric reflux are less likely to survive [50,52].

Prognosis for survival of the mare and return to breeding is general good if the lesions are discovered early and the mare promptly treated.

Uterine prolapse

Uterine prolapse is uncommon but constitutes a high level emergency in the mare [9,23]. Predisposing factors include dystocia, retained placenta or persistent tenesmus due to perivaginal pain. However, it may occur following a normal parturition [55]. Uterine prolapse can occur hours or days after foaling. Client should be instructed to place a mare in a quiet clean area and support the uterus with a large plastic bag to prevent contamination and/or rupture. Ultrasonographic examination of the prolapsed tissue before attempting replacement is important in order to determine if it contains the urinary bladder (eversion or prolapse) or intestines (hernia). The tissue should be examined for increased risk of rupture (edema, friable). Replacement of the uterus may be performed following epidural anesthesia but often general anesthesia is required in order to eliminate abdominal contractions. In the field, placement of a nasogastric tube may help reduce abdominal effort. The uterus is replaced carefully and distended using large volume of fluid. Further medical management is similar to metritis or retained placenta [23]. The mare should be checked for hypocalcemia and monitored for hemorrhage (abdominocentesis). Prognosis is poor prognosis if loops of bowel are entrapped [50,52].

Partial inversion (Intussusception) of the uterine horn

Partial inversion of the uterine horn is the initial step in uterine prolapse and is often due to aggressive traction of a retained placenta, excessive use of oxytocin or tenesmus. The major complaint is postpartum colic. The invaginated tip of the horn can be felt transrectally as a thick short tight tissue. Palpation of the ipsilateral broad ligament often produces a painful reaction in the mare. Abdominocentesis may show an increased TP but stable WBC. The tips of the uterine horn can be pushed back manually per vaginam or by uterine dilation used large quantity of fluid after administration of spasmolytic drug such as Buscopan®. Necrosis of the tip of the horn and peritonitis may ensue if the condition is left untreated. Partial or total hysterectomy should be considered in these cases [50].

Gastrointestinal complications

Examination of the distressed postpartum mare should also rule out complications that are not genital in nature. Postpartum mares may show various degrees of colic, impaction, absence or scant feces (constipation) due to postpartum pain. Manual removal of fecal material, use of analgesics, administration of mineral oil and use of laxative feed helps in the majority of cases. Prevention of colic in the postpartum mare is based on reduction of the amount of roughage prepartum. The most serious conditions that are

not responsive to medical management are large colon torsion. A study on 163 postpartum emergency admission to a referral hospital found gastrointestinal disease involvement in more than one third of the cases including colon volvulus (16.6%), small intestinal diseases (small intestinal volvulus, mesenteris rents, adhesions) (7.4%), primary cecal diseases (6.1%) and small colon disease 3.1% (trauma during parturition, mesenteric rents, perforation). Other gastrointestinal disorders seen in the same study included colitis, large colon impaction, large colon displacement and nephrosplenic entrapment [17].

Mare with postpartum gastrointestinal disease may present with various complaints depending on the duration of the problem and site of lesion. Signs may be vague and include depression, colic, tachycardia, fever, and gastric reflux. Mares with large colon volvulus often show violent impaction colic and obvious abdominal distension. Transrectal palpation, transabdominal ultrasonography and abdominocentesis are often helpful in determining the need for emergency surgery. Prognosis is fair if the diagnosis is made early and surgical intervention is rapid. In delayed cases compression of the small colon, rectum, coecum between the uterus and pelvis may result in bruising of the abdominal viscera rendering the prognosis grave or poor. Complications may include peritonitis, mesentery tears resulting in ischemic necrosis, incarceration of a segment of the bowel through mesenteric rent or broad ligament, and rupture of the tip of the coecum [50,52].

Rectal prolapse and rectal tears have been reported in the postparturient mares and requires immediate referral to a surgical facility [50].

