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Can changes in hoof wall temperature and digital pulse pressure be used to predict laminitis onset?

A Knowledge Summary by

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KNOWLEDGE SUMMARY

PICO question

In horses and ponies at risk of laminitis, does the use of hoof wall temperature and digital pulse pressure as diagnostic techniques for acute laminitis provide a method of detecting acute laminitis in the prodromal stage?

Clinical bottom line

- A palpable bilateral increase in forelimb hoof temperature maintained for longer than half a day may indicate that the horse is 18–24 hours from acute laminitis onset.
- A period of increased digital pulse may also be expected up to 11 hours prior to onset.
- Further studies using larger and more representative cohorts are required to confirm the accuracy of the times at which such changes can be expected.

Clinical scenario

Lameness is regarded as the second most common issue among horse owners in the UK – according to the most recent National Equine Health Survey (Slater & Taylor, 2018) 18.4% of clinical lameness cases are attributed to laminitis. The disease is primarily seen as the result of endocrinopathic disease, with pasture-kept ponies and cases of pituitary pars intermedia dysfunction and equine metabolic syndrome being particularly susceptible. Whilst poorly understood, the pathology of laminitis is characterised by the stretching and distortion of the hoof lamellar epithelium (Patterson-Kane et al., 2018), resulting in an eventual downward movement of the distal phalanx. The clinical outcome is lameness, most often bilaterally of the forelimbs (Rendle, 2006), which can be permanent in the case of chronic mechanical damage. It is widely regarded that the best treatment for laminitis is its prevention and early recognition. However, it is estimated that only half of owners recognise the onset of laminitis (Pollard et al., 2017). Whilst the use of radiography and venogram methods are diagnostic possibilities, it is apparent that in order to reach a decision to deploy such methods, the veterinarian and horse owner must be provided with a reliable noninvasive method(s) of laminitis recognition. This Knowledge Summary will consider hoof wall temperature and palmar digital artery pulse pressure as possible methods.

The evidence

The evidence consists of four experimental case control studies, which document changes in hoof wall temperature and/or digital pulse pressure throughout induced pathological onset. Small sample sizes, nonrepresentational study populations and variable experimental methods challenged comparison between the studies. An epidemiological study is also considered, which investigates the current usage of such changes in the diagnosis of acute laminitis episodes in the UK.

Summary of the evidence

Pollitt & Davies (1998)	
Population:	Mature ex-racing Standardbred horses, aged 2–9 years.
Sample size:	22 horses (17 geldings, five mares).

Intervention details:	• Environment maintained at 10°C to which horses were acclimatised to for 16 hours.
	• Pelleted working horse mix and lucerne chaff fed ad-lib.
	• Two temperature sensors placed in a hole in the dorsal hoof wall of each forelimb, 15 mm below the hairline.
	  ○ Temperature readings taken every 5 minutes.
	  ○ Infrared temperature scanner used to check the implanted sensor readings.
	• Core temperature measured using a thermistor catheter in the right atrium of the heart.
	
	Treatment group (14 horses):
	• Administration of 17.5 g/kg ground wheat slurry in three doses of 8 L every 4 hours via nasogastric tube.
	• Horses which developed colic were given 20 mg I.V. butorphanol and 10 mg I.V. detomidine. Five treated horses were euthanised before 48 hours due to nontreatment-responsive colic and severe fluid imbalance. Data excluded.
	Sham treatment group (seven horses):
	• Administration of three doses of 8 L of room temperature water every 4 hours via nasogastric tube.
	Control group (one horse):
	• No administration.
	
	• Euthanasia and necropsy of remaining nine horses in treatment group and sham treated horses at 48 hours.
	• Laminitis onset defined as weight shifting from one foot to the other.

Study design:	Case-control study.
Outcome studied:	Investigation of changes in sublamellar blood flow following the induction of laminitis by alimentary carbohydrate overload, using hoof temperature as an indicator.
Main findings: (relevant to PICO question):	• Laminitis onset occurred between 32–40 hours after induction, confirmed by histopathology. • 4/7 sham treated horses maintained hoof wall temperature within 5°C of ambient (10°C). 3/7 sham horses and the control horse showed random increases in temperature up to 25°C, lasting 1.4–11.4 hours.

