

The musculoskeletal system

14

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14.1

Introduction – Challenges of lameness and gait abnormalities

The musculoskeletal system consists of structures which move the body or maintain its form: muscles, tendons, ligaments, bones and joints.

Lameness/gait abnormalities are perhaps the most common presenting sign to working equine veterinarians. Data from the welfare assessment of 4,903 working equids (Pritchard et al. 2005) suggest that over 99% of animals surveyed show gait abnormalities. Lameness can be very frustrating to treat, especially as many cases are chronic and may have many contributing factors (Broster et al. 2009). Long-term rest is often the most effective treatment but this is usually impractical for the owners of working equids.

Golden Rule 1 Basic knowledge of anatomy is essential for a confident diagnosis.

Golden Rule 2 Think holistically: 'management' rather than 'treatment'.

Golden Rule 3 The direct cause may be difficult to identify.

1. Think about which structures are under the skin in the affected area.

2. Think about what could be happening to those structures and why:

- Acute or chronic?
- Bone, joint, tendon, ligament or muscle?
- Infected or sterile?
- Single or multiple limbs?

3. Link it back to history and clinical examination to make an informed decision.

The lack of radiography, ultrasonography or other 'high-tech' diagnostic aids is no barrier to a thorough work-up. In most cases, lameness examination coupled with a good knowledge of anatomy and a detailed history will help identify the most likely problem (Figure 14.1.1), with prognosis depending on response to management in the first week. When dealing with this system, be aware of the vast amount of terminology: osteomyelitis, tendonitis, septic arthritis, osteoarthritis, synovitis, cellulitis.

In a medical context, the suffix '-itis' almost always implies inflammation of that particular anatomical area.

Remember that infection may or may not be present with inflammation.



Figure 14.1.1 Lameness in working equids is a common presentation. Here the left forelimb is resting on the toe and not fully weight-bearing, indicating pain in that limb.

Lameness examination and diagnostic techniques

14.2

Lameness is any abnormality in the gait. This may be a **mechanical lameness** due to a healed injury, or poor conformation which has affected function, or lameness due to **pain and dysfunction** of the area. It is a sign that the animal is protecting the area from further damage and injury although, in very chronic or subtle lameness, the owner may not notice anything wrong.

Why should a lameness examination be conducted?

The objective of the lameness examination is to identify whether the animal is lame, where the lameness is coming from and the most likely cause.

Classic lameness examinations are described in most textbooks; however, these are often not feasible for working equids and improvisation is frequently necessary. For example, the notion of 'trotting on a lead rope' is difficult if the animal is normally driven from behind and, unless the trotting movement is fluid and consistent, it is difficult to gain any real diagnostic information. Dragging/pulling the animal along restricts its head movement and will make evaluation more difficult. If the animal is unable to trot in hand do not force it.

History

- Exactly what is the problem that concerns the owner?
- When did it start?
- Was there any specific incident that started it?
- Is the lameness getting better, worse or staying the same? (This is often key to prognosis.)

Physical examination

Perform the following examinations in a systematic, consistent way so that nothing is missed. If the significance of a finding is not obvious, compare both sides of the animal (but remember that the problem may be bilateral).

Look at the standing, resting animal.

- **Unequal weight bearing** It is not normal for an equid to point or rest a foreleg (as seen in Figure 14.2.1); however, it may normally 'rest' a hindlimb. Observe any abnormal stance (pointing forelimb, knuckling of fetlock, dropped elbow) or obvious wounds or swellings whilst the animal is still at rest.



Figure 14.2.1 Unequal foreleg weight bearing. The right forelimb is placed forwards reducing weight bearing and indicating a right forelimb lameness.

- **Conformation** Alignment of limbs with body, cow hocks, unequal foot size, etc. Look at the symmetry of the skeleton and evidence of muscle atrophy.

Note the poor hindlimb conformation of severe sickle hocks in the animal in Figure 14.2.2. Think about the implications for work such

conformation could have, and the possibility for predisposition to wounds and lameness in future.

Broster et al. (2009) showed that sickle hock conformation was significantly associated with lameness in working horses. This study also showed that conformation traits such

as broken forward hoof-pastern axis was associated with pain on flexion of the carpal joint in that limb, and upright pasterns were associated with pain on palpation over the digital flexor tendon area and suspensory ligament in that limb (Broster et al. 2009).



Figure 14.2.2 Poor conformation – severe 'sickle hocks' seen in this horse.

Examine the moving animal from a variety of directions.

Do not examine a moving animal if the lameness is severe/non-weight bearing. Do not cause unnecessary pain, fear and distress in a case where the lameness is obviously severe.

Observe from in front (forelimb lameness) Look for a head-nod as the animal walks or trots forwards (Figure 14.2.3, first picture). If the equid is lame on a fore leg, the head will nod downwards when the sound fore leg hits the ground.

Observe from behind (hindlimb lameness) As the equid trots away, look for asymmetrical movement of the pelvis. The hip will raise more or 'hike up' as the lame leg leaves the ground.



Figure 14.2.3 Examining for lameness from in front (far left), behind (left) and the side (above).

Put tape markers over the hip joint or tuber coxa on both sides – the lame side will have greater movement.

Observe from the side The lame leg will have a shorter stride length. In more severe cases, the toe may drag instead of being lifted properly, or 'dig' into the ground at the end of the stride. A shortened stride may indicate a problem with the upper part of the limb; 'high limb lameness', e.g. shoulder.

Move in a circle Lameness is more pronounced when an equid is led in a circle. The inside leg will be subjected to increased twisting and pressure forces that do not occur when walking or trotting in a straight line.



Figure 14.2.4 Observe the animal moving in a circle in both directions.

Close examination

'The foot is the cause of lameness unless proven otherwise.'
(Hodgson 2000 – original quotation unknown)

Foot Always start at the foot and progress upwards, even if there is obvious muscle atrophy or joint swelling further up the leg. Assessing the hoof demonstrates the effect of lameness on the animal's movement, overgrowth and over wear show the current forces on that leg, despite what is often a chronic lameness, and helps to determine prognosis.

- Discharge
- Smell
- Foreign body
- Size and shape Compare with opposite foot, although beware of bilateral problems (Figure 14.2.5).
- Size, fit, quality of shoes and degree of wear (if present) (Figure 14.2.6 – see over)
- Quality of frog, sole and wall horn and any abnormalities like cracks, bruises, infection



Figure 14.2.5 Examination of the solar surface of the foot.

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- Check digital pulse for rate, strength and quality (Figure 14.2.7 – see opposite).
- To detect pain, use the following three techniques in a methodical manner; digital pressure, hoof testers, and a small percussion hammer (Figures 14.2.8 and 14.2.9).



Figure 14.2.6 Examination of the shoe for uneven wear.



Figure 14.2.8 Testing the sole, heels and external hoof wall for pain, using digital pressure, hoof testers and a percussion hammer.





Figure 14.2.7 Palpation of the digital pulse.



Figure 14.2.10 Examine the limb in a methodical manner, e.g. from distal to proximal.

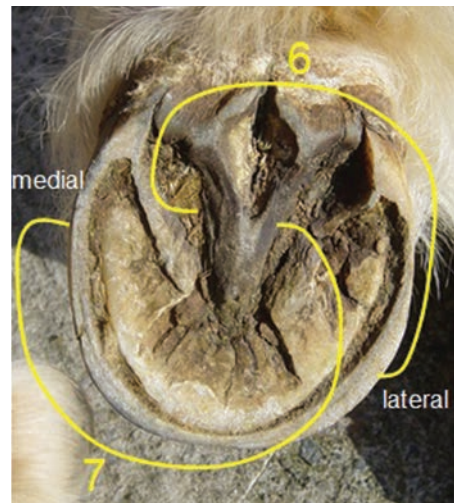
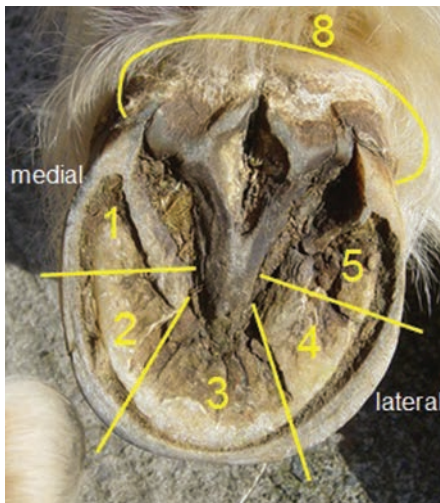


Figure 14.2.9 Systematic examination of the hoof – yellow numbering indicates positioning of the hoof testers across different areas of the foot.

If there are no abnormal findings on examination of the foot then palpate the joints, tendons and muscles of the lame leg for:

- swelling
- pain
- injury.

Tendons Palpate tendons both in a weight-bearing position and off the ground. The latter relaxes the tendons which can allow better examination. Feel for any swelling, heat or pain on gentle palpation. Assess tendon sheaths, particularly the large digital flexor tendon sheath, for swelling (effusion) heat or pain.

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Joints Examine all joints for swelling (effusion) or bony protuberances, both of which can reflect osteoarthritis. If the horse is not in too much pain, flex and extend joints to assess range of motion and/or pain associated with this.

Stop the lameness examination if the animal is feeling excessive pain or showing other signs of distress; analyse the cost to the animal versus the benefit of continuing the diagnosis to a deeper level.

Flexion tests

The purpose of the flexion test is to exaggerate lameness by putting more stress on the joints, ligaments and surrounding structures before asking the animal to move.

Flex the joint for one minute then walk or trot the animal (Figure 14.2.11). Often an animal will be more lame in the affected limb after this, signalling a positive result. The test is not very specific to an individual joint or structure. This is especially true in the hindleg where, due to the reciprocal apparatus, the stifle and hock joints cannot be flexed individually.



Figure 14.2.11 Flexion tests for the lower foreleg, upper foreleg, lower hindleg and full hindleg.

Some would argue flexion tests are of limited use in working equids as many animals have multiple problems and chronic arthritis. Do not attempt flexion tests if the animal shows obvious discomfort as this signals potential arthritis.

Grading of lameness

It is useful to assign a grade to the severity of lameness, using a scale. This gives a more accurate documentation of a lameness examination, thereby allowing changes between subsequent examinations to be recorded more objectively.

Overall Lameness Score (based on the American Association of Equine Practitioners (AAEP) Scoring System)

Grade 0 No lameness

Grade 1 Difficult to observe; not consistently apparent regardless of circumstances (e.g. carrying weight, circling, on an incline, hard surface, etc.)

Grade 2 Difficult to observe at a walk or trotting in a straight line; consistently apparent under certain circumstances (e.g., carrying weight, circling, incline, hard surface, etc.)

Grade 3 Consistently observable at a trot under all circumstances

Grade 4 Obvious lameness, marked nodding, hitching or shortened stride

Grade 5 Minimal weight bearing in motion and/or at rest; inability to move



Figure 14.2.12 Grading of lameness helps with re-assessment to see if there has been an improvement.

