Neurological disease in neonatal foals: beyond dummy foal syndrome

Neonatal encephalopathy (dummy foal syndrome) is the most common cause of neurological signs in neonatal foals. However, there are a wide variety of other causes that may be overlooked, including infections, traumatic, metabolic and congenital causes. There is much overlap between the clinical signs for many of these conditions, highlighting the need for thorough history taking, physical examination, laboratory testing and, in some cases, diagnostic imaging. In most cases, prompt treatment is required if there is to be any chance of survival, further highlighting the need for an accurate diagnosis. The prognosis of these conditions varies from rapidly correctable with appropriate treatment, to euthanasia being the only humane option.

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Weakness and abnormal behaviour are common presenting clinical signs in unwell neonatal foals. This may be the result of a primary neurological disorder, or numerous other conditions. Acquiring a thorough history, including events during gestation, foaling and post-foaling, as well as investigating all body systems, is warranted for any case.

Neonatal encephalopathy (dummy foal syndrome) is a condition that equine veterinarians who see foals will be familiar with. Dummy foal syndrome generally affects foals in the first 48–72 hours of life (although it can be up to 7 days before clinical signs develop) and causes a range of neurological clinical signs from loss of affinity to the mare, to grand mal seizures. Dummy foal disease was extensievly covered in the Novemeber 2020 issue of UK-Vet Equine, so this review focuses on the other neurological conditions that can cause clinical signs in neonatal foals (*Table 1*). For the purpose of this review, neonatal foals less than 30 days of age will be considered.

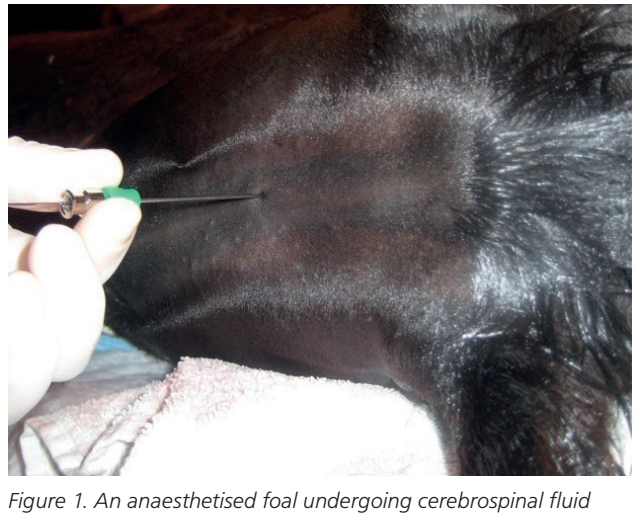

Figure 1. An anaesthetised foal undergoing cerebrospinal fluid sampling from the atlanto-occipital space

Infectious causes

Bacterial meningitis

Although it is still an uncommon condition in equines, bacterial meningitis occurs more frequently in foals than in adult horses (Mitchell et al, 2006), likely because of the increased permeability of the blood–brain barrier, combined with immaturity of the immune system (Koterba et al, 1984; Sanchez et al, 2008; Viu et al, 2012b). The occurrence is highest in foals suffering from sepsis, with a reported prevalence of 2.6–10% (MacKay, 2005), presumably caused by bacteraemic spread. It may also occur as a complication of cranial trauma (MacKay, 2005) and can occur secondarily to osteomyelitis in the axial skeleton in older foals (Morresey et al, 2010).

Cervical pain, rigidity and hyperaesthesia are indicative of this condition. Other neurological abnormalities are non-specific, including altered mental status, reduced suckling, weakness, ataxia, blindness and seizures. A fever may be expected but is not evident in many cases (Viu et al, 2012b). Clinicopathological findings reflect systemic inflammation and include neutropaenia or neutro-

Table 1. Causes of neurological disease in neonatal foals, other than neonatal encephalopathy