	○ Some showed synchronised changes in both front hooves. ○ Mean hoof wall temperature of treated and sham treated horses (14+7) following acclimatisation was 13.3 ± 0.46°C. • 6/14 horses from the treatment group developed laminitis, all of which showed an increase in hoof wall temperature between 4 and 15 hours following carbohydrate administration. ○ In all but one horse, this increase was sharp to 25–32°C. ○ Mean hoof temperature became significantly higher than the sham and control groups at 16 hours. ○ At 40 hours, mean hoof temperature was 25.62 ± 2.15°C (mean ± s.e.) and then sharply decreased. • Core body temperature of horses in the treatment group was significantly higher in laminitis-positive horses at 36 hours following administration compared to laminitis-negative horses (40.17 ± 0.27°C vs 39.26 ± 0.18°C). • 2/6 laminitis-positive and 4/8 laminitis-negative horses (treatment group) developed colic. ○ Laminitis-negative subjects which developed colic received treatment. This was noted to decrease core body temperature for 2–4 hours. No consistent change in hoof temperature resulted. • Most treated horses (number not specified) developed increased digital pulse pressure at 24–32 hours, which then usually disappeared. • 1/6 laminitis-positive horses showed an increased pulse at 48 hours.
Limitations:	• Population not representative of the typical laminitic population (low body condition score (BCS), normal insulin sensitivity, healthy, young, no evidence of endocrine disease). • Long induction period of 12 hours; measurements taken from time of administration are ambiguous with regard to pathological onset. • Treated horses all developed diarrhoea, and 2/6 laminitis-positive horses developed colic. May have accounted for increased core temperature, possibly confounding hoof temperature changes due to laminitis. • Digital pulse changes were not specified as to how many horses in which they occurred, whether they correlated with hoof temperature changes, or their exact time of onset. • Cold-induced vasoconstrictive background may mask changes in lamellar blood flow due to laminitis. • Endotoxaemia may have been a contributing factor to onset due to the induction method. • Assumes that hoof wall temperature changes are caused only by changes in lamellar blood flow. • Only front hooves measured. • Small sample size.

Adair III et al. (2000)	
Population:	Healthy 450–500 kg horses, mean age 8.13 ± 1.29 years. No clinical history or physical/radiographic evidence of laminitis prior to study.
Sample size:	10 horses (minus one horse which failed to develop laminitis and was excluded from the data set).
Intervention details:	• All horses first received 5 L deionised water via nasogastric tube to obtain baseline measurements over 12 hours. • Administration of 5 L black walnut extract via nasogastric tube to all horses. • All horses received 4 mg/kg I.V. phenylbutazone throughout the experiment. • Laser Doppler probe inserted into four 8 mm holes drilled to the junction of the sensitive and insensitive laminae in one forelimb hoof. • Maintained at ambient temperature of 20 ± 1°C. • Offered ad lib water and grass hay, with sweet feed 0.005 kg/kg of body weight per day. • Laminar blood flow measured hourly using laser Doppler flowmetry. • Changes in digital pulse quality and hoof temperature were assessed hourly by palpation. • Laminitis onset defined as lameness of at least Obel Grade I. ○ Clinical signs assessed hourly (weight shifting, digital pulse quality, hoof temperature and ability to lift the limb). • Euthanasia if Obel Grade III lameness maintained for over 72 hours, or at onset of Obel Grade IV lameness. At 12 hours or following onset of Obel Grade III laminitis: • Five horses received administration of 4 mg/kg I.V. phenylbutazone and 15 mg of topical glyceryl trinitrate over the digital arteries. • Five horses received administration of 4 mg/kg I.V. phenylbutazone. • Not relevant to PICO – see second outcome studied.
Study design:	Case-control study.
Outcome studied:	1. To measure changes in laminar microvascular blood flow of the front hooves throughout the prodromal stage of black walnut induced laminitis. 2. To determine the effects of glyceryl trinitrate application on laminar blood flow during this period. • As this is not relevant to the PICO, results from this outcome will not be included in Main Findings.
Main findings: (relevant to PICO question):	• Clinical signs of laminitis developed 8 to 12 hours after administration of black walnut extract (mean ± SEM 10.22 ± 0.43 hours).