Nerve blocks (perineural anaesthesia)

This is the use of local anaesthetic to relieve pain so that lameness will be reduced or hidden in a positive result. See Section 7.2 for further details on local analgesia. To pinpoint the site of pain, nerve blocks can be applied in a logical sequence from distal to proximal regions until the equid becomes sound. These blocks can also be used when suturing wounds, or for desensitisation of the hoof for treatment of abscesses or other painful exploratory procedures in the hoof.

Prepare sites for nerve blocks in a sterile fashion (clip and scrub with antiseptic) to avoid excessive swelling and possible infection post-examination.

It is absolutely imperative that veterinarians carrying out nerve blocks know the exact location of the nerve and are aware of the location of adjacent structures to avoid iatrogenic damage/injection into adjacent synovial structures. Accidental injection of synovial structures is likely to result in synovial sepsis that will not only finish the animal's working life but would warrant euthanasia on welfare grounds. It is important to have practised injection of coloured dye into cadaver limbs before injection into a live animal (Figure 14.2.13 – see over); the skin on the

cadaver can be dissected back to reveal if the injection/dye was placed in the correct position.

Palmar/plantar digital nerve block

This block desensitises the palmar aspect (frog and heel areas) of the sole and is very good for **painful procedures** such as paring out a hoof abscess. Always wait at least 15 minutes and test with a pen prick as it can take a long time to work. The common point of needle insertion for this block is just above the most proximal part of the lateral cartilages, close to the edge of the deep flexor tendon where the neurovascular bundle can be palpated (Figure 14.2.14). Inject 5 mls of lignocaine perivascularly.

Take great care and attention to avoid inadvertent injection of the digital flexor tendon sheath which runs close by this injection site.

Abaxial sesamoid nerve block

This is used to desensitise all structures distal to the fetlock joint, and some of the sesamoids, and is also useful when assessing the hoof and distal phalanx joints for lameness or painful conditions over the whole sole (a palmar digital nerve block will not desensitise the toe).

Feel for the nerve bundle over the distal extent of the abaxial sesamoids either side of the fetlock joint (Figure 14.2.15) and infiltrate SC local anaesthetic via a 25G needle into the area (2–5 mls lignocaine).



Figure 14.2.13 Take advantage of practising on cadaver limbs before injecting into a live animal for the first time.



Figure 14.2.14 Site for injection of local anaesthetic for a palmar digital nerve block.



Figure 14.2.15 Site of injection of local anaesthetic for an abaxial sesamoid nerve block.

Hoof anatomy and conformation

14.3

Hoof anatomy

Figures 14.3.1 and 14.3.2 show the following parts of hoof anatomy:

Sole Seen when the foot is lifted (Figure 14.3.1). Sole horn is softer than hoof horn and more prone to injury caused by stepping on stones and other foreign objects. Ideally the sole itself should not make contact with the ground (it is concave); the frog and the wall should be the structures touching the ground in a healthy hoof.

Frog Elastic 'V'-shaped cushion at the back of the sole (Figure 14.3.1). It should be the first part of the foot to hit the ground. The elastic properties allow the foot to expand during weight bearing. It should be symmetrical and seated within deep grooves either side; known as the frog sulci. Poor conformation of the frog may be due to inadequate foot care or inappropriate farriery, such as excessive trimming of the frog.

Heel bulbs These are caudal to the heel where the frog merges with the skin.

White line Junction where the sole meets the wall on the ground surface of the foot. This represents where the sensitive laminae meet the insensitive laminae and is a natural weak spot of the hoof where bacteria can occasionally penetrate.

Bars Parts of the hoof wall that angle forward at the heel either side of the frog sulci. These should be well developed.

Coronary band Junction between hoof and skin. This area is responsible for hoof growth so, if injured, subsequent hoof growth can be affected.

Wall The hard keratinised outside capsule of the hoof (Figures 14.3.1 and 14.3.2) seen when the foot is on the ground. The wall is formed at the coronary band so injuries to this area will affect its structure and growth rate. It takes 9–12 months for the wall to grow from the coronary band to the ground. For this reason, diseases of the wall often take many months to correct; this should be explained to owners when attempting corrective trimming.

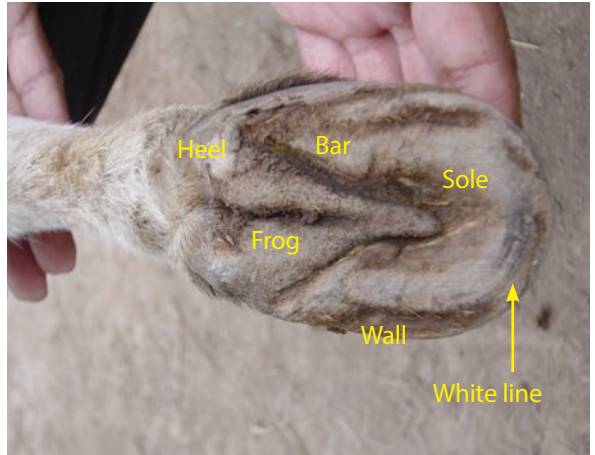


Figure 14.3.1 Solar surface of the forefoot of a working donkey.



Figure 14.3.2 Lateral view of the hoof wall of a working donkey.

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Internal structure of the foot

There are many important structures inside the hoof. A good knowledge of anatomy is important; if a nail penetrates the sole of the foot it is essential to know what might be damaged.

Bones

- P1 First/proximal phalanx or long pastern bone
- P2 Second/middle phalanx or short pastern bone
- P3 Third/distal phalanx or pedal bone
- Distal sesamoid or navicular bone

Joints

- Pastern joint Proximal interphalangeal joint
- Coffin joint Distal interphalangeal joint

Tendons

- SDFT Superficial digital flexor tendon
- DDFT Deep digital flexor tendon

Laminae

- Highly vascular interlocking ridges that hold the hoof wall on to P3

Blood vessels

- Digital plexus and associated veins and arteries which supply the highly vascular laminae

Foot conformation

Foot conformation is absolutely critical as any imbalance will impact the rest of the limb.

Foot axis

The angle of the dorsal hoof wall

- Forelimb = 45–50 degrees
- Hindlimb = 50–55 degrees

Hoof pastern axis

A line drawn through the centre of a metacarpophalangeal (MCP)/metatarsophalangeal (MTP) (fetlock) joint passes straight through the centre of the proximal interphalangeal (pastern) and distal interphalangeal (coffin) joints.

- This should be a straight line with no angulations between each joint.
- It should also be parallel with the hoof axis.
- Feet should be trimmed to keep the hoof pastern axis (HPA) as straight as possible – even if the hoof axis is not at the correct angle as a result.

■ An ideal HPA:

- The HPA is parallel with the dorsal hoof wall and the heels.
- A vertical line from the middle of the fetlock joint should align with the back of the heels.
- A vertical line from the centre of the coffin joint should divide the hoof into two equally sized halves; this allows weight to be carried evenly.
- Figure 14.3.3 shows the HPA in a horse, with almost straight HPA.

A broken forward HPA has been significantly associated with pain on flexion of the carpus of the same limb (Broster et al. 2009) in a study of 224 working horses in India and Pakistan.



Figure 14.3.3 Hoof pastern axis shown by the yellow-dashed line.

Broken back HPA

The angle of the dorsal hoof wall is behind the slope of the pastern (Figure 14.3.4).

This is a very common malformation which results in a number of different pathologies and lameness as a result of the incorrect strains placed on a limb. This foot conformation predisposes to navicular disease.

In a study by Broster et al. (2009) of 224 working horses from India and Pakistan, 81% of forelimbs and 47% of hindlimbs showed this conformation.

- The long toe delays break over.
- The low heel strains flexor tendons.
- Heel bruising
- Navicular disease
- Quarter and heel cracks
- Dorsal sole bruising
- Separation of the toe from the wall
- A broken back HPA should always be corrected even if the animal is currently sound. If left it will lead to pathology and lameness.
- This cannot be corrected in one trimming and requires multiple small trims to improve the conformation.



Figure 14.3.4 A broken back HPA

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- Trim the toe to reduce the toe length, leave the heels as much as possible to allow them to grow.
- If shoeing, ensure heels of shoe are extended to the position they are meant to be – e.g. level with the bulbs of the heel – not where the foot stops.

Broken forward HPA

The angle of the dorsal hoof wall is in front of the slope of the pastern (Figure 14.3.5).

- Short toe
- High heel
- Places strain on the deep digital flexor tendon, proximal suspensory ligament, navicular bone and navicular bursa
- Injury to the extensor process of the pedal bone
- Osteoarthritis of the coffin joint
- Pedal osteitis (see Section 14.5 The foot – Care and disease)
- This cannot be corrected in one trimming and requires multiple small trims to improve the conformation.
- Gradually reduce heel to correct the conformation.



Figure 14.3.5 A broken forward HPA.

'Toe-in' and 'toe-out' conformation'

A line drawn through the centre of the dorsal aspect of the fetlock to the centre of the toe (in hindlimbs – plantar aspect of the fetlock to the centre of the heel) dividing the hoof into two equal parts should normally be a straight line (Figure 14.3.6). Problems arise with the toe-in and toe-out conformation.

Toe-in The axis slopes inwards, the medial side being longer and more sloping than lateral side.

Toe-out The axis slopes outwards, the lateral side being longer and more sloping than medial side (Figure 14.3.7).

Toe-out conformation has been reported by Broster et al. (2009) as significantly associated with pain on flexion of the MCP joint (in a study of 224 working horses in India and Pakistan).

Medial lateral foot balance

This is the balance of the medial (inside) hoof wall compared to the lateral (outside hoof wall) (Figure 14.3.8). Ensuring there is good medio-lateral balance is challenging and is an art that good farriers must master.

The aim of good medio-lateral balance is to ensure the animal's weight is loaded centrally over the foot.

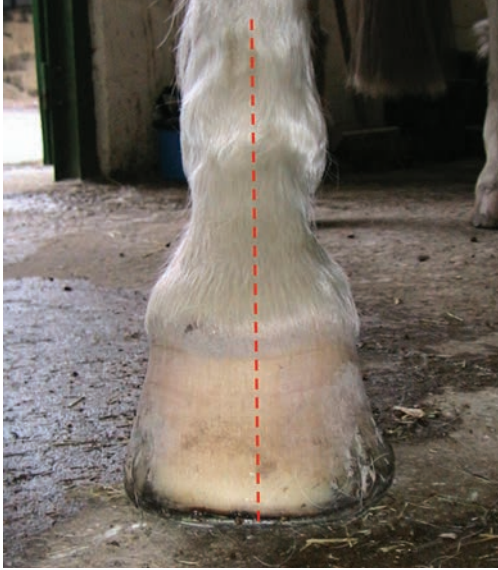


Figure 14.3.6 HPA of the forelimb from the dorsal (cranial) view.



