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# *Clinical Commentary*

## The Friesian horse breed: A clinical challenge to the equine veterinarian?

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**Keywords:** horse; Friesian; genetic trait; dwarfism; insect bite hypersensitivity; hoof cartilage ossification

### Summary

**The Friesian horse breed is a beautiful breed for showing, riding and driving. However, some clinical problems seem to have a higher incidence in the Friesian breed compared to other breeds and this raises suspicions that these clinical entities may have a genetic basis.**

### Introduction

This article accompanies the report by Viljoen *et al*. (2012) and reviews the clinical entities that seem to occur in Friesian horses more often than in horses of other breeds, and discusses whether the vascular ring anomaly described in the report was 'the chicken or the egg'.

Viljoen *et al*. (2012) expressed the opinion that the primary cause of the megaoesophagus in their 11-year-old Friesian gelding was a vascular ring anomaly and they illustrate this very nicely with *post mortem* macroscopic illustrations. Oesophageal obstruction in Friesian horses, however, is in most cases the result of the development of megaoesophagus (Boerma and Sloet van Oldruitenborgh-Oosterbaan 2008; Van der Kolk *et al*. 2011). The question is 'was the trotting sound heard behind these authors really caused by a zebra, or was it nevertheless a horse'?

### Clinical, possibly genetic disorders in Friesian horses

A variety of clinical problems occur in many breeds but some seem to have a remarkably high incidence in Friesian horses. In The Netherlands, around 7% of the horse population is the Friesian breed and, during the period 1995–2003, 7% of the caseload of the university clinic were Friesians (Van Vliet and Back 2006). When thus overall considerably more than 7% of a disease is prevalent in Friesian horses, suspicion is raised that genetics may play an important role in the prevalence of that particular disease (Van Vliet and Back 2006; Orr *et al*. 2010).

#### *Developmental disorders*

Within the Friesian horse population, dwarfism (**Figs 1** and **2**) has been recognised for many years. However, more detailed information became available in 2008, showing that growth retardation occurs mainly in the limbs (25% shorter than normal) and ribs (Back *et al*. 2008). The bodyweight of dwarfs is about 50% lower than that of aged-matched normal Friesian foals (Back *et al*. 2008). Dwarfs grow after birth, albeit some parts of the body at a slower rate and thus mature dwarfs show a typical phenotype: normal, but relatively larger head conformation, a broader chest with narrowing at the costochondral junction, a disproportionally long back, abnormally short limbs, hyperextension of the fetlocks and narrow long-toed hooves (Back *et al*. 2008).

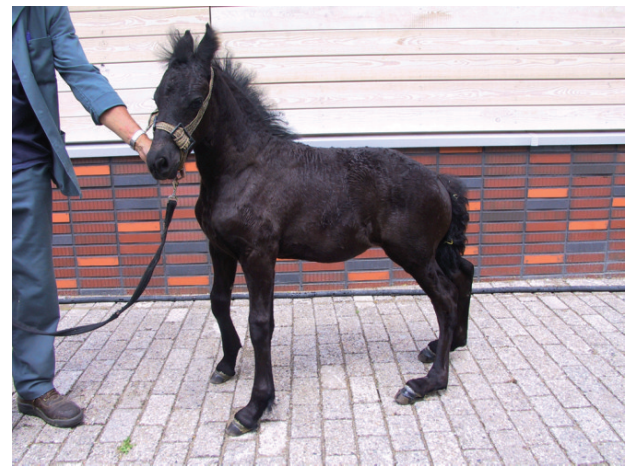

***Fig 1: A 7-week-old Friesian foal with dwarfism.***

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***Fig 2: A 4-year-old Friesian mare with dwarfism and her normal foal with diarrhoea (this picture dates from 1995, nowadays dwarfs are no longer used for breeding).***