Urinary tract complications

The most common urinary tract postpartum complications are bladder rupture, bladder eversion/prolapse and bladder atony (paralysis). Bladder rupture is often a consequence of tissue necrosis due to compression against the pelvis during a dystocia. Mare with a bladder rupture often present with a complaint of depression or mild colic, progressive abdominal distension and tachycardia. Transrectal palpation and ultrasonography often reveal very small bladder and thickened small intestine. Transabdominal ultrasonography will reveal various amount of free fluid (uoperitoneum). Diagnosis is confirmed by analysis of peritoneal fluid which should increased WBC, nucleated cells, and high creatinine (ratio of peritoneal to plasma creatinine of more than 4 to 1). The exact site of the tear can be determined by endoscopy of the bladder or by laparoscopy. Direct manual examination of the bladder may be possible in large mares immediately following parturition. Surgical management is the only viable option [50].

Eversion of the bladder (visible mucosal surface of the bladder) may result from persistent tenesmus and invagination through the large urethra. Prolapse of the bladder (visible serosal surface of the bladder) may occur through a vaginal rent. Ultrasonographic examination of the tissue should be performed to evaluate the content and possible involvement of small intestines. Management of these cases requires replacement

of the bladder after epidural anesthesia and surgical correction of the defect. Mares should be placed on systemic antimicrobial and anti-inflammatory therapy [50].

Bladder paralysis and distension is often a complication of painful postpartum conditions (large perivaginal masses due to trauma, broad ligament hematomas etc.). Management requires the use of indwelling catheter and enhancement of detrusor muscle function (Bethanechol 80 mg PO, TID or 0.07 mg/kg SQ TID). Cystitis is often a complication and required specific medical managed (urinary bladder flushing and antibiotics) [50].

Necrotic vaginitis, vaginal masses and vaginal adhesions

Necrotic vaginitis should be suspected in any mare with severe perineal swelling, straining or urinary problems. Necrotic vaginitis may occur following a presumably normal parturition (Figure 22). However most commonly, a history of dystocia and obstetrical manipulation is present. Vaginal pressure necrosis may occur following severe swelling and contusions. Severe vaginal inflammation may also be a consequence of fetotomy. If not treated promptly, laceration of the vagina can become necrotic and may be fatal. In other cases vaginal adhesions may develop. The abraded vaginal walls tend to heal and seal over together forming a complete wall. These mares often develop pyometra. Bladder paralysis and atony may result from inability to posture and evacuate the bladder due to the vaginitis or presence of vaginal masses. Regular catheterization of the bladder or even placement on an undewelling bladder catheter is often warranted until the swelling goes down. Systemic antimicrobial and non-steroidal anti-inflammatory treatment is recommended in severe cases. Vaginal adhesions may be prevented by application of a tampon covered with a lanoline based cream containing antibiotics and anti-inflammatory drugs [52].

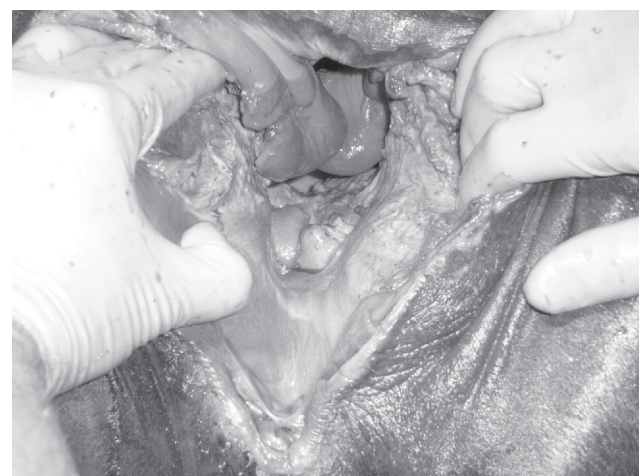


Figure 22: Necrotic vaginitis following a dystocia.

Rectovaginal Tears and Perineal Lacerations

Perineal lacerations are common injuries of foaling. First degree lacerations involve mainly the mucous membrane of the vestibule and skin of vulvar lip. Second degree laceration involves deeper tissue of the perineal body. Both these conditions are not

life-threatening and can easily be managed with reconstructive surgery (Caslick's or vulvoplasty). Medical management may include antimicrobials, NSAID's and tetanus immunization depending on severity of the lesions. Fecal softener (mineral oil, bran mash) helps in rapid healing. Mare should be bred on foal heat if no further complication using artificial insemination if it is an option. Surgery may be delayed for mares requiring natural cover until after ovulation [50].