Figure 14.3.7 Bilateral toe-out in a working horse.

Altering the relative heights of the sides of the foot shifts the position of the hoof beneath the limb and changes the loading of the foot.

Lowering the inside of the foot will increase the load on the outside wall and, conversely, lowering the outside wall will increase the load on the inside wall.

Uneven loading of the foot has repercussions for the entire limb as the forces up the limb will also be uneven; this puts increased strains on joints, tendons and muscles.

The medial and lateral walls of the foot should strike the ground at the same time.

To achieve this, trim the hoof so the ground surface is perpendicular to the midline of the horse.

Make the following checks:

- Pick up the foot and hold the leg by resting the cannon gently in one hand – so the foot is not being supported but is hanging down. This allows the trimmer to **assess the weight-bearing surface of the foot** and takes into account any natural conformational deviations the horse may have.
- The medial and lateral sides of the foot should look even – so the weight-bearing surface is flat.

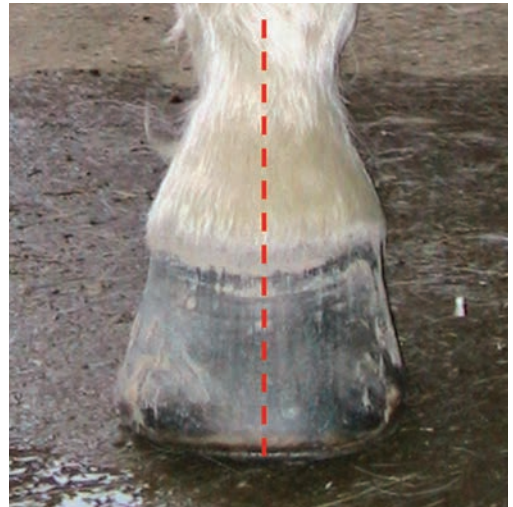


Figure 14.3.8 Foot showing medio-lateral foot balance – remember the foot should land as flat as possible to encourage even weight bearing.

- Always watch the equid walking.
- Watch the equid walk away and back to see if one side of the foot is landing before the other. If this is happening, trim the side that is landing first slightly shorter so the foot lands flat on the floor.
- Re-check the hoof landing when walking.
- Some equids' conformation means their foot will never land flat. In these cases overzealous trimming to achieve this can be very counterproductive. Therefore, correct the foot as far as possible but do not necessarily expect it to be perfect. Make any changes gradually over a number of trimmings.
- Assessing the hoof wear prior to trimming can also be helpful – if one side is much shorter than the other side make sure less is trimmed off this side.

Imbalance of the feet of working equids has been reported in one study by Upjohn et al. (2012). Of 312 working horses in Lesotho, 52% in the forefeet and 37% in the hindfeet had either medio-lateral or dorsopalmar/plantar imbalance. In another study of 214 working horses in India and Pakistan by Broster et al. (2009) hoof imbalance was reported in as high as 92% of forefeet examined. Differences in these studies were seen in a number of areas: fewer than 25% of horses in Lesotho were shod, whilst 85% of horses in India and Pakistan were shod, possibly indicating a link between shoeing and poor foot balance. Also the horses in India and Pakistan were urban and more likely to be working longer, harder hours and over tougher terrain than the rural horses in Lesotho.

Donkey and mule feet

Differences exist between the feet of horses and donkeys (Walker et al. 1995; Thiemann et al. 2013). A donkey's feet are smaller and more 'boxy' (Figure 14.4.2); the angle of the hoof wall is more upright than that of the horse. A donkey's hoof is more prone to becoming deformed as the result of a higher moisture content in the hoof wall. Radiographic anatomy differs between horses and donkeys (Collins et al. 2011). There is a paucity of information available on mule feet; however, texts indicate that they should be cared for in the same way as donkey feet (Svendson 2008).

Study the anatomy and conformation of the hoof first (see Section 14.3).

Responsibility of the owner

A healthy foot is vital to the fitness of an equid and its ability to work. Encourage owners to carry out daily foot checking and cleaning as part of a complete preventative healthcare programme.

Owners need to be aware of what signs to look out for to identify foot problems early; emphasise that any penetrating injury to the sole requires immediate attention and a tetanus prophylaxis (consisting of tetanus antitoxin and tetanus toxoid). Repeat the tetanus toxoid 4–6 weeks later in animals with no known vaccination history.

The importance of good farriery cannot be overstated. If animals are shod, encourage owners to replace shoes that are worn, broken or too small. Regular inspection by a good farrier for trimming/shoeing should be encouraged; urge owners to seek out and strengthen existing farriery services in the local areas.

Responsibility of the veterinarian

The longer a foot problem is present the more difficult it is to correct. Advice given to owners and farriers when foot abnormalities are mild can prevent painful lameness and irreversible arthritis in the future. This is a crucial time when welfare can be improved.

Examine the feet of every equid irrespective of its presenting problem.

Early identification and correction of foot abnormalities may prevent chronic changes. Many causes of lameness affect more than one foot even if the animal only appears to be lame on one leg. When examining an animal check all four feet so that early signs of disease in the other feet are treated at the same time.

Demonstrate foot care to owners and farriers as this has more impact than verbal instructions. Clear advice to farriers can help prevent problems from recurring although, in some cases, it may be impossible to return the foot to a pain-free and functional state. Have a good knowledge of the availability and quality of farrier services in the area and, if necessary, advise the regional government or animal health NGOs of the potential for strengthening local services.

Foot problems and lameness must be tackled on a holistic basis. In order for there to be a decrease in such problems, owners and farriers need to be able to apply many of the preventative measures available. If owners do not have the option of a good farriery service, the local animal health provider has a responsibility to help rectify the situation.

Trimming the foot

Farriery is not a task routinely carried out by veterinarians; however, it is essential that poor trimming is recognised and addressed. **Trimming should be done regularly every 6–12 weeks** depending on the rate of hoof growth and wear. The trimming approach for each hoof differs, and it is important to attend a farriery course if this work is to be carried out regularly.

Outlined below are the basic steps to trimming a normal hoof. For further information consult other texts (e.g. Ross and Dyson 2003).

1. Sole Remove the flaky/chalky material of the sole so that the sole callous is observed in the toe area. The sole callous is the raised area just inside the hoof wall. The sole is naturally cupped from side to side as well as from front to back. The shape should be slightly concave and the correct thickness is when very slight movement can be felt with thumb pressure.

Trimming the sole is critical to foot balance and it is essential that the correct amount is removed. Remove too much and the sole will be thin resulting in bruising, remove too little and the sole will be too thick. This limits foot expansion and the hoof's ability to act as a shock absorber. In normal feet very little sole needs to be removed; continuously check with digital pressure to ensure that the sole is not too thin. The sole should never bleed during routine trimming. The bars are trimmed only to remove overgrown or deformed horn.

2. Hoof wall Rasp or nip back the hoof wall to just above the level of the sole callous. Continue this round on each side of the toe. At this point if the toe is overly long it, too, should be trimmed back appropriately. Trim the toe last. Trim the wall using the hoof pastern axis and the angle of the wall at the coronary band as a guide. Always keep the plane of the cut parallel to the ground – a common fault is to cut the wall at 45 degrees or more so that the sole inside the white line is bearing all the weight. This is called 'dumping the toe' (the second photo Figure 14.4.6), and will cause pain and lameness.

When trimming the wall of the foot ensure that both sides are the same height by carefully comparing one to the other; this will keep the foot level and symmetrical.

3. Heel Flatten the heel so that each side is equal (and at the same level as the frog buttress). The sole is then rasped back for one or two strokes only so it is flat and level ensuring medio-lateral balance.

4. Frog The frog is trimmed to remove loose and overgrown flaps which trap dirt and are prone to thrush. The cleft of the frog can be trimmed to reduce bacterial infection. Trim back the sulci to the frog so that mud can fall out and isn't trapped. Trimming the rest of the frog is not recommended as the frog should touch the ground when the animal is standing although the majority of the weight bearing is through the wall and the bars. **The frog should contact the ground first**; over-trimming the frog is a common and serious problem which leads to pain, thrush and contracted heels and contributes to arthritis by increasing concussion.

5. Dorsal hoof wall Rasping of the outer hoof wall may be necessary to form a straight line from top to bottom but it is important that the white line is not breached. The wall should be straight from the coronet to the floor surface with no indents or curves (wall flare). Bring the foot forward to dress wall flare. Use a rasp to smooth the edges but do not rasp a lot of horn from the front of the wall or it will be too thin and brittle to bear weight. If the dorsal hoof wall is so overgrown that it is turned up at the toe it will require several trimmings to correct it. Never cut it all back at once as this will cause imbalance and pain; the sole needs time to grow out to allow normal weight bearing on the dorsal wall.

6. Rasp the rim of the hoof wall to remove sharp edges.

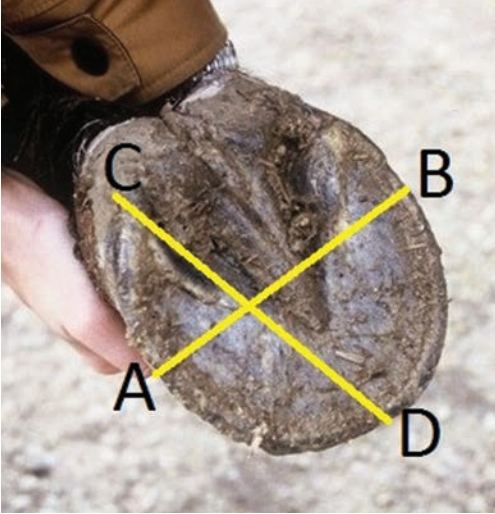


Figure 14.4.1 The line A-B indicates the width of the foot at the widest part, and line C-D indicates the length of the foot from the corner of one heel to the midpoint of the toe.



Figure 14.4.2 A normal donkey foot.

When trimming a hoof wall, shape the underside of the foot and follow the natural outline of the coronet band. As an approximate guide to the shape of the solar surface (Figure 14.4.1):

- Front feet The line A-B should be equal to C-D.
- Hind feet The line A-B should be slightly shorter than C-D (as hindfeet are slightly narrower).
- Donkeys The feet are always narrower (see Figure 14.4.2).

Work in the field

Trimming in the field (Figure 14.4.3) can present a number of challenges:

- Animals may have received very little or no farriery or trimming and therefore hoof conformation can be extremely poor and overgrowths dramatic.
 - It is not possible to correct this in one trimming – changes need to be made gradually as dramatic trimming can cause increased stress and lameness/injuries.
 - Owner education is critical – encouraging owners to have feet trimmed regularly is essential.



Figure 14.4.3 A hoof being trimmed in the field in India.

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■ Feet can be very hard and difficult to trim.

- Source sharp hoof knives, locally whenever possible, and keep them in good condition.
- If feet are very hard, soaking them in warm water for 10 minutes prior to trimming can help soften them.