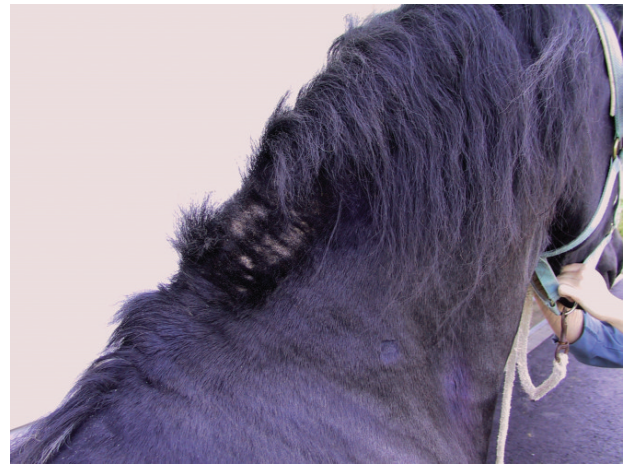

***Fig 4: Severe pruritus lesions as result of insect bite hypersensitivity in a 13-year-old Friesian mare.***

***Fig 3: Hydrocephalus in an aborted fetus (the Friesian mare underwent a partial fetotomy to allow the fetus to be extracted).***

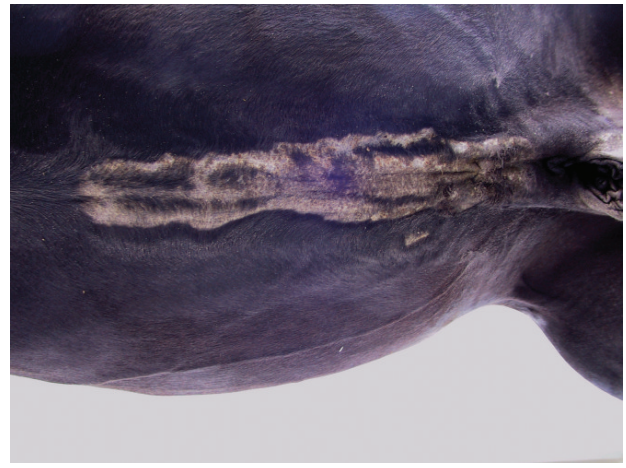

***Fig 5: Severe pruritus lesions as result of insect bite hypersensitivity under the belly in a 2.5-year-old Friesian gelding.***

Hydrocephalus is an uncommon disorder (**Fig 3**) in horses but the Friesian horse has shown a higher incidence than other breeds with an estimated incidence rate of approximately 2.5 affected foals per 1000 births (Sipma *et al*. 2011). A dyschondrodysplasia could be the cause, leading to malformation of the *os petrosum* and thus to a distorted, nonfunctional jugular foramen; this would lead to internal jugular vein compression, disturbing CSF drainage and enhancing its accumulation.

## *Immune-mediated disorders*

Retained placenta is often defined as a failure to expel all fetal membranes within 3 h of delivery. In the general horse population, the incidence of retained placenta is estimated to be 2–10% (Vandeplassche *et al*. 1971; Provencher *et al*. 1988). However, in Friesian mares, the incidence of retained placenta after normal foaling is much higher (54%) (Sevinga *et al*. 2004a). Sevinga and co-authors showed that there are indications that this high rate of retained placenta is at least partly a result of inbreeding (Sevinga *et al*. 2004b).

Insect bite hypersensitivity (**Figs 4** and **5**), involving *type I* and *IV* hypersensitivity reactions, is recognised in The Netherlands and occurs in about 18% of the Friesian horse population (Van Grevenhof *et al*. 2007). This incidence is much higher than in most other breeds, even in the same country. The incidence of insect bite hypersensitivity in The Netherlands in Shetland ponies studied in the same period was about 8% (Van Grevenhof *et al*. 2007). Further studies to elucidate the genetic background have shown that insect bite hypersensitivity is a familial disease with a polygenetic background (Van Grevenhof *et al*. 2007; Schurink *et al*. 2009).