Infectious	Bacterial meningitis
	Sepsis-associated encephalopathy
	Tetanus
	Botulism
Traumatic	Head trauma
	Spinal trauma
	Peripheral nerve and plexus injury
Metabolic	Hypoglycaemia
	Hyponatraemia
	Hypocalcaemia
	Hyperbilirubinaemia
	Hyperammonaemia
Congenital	Hydrocephalus
	Occipitoatlantoaxial malformation
	Cerebellar abiotrophy
	Epilepsy

philia and hyperfibrinogenaemia (MacKay, 2005). Definitive diagnosis requires cerebrospinal fluid (CSF) sampling (MacKay, 2005; Furr, 2008) and likely a general anaesthetic (*Figure 1*), although in a young foal sampling can often be performed safely under heavy sedation. The non-specific nature of the clinical signs means that in human infants, CSF sampling is routinely performed as analysis reveals neutrophilic pleocytosis (total nucleated cell count >7 cells/μL), increased total protein (>1.2 g/L) and intracellular bacteria (Viu et al, 2012b). The bacteria isolated are commonly the same as those associated with neonatal sepsis (Koterba et al, 1984; Viu et al, 2012b), including *Escherichia coli*, *Actinobacillus* spp., *Klebsiella* spp., *Streptococcus* spp. and *Salmonella* spp. (MacKay, 2005).

Imaging does not play a significant role in the routine diagnosis of meningitis, beyond ruling out causes such as skull fracture. However, in human medicine, magnetic resonance imaging (MRI) examination is used to detect and monitor intracerebral complications of meningitis, such as hydrocephalus or haemorrhage, and there is evidence to suggest that localisation of pathological signal alterations may provide insight into the aetiology. Therefore, there may be a place for MRI examination in the initial diagnosis but further investigation is required (Jaremko et al, 2010). In equine medicine, there are limited reports on the MRI findings associated with meningitis, although a case report of a foal with meningoencephalitis described similar findings to those seen in humans (Viu et al, 2012a).

Treatment is primarily with intensive antimicrobial therapy. This would be ideally guided by culture and sensitivity, but the urgency of initiating treatment often means empirical treatment is required (MacKay, 2005). Penetration of the blood–brain barrier is a key consideration, although the permeability of the inflamed blood–brain barrier is likely to be increased. The third-generation cephalosporins cefotaxine (40 mg/kg IV, every 6 or 8 hours) or ceftriaxone (25 mg/kg IV, every 12 hours) have good penetration of the central nervous system (CNS) (MacKay, 2005; Mitchell et al, 2007), although lack of availability and cost may prohibit their use. Chloramphenicol sodium succinate is considered to have good penetration into the CSF and is broad spectrum, making it an ideal drug in foals that can tolerate oral drug therapy. Trimethoprim-sulfamethoxazole has also been recommended (Chaudhuri, 2004; Corley and Hollis, 2009; Ogunlesi et al, 2015). Aminoglycosides and ceftiofur are considered to have poor blood–brain barrier penetration (Corley and Hollis, 2009) and as such, are rarely recommended. Since much of the CNS damage is caused by the inflammation rather than the infection itself, anti-inflammatories are often indicated. Dexamethasone has been shown to be beneficial in treating bacterial meningitis in humans, especially if it is administered early in the clinical course (MacKay, 2005), although it should be noted that it has not been shown to alter mortality. Extrapolating from data in children, it has been suggested that foals be given dexamethasone sodium phosphate, 0.4 mg/kg intravenously, every 12 hours for 2 days, with the first dose given with or before the first dose of an antimicrobial agent (Viu et al, 2012b). More conservative doses are often used in horses. The prognosis for survival depends on the severity of the presenting signs (MacKay, 2005) and is considered fair in foals that remain relatively alert, but guarded to poor if the foal is obtunded (Zhang et al, 2014).