■ Hooves are often in very poor condition with brittle, poor quality hoof horn.

- Owner education about proper nutrition is essential as this will greatly improve horn quality.
- Teaching owners how to pick out and clean hooves can make a marked difference to hoof health. Encourage them to incorporate it as part of a daily grooming routine – simple prevention and management can greatly reduce the incidence of diseases such as thrush.

Figure 14.4.4 depicts untrimmed feet with poor foot care showing overgrowths which will be putting increased stress on the joints. Notice that the horn quality is also poor and brittle with cracks in both toes. Figure 14.4.5 shows a well-trimmed, balanced foot where the horn quality looks good and healthy with no cracks.



Figure 14.4.4 These hooves are overgrown and show poor conformation.



Figure 14.4.5 A hoof that is well balanced after trimming.

'To shoe or not to shoe?'

■ While many people rely on shoeing, poor or incorrect shoeing can do a great deal of damage (Figures 14.4.6, 14.4.7 and 14.4.8).

- Nail placement must be very accurate and go via the insensitive laminae only. If a nail pricks the deeper sensitive laminae underneath the horn this will be very painful and allow the introduction of infection, ultimately resulting in a foot abscess. For more information on nail bind and hoof abscess see Section 14.5.
- Shoes must be fitted accurately. Shoe placement determines where on the foot weight is distributed. If the shoe is in the wrong place, or is too large or too small, weight will not be distributed evenly around the wall, and areas such as the sole might, as a consequence, be weight bearing. This will inevitably lead to lameness and will also distort future growth of the hoof resulting in deformed hooves with very poor conformation.

- Shoes must be changed regularly. To avoid complications, shoes should be replaced every 4–6 weeks. Shoes need to be removed as the hoof grows so that the feet can be trimmed to maintain an accurate fit. Moreover, in working animals shoes will wear out and, once wear has occurred, they should be replaced.

Many equids cope well without shoes and, if they are coping well and are not working a lot on tarmac roads, most are best left unshod.

If owners are already using shoeing it is vital that they are educated about hoof care and the requirement for regular replacement and trimming.

Figure 14.4.6 shows a shoe that is placed too far palmar (caudal/to the back) on the foot and does not follow the circumference of the hoof wall. This will be distributing the horse's weight incorrectly and will result in foot pain and lameness.



Figure 14.4.6 Poorly fitted shoes.

Figure 14.4.7 shows shoes that have been half worn away. This will result in unbalanced loading of the hoof. Shoes should be replaced regularly to avoid reaching this stage of wear. This will greatly reduce foot pain and lameness and therefore is a great benefit for the equid and also the owner.



Figure 14.4.7 Shoes that have been half worn away or cracked.

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Figure 14.4.8 shows a horse shod in Afghanistan with a shoe that crosses over at the heel. This will produce an imbalanced foot, placing unequal stress on the limb.

Figure 14.4.9: Note in this well-trimmed and shod foot the frog has not been trimmed back too far but the frog sulci (grooves either side of the frog) are clean and tidy. The frog expands into these sulci when the foot bears weight and this is part of the shock absorbing mechanism that reduces the impact on the joints in the limb.



Figure 14.4.8 A horse shod in Afghanistan with a shoe that crosses over at the heel.



Figure 14.4.9 A well-trimmed foot and correctly placed shoe.

Conditions affecting the hoof

14.5

'No hoof, no horse' (or mule, or donkey!)

The equine foot is subject to continuous concussion which causes wear on both internal and external structures.

Poor hoof care and farriery predisposes the animal to injury and infection, and can result in changes to the size, shape and function of the hoof.

The foot is a common site of both acute and chronic pain leading to lameness. Untreated diseases and abnormalities of the foot can create long-term problems in other parts of the leg due to changes in gait and weight bearing.

Conditions affecting the hoof wall

Overgrowth/overwear

If an animal has normal conformation and no shoes (unshod), there should be a balance between growth and wear of the wall.

Overgrowth occurs:

- when unshod animals are kept on soft ground
- in shod animals, when shoes are left on too long or the feet are not trimmed properly (Figure 14.5.1)
- in animals with poor conformation/injuries/lameness as the weight is not distributed evenly over the whole hoof.

Overwear occurs:

- when unshod animals are worked excessively on hard ground (Figure 14.5.2)
- when pain, poor conformation, poor hoof balance or bad shoeing cause the animal to land unevenly on the foot
- when the shoe frequently wears or cracks in one particular area.



Figure 14.5.1 The shoe has been left on too long and the hoof wall is overgrown. This shoe should be removed and the hoof trimmed.



Figure 14.5.2 An example of an overworn hoof.

Treatment

- Identify and correct the underlying problem. Removing the cause of pain will correct mild overwear as the animal starts to bear weight evenly again.
- Correct moderate to severe uneven wear with trimming. If the problem is permanent (e.g. arthritic pain or poor conformation) trim the feet regularly (see Section 14.4 Trimming and shoeing)
- Returning the hoof to a perfect balance may not be possible.
- Never over-trim the wall to compensate for abnormal foot wear.

Hoof cracks: 'Grass cracks' or 'sand cracks'

Hoof cracks are classified as '**grass cracks**' if they start from the ground (Figure 14.5.3) or '**sand cracks**' if they start from the coronary band. Sometimes cracks can cover the whole extent of the hoof wall from the coronary band to the ground surface (Figure 14.5.4).

Causes

- Dehydrated and brittle hoof horn
- Damage from shoe removal
- Excessively overgrown feet
- 'Seedy toe' (see 'laminitis' later in this chapter)
- Abnormal foot shape or poor shoe placement
- Damage to the coronary band

Diagnosis

Most cracks do not cause lameness as the full thickness of the wall is not penetrated. Lameness and a pain response are seen with deeper cracks as these can become infected or pinch the sensitive laminae. Swelling, heat and pain are present at the coronary band as infection tracks up the wall.

Treatment

The aim of treatment is to stabilise the crack and keep it clean and dry. If dirt fills the crack it will put pressure on the edges forcing it further apart. For deep hoof cracks, with infection and lameness, treat as for a hoof abscess (see later in this chapter). Stabilise the crack using quarter clips either side or with the use of adhesives to limit hoof expansion during weight bearing to reduce movement across the crack. When cracks occur in the area of the quarters, trim the wall near the heels so that the wall



Figure 14.5.3 A grass crack in the lateral quarter of the hoof extends from the ground to approximately halfway up the hoof wall.



Figure 14.5.4 A severe crack passing from the ground surface through the coronary band.

either side of the crack is non-weight bearing. This allows the heel and the frog to bear the weight rather than the cracked area. Do not make a horizontal groove in the horn across the top of the crack as this weakens the hoof.

Prognosis

Good for cracks that are not infected and do not reach the coronary band. Healing of infected cracks depends on complete removal of all necrotic tissue. Permanent damage to the laminae can result in abnormal horn growth, separating the wall from the underlying tissue and causing further cracking. Cracks that originate from the coronary band have a poor prognosis as normal horn will not re-grow. Prevent hoof cracks with good farriery and good nutrition. Soak dry or brittle hooves in water, and coat in hoof grease to retain moisture.

Hoof avulsion

Avulsion of the hoof occurs when there is separation of the hoof wall from the internal structures. Exposure of the sensitive laminae and pedal bone results in severe pain and lameness, infection and osteitis.

Causes

- Trauma
- Severe febrile diseases
- Laminitis
- Administering corticosteroids to a laminitic patient (iatrogenic)

Treatment

Damage to the coronary band affects horn growth so the prognosis is poor. If the area of separation is small, attempt treatment by flushing the area, bandaging and administering antibiotics and NSAIDs.

The most humane treatment, especially in total hoof avulsion, is euthanasia. Explain to the owner that the horn will not re-grow.

Club foot

An abnormally shaped upright foot
(Figure 14.5.5)

Causes

- Congenital or acquired flexural deformity of the deep digital flexor tendon
- Prolonged disuse or chronic limb pain (a common example is from a hoof abscess) causing tendon contraction, pulling the pedal bone into a more upright position



Figure 14.5.5 An upright or club foot.

Diagnosis

Club foot is diagnosed if the axis of the toe exceeds 60 degrees; the foot will also have high heels as a result. Often only one foot is affected; bilateral conditions tend to be congenital. In acute cases the heel will not touch the ground – ‘walking on the toe’. If the club foot is chronic, the heels become overgrown and contracted and the dorsal hoof wall will be concave. Lameness is variable; some animals adapt their gait and are not lame.

Treatment

For acute cases, identify and remove the source of pain (e.g. hoof abscess) and use NSAIDs to encourage relaxation of the tendons. The prognosis for return to normal shape is guarded. If the club foot is chronic, trim the hoof regularly and keep the foot balanced. Do not try to return it to the ‘correct’ shape, as this will cause lameness; when the heel is lowered dramatically this puts a great deal of pressure and stretch on the flexor tendons. Toe abscesses are common, so protect the toe region from over wear.

Conditions affecting the sole and frog

All of these conditions are exacerbated by inadequate attention to:

- hoof hygiene; removing stones, mud or faeces (Figure 14.5.6)
- contamination of standing areas with sharp objects and stones, dirt, faeces and urine.

Demonstrate to owners how to check and clean the feet and keep the frog area clean and dry. Address any signs of frog abnormality or a foul smell immediately.



Figure 14.5.6 Basic hoof hygiene is simple but often neglected.

Puncture wounds and hoof abscesses

Causes

- Stone and foreign bodies, e.g. nails (Figure 14.5.7), can pierce the sole, leading to the introduction of anaerobic bacteria and abscess formation.
- Faulty/defective attachment of the shoe can result in ‘nail prick’ when the nail punctures the sensitive tissues of the hoof, or ‘nail bind’ when the nail applies pressure to the lamellae without directly touching the tissues.



Figure 14.5.7 Nail found penetrating the frog.

Diagnosis

Severe, acute lameness; this can be mistaken by the owner as a fracture. There is often increased heat in the foot with a 'bounding' digital pulse. Following a puncture wound, infection may run up the white line or under the sole and break out at the coronary band or heel bulbs. With a penetrative injury the foreign object may still be in the sole when the equid is examined.

The location and depth of penetration will greatly affect prognosis.

A penetrative injury to the middle third of the frog is the most serious as it can easily pierce the **navicular bursa** and/or **coffin joint**. This may lead to an unresponsive infection resulting in severe lameness. In such cases recommend euthanasia on welfare grounds.

Treatment

Carefully remove the foreign body if present; ensure adequate drainage through the site of penetration. Pare out the hole if the diameter is very small to prevent sealing over and abscess formation. For a suspected abscess remove the shoe and use hoof testers around the wall, sole and frog to localise the site of pain. Pay particular attention to the nail-holes, which may be the site of pain or infection ('nail-prick' or 'nail-bind').