Neonatal isoerythrolysis is an uncommon disease in foals (**Fig 6**), based on maternal alloantibodies related to blood group factors. In 3 Friesian foals, an unusual form of neonatal isoerythrolysis was demonstrated where the mares had haemolytic alloantibodies not attributable to a

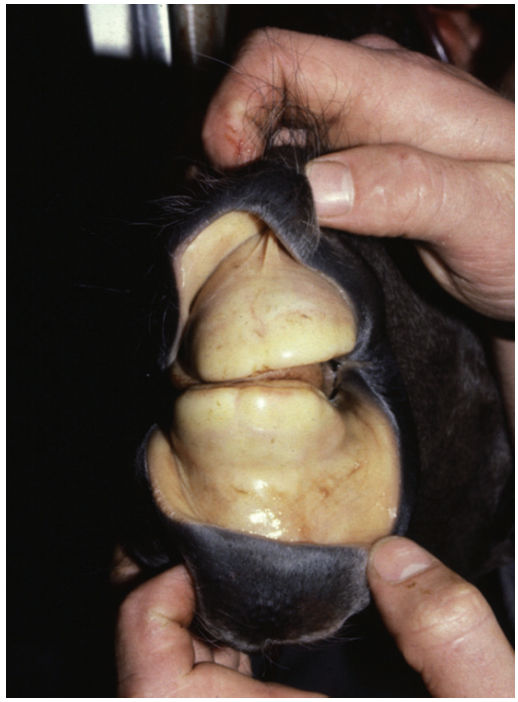

*Fig 6: A 5-day-old Friesian foal showing jaundice as a result of neonatal isoerythrolysis.*

specific antigenic group (De Graaf-Roelfsema *et al*. 2007). Further research is necessary to evaluate whether it was a coincidence that all 3 cases were Friesian foals or whether this is another specific Friesian breed problem.

## *Soft tissue disorders*

Megaoesophagus is a chronic dilatation and atony of the body of the oesophagus. The atony results in accumulation of food and saliva in the dilatated oesophagus. This often results in obstruction/impaction of the oesophagus and subsequent regurgitation (**Figs 7–9**) and, in some cases, aspiration pneumonia (Boerma and Sloet van Oldruitenborgh-Oosterbaan 2008). At endoscopy of the trachea, a dorsoventral compression may occur as result of compression by the very enlarged oesophagus (Gehlen *et al*. 2005). Megaoesophagus is mainly diagnosed in Friesian horses between 1 week and 19 years of age (Boerma and Sloet van Oldruitenborgh-Oosterbaan 2008) and is presumed to be a genetically determined neuromuscular disorder (Van der Kolk *et al*. 2011).

Other abnormalities of the digestive tract seem to occur more in Friesian horses compared to other breeds. Knowles and Mair (2009) described an unusual case of colonic volvulus associated with multiple mesenteric abnormalities. The colt was subjected to euthanasia due to the extent of the intestinal damage and likelihood of recurrence. The authors speculated that the anomalies may have been of genetic aetiology associated with a restricted gene pool.

*Fig 7: Excessive loss of saliva in a 2-year-old Friesian stallion suffering from an oesophageal obstruction as result of a megaoesophagus.*

*Fig 8: Endoscopic view of a megaoesophagus in an adult Friesian horse.*

Verrucous pastern dermatopathy or chronic proliferating lymphangitis (CPL) is a well recognised chronic pastern dermatitis with hyperkeratotic, hyperplastic nodules that may become ulcerated and painful in the course of the disease (**Fig 10**). The entity has several names including 'greasy heel syndrome', 'condylomatous pastern dermatitis', 'granulomatous pastern dermatitis', 'grapes' and 'chronic progressive lymphoedema'. Heavy cold-blooded horses with long feathered fetlocks such as the Belgian, Dutch and German draught horse breeds are overrepresented. In The Netherlands, it is also a very common problem in the Friesian horse. Although a genetic background is suspected, this has not yet been proven in detail (De Cock *et al*. 2009).