Sepsis-associated encephalopathy

Although sepsis can result in overt infection of the CNS, it can also cause neurological dysfunction as a result of severe systemic inflammation (Chaudhry and Duggal, 2014). The pathophysiology is believed to involve numerous mechanisms including direct neuronal cell injury, mitochondrial and endothelial dysfunction, derangements of neuronal calcium homeostasis and disturbances of neurotransmission (Cook et al, 2001). This results in a spectrum of signs ranging from mild behavioural abnormalities to severe

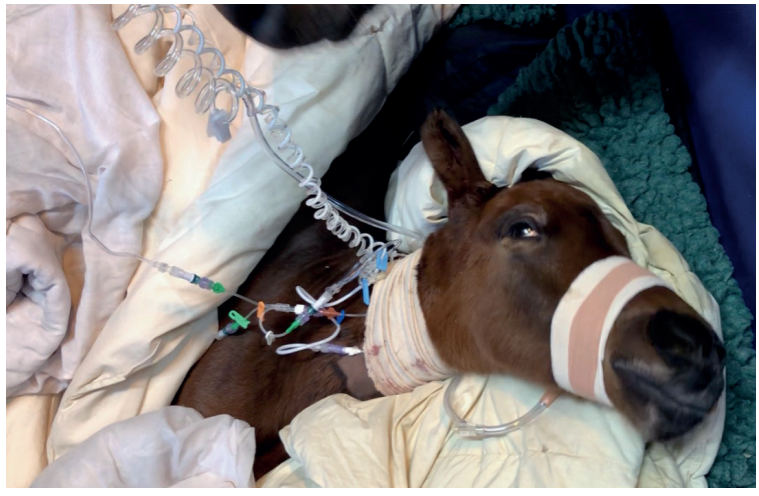

Figure 2. A foal with sepsis-associated encephalopathy demonstrating seizure activity.

disturbances of consciousness, seizures and coma (*Figure 2*). Diagnosis and treatment is as for neonatal sepsis, which is beyond the scope of this review. Sepsis-associated encephalopathy can be differentiated from meningitis by CSF analysis; unlike with meningitis, with sepsis-associated encephalopathy there is no evidence of CNS infection in the CSF, although protein levels may be mildly elevated because of the increased permeability of the blood–brain barrier (Gofton and Young, 2012). Neurological signs caused by sepsis have been associated with poorer survival rates in hospital (Corley, 2004).

Tetanus

The neurotoxin tetanospasmin, produced by proliferating *Clostridium tetani*, circulates to peripheral nerve terminals and then travels via axons to presynaptic inhibitory terminals in spinal interneurons. Here it acts to prevent the release of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA), resulting in disinhibition of spinal motor neurons and a resultant syndrome of spastic paralysis (MacKay, 2005). Toxin production occurs when *C. tetani* proliferates, which requires anaerobic conditions. In foals this is most commonly associated with the umbilical remnant (Mykkänen et al, 2011). Horses are highly sensitive to tetanus toxins but disease is rare as a result of widespread use of tetanus toxoid vaccines. Cases in foals are typically seen in those older than 7 days (MacKay, 2005; Mykkänen et al, 2011).

Affected foals initially have difficulty nursing because of spasms of the masticatory muscles (trismus) and neck. If the foal manages to latch on, pharyngeal spasms typically result in dysphagia. The limbs and back are in rigid extension resulting in a classic 'sawhorse' posture. The face often assumes a fixed, anxious expression (risus sardonicus) with intermittent prolapse of the third eyelid in response to external stimuli. Attempts to move may provoke muscle spasms resulting in the foal falling. Eventually the foal becomes laterally recumbent in a rigidly extended position and is subject to rounds of severe tonic muscle contractions. Sympathetic overactivity may cause tachycardia and profuse sweating and ultimately, respiratory failure which results in death (Cook et al, 2001). Diagnosis is based on clinical signs, combined with the history of incomplete maternal vaccination or failure of passive transfer of immunoglobulin from the colostrum (Mykkänen et al, 2011).

The mainstay of treatment is tetanus antitoxin (10,000 units intramuscularly) (Mykkänen et al, 2011), to neutralise the circulating toxin, combined with intensive supportive care. Muscle spasms may be controlled with benzodiazepines or phenobarbital (MacKay, 2005). Penicillin is the standard treatment for attempting to reduce further toxin production, but its use has been questioned because of its potential GABA antagonism (Mykkänen et al, 2011). If possible, surgical excision or drainage of the anaerobic site (such as the umbilicus) may be considered. The prognosis is guarded if treated early and aggressively. The prognosis is considered poor when recumbency ensues, although successful outcomes have been reported (MacKay, 2005).