Use a hoof knife to pare the sole and white line until they are clean, looking for a black spot at the site of penetration. Follow the black area until the origin of the abscess is opened (Figure 14.5.8). Paring the sole can be extremely painful; in this case an abaxial nerve block may be given. The foot will then be desensitised so hoof testers are no longer useful. Ensure that the location of the tract is clear before the block is performed.

Stop paring the foot if fresh blood appears. Paring out the wound removes contamination and allows aeration, thus reducing anaerobic bacterial infection.

Antibiotics are not necessary and will not work if the infection is localised, due to poor blood supply to the sole tissue. The equid must be vaccinated against tetanus as the hoof could easily become contaminated with dirt containing *Clostridium tetani*.

If the animal is not vaccinated, or the vaccination history is unknown, then administer tetanus prophylaxis.

In all cases, drainage is most important for successful treatment.

Soak the whole foot for 10 minutes in a bucket of water containing magnesium sulphate or dilute povidone-iodine. Ask the owner to repeat this twice a day until the animal becomes



Figure 14.5.8 Foot abscess with sole pared to release purulent material.

sound. It may be appropriate to keep the wound covered/bandaged until fully healed to prevent re-infection. Change this dressing daily to keep the wound fresh and dry.

In rare cases, a deep puncture wound to the central zone of the foot, near the frog, can cause fracture of the navicular bone or septic navicular bursitis (see Section 14.6). If an animal develops severe lameness within a few days following a puncture of this nature, and does not respond to intensive therapy, the prognosis is very poor; discuss euthanasia with the owner.

White line disease

This is an infection of the white line – the junction of the insensitive laminae of the hoof wall and the horn of the sole. This is a natural weak region of the hoof; poor, brittle or soft horn will result in a defect at the white line and subsequent infection. Damp or very dry conditions will also predispose the animal to white line disease.

Diagnosis

The white line may appear widened and is often packed with necrotic material. If left untreated, infection can track up to the coronary band.

Treatment

Remove the necrotic horn. The foot can then be stood in magnesium sulphate solution or dilute povidone-iodine as for foot abscesses. Keep the foot clean and dry. Show the owner how to clean the white line and repeat the soaking if necessary.

Solar bruising

Solar bruising is the result of an impact to the sole that causes haemorrhage in the solar chorium. This may occur if the animal is working on stony or rocky ground or if the soles are thin as the result of poor farriery or regular work on hard surfaces.

Diagnosis

- A dark red or purple area on the white sole, seen after light paring with a hoof knife
- Pain response to hoof testers
- Mild to moderate lameness, usually worse on hard/rough ground. Lameness may be unapparent when the ground is soft.

Treatment

NSAIDs and rest to reduce pain. Treatment with formalin will toughen the solar surface; however, this chemical is dangerous to human and environmental health. Good prognosis for full recovery if the initial cause can be identified and managed.

Prevention

Discourage fast work over rough or stony ground as this exacerbates the problem. Excessive paring of the sole by the farrier makes it thin and susceptible to bruising.

Corns

Corns are bruises between the bars and the hoof wall, usually as a result of poor shoe maintenance, and are most common on the medial side of the front feet. Corns are caused when the shoe is too small or narrow at the heel – common when standards of farriery are poor. Also, when shoes are left on too long, the growth causes the shoe to be pulled forward and the shoe branch then impacts on the region most prone to corns.

Diagnosis

Lameness of varying degrees, and pain when pressing the area. Paring will reveal a soft, red/purple area between the bars and wall (Figure 14.5.9). Secondary bacterial infection may occur.

Treatment

Remove the old shoe and replace; the corn will self-heal if there is no infection. If infection is present, treat as a sole abscess (described earlier in this chapter).

Prognosis

Good, but the discoloured horn will take several weeks to grow out. Demonstrate the cause of corns to owners and farriers. Emphasise the importance of using large shoes with wide-set heels, fitting shoes correctly and replacing them regularly.



Figure 14.5.9 Corns present on medial and lateral heel angles.

Thrush

This is a bacterial infection of the grooves (central or collateral sulci) of the frog (Figure 14.5.10). The infection usually occurs when the condition of the frog degenerates as the result of a dirty environment and irregular cleaning. Excessive trimming of the frog and contracted heels result in a small deep-set frog with poor air circulation to the area. The predominant infective pathogen is *Fusobacterium necrophorum*.

Diagnosis

Lameness occurs only in severe cases; milder forms are usually unnoticed by owners. A characteristic foul smell comes from the frog due to anaerobic bacteria. The thick black discharge in the sulci may be hidden by flaps of overgrown frog.



Figure 14.5.10 A thrush infection in the central and collateral sulci of the frog.

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Treatment

Trim any horn flaps and improve the foot shape to allow more air to reach the sulci. Clean with dilute povidone-iodine, repeat daily until the infection is cleared. Do not bandage the foot as it is important to aerate the affected area. Antibiotics are unnecessary unless deeper structures are affected.

Prognosis

Good, but thrush will recur if the underlying problems are not corrected. Demonstrate the cause to owners and farriers. Encourage farriers to trim heels correctly and ensure that the frog comes into contact with the ground/is bearing weight.

Advise the owner to clean the feet daily and keep the horse on a dry standing area (remove faeces, urine and damp bedding daily).

Canker

Cause

Canker is a proliferative dermatitis that occurs as the result of a chronic infection of the frog and surrounding tissues by *Bacterioides* spp., spirochetes and possibly bovine papilloma virus. This condition may initially be confused with thrush but can be differentiated by a foul, necrotic odour and the presence of granulation-like tissue. It is common in animals standing for long periods in wet, dirty conditions and in warm climates.

Diagnosis

- Frog horn loosens to reveal foul-smelling, proliferative granulation tissue (Figure 14.5.11).
- Instead of the usual flat, uniform horn, filamentous fronds of horn develop.
- Thick, cream-coloured exudate
- Pain and associated lameness
- Bleeds easily and is very susceptible to screw-worm maggots

Treatment

The treatment is likely to be difficult and prolonged. Debride the granulation tissue thoroughly; this may require a nerve block and a tourniquet. Apply topical metronidazole and bandage to keep the foot dry. Change daily for 10 days.

Prognosis

Recurrence is common and treatment requires veterinary attention over a long period. Treatment may be successful if initiated early in disease course.



Figure 14.5.11 Canker present in a foot of a working equid.

Conditions affecting the heels

Traumatic heel wounds

The heel is susceptible to traumatic injuries such as laceration with sharp sheet metal, or from kicking a sharp object. Often there is profuse bleeding, especially if a digital artery has been severed at the pastern.

Treatment

Ligate the artery if there is severe bleeding. Clean, debride and apply a pressure bandage; 'proud flesh' is very common following injuries in this location. Decrease motion and stabilise the hoof. Suturing is contra-indicated due to the high rate of wound breakdown in this area.

Prognosis

Usually good unless cartilage has been damaged. If the lateral cartilages are damaged there may be delayed healing or **quittor** (chronic infection of the cartilaginous extension of the pedal bone). If the digital flexor tendons are involved and have been cut, the prognosis for return to work is extremely poor (see Section 14.8).

Contracted heels

Cause

- Shoes that are too small and narrow at the heel prevent natural expansion as the foot hits the ground.
- Excessive paring of the frog prevents it from contacting the ground and induces frog atrophy.
- Inadequate trimming of the heels leads to an upright foot with a broken-forward hoof-pastern axis.

Over time, repeated placement of a small, tight shoe causes the foot to become smaller and more upright, with a very concave sole and small ineffective frog (Figure 14.5.12). This contributes to other foot and leg abnormalities.

Treatment

Only good regular farriery will improve contracted heels. Shoes which are slightly larger laterally at the quarters and heels encourage hoof wall expansion (they may need to be custom-made for an individual). The frog should not be trimmed except to remove small ragged pieces.

Prognosis

Depends on severity and duration of heel contraction. It is poor in severe chronic cases where the joints and tendons have been secondarily affected by the abnormal hoof shape.



Figure 14.5.12 The heels have narrowed creating a more oval-shaped hoof in this horse, with a diminished and ineffective frog.

Sheared heels

When one heel is longer than the other, the heel bulbs are subjected to a shearing force which can cause a breakdown of the tissue that normally holds the two heels together. This is usually the sequelae of faulty shoeing/trimming where one heel is left longer than the other.

Diagnosis

When an equid loads the foot, the heels move independently of each other as one heel bears all the weight. In severe cases the skin between the heels can be damaged by this movement and lesions or granulomas may develop. This imbalance can also lead to heel or quarter cracks.

Treatment

Allow the shorter heel to grow longer and match the other side. Good farriery can help support the heels and minimise further movement.

Heel dermatitis/'greasy heel'

Causes

- Wet, dirty standing conditions
- Hobbling with dirty rope or cloth
- Chorioptic mange (caused by *Chorioptes* mites)
- Photosensitisation on white lower legs

Diagnosis

Skin lesions are seen at the palmar or plantar aspect of the pastern and heel. The dermatitis is characterised by scab formation, moist/greasy exudate and erythema (Figure 14.5.13). The skin lesions are irritating and painful, although this condition rarely causes lameness. In chronic cases granulomatous round grape-like growths can develop, triggered as a result of persistent inflammation.

Treatment

Early diagnosis and treatment is essential to prevent the involvement of deeper structures. Clip hair from the area and clean twice a day with dilute povidone-iodine, removing all grease and scabs. Dry thoroughly and do not bandage; the aim is to dry out the skin. Antibiotics are not necessary as pharmacological penetration to this area is limited and good hygiene should resolve secondary infection. Administer antibiotics if cellulitis is present (see Section 15.3).

Prognosis

Good if the underlying cause is removed. Encourage owners to provide a clean, dry standing area and groom the legs daily to remove dirt. Dry heels with a cloth after equids have worked in wet conditions. It is important that hobbles, if used, are kept clean and well maintained.



Figure 14.5.13 Greasy heel affecting a limb.

Brushing and over-reaching

Both conditions are due to poor conformation or poor hoof balance resulting in an abnormal gait.

Brushing is an injury to the inside of the pastern/fetlock as the result of one hoof striking the other during movement (Figure 14.5.14). This may be exacerbated by poor placement of nails on shoes or poor conformation. Equids with 'cow hocks' will have brushing on the medial hocks.

Over-reaching is when the front heels are struck by the toes of the hindfeet during movement.

Treatment

Clean the area. These injuries often lie close to the fetlock joint; think about the potential for joint infection with chronic injury. Correct the underlying cause to prevent recurrence.

Ensure the feet are balanced, the shoe is correctly placed, and the nails are knocked flat. In cases of over-reaching, shortening the toe of the front feet may speed break-over allowing the front feet to 'get out of the way' more quickly (Ross and Dyson 2003). Fit locally-made protection over the fetlocks and pasterns, such as a 'brushing ring' of twisted cloth or rubber around one fetlock to protect it from contact with the opposite foot. Working the animal too young (< 3 years) will contribute to poor carpal and hock conformation, as the bones are still developing.