	• Hoof wall temperature:
	  ○ 9/18 hooves showed a palpable decrease 5 hours after administration.
	  ○ All hooves showed a palpable increase 6–12 hours after administration.
	  ○ 5/18 hooves showed a palpable decrease 13–17 hours after administration.
	• Digital pulse pressure:
	  ○ 3/18 hooves showed a palpable increase 1–¬3 hours after administration, increasing to 16/18 at 7–¬12 hours.
	  ○ 14/18 hooves showed a palpable decrease 13–23 hours after administration.

Limitations:	• Endotoxin induction potentially nonrepresentational of the endocrinopathic case. • No specific documentation of individual hooves to allow correlation of surface temperature and pulse quality changes. • Subjective measures of pulse pressure and hoof wall temperature used, with no indication of consistency in personnel used to make the measurements. • No blinding of the personnel evaluating pulse quality and hoof wall temperature indicated. • Changes recorded around point of onset of laminitis, not accounting for individual variation. • Study was primarily concerned with recording changes in laminar blood flow and not with changes in hoof wall surface temperature/digital pulse pressure ¬therefore limited data are available. • Only front hooves measured. • Small sample size.

Hood et al. (2001)	
Population:	Healthy adult horses with no evidence or recent history of lameness.
Sample size:	30 horses split across three separate experiments.

Intervention details:	1. Measurement of hoof wall temperature changes in hot versus cold-acclimatised horses (n=12) (not relevant to PICO).
	2. Measurement of hoof wall temperature before and after tourniquet application (n=6) (not relevant to PICO).
	3. Measurement of hoof wall temperature before and during the prodromal and acute phases of carbohydrate-induced laminitis (n=12).
	  • Ambient temperature controlled at 19 ± 1°C with at least 3 hours acclimatisation.
	  • Hoof wall temperature measured every 4 hours using thermistors on the dorsal surface of the hoof wall one third of the distance from the coronet to the ground. Only front hooves were measured.

	• Laminitis induced by administration of a high carbohydrate diet (starch + wood flour gruel). • Assessment of lameness made every 4 hours – laminitis onset was determined by an unwillingness to walk from rubber mat to concrete.
Study design:	Case study.
Outcome studied:	1. To investigate hoof wall surface temperature variations as an indicator of digital perfusion throughout the onset of carbohydrate induced acute laminitis. 2. To investigate hoof wall surface temperature during vascular occlusion of the forefeet. • As this is not relevant to the PICO, results from this outcome will not be included in Main Findings. 3. To investigate hoof wall surface temperature during cold-challenge of hot-acclimatised horses. • As this is not relevant to the PICO, results from this outcome will not be included in Main Findings.
Main findings: (relevant to PICO question):	• Lameness (positive laminitis result) occurred an average of 10 ± 3 hours following onset of digital hypothermia, and 33 ± 7.46 hours post-administration. • Significant decrease in hoof wall temperature seen 8–12 hours post-administration. ○ Only significant when normalised to lameness onset time. ○ 32.31 ± 1.32°C (mean ± s.d.) control temperature before administration. ○ 29.71 ± 3.85°C 12 hours prior to lameness onset. ○ 30.02 ± 4.3°C 8 hours prior to lameness onset. • Hoof temperature not significantly different to pre-administration at lameness onset.
Limitations:	• Digital pulse pressure not measured. • Lameness was not scored on a standardised scale, making cross-comparison with other studies difficult. • High ambient temperature may have masked increases in temperature due to pathological vasodilatation. • Core body temperature was not measured or accounted for. • No control group. • No attempt to measure hindlimb hooves was made. • Small sample size.

De Laat et al. (2010)	
Population:	Clinically normal ex-racing Standardbred horses. Mean age 5.4 ± 1.95 years. Light-moderate body condition score 2.5–4.5/9.
Sample size:	Eight horses (seven geldings, one filly).