Botulism

Unlike tetanus, the neurotoxins produced by *Clostridium botulinum* result in a syndrome of flaccid paralysis. Although common in certain regions of the USA, disease occurs only sporadically elsewhere. In foals, the disease is most commonly thought to occur following ingestion of *C. botulinum* spores from the environment, that germinate in the gastrointestinal tract releasing a toxin that is then absorbed (Stratford et al, 2014). This is unlike the situation typically seen in adults where the preformed toxin is typically ingested (Wilkins and Palmer, 2003). Toxin types B and C are typically implicated, although type A cases have been reported (Wilkins and Palmer, 2003). Botulism toxins act presynaptically at the neuromuscular junction to inhibit release of the neurotransmitter acetylcholine. The neurotoxin does not affect the CNS or sensory nerves, which can often aid in distinguishing botulism from other neurological disorders. The disease typically presents as the sudden development of weakness, trembling and dysphagia (Wilkins and Palmer, 2003), although severely affected foals may be found dead. The disease typically progresses and ensuing respiratory paralysis results in death, although the prognosis is better in foals compared to adults.

Diagnosis is generally presumptive as a definitive diagnosis is challenging. Demonstration of the spores or toxin in the faeces using a mouse bioassay has high specificity but low sensitivity (Stratford et al, 2014). Unlike tetanus, in which cases tend to occur in foals from dams with poor vaccination status, one study in the USA found 20% of botulism cases in foals were in those from dams with reported vaccination history against botulism, suggesting that transfer of immunity cannot be relied upon (Stratford et al, 2014). Treatment requires administering antitoxin and intensive supportive care. Administering antitoxin quickly is key as the circulating toxin must be neutralised before it binds to the motor endplate, as once this occurs the situation is irreversible. However, acquiring the antitoxin in the UK is likely to be challenging (Wilkins and Palmer, 2003). Although antitoxin administration is associated with vast improvements in survival rates (Marr, 1999), survival without the antitoxin has been reported in foals with mild disease that progresses slowly, so management can be attempted (Wilkins and Palmer, 2003). If the foal becomes recumbent, mechanical ventilation is required for the foal to have any chance of surviving (MacKay, 2005). Survival for type B and C cases can be good if aggressive care is instigated early in the disease course (Prutton et al, 2016), with up to 96% chance of survival being reported (Wilkins and Palmer, 2003). Type A is typically associated with poorer survival in horses, with a reported fatality rate of 90%. However, in one study, none of the affected foals survived (Johnson et al, 2010). Survival has been reported in one foal with type A botulism (Prutton et al, 2016).

Traumatic causes

Head trauma

Regardless of whether there is a reported history of trauma, this should always be considered in foals with acute onset of neurological signs. Brain trauma may occur following blunt force trauma to the frontal or parietal bones (typically as the result of a kick), or to the poll or temporal region, which typically occurs if the foal flips over backwards (MacKay, 2005). The immediate injury and the subsequent tissue reaction both result in neurological signs. There

may be signs suggestive of trauma such as abrasions, swollen or painful areas on palpation, crepitus or evidence of bleeding from the nose or ears. However, in many cases there will be no outward signs and imaging may be required for confirmation (*Figure 3*).

Clinical signs will depend on the area of the brain that is affected. Trauma to the rostral skull typically results in disorientation, head-pressing, lethargy and a tendency to walk compulsively, often in circles. Poll impact more commonly results in signs attributable to brain stem dysfunction, including profound obtundation, head tilt, facial paralysis and ataxia (MacKay, 2005) (*Figure 4*). Imaging, including computed tomography (CT) and MRI where available, and endoscopic examination of the guttural pouches, provides further localisation and potentially prognostic information, although care must be taken when sedating or anaesthetising foals for these procedures.