Figure 14.5.14 An example of a brushing injury.

Pedal osteitis

Pedal osteitis strictly refers to inflammation of the distal phalanx. However, it has been used to describe radiographic changes, principally demineralisation at the solar margin of this bone. Whether this is a proven disease and a cause of lameness is controversial (Ross and Dyson 2003).

Causes

Pedal osteitis is a result of persistent inflammation of the foot. It can be caused by a number of problems including: chronic solar bruising, chronic corns, laminitis, puncture wounds, deep untreated abscesses, etc. In some cases where infection is present, e.g. with a deep, chronic abscess, the pedal bone can be infected. However, the condition can occur without infection as a result of long-term persistent inflammation.

Diagnosis

Persistent lameness with a persistent reaction to hoof testers. Confirmation of diagnosis can be achieved with radiographs; however, observation of clinical signs can support an initial suspicion, without requiring further diagnostic tools.

Treatment

Prevent by the diagnosis and treatment of inflammatory conditions of the foot. Once pedal osteitis has developed treatment is limited so identify and treat the inciting cause.

Laminitis

Severe inflammation and necrosis of the sensitive tissues (laminae) attaching the hoof to the pedal bone (P3)

Causes

■ Mechanical overload

Over-exercising on hard ground. Excessive weight bearing on one leg following lameness on the opposite limb. With any lameness examine the contra-lateral limb for signs of laminitis.

■ Bacterial endotoxin release into the blood

High carbohydrate intake. **iatrogenic** veterinary-administered corticosteroids to treat a different condition or owner-administered corticosteroids to increase muscle and condition of animals.

Systemic infection (toxaemia) from retained placenta (septic metritis), mastitis, enteritis, colitis, gastrointestinal torsion, pleuropneumonia, etc.

Pathogenesis

Laminitis is characterised by a failure of attachment of the epidermal laminae to the dermal laminae. The precise pathophysiology is yet to be determined. A vasoconstriction theory of laminitis aetiology, based on vascular pathology resulting in ischemia of the lamellar tissues, has been largely supplanted by the **enzymatic** theory in which matrix metalloproteinase enzymes (MMPs) are activated. MMPs at normal levels are responsible for enzymatic remodelling; when over-active, this enzyme causes destruction of lamellar tissues and separation. There are many experimental studies which attempt to determine the pathophysiology of laminitis. Reviews by Pollit (2004) and Eades (2010) provide a good overview.

Diagnosis

Characterised by an acute onset of severe lameness. Affected equids adopt a characteristic stance, most often the front limbs are affected and so the hindfeet are placed as far forward as possible with the **forelimb pushed forwards** to relieve weight bearing (Figure 14.5.15). The lameness in acute cases is severe; weight will be shifted from one side to the other, and affected equids are likely to spend long periods of time recumbent, sometimes with the legs stretched out. It can be difficult to examine the animal as there is often a refusal to walk or to lift the foot as this will increase pressure on the affected contra-lateral limb. Extreme pain is manifested as elevated respiration and heart rate.

On examination of the foot with hoof testers there is pain all over the sole,



Figure 14.5.15 This horse has signs of chronic laminitis in all four feet. However, the typical leaning back stance is not seen due to joint changes in the forelimb fetlocks preventing this; note the firing over the four fetlock joint areas.

especially at the point of the frog. **Bounding digital pulses** (increase in amplitude) are palpable in most cases, and the feet will be hot in the early stage. Separation between the wall and underlying pedal bone causes sinking and rotation of P3 which can penetrate the sole in severe cases, seen as a creamy-white, blood-stained area in front of the frog. A **depression at the coronary band** may be observed as a result of the sinking and shearing forces as the pedal bone drops or rotates. In the working equid context, detection of this sign can give a useful indication of pedal bone movement when radiography is not available.

It is important to differentiate cases of acute laminitis from colic, both of which are painful conditions. Conduct a full examination. Hoof abscesses may also present with similar signs to laminitis; however, an abscess is likely to be unilateral.

Treatment

Acute laminitis is an emergency. If left untreated, the pedal bone may rotate through the sole in a few days, necessitating euthanasia.

- Treat the **underlying cause** (retained placenta, systemic infection, grain overload, etc.).
- **Do not move** the animal anywhere, even to the mobile clinic, as concussion on the foot during movement will cause more sinking and rotation of the pedal bone.
- **Complete rest is essential**, in a confined area on a thick bed of sand, straw or any soft, conforming material to cushion the feet. It is imperative that rest is continued until soundness has completely returned once NSAID treatment has ceased.
- Working an animal too soon will re-trigger the laminitis which is likely to be more serious if it recurs. Convey this message clearly to owners.
- Mechanical **foot supports** for the distal phalanx; reduce concussion by applying thick padding firmly taped onto the frog/sole. Replace the foot supports as they become flattened. Frog supports are probably inappropriate for donkeys; cover the entire sole with a thick soft dressing.
- It may be necessary to **nerve block** the foot (abaxial sesamoid block) in order to apply frog supports.
- Do not walk the animal whilst feet are anaesthetised.
- In shod equids, ideally **remove the shoes**, as they concentrate the weight loading on the outside of the foot, closer to the laminae. Remove the shoes carefully to minimise concussive forces.
- **Administer NSAIDs** to ameliorate foot pain and suffering. Animals should be on complete rest whilst on medication.
- Corticosteroids have been linked to the development of laminitis in certain situations; avoid the use of these medications.
- Sedatives such as **acepromazine** can be administered to encourage recumbency and reduce weight bearing and concussion within the hooves (van Eps 2010). A dose of 0.02–0.04 mg/kg intramuscularly or intravenously 4 times a day has been recommended for 3–5 days after the acute onset. It has been suggested that acepromazine is beneficial as a vasodilator. However, as the vasoconstriction theory of laminitis aetiology is unproven, this beneficial effect remains hypothetical.

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- Administer **IV fluid therapy** if there are signs of shock.
- If the laminitis has been induced by a **grain overload**, pass a stomach tube to allow for reflux and to prevent gastric rupture. Then administer mineral oil/liquid paraffin. Ensure all grain is removed from the diet, feed a high fibre diet. Do not starve.
- **Cold-therapy** of the legs and feet has proven preventative experimentally. It appears to provide some analgesia and prevent progression of the laminitis. This can be achieved by standing an animal in buckets of cold or iced water or standing in a nearby stream.
- **Euthanasia** is recommended if the pedal bone has come through the sole, or if prolonged signs of pain cannot be alleviated by NSAID medication.

For further information on treatment refer to van Eps (2010).

Prognosis

A severely rotated pedal bone is a very serious condition that is life threatening.

The outcome of treatment depends on many different aspects including:

- The animal's weight
- Degree of pain
- Degree of pedal bone rotation
- Degree of white line separation
- Sub-solar infection development
- Thickness of sole
- Environment animal is kept in
- Willingness of owner to treat and rest the animal

Permanent changes to the foot structure from a single bout of laminitis make the individual prone to repeated or chronic laminitis. Classic signs of chronic laminitis include: 'slipper-shaped' hoof, with low heels and a concave dorsal wall, and distinct rings of wall horn representing bouts of laminitis. Hoof growth is affected by laminitis – the heel is spared and grows faster than the toe. Divergent growth rings appear (Figure 14.5.16) which are normally parallel to the coronary band. A flat sole with indentation around the coronary band indicates movement of the pedal bone. A dark purulent discharge from the coronary band occurs after abscess formation between the separated lamellae. Bruising of the sole occurs due to movement of the pedal bone resulting in compression of the solar tissue. 'Seedy toe' appears as a wide area of crumbly horn at the dorsal white line which can become impacted with dirt and small stones. Animals with seedy toe are prone to white line abscesses and cracks in the dorsal wall. Pare out and trim the area regularly until the damaged horn has grown out completely.



Figure 14.5.16 Typical growth patterns of chronic laminitis, where growth is faster at the heels than the toes leading to diverging lines around the hoof.

Trimming a chronic laminitic foot

The basic principle of trimming a laminitic foot is to realign the pedal bone with the hoof wall after rotation. Trimming alone cannot force realignment but it does enable normal growth patterns to be re-established.

The main aims of therapeutic trimming are to reduce the distractive forces on the dorsal laminae in weight bearing and break over; to stabilise the pedal bone within the hoof capsule and unload the areas of pressure. Any abscesses that have formed must be drained.

Key principles

Reduce the heel area very gradually. If too much is removed in one trimming it will result in a great strain on the deep digital flexor tendon; this can lead to damage and encourage further rotation. It can be helpful to trim the toe from the ground surface to reduce the pressure on this region. The area just in front of the apex of the frog should not be trimmed. Therapeutic trimming needs to be regularly repeated every 4–6 weeks, sometimes for the rest of an animal's life.

Prevention of laminitis

Inform owners how to recognise the early symptoms of acute laminitis, as treatment in the first 24 hours will greatly decrease the likelihood of permanent damage or euthanasia. Avoid excessive or fast work on hard roads. Keep feet well-trimmed and balanced. Keep a high ratio of roughage to grain in the diet, with changes introduced slowly. Avoid heavy or long-term use of corticosteroids, a major cause of laminitis in some countries is due to owner administration of long-acting steroids (for example, this occurs with the wedding horses in India). This is a good example of the importance of working with all stakeholders on appropriate drug usage as a means of prevention.

Conditions affecting the bones

14.6

Conditions affecting bones are usually serious. All bone conditions require complete long-term (weeks to months) rest, with a thick bed of sand or straw to lie on, to allow the bone to heal.

Even after recovery, the equid will need to be worked slowly and for shorter periods to prevent the problem recurring.

Young animals should pull a lighter load, work more slowly and for shorter periods than mature animals, to allow bones to grow and adapt to the work. Do not introduce an animal to a full load and full day's work until it is at least 3 years old.

Fractures

Causes

Most commonly, fractures are caused by **trauma** from accidents, kicks or falling. Fractures may occasionally be **pathological** due to tumours, infection or poor nutrition. However, this is rare in equids.

Diagnosis

Although radiographs will definitively diagnose fractures, observation of one or more of the following signs can support an initial suspicion, without requiring further diagnostic tools:

- Sudden onset of lameness, often severe or non-weight-bearing
- Heat, swelling and pain on palpation
- Crepitus (grating noise from the fractured ends of the bone). Auscultate with a stethoscope while gently manipulating affected limb.
- Deviation or alteration in the normal shape of the limb
- Fractured ends of the bone penetrating through the skin

How do bones heal?