Ruptures in the aortic arch near the *ligamentum arteriosum* are uncommon (Van der Linde-Sipman *et al*. 1985; **Fig 11**). Recently, 31 cases were described in Friesian horses, all showing a persistently high heart rate

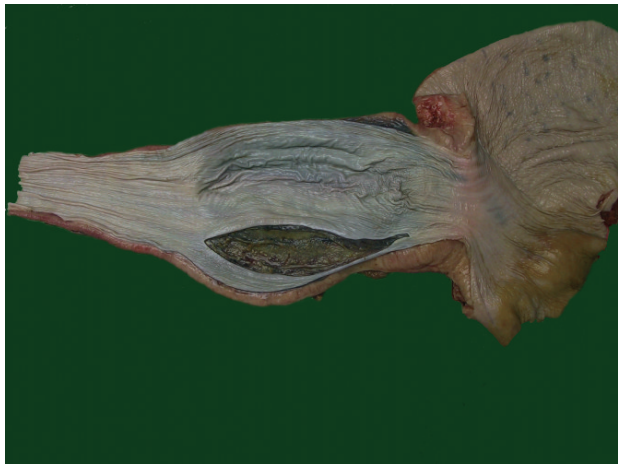

***Fig 9: Megaoesophagus with a longitudinal rupture in a 12-year-old Friesian gelding (courtesy of Veterinary Pathology, Utrecht).***

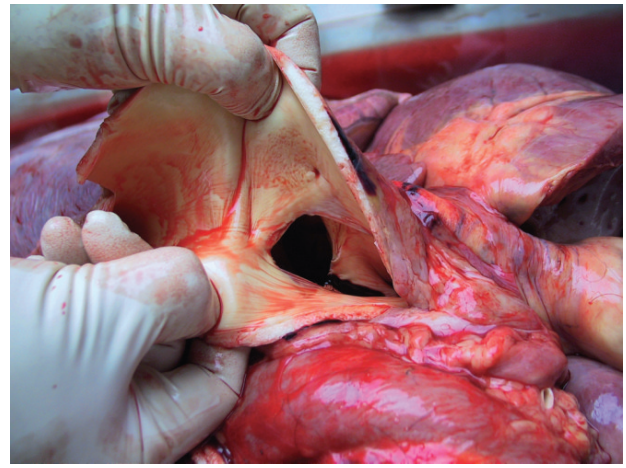

***Fig 11: Aortopulmonary fistula in a Friesian mare age 25 months, seen from the aortic side (courtesy of Veterinary Pathology, Utrecht).***