Treatment is supportive only, unless stabilisation of fractures is possible. Anti-inflammatories may be of some benefit in the acute phase. The use of corticosteroids is controversial and is no longer recommended (CRASH trial collaborators, 2004; Reed et al, 2018). A large prospective study in humans suggests that corticosteroids are contraindicated in cases of traumatic brain injury as they are associated with increased mortality (CRASH trial collaborators, 2004); the mechanism for this is not understood, although it is postulated that it is associated with the increased risk of hyperglycaemia, which is considered especially hazardous in cases of cerebral oedema (Jeremitsky et al, 2005). It should however be noted that equine-specific data are lacking and the doses of steroids used in human medicine far exceed those typically used in equine practice. Broad spectrum antimicrobial therapy is typically indicated as there is a risk of CNS infection. The second phase of CNS injury may be addressed using treatments similar to those implicated in hypoxic ischaemic encephalopathy, including antioxidants, although evidence is lacking (Bayir et al, 2009). Therapeutic hypothermia has been shown to decrease oxidant injury in human neonates suffering from traumatic brain injury (Feldman et al, 1992) and may prove useful in foals, although current practical difficulties have limited use in the equine setting. Elevating the head has also been shown to be useful in humans to reduce intracranial pressure (MacKay, 2005), although this is not specifically possible in foals.

Spinal trauma

Spinal trauma is most commonly seen in foals older than one week of age, although it has also been reported following dystocia (Mackenzie et al, 2018). Typically, injuries occur when foals collide with immobile objects, resulting in fractured or dislocated vertebrae (MacKay, 2005). Clinical signs range from weakness to ataxia to total paralysis in extreme cases, with the affected limbs being those caudal to the site of injury. If the cranial neck is involved, reluctance to move the head may be the only clinical sign. Radiography is typically sufficient for diagnosis although advanced imaging and myelography may be used, particularly if cord compression is debated. Treatment is similar to that for head trauma, although steroids have been suggested to be of some benefit in these cases (MacKay, 2005). The prognosis depends on the severity of cord damage.

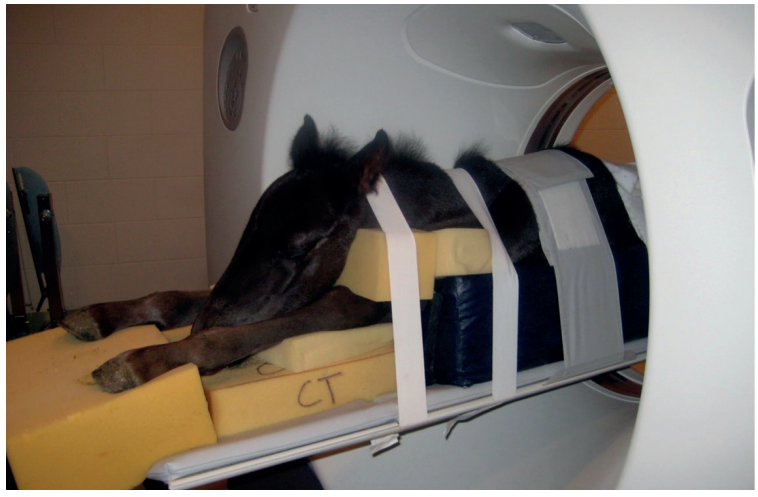

Figure 3. A foal undergoing a computed tomography examination.

Peripheral nerve and plexus injury

Iatrogenic injury can occur in the brachial plexus or femoral nerve following forced extraction of the foal in the event of dystocia, or to the sciatic or tibial nerve after injections in the gluteal muscles or hamstrings. Injury can also occur secondary to soft tissue trauma or fractures. Injuries are classified as type 1 or type 2 depending on whether or not axonal continuity is disrupted. Clinical signs include weakness and hyporeflexia of the affected limb, with or without loss of skin sensation. Roughly 2 weeks after injury, muscle atrophy will be evident (MacKay, 2005). The diagnosis is typically presumptive because confirmation requires electrodiagnostic

Figure 4. A foal with a head tilt following head trauma. A subpalpebral lavage system is present in the left upper eyelid for management of a corneal ulcer.

studies, including nerve conduction and needle electromyography, which are not typically available in general practice.