An understanding of the fracture healing process will help in decisions about treatment and prognosis when dealing with a fracture:

1. Reactive phase Formation of blood clot and granulation tissue in the first few days post-fracture, similar to tissue healing
2. Reparative phase
 - a. '**Bony callus**' formation where the periosteum provides two important precursor cells for callus formation
 - a.i. Chondroblasts – precursor to hyaline cartilage
 - a.ii. Osteoblasts – precursor to woven bone
 - b. The hyaline cartilage and woven bone knit together and unite, eventually covering the bone defect between the two ends of the fracture.
 - c. Endochondral ossification is the 'mineralisation' of the woven bone/hyaline cartilage scaffolding.
3. Remodelling phase Trabecular bone formed during endochondral ossification is replaced by permanent, strong 'compact bone'. Eventually the healed bone remodels to a shape and strength closely resembling the original bone.

In order for a bone to heal the following requirements must be met:

- Immobilisation of the whole area (including the joint at either side)
- Fracture ends in contact for extended periods (6–8 weeks). Constant movement will continuously break the callus and prevent healing.
- Good blood supply to provide sufficient nutrients and oxygen for bone healing
- No infection (implication for open fractures)
- Limited contact of fracture ends with other structures such as joints
- Age – a younger animal is more likely to heal.

It is usually unrealistic to fulfil these requirements, particularly in the context of a less developed country and in equids required to work.

Long bone fractures (femur, tibia, humerus, and radius)

Adult (mature) equids

These will never heal effectively because of the weight of the animal and difficulty immobilising the fracture site for long periods.

Surgical treatment requires specialist hospital facilities and there is a high likelihood of contra-lateral limb laminitis if treatment is attempted. **Limb amputation is not acceptable** for a working equid. **Euthanase** to prevent prolonged suffering (Figure 14.6.1).

Foals (immature) equids

Closed, non-displaced long bone fractures can be contained within a **Robert-Jones bandage** or cast, extending from the ground as high up the leg as possible. Ensure thick padding under the heel and keep lower joints flexed. This prevents normal weight-bearing forces from displacing the fracture. Plenty of thick padding must be used over the whole limb, especially tendons and joints, to prevent damage and pressure sores from the cast or splint. The splint will need to be in place for **at least 6 weeks** with deep bedding for the foal to lie on. If the animal is older, e.g. a yearling, it could possibly be cross-tied standing, but young animals are unlikely to tolerate this and it may cause other developmental orthopaedic problems. The animal must not work for **4–6 months**. The **prognosis is guarded**, as the fracture may fail to heal or, when healed, the limb may be weak or deviated preventing a good working life in future.

When considering whether to treat a long bone fracture in a young animal, always remember welfare cost versus benefit.

- What are the physical/mental welfare implications of long-term confinement?
- Will regular veterinary visits be possible to ensure the bandage/cast is not causing pressure sores, infection, swelling or even necrosis of the encased limb?
- Is the owner willing to allow the animal to rest for the long period required for healing?

Never attempt to treat an open fracture. Euthanase these cases straight away.



Figure 14.6.1 Fractured right hindleg with owner-applied splint (this horse was euthanased on humane grounds).

Non-displaced fractures

If the fracture is not displaced (for example in a metacarpal bone), diagnosis can be difficult without radiography. Consider the following when reaching an informed decision about how to proceed:

- Severity of lameness
- Duration of lameness
- History of how it developed
- Physical and mental welfare status of the animal – Alert? Eating?
- The owner's attitude
- Response to treatment

In most cases if there is a **positive response to complete rest and pain relief** it is unlikely that a fracture has occurred. Table 14.6.1 demonstrates the non-displaced fractures that can be confused with other conditions.

Type of fracture	Can look like...	Suspect fracture if...
Pedal bone	Laminitis, hoof abscess	Trauma to the foot
Navicular bone	Laminitis, hoof abscess	Penetrating injury in frog area, trauma, severe pain on foot manipulation (due to movement of the deep flexor tendon)
Splint bones	Discharging sinus, generalised fetlock/cannon swelling	Trauma, brushing injuries
Carpal bones	Carpal osteoarthritis, joint infection	Carpal hyperextension/fatigue, e.g. from fast work, hard ground Evidence of falling, e.g. knee lesions
Olecranon (elbow)	Capped elbow	Direct trauma (kick, accident) 'Dropped elbow' stance Fatigue – slipping when forelimb out in front (avulsion fracture)
Scapula	'Sweeney' (muscle atrophy of the shoulder area due to damage to the suprascapular nerve, if chronic)	Direct trauma to the area
Patella	Stifle osteoarthritis, joint infection	Direct trauma, stiff hindlimb gait
Pelvis	Early tetanus, neurological problems	Direct trauma/falling/knocking pelvis on cart or gate
Spine	Neurological disease, collapse	Very severe trauma

Table 14.6.1 Differential diagnoses for fractures accompanied by aspects of history and clinical signs.

If treatment is not an option, which will be very common with the working equid, then advise euthanasia as early as possible to **alleviate pain and suffering**. If an injured equid needs to be moved, ensure that adequate support and protection is given to the limb to avoid further pain, and that analgesia is administered.

Emergency splinting and bandaging

If a fracture is suspected, splint the leg immediately following examination (Walmsley 1999); the position of the splint depends on the site of the fracture.

Light sedation may be required. The joints above and below the fractured bone need to be immobilised. A 'Robert-Jones' is a multi-layered bandage that will provide a degree of support. This bandage comprises many layers of cotton wool applied sequentially and held in place with an elastic bandage. Each layer should be at least 2.5 cm thick and should be applied more tightly than the last. The bandaged limb should be three times the thickness of the contra-lateral limb. Once a Robert-Jones is completed a splint can be applied, the position of which depends on the site of the fracture. A splint should be light but strong and not bend; a broom handle cut to an appropriate length is ideal. Firmly tape to the leg with non-elastic tape.

Sequestra

A sequestrum is a small piece of bone that has become detached from its blood supply usually as the result of trauma, e.g. avulsion or a comminuted fracture.

Diagnosis

- History of trauma
- Evidence of a sinus tract discharging pus through the skin, often recurring

Treatment

- Identify bone piece(s) by palpation.
- Remove the bone, if easy to access, under local, regional or general anaesthesia.
- Extreme care must be taken to avoid synovial structures when removing sequestra.
- Debride and flush the wound thoroughly. Administer a 7–10 day course of penicillin, and NSAID medication as required.
- Tetanus prophylaxis is essential.

Prognosis

Depends on the site of the sequestra and how effectively all necrotic tissue has been removed. In some cases (e.g. fractures of the pelvis) **it will not be possible to remove the sequestra**. There is likely to be a continuous discharging tract, and infection may spread to neighbouring tissues.

Periosteal regrowth – ‘splints’ and ‘bucked shins’

‘Splints’ refer to periostitis or fractures of the 2nd or 4th metacarpal/tarsal bones (‘splint bones’ Mc/t II and IV).

‘Shin splints/bucked shins’ refer to dorsal periostitis (stress microfractures) on the dorsal aspect of the 3rd metacarpal (cannon bone, McIII).

Periosteal new bone growth in both of these areas occurs as the result of inflammation (periostitis); this can be due to abnormal **stress**, direct **trauma** (e.g. brushing) or excessive **work**. Stress microfractures in McIII occur commonly in young horses running at speed. This work exceeds the ability of the bone to re-model and adapt to this stress. Large amounts of new bone growth on McII and IV may interfere with the **suspensory ligament** (interosseous) and can even lead to fractures of the splint bones if severe.

Clinical signs of forelimb periosteal growth

- Acute phase Variable amount of swelling, heat and pain on palpation; the animal may show lameness which increases with further work.
- Chronic phase Mild to severe, cold swelling; the animal shows no lameness.
- If severe signs are present, a splint bone may be fractured. Microfractures predispose McIII to complete fractures.

Treatment

In the acute phase, rest the animal for as long as possible, ideally **one month**, to minimise the periosteal reaction. Use systemic (and topical, if available) NSAID medication to reduce the lameness and soft-tissue swelling. Chronic splints are a cosmetic problem only and do not require treatment. Correct the underlying cause if possible.

Sesamoiditis

Cause

Inflammation of the proximal sesamoid bones as the result of tearing of the suspensory ligament fibres at the insertion. The suspensory ligament undergoes severe strain during fast work. The other structures of the fetlock (ligaments, tendon sheath, cartilage and soft tissue) may also be affected.

Clinical signs

- Sudden onset of moderate to severe lameness
- Diffuse swelling around the sesamoids, with pain on palpation and fetlock flexion
- Distension of the fetlock joint capsule

The ideal treatment is to apply a strong support bandage for 2 months and rest the animal for up to a year. This, however, is impractical in working equids which cannot be rested for such a long period; in this case rest as long as possible and use support bandaging and NSAIDs to reduce inflammation and pain.

Pathology involving the navicular bone and bursa

Navicular syndrome is chronic idiopathic osteitis of the navicular bone.

Acute navicular bursitis/fracture is infection and inflammation of the navicular bursa due to a penetrating injury in the frog area.

Cause

Different theories have been discussed about the underlying cause and pathogenesis of navicular syndrome; however, acute navicular bursitis or fracture is often the more likely presentation in working equids. The end result is degeneration and pain in the navicular bone with accompanying lameness.

Clinical signs of navicular pathology

- Mild, slow-onset forelimb lameness
- Unequal foot size
- Positive response to palmar digital nerve block
- Pointing of the toe in the front feet

Expert radiography and interpretation is needed to confirm diagnosis. Without access to further diagnostic aids an informed decision is based on history, clinical signs and knowledge of anatomy.

Treatment

There is no cure for navicular disease; treatment is management-based with the use of long-term daily pain relief and hoof trimming to achieve a correct hoof-pastern-axis. Treatment of navicular bursitis is rarely successful, and euthanasia is recommended if the lameness and pain is severe.

Osteomyelitis/osteitis

Osteomyelitis is inflammation and infection of bone and marrow.

Osteitis is inflammation of bone only.

Pedal osteitis Inflammation of the pedal bone P3. The significance of this condition is controversial.

Causes

Haematogenous spread (via the bloodstream). This occurs in young foals at the physis (growth plate) of subchondral bone, as the blood flow in these vessels is slow. A localised osteitis can develop after trauma, from the spread of infection from local structures.

Diagnosis

- Lameness is seen in the initial phases. However, if the condition becomes chronic, lameness may no longer be apparent.

- In **osteitis secondary to trauma**, there is often a purulent discharging sinus with a non-healing wound. There may be a history of an intermittent discharge without resolution.
- In **haematogenous osteomyelitis**, the affected limb will be swollen and lame; several sites may be infected including both bones and joints.

Treatment

- If a localised bone infection is present secondary to an **open wound**, antibiotic therapy is of little or no value. The lesion may improve following treatment but, once this is ceased, the signs will recur.
- Debride **necrotic bone**, if present, with appropriate analgesia, e.g. regional anaesthesia and systemic analgesia. Only perform this operation if the sequestra are in a safely accessible area, without a large risk of iatrogenic damage. A good knowledge of local anatomy is essential.
- In haematogenous osteomyelitis, prolonged antibiotic therapy is warranted and, if the treatment is started early enough in the course of the disease, there may be a resolution. This is likely to take 3–4 weeks. In severe cases it may not be successful so warn owners before considering treatment. If an animal is not going to be treated, recommend euthanasia on welfare grounds.