Third degree perineal lacerations involve all tissues between the rectum and dorsal wall of the vagina forming a cloacae or rectovaginal fistula. These lesions are best left to heal by second intention and are surgically corrected after 4 to 6 weeks. Most mares will breed successfully after repair of a 3rd degree laceration [50].

Perineal bruising and vulvar hematoma

Mare with perineal protrusion should be examined carefully to determine if they extend into the vagina. Hematomas in this area are the result of ruptured obturator or internal pudendal artery due to delivery of a large foal or inexperience forced extraction. Small hematomas will resolve within a couple of weeks. Hematomas may be associated to bladder atony. Large contained hematomas may dissect along the fascia plane within the pelvic cavity (retroperitoneal hemorrhage) and presents as a large unilateral vulvar swelling. In these cases peritoneal fluid may show increased TP and WBC (TP 30-60g/L, WBC up to 100 x 10⁹/L). These conditions can be complicated by abscess formation and drainage either into the vagina or the retroperitoneal cavity [50].

Eclampsia (Lactation tetany)

Hypocalcemia is usually found in draught breeds, miniature horses and ponies. The mare shows restlessness, tachypnea, staring eyes, twitching, trembling, clonic spasm and recumbency. The condition should be differentiated from tetanus. Diagnosis is based on determination of blood calcium level. Administration of calcium gluconate IV resolves this situation [50].

Mastitis

Acute mastitis is relatively uncommon in the postpartum period in the mare. However, examination of the udder should be part of any complaint of lameness, foal rejection or mild colic with increased rectal temperature. 50

CONCLUSION

Reproductive emergencies in stallions and mares can arise at anytime. Veterinary practitioners involved with studfarm medicine should be equipped and have a clear plan for handling these emergencies and stabilization of the patient for eventual referral to surgical facilities. Most emergencies in stallions are traumatic of origin and can be avoided by proper management during handling and breeding. Stallions with sudden onset of colicky episode should be evaluated for testicular torsion or scrotal hernia as these can be life-threatening.

In pregnant mares, emergencies may be due to an abnormal pregnancy or accidents. Mares undergoing surgery should be considered at high risk and monitored closely. Most of the reproductive emergencies in mares occur in the last trimester of pregnancy and early postpartum. Proper monitoring during these period and management thru foaling as well as adoption of strict protocols for the examination of the postpartum mare improve the prognosis.