In acute cases, systemic anti-inflammatories should be administered and consideration can be given topical agents drugs such as dimethyl sulfoxide (DMSO). If the nerve is severed, anastomosis is possible, although this is rarely performed in horses (Eberstein and Eberstein, 1996). The affected limb should be protected against further injury, and joints can be kept flexible with passive flexion and extension several times a day. The use of electrical stimulation of affected muscles may prevent denervation atrophy (Hollis et al, 2008). For type 1 injuries (where there is no disruption to the axon), clinical signs should resolve within several weeks. Regrowth by axonal sprouting from the proximal stump of a severed nerve should occur at the rate of about 2.5 cm per month (MacKay, 2005).

Metabolic causes

A variety of metabolic derangements can result in neurological dysfunction, which can be reversed by correcting the imbalance in many cases. It should be noted that, since these abnormalities typically occur secondary to other disease processes, the isolated metabolic derangement may not be solely responsible for the exhibited clinical signs.

Hypoglycaemia

Derangements in glucose levels commonly occur in critically ill foals (Johnson et al, 2012). In a foal demonstrating neurological signs with a glucose concentration <2.2 mmol/L, hypoglycaemia may be responsible (Costa et al, 2020). Clinical signs include muscle twitching, disorientation, collapse and seizures (Trefz et al, 2016), although it should be noted that a study of severely hypoglycaemic calves demonstrated that neurological signs occurred in just 10% of individuals (Johnson, 2020). The brain is unable to synthesise glucose and relies on a continuous supply. Therefore, severe hypoglycaemia is associated with a shortage of glucose delivery to the brain; a state called neuroglycopenia, which is characterised by alterations in activity on an electroencephalogram (Johnson et al, 2012). Short-term hypoglycaemia is not believed to cause permanent injury and clinical signs can be rapidly reversed by supplementing dextrose (Johnson, 2020). However, persistent severe hypoglycaemia is associated with neuronal degeneration. It was previously believed that this was caused by neuronal energy depletion, but it is now understood to be a form of cerebral excitotoxic neuropathology, whereby neurons are destroyed by excitatory amino acids (MacKay, 2005). Therefore, hypoglycaemic foals should receive glucose supplementation without delay.

Hyponatraemia

Disorders in serum sodium levels are fairly common in critically ill foals, with hyponatraemia occurring more commonly than hypernatraemia. The cause of hyponatraemia is most commonly enterocolitis, although it is also seen with uroperitoneum, renal disease and sepsis (Lakritz et al, 1992). Clinical signs include depression, abnormal gait, blindness and seizures (Johnson et al, 2012), although these typically occur when the derangement is severe (<120 mEq/L) or occurs acutely (Johnson et al, 2012). The reduced sodium concentration changes the plasma osmolality, resulting in dramatic shifts of fluids into the CNS tissues and subsequent cerebral oedema (Verbalis et al, 2013).

Treatment requires correction of the deficit. Since there is a free water excess, correction requires the administration of fluids that are relatively hypertonic to plasma. Rapid correction of chronic hyponatraemia can result in osmotic demyelination, so treatment should be considered carefully. Human guidelines, which have been extrapolated for foals, suggest limiting the increase in plasma sodium to <0.5–1.0 mmol/L/h or <10 mmol/L/day (Dunkel et al, 2020).

Prognosis is typically considered to be related to the underlying disease, although the presence of neurological signs in hyponatraemic foals has been associated with death (Beyer et al, 1997). Hypernatraemia can also occur, although it is often iatrogenic in nature. This can also result in osmotic demyelination and should be corrected gradually.

Hypocalcaemia

When considering calcium levels, it should be remembered that ionised calcium is the biologically active form and the form that should be assessed. Tetany and seizures have been associated with hypocalcaemia (Johnson et al, 2012), although clinical signs are typically only seen in foals with ionised calcium levels less than 0.7 mmol/L and, again, the rapidity of the decrease is often more important than the value (Schwarz and van den Hoven, 2012). A syndrome of hypocalcaemic seizures has been described in foals aged 2–5 weeks and is thought to be caused by a primary hypoparathyroidism (Johnson et al, 2012).