Intervention details:	Treatment group (four horses):
	• Administration of insulin and glucose infusions to create hyperinsulinemia and euglycaemia.
	  ○ Initial insulin bolus administration (45 mIU/kg BWt).
	  ○ Insulin infusion (6 mIU/kg BWt/min) with 50% glucose solution infusion (10 µmol/kg BWt/min) for remaining duration of study.
	  ○ Alteration of rate of glucose infusion as required to maintain euglycaemia.
	Control group (four horses):
	• Received constant infusion of a balanced electrolyte solution (0.57 ml/kg BWt/h).
	• Ambient temperature maintained at 15.9 ± 0.4°C with a 24 hour acclimatisation period before commencing interventions.
	• Fed ad lib lucerne chaff, hay and fresh water 72 hours prior to and throughout the study.
	• Hoof wall surface temperature recorded continuously using noninvasive thermistor probes on the dorsal hoof wall of both forelimbs, 25 mm below the hairline.
	  ○ Recorded as hoof wall surface temperature – ambient temperature.
	  a) Pulse pressure manually palpated.
	  b) Laminitis onset distinguished as onset of Obel Grade II lameness.
	  ○ Euthanasia and necropsy following onset.

Study design:	Case-control study.
Outcome studied:	1. Investigation of hyperinsulinemia as a cause of laminitis. 2. Investigation of the association between front hoof wall temperature, vascular activity and laminitis development.

Main findings: (relevant to PICO question):	• All treated horses developed Obel Grade II laminitis within (mean ± s.e.) 46 ± 2.3 hours.
	  ○ Palpably increased digital pulses, restlessness and shifting of the hooves seen at 31.5 ± 4.65 hours.
	  ○ Consistent shifting of weight and turning to look at the hind hooves seen at 40.5 ± 3.87 hours.
	• No significant changes in digital pulse seen in control horses.
	• Following achievement of hyperinsulinemia hoof wall surface temperature minus ambient temperature was significantly higher from 11 hours onwards and less variable in treated horses than in controls.
	  ○ Approximately 11–13.5°C (treated) vs 4.5–¬9°C (control).
	  ○ Coefficient of variation 4% (treated) vs 42% (control).
	  ○ No concordant significant changes in body temperature seen.

	• No evidence of distal phalanx (PIII) movement relative to the dorsal hoof wall seen in any horse (i.e. chronic onset did not occur).
Limitations:	• Population not representative of the typical laminitic population (low BCS, normal insulin sensitivity, healthy, young, no evidence of endocrine disease). • Insulin concentrations induced were higher than typically seen in the endocrinopathic case. ○ May not be pathologically representative of natural laminitis onset – assumes it is entirely insulin induced. • No reproducible method of digital pulse sensing. No details as to whether there was consistency in the personnel used to make measurements. • Changes recorded around point of onset of laminitis, not accounting for individual variation. • Only front hooves measured. • Small sample size.

Pollard et al. (2018)	
Population:	Horses and ponies in England, Scotland and Wales.
Sample size:	1070 horses.
Intervention details:	• Online laminitis reporting form completed by self-enrolled horse owners between August 2016 and December 2016 for all active laminitis cases. ○ Active laminitis cases were defined as veterinary diagnosed and/or owner-recognised. • Clinical signs observed during active episodes recorded. • Incidence estimated using first and repeat episodes reported during the study period.
Study design:	Cohort study.
Outcome studied:	To investigate owner-reported incidence and clinical signs of laminitis in horses and ponies in the UK.
Main findings: (relevant to PICO question):	• A total of 123 active laminitis episodes were reported, affecting 97 animals. • Most prevalent owner-reported clinical sign of laminitis was lameness (associated with a short, stilted walk (73.5%, 83/113 cases), lame walk (71.1%, 81/114 cases) and difficulty turning (80.4%, 90/112 cases)). • Laminitis was most common in both forelimbs (62.9%; 73/116 cases). • Mean age of reported cases was 14.7 years (± 6.9 years). • Previous laminitis history reported in 39.1% of cases. • 99.1% (115/116) of cases were assessed by their owner for increased hoof temperature.

	○ 57.8% (67/115) of owners of assessed cases reported increased hoof temperature as an indicator of laminitis. • 88.8% (103/116) of cases were assessed by their owner for a bounding digital pulse. ○ 50.9% (59/103) of owners of assessed cases reported a bounding digital pulse as an indicator of laminitis. • 31.9% (37/111) of owners reported an overweight body condition as an indicator of laminitis (only 1.7% (2/111) of cases were described as underweight).
Limitations:	• No detail regarding the timing/timespan of changes seen in hoof temperature or digital pulse in relation to other clinical signs. • No standardised/proven methods of monitoring were outlined. ○ Digital pulse can be challenging to monitor¬ no indication of degree of success given. • Subjectivity in owner reporting.