REFERENCES

1. Alves GES, Santos RL, Henry M, Ribeiro AG, Rothschild CM. 2000. Acquired bilateral inguinal hernia in a stallion. *Equine Veterinary Education*;12: 256-259.
2. Arnold CE, Brinsko SP, Love CC, Varner DD. 2010. Use of a modified Vinsot technique for partial phallectomy in 11 standing horses. *Journal of the American Veterinary Medical Association*;237: 82-86.
3. Bartmann CP, Klug E. 2012. Colic in the pregnant and periparturient mare - distribution, survival and abortion rate. *Pferdeheilkunde*;28: 406-412.
4. Blanchard TL, Schumacher J, Edwards JF, Varner DD, Lewis RD, Everett K, Joyce JR. 1991. Priapism in a stallion with generalized malignant melanoma. *Journal of the American Veterinary Medical Association*;198: 1043-1044.
5. Boerma S, Back W, van Oldruitenborgh-Oosterbaan MMS. 2012. The Friesian horse breed: A clinical challenge to the equine veterinarian? *Equine Veterinary Education* 24: 66-71.
6. Boller M, Fürst A, Ringer S, Dubs M, Bettschart-Wolfensberger R. 2005. Complete recovery from long-standing priapism in a stallion after propionylpromazine/xylazine sedation. *Equine Veterinary Education*;17: 305-309.
7. Brinsko SP, Blanchard TL, Varner DD. 2007. How to treat paraphimosis: 580-582.
8. Burns JJ, MacMillan K, Uehlinger FD, Riley CB. 2011. Concurrent nephrosplenic entrapment and acquired inguinal herniation of the jejunum in a Standardbred stallion. *Canadian Veterinary Journal-Revue Veterinaire Canadienne*;52: 295-296.
9. Causey R, Ruksznis D, Miles R. 2007. Field management of equine uterine prolapse in a Thoroughbred mare. *Equine Veterinary Education*;19: 254-259.
10. Chaney KP, Holcombe SJ, LeBlanc MM, Hauptman JG, Embertson RM, Mueller PE, Beard WL. 2007. The effect of uterine torsion on mare and foal survival: a retrospective study, 1985-2005. *Equine Veterinary Journal*;39: 33-36.
11. Crabtree J. 2012. Peripartum problems in mares 1. Prepartum problems. *In Practice*;34: 400-410.
12. Crabtree J. 2012. Peripartum problems in mares 2. Postpartum problems. *In Practice*;34: 462-471.
13. Cuervo-Arango J, Newcombe JR. 2009. The Effect of Manual Removal of Placenta Immediately after Foaling on Subsequent Fertility Parameters in the Mare. *Journal of Equine Veterinary Science*;29: 771-781.
14. da Silva LAF, Rabelo RE, de Godoy RF, da Silva OC, Franco LG, Coelho CMM, Cardoso LL. 2010. Retrospective study of traumatic phimosis in horses and treatment with penis shortening surgery circumcision (1982-2007). *Ciencia Rural*;40: 123-129.