Hyperbilirubinaemia

Bilirubin is capable of crossing the blood–brain barrier, especially in neonates where the barrier is more permeable, and when the diffusion gradient is high (bilirubin concentration exceeding 428–513 µmol/L) (Loynachanet al, 2007). Once in the CNS, bilirubin is neurotoxic and in rare cases can cause the neurological syndrome 'kernicterus', characterised by severe icterus and seizures (Broux et al, 2015). The most common cause of hyperbilirubinaemia in neonates is neonatal isoerythrolysis, with severe anaemia often being seen. Finding and McSloy (2011) offers a review of this condition.

The damage hyperbilirubinaemia causes to the brain is irreversible and as such, the prognosis is poor. The definitive diagnosis is histological and post-mortem only. The only treatment to address hyperbilirubinaemia is therapeutic plasma exchange (Dunkel et al, 2011; Johnson et al, 2012). However, this is rarely performed. Since the damage caused by bilirubin is irreversible, therapeutic plasma exchange should be performed before signs of kernicterus develop.

Hyperammonaemia

Hyperammonaemia may occur secondary to hepatic insufficiency, portosystemic shunts or with increased gastrointestinal production (McConnico et al, 1997). Clinical signs include depression, circling, ataxia, head pressing and recumbency. Further abnormalities should be expected, such as increased hepatic enzymes in cases of hepatic insufficiency, and diarrhoea or colic in cases of gastrointestinal disease. A genetic defect in the urea cycle has been reported in Morgan foals (MacKay, 2005) resulting in 'HHH' syn-

Case example: head trauma resulting in basisphenoid fracture and meningitis

History

A 2-week-old Warmblood filly with a 6-hour history of neurological signs that developed shortly after flipping over backward during handling. Before this incident the foal was reported to be completely normal, with successful passive transfer of maternal immunity and no medications administered. The foal was kept on a large commercial stud. All other animals were reported to be healthy.

Physical examination

- Depressed but responsive. Mild tachycardia (100 beats per minute), otherwise vital parameters were within normal limits
- No blood was noted from the nose or mouth and no cerebrospinal fluid from the ears
- Cranial nerve examination revealed muzzle deviation to the left side, head tilt and ear droop to the right and ptosis of the right eyelid. No nystagmus was noted and the filly appeared to be visual. Ophthalmo-neurologic reflexes were deemed normal
- Grade 2/5 ataxia was noted. No weakness was appreciable
- Panniculus reflex, proprioception, tail tone and anal tone were normal.

Neurolocalisation: right-sided peripheral vestibular syndrome and associated central nerve VII signs

Laboratory investigations

- Mild hyperglycaemia only.

Imaging

- Skull radiographs revealed a basisphenoid fracture with a step misalignment.
- Upper airway endoscopy including guttural pouches; severe bruising within the wall of the right guttural pouch, mostly in the lateral compartment.

Diagnosis: basisphenoid fracture with damage to central nerve VII and VIII

Treatment

- Flunixin meglumine 1.1 mg/kg intravenously twice daily.
- Trimediazine sulfamethoxazole 30 mg/kg by mouth twice daily.
- Box rest.

Case progression

Initially the filly was stable, eating and drinking unimpeded and neurological status was improved.

On the fourth day the filly became quiet with an increased rectal temperature (40°C) and reduced neck flexion. A blood sample was within normal limits except for elevated fibrinogen (5.0 g/L).

Further investigations

- Cerebrospinal fluid analysis demonstrated a markedly increased nucleated cell count and total protein.

Diagnosis: meningitis secondary to skull fracture

Treatment

- Chloramphenicol 50 mg/kg by mouth four times daily and rifampicin 10 mg/kg by mouth twice daily.
- Flunixin meglumine was continued.

Case progression

The filly remained pyrexic for 36 hours and then showed gradual improvement in clinical signs and fibrinogen concentrations.