Appraisal, application and reflection

Laminitis can be subcategorised based on its causative factors. Endocrinopathic laminitis accounts for 90% of cases (Patterson-Kane et al., 2018) in which onset is linked with insulin dysregulation as the result of equine metabolic syndrome, pituitary pars intermedia dysfunction or glucocorticoid administration. Sepsis-associated laminitis makes up a smaller percentage of cases in which a systemic inflammatory state is achieved through enterocolitis or toxin absorption. Supporting limb laminitis is occasionally seen in lame horses (Wylie et al., 2015). In all cases, laminitis progression can be considered in several phases. The 'prodromal' phase is described as the first 72 hours prior to the onset of clinical lameness, which marks the start of the 'acute' phase (Hood et al., 1993; and Rendle, 2006). Up to 72 hours following acute onset, laminitis can either enter the 'subacute' phase and resolve upon removal of the inducing factor, or it can enter the 'chronic' phase, whereby irreversible mechanical detachment and pedal bone rotation occurs (Rendle, 2006). In sepsis-associated laminitis, these stages have been shown to be characterised by changes in lamellar blood flow and vascular resistance (Baron, 2002; and Adair III et al., 2000). Ischaemia of the digit is seen in the prodromal phase, followed by a reactive hyperaemia upon reperfusion, constituting the acute phase (Bailey et al., 2004). These changes are also suspected to underlie the endocrinopathic case, but they have yet to be fully evaluated.The arteriovenous anastomoses supplied by the digital arteries are involved in such changes and in regulation of hoof temperature (Pollitt & Davies, 1998), with their dilation resulting in a warming of the hoof (Pollitt & Davies, 1998). By consideration of these circulatory changes, palpation of hoof wall temperature and of the lateral digital arterial pulse could provide the clinician or horse owner with an indication of the stage of laminitis onset.

The pathological presentation of laminitis appears to vary depending on the subtype, with endocrinopathic cases recently shown to display epithelial stretching as opposed to the epithelial detachment from the basement membrane demonstrated in sepsis-associated laminitis (Patterson-Kane et al., 2018). Differing pathological processes may affect the measurable changes in hoof circulation and so it is important to consider the methods by which laminitis was induced in the studies considered. The administration of toxin extract and carbohydrate overload can be regarded to cause sepsis-associated laminitis, differing from the endocrinopathic case in which the horse shows insulin dysregulation. However, whilst the increased intestinal fermentation caused by a carbohydrate overload likely contributes a sepsis-associated release of bacterial compounds, Tóth et al. (2009) also showed that carbohydrate overload reduces insulin sensitivity. Hence it is possible that such a model bears some representability of the endocrinopathic case. Given the limited availability of endocrinopathic laminitis studies, it is necessary to assume that any such pathological differences are of insignificant impact in the context of forming a diagnostic framework based upon palpable changes.

Hoof Wall Temperature

Pollitt & Davies (1998) administered a carbohydrate gruel to a group of 14 horses, of which six developed laminitis 32–40 hours later. This was preceded at 4–15 hours by a sharply attained maximum hoof temperature in both front hooves of each horse, distinguishing the laminitis-positive from the laminitis-negative horses. Mean hoof temperature remained higher than the control horses from 16 hours onwards. Variability in hoof temperature was seen in sham treated and control horses, with some showing little fluctuation about the ambient temperature and others displaying random increases in both or either front hooves, lasting between 1.4 and 11.4 hours. All changes were independent of ambient temperature, but core body temperature was significantly higher in laminitis-positive horses from 36 hours, around the time of onset. This was likely to be due to endotoxaemia and so not reflective of digital blood flow.

Hood et al. (2001) also looked at the hoof wall surface temperature following the administration of a carbohydrate gruel. No statistically significant changes to hoof wall temperature were observed prior to the onset of lameness, but when the data were normalised to the point at which lameness became evident, a decrease between 8 and 12 hours prior to the onset of lameness was seen. No significant difference in temperature was recorded at this point.