15. De Bock M, Govaere J, Martens A, Hoogewijs M, De Schauwer C, Van Damme K, De Kruif A. 2007. Torsion of the spermatic cord in a Warmblood stallion. *Vlaams Diergeneeskundig Tijdschrift*;76: 443-446.
16. Delling U, Stoebe S, Brehm W. 2012. Hand-assisted laparoscopic adhesiolysis of extensive small intestinal adhesions in a mare after breeding injury. *Equine Veterinary Education*;24: 545-551.
17. Dolente BA, Sullivan EK, Boston R, Johnston JK. 2005. Mares admitted to a referral hospital for postpartum emergencies: 163 cases (1992-2002). *Journal of Veterinary Emergency and Critical Care*;15: 193-200.
18. Driessen B, Zarucco L, Kalir B, Bertolotti L. 2011. Contemporary use of acepromazine in the anaesthetic management of male horses and ponies: A retrospective study and opinion poll. *Equine Veterinary Journal*;43: 88-98.
19. Feary DJ, Moffett PD, Bruemmer JE, Southwood L, McCue P, Niswender KD, Dickinson C, Traub-Dargatz J. 2005. Chemical ejaculation and cryopreservation of semen from a breeding stallion with paraphimosis secondary to priapism and haemorrhagic colitis. *Equine Veterinary Education*;17: 299-304.
20. Filippo PAd, Pereira RN, Perotta JH, Alves AE, Dias DPM, Santana ÁE. Retrospective study of 50 equine colic cases presented to the veterinary hospital of FCAV-UNESP between 2004 and 2005. *Ciência Animal Brasileira*; 11: 689-694.
21. Gerhards H. 2003. Complications associated with castration of stallions and their prevention. Surgical and legal aspects. *Pferdeheilkunde*;19: 37-44.
22. Gonzalez M, Tibary A, Sellon DC, Daniels J. 2008. Unilateral orchitis and epididymitis caused by *Corynebacterium pseudotuberculosis* in a stallion. *Equine Veterinary Education*;20: 30-36.
23. Hewes CA, Johnson AK, Kivett LE, Stewart AJ, Weisman JL, Caldwell FJ. 2011. Uterine prolapse in a mare leading to metritis, systemic inflammatory response syndrome, septic shock and death. *Equine Veterinary Education*;23: 273-278.
24. Karzenski S, Crisman MV, Robertson J. 1997. Case report - Endocarditis in a stallion with a history of testicular torsion. *Equine Practice*;19: 24-27.
25. LeBlanc MM. 2008. Common Peripartum Problems in the Mare. *Journal of Equine Veterinary*;28: 709-715.
26. Love CC, McDonnell SM, Kenney RM. 1992. Manually Assisted Ejaculation in a Stallion with Erectile Dysfunction Subsequent to Paraphimosis. *Journal of the American Veterinary Medical Association*;200: 1357-1359.
27. Marino G, Zanghi A, Quartuccio M, Cristarella S, Giuseppe M, Catone G. 2009. Equine Testicular Lesions Related to Invasion by Nematodes. *Journal of Equine Veterinary Science*;29: 728-733.
28. McDonnell SM. 2005. Managing the paralysed penis, priapism or paraphimosis in the horse. *Equine Veterinary Education*;17: 310-311.
29. Mendoza FJ, Perez-Ecija A, Estepa JC. 2010. Inguinal-scrotal herniation and torsion of the large colon in an adult Andalusian stallion: a case report. *Veterinarni Medicina*;55: 281-284.
30. Mezerova J, Zert Z, Kabes R, Jahn P. 2003. Hernia inguinalis incarcerata in horses: 43 cases. *Pferdeheilkunde*;19: 263-268.
31. Mohindroo J, Singh S. 2011. Successful Treatment of Penile Hematoma in a Colt. *Journal of Equine Veterinary Science*;31: 615-617.
32. Morley S, Rodriguez J, Pearson L, Sandoval S, A. T. 2010. Post-dystocia bladder paralysis and cystitis in a mare: Medical management and outcome. *Clinical Theriogenology*;2: 401.
33. Papa F, Leme D. 2002. Testicular fine needle aspiration cytology from a stallion with testicular degeneration after external genitalia trauma. *Journal of Equine Veterinary Science*;22: 121-124.
34. Pauwels F, Schumacher J, Varner D. 2005. Priapism in horses. *Compendium on Continuing Education for the Practicing Veterinarian*;27: 311-315.
35. Pearson LK, Rodriguez JS, Tibary A. 2011. How to obtain a stallion testicular biopsy using a spring-loaded split-needle biopsy instrument. *Lexington: American Association of Equine Practitioners (AAEP)*, 219-225.
36. Ragle CA, Yiannikouris S, Tibary AA, Fransson BA. 2013. Use of a barbed suture for laparoscopic closure of the internal inguinal rings in a horse. *Journal of the American Veterinary Medical Association*;242: 249-253.
37. Rochat MC. 2001. Priapism: a review. *Theriogenology*;56: 713-722.
38. Schlafer DH. 2004. Postmortem examination of the equine placenta, fetus, and neonate: methods and interpretation of findings. *Lexington: American Association of Equine Practitioners (AAEP)*;144-161.
39. Schumacher J, Hardin DK. 1987. Surgical Treatment of Priapism in a Stallion. *Veterinary Surgery*;16: 193-196.
40. Schumacher J, Varner DD, Crabill MR, Blanchard TL. 1999. The effect of a surgically created shunt between the corpus cavernosum penis and corpus spongiosum penis of stallions on erectile and ejaculatory function. *Veterinary Surgery* 1999;28: 21-24.
41. Threlfall WR, Carleton CL, Robertson J, Rosol T, Gabel A. 1990. Recurrent Torsion of the Spermatic Cord and Scrotal Testis in a Stallion. *Journal of the American Veterinary Medical Association*;196: 1641-1643.
42. Tibary A. 2004. Testicular diseases in the stallion. *Gainesville: Eastern States Veterinary Association*;234-237.
43. Tibary A. 2005. Diseases of the reproductive tract in stallions. In Tibary A, Bakkoury M (eds): *Reproduction Equine: Tome II: L'étalon (Equine reproduction, Vol. II The Stallion)*. (in French) *Actes Edition*, Rabat, Morocco, ISBN: 9981-801-62-3 Pages 185-316. 2005.
44. Tibary A. 2006. Postpartum complications in the mare. *1st Jornada Internacional de Reproduccion Equine*, Buenos Aires, October, 2006.
45. Tibary A, Chabchoub A, Sghiri A. 2007. Conduite à tenir diagnostique: face à une augmentation de volume des bourses scrotales: chez l'étalon. *Le Nouveau Praticien Vétérinaire - Équine*. 21-27.
46. Tibary A. 2011. Chapter 52. Postpartum Care of the Mare and Foal. In. Carla L. Carleton (ed). *Blackwell's Five-Minute Veterinary Consult, Clinical Companion. Equine Theriogenology*, Wiley-Blackwell Publishing, Ames, Iowa, 2011. Pages: 440-447. 2011.
47. Tibary A, Pearson L. 2012. Dystocia and obstetrical manipulation in the mare. *XIII conference of Brazilian Equine Veterinary Association, Brazilian Journal of Equine Medicine, Supplement III- Vol 41*. pp. 72-89.