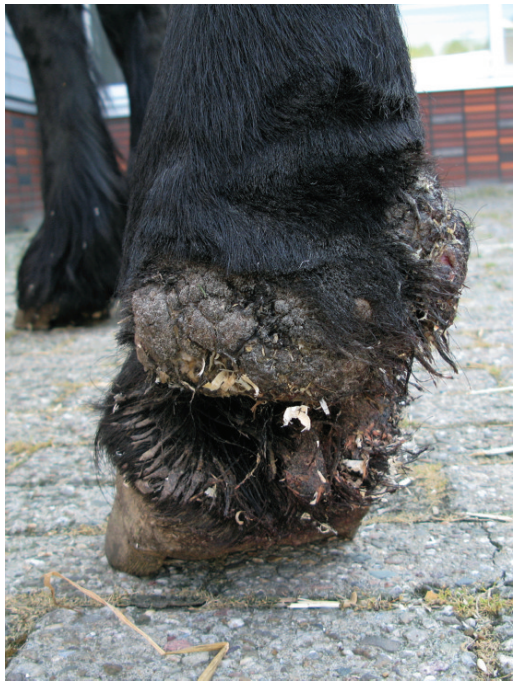

***Fig 10: Severe verrucous pastern dermopathy in a 15-year-old Friesian gelding.***

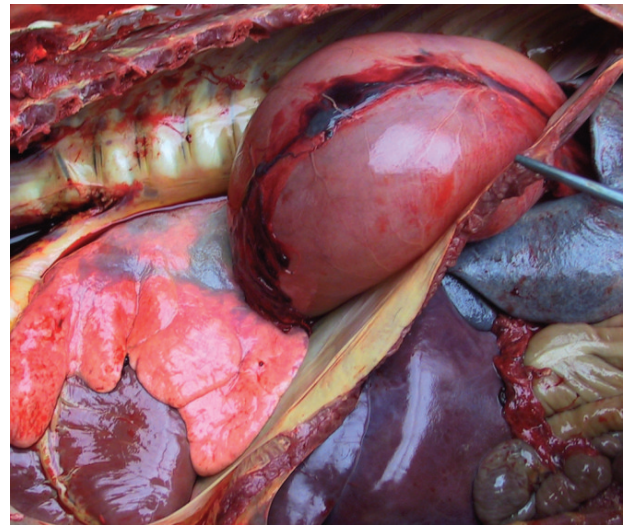

***Fig 12:* Hernia diaphragmatica *in a 1-day-old Friesian foal: the stomach is positioned in the thorax.***

(60–80 beats/min) with a bounding arterial pulse (Ploeg *et al*. 2011). Rupture of the aorta may lead to an aortic-pulmonary fistula causing left to right shunting of blood and/or rupture of the aorta causes a blood tamponade in the mediastinum that may rupture and lead to the immediate death of the patient (**Fig 11**).

Although there is no evidence available, it is the clinical impression that all kinds of hernia and cryptorchids seem to occur more often in Friesian horses (**Figs 12** and **13**) than in other breeds. However, there is only limited literature available, including descriptions of umbilical hernias (Weigand *et al*. 1997; Voermans *et al*. 2004), diaphragmatic, inguinal and ventral abdominal hernias (Hendriks *et al*. 2007).

## *Orthopaedic disorders*

Tendon/ligament laxity has been proven to be different in Friesian horses as there is a significant difference in tendon properties between dwarf Friesians and normal ponies leading to load failure of the stay apparatus (Gussekloo *et al*. 2011). In contrast, normal Friesian horses had properties in between dwarfs and ponies with their tendons appearing more elastic than has previously been reported in Thoroughbreds.

Tendon/ligament laxity can lead to a more extended fetlock position, resulting in a more horizontal position of

*Fig 13: Ventral hernia in an 11-year-old pregnant Friesian mare: she delivered a normal foal 6 days after this picture was taken but had to be subjected to euthanasia 7 days after delivery.*

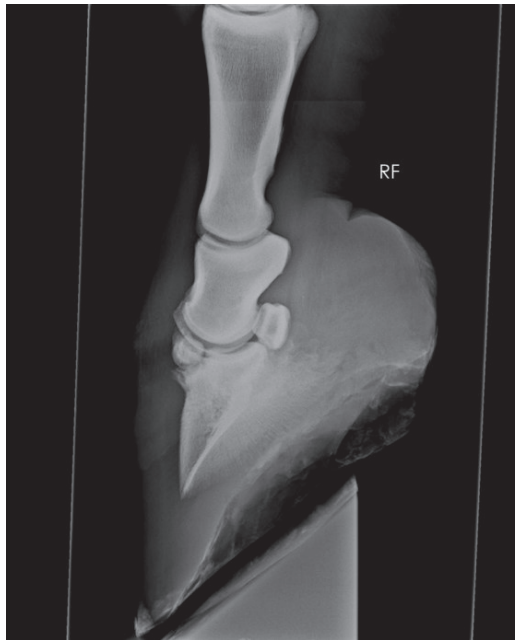


***Fig 14:* Processus extensorius *fracture in a 3-year-old Friesian stallion.***

the pastern bone, thereby facilitating the development of a *processus extensorius* fracture (**Fig 14**) (Viitanen *et al*. 2003) and more collateral ligament laxity, thereby facilitating ossification of the hoof cartilages (**Fig 15**) (Dakin *et al*. 2009). On the other hand, the concurrent more upright foot and convex navicular bone shape might protect Friesian horses from the development of navicular disease (Dik *et al*. 2001).