Perhaps the discrepancy seen in the results of these carbohydrate overload studies is attributable to the ambient temperature; the Pollitt & Davies (1998) study maintained an ambient temperature of 10°C and the Hood et al. (2001) study maintained 19°C. Thermoneutrality in the Pollitt & Davies (1998) study was not indicated, and several of the horses were noted to shiver, with control horses maintaining hoof temperatures near ambient at 13.3 ± 0.46°C. Alternatively, this shivering may have been a consequence of endotoxaemia. In the Hood et al. (2001) study, subjects maintained hoof temperature above ambient at 32.31 ± 1.32°C. It is therefore possible that increases in hoof temperature due to pathological vasodilation were undetectable in the Hood et al. (2001) study due to thermostatic vasodilation. Likewise, the vasoconstrictive background seen in the Pollitt & Davies (1998) study may have masked a pathological decrease in temperature.

De Laat et al. (2010) induced hyperinsulinemia in Standardbred horses via a continuous intravenous administration of insulin and glucose, with consequent acute laminitis. Approximately 35 hours before onset there was a significant increase in hoof wall temperature independent of ambient and core body temperature. Hoof temperature was less variable than seen in the controls, as noted in the Pollitt & Davies (1998) study.

A study by Adair III et al. (2000) instead mimicked endotoxaemia induced laminitis via the administration of black walnut extract. Palpation was used as a measure of hoof wall temperature as opposed to thermistors, and yet a notable cooling was observed within 5 hours of induction in half of the tested hooves, followed by an increase between 6 and 12 hours in all hooves. At this point, lameness of at least Obel Grade I was seen. These changes were concordant with those proposed by the vascular theory (Hood et al., 1993) and supported by capillary perfusion measurements via Doppler sonography. However, the rate of onset of acute laminitis seen in this study is significantly faster than seen in the carbohydrate/hyperinsulinemia induction studies considered. Given its overall resemblance to sepsis associated laminitis, the question is raised as to the relevance of the data with regard to the typical endocrinopathic case.

Digital Pulse Pressure

In the normal horse, the digital arterial pulse is faint or absent. In acute laminitis it often presents as a 'bounding' pulse (Pollard et al., 2018), a universally recognised diagnostic criterion of acute laminitis. De Laat et al. (2010) described a significant increase in palpated digital pulse pressure which coincided with the onset of weight shifting. This is a feature of Obel Grade I lameness, an early clinical sign of acute laminitis. This shifting began at 31.5 ± 4.65 hours following commencement of the euglycemic hyperinsulinemic clamp, becoming more

consistent at 40.5 ± 3.87. Therefore it can be approximately calculated that up to 9 hours may have separated the observable increase in digital pulse pressure and the appearance of consistent signs of acute laminitis. Pollitt & Davies (1998) also described an increase in digital pulse pressure between 24 and 32 hours after induction in laminitis-positive horses, which disappeared before the increase in hoof wall temperature in all but one horse. Taking the onset of laminitis to be 32–40 hours post-carbohydrate administration, this increase in digital pulse pressure can be said to have occurred approximately 8 hours before onset (calculated using the midpoints of the 8 hour reporting intervals).

The Adair III et al. (2000) study showed a palpable increase in pressure 1–3 hours post-induction in 17% (3/18) of front hooves, increasing to 89% (16/18) at 7–12 hours post-induction. These changes were not always concordant for both limbs. 78% (14/18) of front hooves then showed a decrease 13–23 hours after black walnut extract administration.

Whilst the presence of indicators seen at different timepoints during laminitis onset was not examined by Pollard et al. (2018), a bounding pulse alongside bilateral lameness was reported as an indicator of active laminitis in 57.3% (59/103) of cases in which it was assessed. This is consistent with previous data which found 91% of veterinary diagnosed acute and chronic cases presented with an increased digital pulse (Wylie et al., 2016). It contrasts with the data provided by Pollitt & Davies (1998) and Adair III et al. (2000), which indicates a decrease in digital pulse pressure from the onset of lameness. A second increase or a difference in pathogenesis between experimentally induced and 'naturally occurring' laminitis is therefore indicated.

The measure of pulse intensity is subjective due to a lack of available instrumentation. The studies do not detail whether the same person was used in making such measurements, and so the reliability of change detection cannot be commented upon.