On day 14 the filly was discharged for continued box rest and antimicrobial treatment for a further 3 weeks.

Outcome

The foal was reported to be normal at follow-up 3 months later, and there were no ongoing concerns.

drome, characterised by hyperornithinaemia, hyperammonaemia and homocitrullinuria, although this is considered extremely rare.

Congenital causes

Congenital abnormalities are uncommon, but should always be considered in the neonate presenting with neurological disease.

Hydrocephalus

Hydrocephalus is a rare condition that results from impaired drainage of CSF, leading to abnormally distended ventricles and increased intracranial pressure, ultimately causing compression of the neural tissue. The inciting cause can either be a congenital malformation of the ventricular system or inflammation, such as that caused by meningitis (Sipma et al, 2013). There may be a genetic component, since it is thought to be more frequent in Friesian horses (MacKay, 2005). Most foals appear normal physically, although doming of the skull may be appreciable. Clinical signs include dullness, head pressing, compulsive walking, seizures and the variable development of menace responses. Involvement of the brain stem is indicated by strabismus or ataxia (Oey et al, 2011). Definitive diagnosis is rarely made in a live foal as it requires CT or MRI (MacKay, 2005). It is universally fatal and is typically definitively diagnosed at postmortem.

Occipitoatlantoaxial malformation

A rare condition that presents with a spectrum of clinical signs, from mild paresis and ataxia to quadriplegia (MacKay, 2005). Some foals have a head tilt and an audible click may oc-

KEY POINTS

- Although neonatal encephalopathy is the most common cause of neurological disease in neonates, there are other causes that should not be forgotten.
- Infectious, traumatic, metabolic and congenital causes should all be considered.
- Metabolic causes are often secondary to other disease processes.
- There is much overlap between clinical signs for different conditions.
- Prompt treatment is generally essential for a successful outcome.

cur with head movement. An inherited form is recognised in Arabian foals but it has also been reported in other breeds (Mayhew et al, 1978). The diagnosis is typically made with radiography, where fusion of the atlas and occiput is seen along with hypoplasia of the dens. Advanced imaging may be useful (MacKay, 2005). Foals are typically euthanased as a result of poor prognosis.

Cerebellar abiotrophy

This is a neurodegenerative condition that affects the cerebellum. Although it can occur in multiple breeds, it is most common in Arabians with an autosomal recessive mode of inheritance (Brault and Penedo, 2011; Brault et al, 2011). The cerebellum initially undergoes full development but then degenerates before, or shortly after birth. Clinical signs may be present at birth or begin within the first few months of life. Signs include ataxia, jerky movement and head tremors. Although these foals are visual, they never develop a menace response (MacKay, 2005). The diagnosis is usually presumptive, although advanced imaging may reveal a small cerebellum. A genetic test does exist and genetic screening has been performed in some Arabian populations (Tarr et al, 2014, Bugno-Poniewierska et al, 2019). Affected foals are typically euthanised and carrier status should be considered when making breeding decisions.

Epilepsy

Seizures can occur with many of the conditions discussed above. However, they can also be idiopathic, in which case they are then referred to as epilepsy. This diagnosis is made following exclusion of known intra- and extracranial causes of seizures. A syndrome of juvenile idiopathic epilepsy has been described in Arabian foals (Aleman et al, 2006) and is characterised by recurrent tonic-clonic seizures that present in the first day to month of life. Unlike many other of the congenital conditions, epilepsy appears to be self-limiting and typically resolves by the time the foal reaches 1–2 years of age (MacKay, 2005; Aleman et al, 2006).

Conclusions

The causes of neurological disease in foals are highly varied, supporting the need for full and thorough history taking and physical examination in every case. The prognosis varies from rapid resolution of clinical signs with correct treatment to hopeless, despite every effort being made. This highlights the importance of identifying the cause wherever possible, so that effective treatment can be quickly initiated or euthanasia performed. The wider availability of CT and MRI will likely aid diagnosis. EQ

Conflicts of interest

The author has no conflicts of interest to declare.

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