48. Tibary A, Pearson L, Rodriguez J. 2012. Common reproductive problems in breeding stallions. *XIII conference of Brazilian Equine Veterinary Association, Brazilian Journal of Equine Medicine, Supplement III- Vol 41*. pp.103-122.
49. Tibary A, Pearson L, Rodriguez J, Sghiri A. 2012. Gestation à haut risque chez la jument et complications. In : *Tibary A and Sghiri A (ed). Jument et Poulain : Suivi de la gestation, du poulinage et du nouveau-né, Actes Edition, Rabat- Instituts, Morocco*. 2012, pp.103-162. 2012.
50. Tibary A, Pearson L, Sghiri A, Rodriguez J. 2012. Urgences et autres complications du post-partum chez la jument (Emergencies and postpartum complications in the mare). In : *Tibary A and Sghiri A (ed). Jument et Poulain : Suivi de la gestation, du poulinage et du nouveau-né, Actes Edition, Rabat- Instituts, Morocco*. 2012, pp.251-304. 2012.
51. Tibary A, Pearson LK. 2012. Mare problems in the last month of pregnancy. : 350-358.
52. Tibary A, Pearson LK. 2012. Medical problems in the immediate postpartum period. 362-369.
53. Tibary A, Chabchoub A, Sghiri A. 2008. Chirurgie conduite à tenir thérapeutique: face à une augmentation de volume: des bourses scrotales: chez l'étalon. *Le Nouveau Praticien Vétérinaire - Équine*, : 51-54.
54. Torre F. 2000. Surgical treatment and complications in a case of priapism in a stallion. *Ippologia*;11: 5-9.
55. Torres EB, Abalos JHA, Gicana KRB. 2009. Uterine Prolapse in a Mare Following Normal Delivery: Clinical Case Management. *Philippine Journal of Veterinary Medicine*; 46: 119-122.
56. Van Harreveld PD, Gaughan EM. 1999. Partial phallectomy to treat priapism in a horse. *Australian Veterinary Journal*;77: 167-169.
57. Vasey JR. 1981. Simultaneous Presence of a Direct and an Indirect Inguinal-Hernia in a Stallion. *Australian Veterinary Journal*;57: 418-421.
58. Weiermayer P, Richter B. 2009. Simultaneous presence of a seminoma and a leiomyoma in the testes of a horse. *Equine Veterinary Education*;21: 172-176.
59. Wilderjans H, Meulyzer M, Simon O. 2012. Standing laparoscopic peritoneal flap hernioplasty technique for preventing recurrence of acquired strangulating inguinal herniation in stallions. *Veterinary Surgery*;41: 292-299.
60. Wilson DV, Nickels FA, Williams MA. 1991. Pharmacologic treatment of priapism in two horses. *J Am vet med Ass*;199: 1183-1184.
61. Wilson KE, Dascanio JJ, Duncan R, Delling U, Ladd SM. 2007. Orchitis, epididymitis and pampiniform phlebitis in a stallion. *Equine Veterinary Education*;19: 239-243.
62. Zhang J, Ricketts SW, Tanner SJ. 1990. Antisperm Antibodies in the Semen of a Stallion Following Testicular Trauma. *Equine Veterinary Journal*;22: 138-141.