Conclusions

To allow cross-comparison between the studies, the time taken between induction and laminitis onset must be consistent. One of the hallmarks of Obel Grade I lameness is foot lifting at rest (Vinuela-Fernandez et al., 2011) which is consistent with the onset described by Pollitt & Davies (1998), Adair III et al. (2000) and De Laat et al. (2010). Laminitis onset in the De Laat et al. (2010) study was defined as Obel Grade II, but data was also provided at the onset of weight shifting, corresponding with Obel Grade I lameness. Despite Hood et al.'s (2001) lack of use of a specified scale, it is likely that the described lameness markers also fall under the same subjective categorisation. For this reason, it could be considered that the points of onset are comparable. However, the method of induction remains a point of discrepancy; Pollitt & Davies (1998) used wheat slurry administrations over 12 hours, whereas Hood et al. (2001) used a single administration (Garner et al., 1975). The De Laat et al. (2010) study used the continuous I.V. infusion of insulin, to mimic insulin dysregulation. Adair III et al. (2000) used a single dose of black walnut extract. There is no defined speed of induction of acute laminitis therefore consideration of changes around the time of common onset may be more appropriate, and would account for individual variation.

The studies considered report an increase in hoof wall temperature of between approximately 5°C (De Laat et al., 2010, estimated from graphed data) and 12.32°C (Pollitt & Davies, 1998). This is the difference in hoof wall temperature between the times of induction and onset of laminitis, as calculated by the author of the Knowledge Summary. In the De Laat et al. (2010) study, the increase in hoof wall surface temperature became statistically significant compared to controls between 11 and 15 hours post-induction. Taking the mean onset time of Obel Grade I lameness (defined as the appearance of weight shifting) to be 31.5 ± 4.65 hours post-induction, this increase in temperature can be calculated to be approximately 18.5 hours before onset, using the midpoint of the hoof wall temperature measurement interval. In the Pollitt & Davies (1998) study, a statistically significant increase in temperature was seen at 16 hours post-induction. Since Obel Grade I lameness was seen at 32–40 hours, it can be deduced that this was 16–24 hours before onset. Hood et al. (2001) and Adair III et al. (2000) showed a prodromal decrease in hoof temperature. The decrease seen in the Hood et al. (2001) study was from

30.02 ± 4.3°C to 29.71 ± 3.85°C and was significant only when normalised to the onset of lameness. This scale of change is unlikely to be palpably detectable by an owner or veterinarian.

The changes in temperature and digital pulse pressure demonstrated by Adair III et al. (2000) appear to follow the expected pattern of hypoperfusion and hyperaemia as described in the vascular theory (Hood et al., 1993), but given its likeness to sepsis associated laminitis and short onset time (average 36.5 hours (De Laat et al., 2010; Hood et al., 2001; and Pollitt & Davies 1998) versus 10.22 hours (Adair III et al., 2000)), it is uncertain as to whether a comparison to the other case studies and to endocrinopathic laminitis can be made.

In the UK's most recent laminitis reporting study, Pollard et al. (2018) showed that whilst 99.1% (115/116) of horse owners used raised hoof wall surface temperature in their assessment of laminitis, only 58.3% (67/115) reported it as a diagnostic sign. This indicates that it either has poor predictivity or that it is being recorded at an inappropriate time. The reviewed data supports the latter case given the rise in temperature consistently associated with the onset of lameness (De Laat et al., 2010; Adair III et al., 2000; and Pollitt & Davies 1998).