Last, but not least, hyperextension of the fetlock together with poor hind limb propulsion and hyperrotation of the hind foot may facilitate desmitis of the *ligamentum intersesamoideum* due to interbone ligamental laxity (Voermans *et al*. 2009).

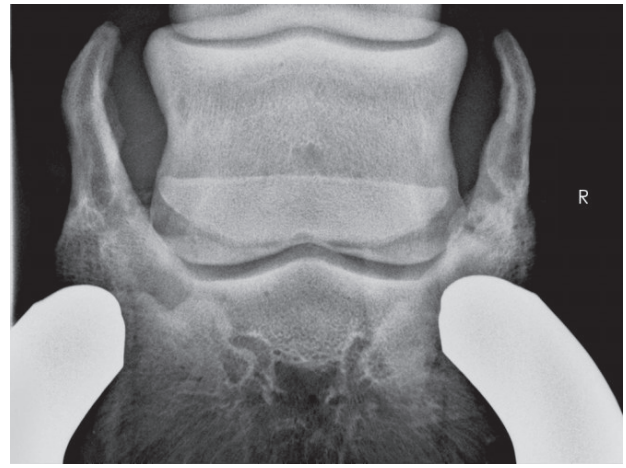


*Fig 15: Excessive ossification of the hoof cartilages in a 6-year-old Friesian mare.*

## Discussion

Many of the problems described above occur mainly or predominantly in the Friesian horse and may be related to the relatively small gene pool of the Friesian horse and/or the high reproduction rate when this breed became very popular in the eighties.

It may be that several of these disorders are related to one common feature which has been intensively selected to obtain the breed specific postural characteristics: a baroque type appearance with a vertical neck and a hyperflexing and hyperelastic 'dancing' locomotor pattern (Halper *et al*. 2006). In particular, the latter fits with the suggestion that many of the problems are collagen-related and a systemic collagen-linked abnormality plays an important role. This supposition is supported by a recent study of tendon properties. It has been shown that there is a significant difference in tendon properties between dwarf Friesians and normal ponies leading to load failure of the stay apparatus (Gussekloo *et al*. 2011); normal Friesian horses had properties in between the dwarfs and ponies and tendons were more elastic than reported in Thoroughbreds. Another indication of a possible abnormality of collagen is demonstrated by the predisposition to rupture of the aortic wall: H&E staining of these lesions revealed significant presence of degeneration, collagen fibre fragmentation, necrosis and inflammation (Ploeg *et al*. 2011). Abnormal collagen formation together with aberrant elastin properties has previously been demonstrated in Belgian draught horses with verrucous pastern dermatitis (De Cock *et al*. 2009).

With regard to the horse described by Viljoen *et al*. (2012), it may be that the trotting sound nevertheless was from a horse and not from a zebra. Previously described vascular ring anomalies in horses have mainly occurred at a young age (Clabough *et al*. 1991; Smith 2004), while signs of a megaoesophagus can be seen in foals or adults up to the age of 19 years (Boerma and Sloet van

Oldruitenborgh-Oosterbaan 2008). Considering the fact that the problem in the case of Viljoen *et al*. (2012) occurred at the age of 11 years, it is possible that the developing megaoesophagus was the primary problem and only when the oesophagus became enlarged did the vascular ring anomaly become evident.

## Authors' declaration of interests

No conflicts of interest have been declared.

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