By considering the timepoints associated with changes in hoof wall temperature and digital pulse pressure across these studies, a palpable bilateral increase in forelimb hoof temperature which is maintained for longer than 1.4–11.4 hours may indicate that the horse is 16–24 hours away from acute laminitis onset, accounting for random daily fluctuations. A period of increased digital pulse pressure up to 9 hours prior to onset would also be expected, potentially disappearing transiently at onset of clinically apparent laminitis. However, the reliability of the data studied poses several limitations to these conclusions. As demonstrated by Pollitt & Davies (1998) and Hood et al. (2001), the ambient temperature may account for changes in hoof temperature, limiting its potential as a diagnostic indictor. The regularity by which random daily fluctuations in hoof wall temperature occur also lacks evidence, and so further investigation of such changes in normal horses would be useful. All of the considered studies only measured forelimb hooves, and so whilst laminitis is most commonly seen in the forelimbs (Rendle, 2006), it cannot be determined whether the changes seen in these studies also occurred in the hindlimbs. This would be worth investigating since hindlimb measures could be used to normalise for core/ambient temperature changes if they are pathologically less affected/unaffected. The coupling of hoof temperature measure with arterial digital pulse may also lend a more accurate diagnosis. The cohorts used were small and in the Pollitt & Davies (1998) and De Laat et al. (2010) studies consisted of young, healthy Standardbred horses of low to moderate body condition score and as such, the study populations were highly likely to be insulin sensitive. These are not considered representative of the typical endocrinopathic case, which presents at an average age of 14.7 ± 6.9 years, with only 1.8% (2/111) reported as underweight (Pollard et al., 2018). As such, the models were insulin sensitive – De Laat et al. (2010) noted that insulin resistance may prolong the actual onset time in the endocrinopathic case; a period which is yet to be defined.

Since the clinical patient will not likely be the subject of an induced laminitis, further studies will need to be carried out to investigate whether such changes in digital pulse pressure and hoof wall temperature apply to the true risk population for endocrinopathic laminitis. Continual measurement of hoof wall temperature and digital pulse changes should be made in a reproducible manner by consistent persons or by instrumentation until there is a development of laminitis in a large cohort of horses and ponies. This onset must be determined by common clinical endpoint, such as the onset of Obel Grade I/II lameness. Horses should be categorised depending on the likely causative factor. Such a study may be possible to carry out on a large scale via owner and vet correspondence, but a controlled experimental approach to natural onset endocrinopathic laminitis will likely be impossible. Nonetheless, a larger dataset will allow the use of statistical analyses to either redefine or strengthen the timepoints indicated by the studies considered in this Knowledge Summary.

If the data from the considered studies is to be applied to the clinical laminitis case, the feasibility of using hoof wall temperature and digital pulse pressure to predict acute laminitis onset in a clinically sound horse relies on their consistent measurement by the owner. To maximise the probability of its detection in the prodromal phase, twice-daily documented measures are recommended. The observation of a maintained bilateral increase in forelimb hoof temperature may indicate that the horse is approximately one day from onset, and a period of

increased digital pulse pressure approximately half a day. Given the variability in such events seen in the studies considered, the use of such palpable changes should only form a crude diagnostic indicator of onset. Nonetheless, in at-risk horses and ponies this indication may be sufficient to warrant removal from the inciting factor (i.e. pasture or high carbohydrate intake), encouraging a more favourable clinical outcome.

Methodology Section

Search Strategy	
Databases searched and dates covered:	CAB Abstracts on OVID Platform (1973 – Week 21 2019) PubMed (1973 – Week 21 2019)
Search terms:	CAB abstracts: 1. (laminitis or equine laminitis or acute laminitis or chronic laminitis or founder or laminit*) 2. (pulse or digital pulse or hoof temperature or pulse pressure) 3. 1 and 2 PubMed: 1. ((laminitis) OR (equine laminitis) OR (acute laminitis) OR (chronic laminitis) OR (founder) OR (laminit*)) 2. ((pulse) OR (digital pulse) OR (hoof temperature) OR (pulse pressure)) 3. ((diagnosis) OR (diagnostic) OR (onset) OR (prodromal) OR (developmental)) 4. ((digital artery) OR (circulatory) OR (blood flow)) 5. 1 and 2/3/4
Dates searches performed:	04/06/2019

Exclusion / Inclusion Criteria	
Exclusion:	Articles with content irrelevant to PICO. Articles published before 1994 (older than 25 years). Studies which did not measure either hoof temperature or digital pulse.
Inclusion:	Papers related to equine laminitis diagnosis using noninvasive techniques. Epidemiological studies related to diagnostic measures of laminitis.

Search Outcome				
Database	**Number of results**	**Excluded – before 1994**	**Excluded – irrelevant to PICO**	**Total relevant papers**
CAB Abstracts via the Ovid platform	35	5	29	1
PubMed	7750	333	7412	5
Total relevant papers when duplicates removed				**5**

CONFLICT OF INTEREST

The author declares no conflict of interest.

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