14.7 Conditions affecting the joints

Joints are complex structures that are extremely susceptible to injury in working equids (Broster et al. 2009).

All joints have the following characteristics:

- Cartilage Elastic 'shock absorber'
- Synovial fluid Highly viscous 'lubrication'
- Joint capsule/tendons/ligaments Vary according to which joint is involved but generally act as 'stabilisers'

Injury and damage to the joint results in **pain**, **inflammation** and **decreased flexibility** (Figure 14.7.1), all of which contribute to lameness. When treating joint disorders with parenteral medication, the limited blood supply makes it difficult to achieve articular therapeutic concentrations.



Figure 14.7.1 Ensure a full examination of all limbs and flexion of joints.

Osteoarthritis (OA) and degenerative joint disease (DJD)

The most common cause of lameness reported in horses is **joint disease** (Rossdale et al. 1985) and **osteoarthritis** is the most common condition. One report found that the highest cause of death or euthanasia was joint disease (Egenvall et al. 2006).

OA/DJD = chronic inflammatory changes in the joint over time leading to a progressive deterioration of articular cartilage.

Causes

- 'Wear and tear' as part of the ageing process
- Secondary to joint injury – joint strain, articular chip fractures, joint infection
- Long-term, excessive work on hard roads
- Poor limb conformation – resulting in asymmetric stresses to joints
- Uneven weight loads or inappropriate weight loads
- Poor foot care
- Iatrogenic – poor, incorrectly administered joint injections

Changes that occur in the joint during development of osteoarthritis

- Cartilage Cracks (fissures) and decreased elasticity over time reduces the 'shock-absorber' effect.
- Synovial membrane Twisting, stretching or direct trauma over time results in a continuing cycle of joint damage:
 - Increased production of synovial fluid which is less viscous, resulting in less effective joint lubrication and protection
 - Increased production of enzymes and inflammatory cells, causing further tissue destruction and inflammation

Clinical signs of early osteoarthritis/DJD

- Varying degrees of lameness, usually in multiple limbs
- Pain on joint manipulation
- Decreased range of motion
- Positive response to flexion tests
- Variable joint capsule distension with synovial fluid

With advanced osteoarthritis/DJD, clinical signs may also include rough bony swellings (**osteophytes**) around the joint margins which are easily palpated and externally visible in more advanced cases (Figure 14.7.2). The metacarpophalangeal (MCP) or fetlock joint has been reported as the most commonly affected joint with OA in the horse (Cantley et al. 1999). There is reduced

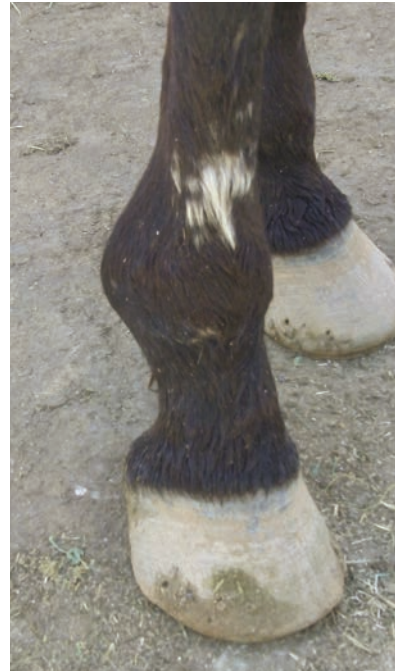


Figure 14.7.2 Osseous growth around the metacarpophalangeal joint of the right forelimb.

New bone formation (common name)	Location	Example
Low ringbone	Distal interphalangeal joint	
High ringbone	Proximal interphalangeal joint	
Bone spavin	Distal hock joint (distal intertarsal and tarsometatarsal joints)	
Fluid Distension (common name)		
Wind gall/wind puffs	Digital flexor tendon sheath (encases the flexor tendons) Incidental, unassociated with lameness	
Bog spavin	Tarso crural joint (hock)	

Table 14.7.1 Examples of other common terms that refer to osteoarthritis.

flexion of the joint. In some cases the joint cannot be moved at all because it has become fixed (**arthrodesed**) by new bone formation around the joint.

'One condition, many names' – Different terms are used for osteoarthritis depending on the site (Table 14.7.1). The pathology, however, remains the same.

Treatment

Osteoarthritis cannot be cured. Changes in the joint structure and function are irreversible, so emphasise pain management and prevention to owners: A strategic, multimodal approach is often required. It is essential that everyone (owner, vet, user, carer, animal) works as a team. Always keep the animal's best interest in mind.

Owner communication is essential for management and prevention, emphasising that osteoarthritis cannot be cured, and any visible changes to the joints (bony swellings, fluid pockets) are permanent. Instead, encourage the following:

- Reduce the speed and duration of work on hard roads.
- Attempt to rest animals for long periods if they are lame.
- Take good care of hooves – especially the frog.
- Start young working equids slowly and gently – and do not start work before 3 years of age.

Rest reduces the aggravation of the inflammation process allowing time for natural healing. When lameness and pain reduce, slow and steady exercise can begin.

Medical treatment options

NSAIDs such as phenylbutazone (PBZ) and flunixin are commonly used to alleviate the pain associated with OA. Daily NSAIDs can be administered but, in advanced cases, this is often ineffective. Remember the side-effects of long-term use on internal organs. There has also been concern regarding the negative effects of NSAIDs on cartilage (Beluche et al. 2001). NSAIDs will not treat the condition, may mask the clinical signs and make the animal more comfortable. The owner should be warned of this and it should be remembered that working an animal on NSAIDs is likely to speed the progression of the disease by increasing the wear and tear of the joint. An animal's natural instinct to protect an injured limb will decrease with pain relief, often resulting in further damage if the owner is not careful. This should not prevent the use of these medications as they have both analgesic and anti-inflammatory effects. In the early stages of disease NSAIDs should be used to full effect to stop progression.

Corticosteroids are very effective drugs for reducing inflammation. Intra-articular corticosteroid injections are only recommended if used very carefully in the early stages. This is usually not feasible in working equids due to the presentation of mostly chronic cases to clinics. Joint injections should be followed by 3–4 weeks of complete rest. This medication is ineffective where a lot of bony changes are already present; do not use if the joint capsule is already distended. Avoid steroids if there is a suspicion of septic arthritis. Injections must be given under strict aseptic conditions (see Section 4.1 Drug administration techniques).

Weigh up the cost versus benefit – an iatrogenic joint infection as the result of poor hygiene will outweigh any benefits from the treatment.

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- **Correct farriery** Correct trimming or shoeing of the feet is essential in the management of locomotion system pathology.
- **Firing and burning** do not benefit arthritic joints (Table 14.7.1 Bog spavin photograph). Offer owners alternatives such as massage or simple physiotherapy techniques.

Problems with treatment

There can be inter-animal variation of response to treatment as the result of heterogeneity of the disease. The stage of the disease affects the outcome of the treatment. Different preparations of the same drug are available; check concentration and dose rate. Expectations of the owner may be higher than is possible.

There is a poor correlation between imaging and the disease process. The use of radiography does not tend to enhance the ability to diagnose and treat. Once changes are seen on a radiograph the disease is at a very advanced stage and easily diagnosed by clinical examination. Radiography will not affect the treatment regime and management in most cases.

Synovitis

Synovitis is the inflammation of the synovial membrane resulting in excessive synovial fluid production; cartilage is usually unaffected.

Causes

- Excessive concussion of the joints of young animals
- Trauma
- Poor conformation, especially hoof conformation and trimming
- Working on uneven surfaces

Diagnosis

Large fluid swellings in the joints, most commonly seen as 'bog spavin' (dorsomedial and plantar pouches of the hock joints, Table 14.7.1).

Treatment

Reduce the work load. Treat with NSAIDs in the early stages; it is important to stop inflammation early to avoid cartilage damage leading to osteoarthritis.

Septic arthritis

Bacterial infection of the joint(s) results in a septic arthritis.

Causes

- Penetrating joint injury
- Iatrogenic when infection is introduced by a veterinary procedure, e.g. joint injection (especially when corticosteroids are used). It is absolutely essential that joint injections are only done aseptically in a suitable environment.

- Tracking of infection from superficial wounds or abscess in the area (e.g. brushing wounds)
- Haematogenous (joint-ill in foals)

Bacteria in the joint stimulate a severe inflammatory reaction of the synovial membrane. This produces increased amounts of synovial fluid, inflammatory cells and enzymes which further inflame the synovial membrane and rapidly destroy the cartilage. When large areas of articular cartilage are lost, the underlying bone is susceptible to bacterial exposure, leading to **secondary osteomyelitis**.

Clinical signs

- Severe pain and lameness which may not appear for several days after a suspected injury/corticosteroid injection (may be mistaken for a fracture)
- Marked fluid swelling of the joint, which can progress to soft tissue swelling and heat in the surrounding area
- Dullness, poor appetite, fever

Diagnosis

If trained in this procedure, collect a joint fluid sample (see Section 4.1 Drug administration techniques) under **strict aseptic conditions**. Prior to collection ensure that facilities for analysis are available, either within the clinic or at a local laboratory. The joint fluid may appear **discoloured, blood-stained or cloudy**. Septic joint fluid, when shaken in a tube, froths more than normal joint fluid as there is a higher protein content. If a microscope is available, examine a stained smear. A WBC exceeding 30,000/μl with a total protein exceeding 45 g/L is suggestive of an infected joint (Caron 2011). Differential cell counts are valuable; normally the neutrophils make up less than 10% of the WBC count. If this proportion increases, particularly to around 80%, the diagnosis of a septic arthritis is strongly supported.

To determine whether a laceration close to the joint has **penetrated the synovial capsule**, the joint should be aseptically prepared at a distant site to the injury. Following retrieval of a sample for analysis, a small volume of sterile saline can be injected into the joint capsule. If fluid flows from the site of trauma the joint capsule has been breached and should be treated as septic. Once completed, the synovial structure is typically injected with an antibiotic (e.g. 125–500 mg amikacin sulphate).

Collecting a synovial fluid sample

1. With the site identified and prepared (see Section 4.1 Drug administration techniques), appropriately restrain the animal and identify the location for needle insertion. Some joints may be easier to sample with the limb flexed (e.g. carpus).
2. Use a 20/21G, 25- or 40-mm needle, depending on the size of the joint and amount of soft tissue swelling. Introduce it quickly through the skin, then advance slowly until synovial fluid appears in the hub. In inflamed/infected joints, the synovial fluid is under pressure and will usually drip out, otherwise attach a 5-ml syringe and apply gentle suction to aspirate sample. This needs to be performed under strict asepsis. Do not attempt to collect synovial fluid through infected skin.
3. Immediately transfer the sample to two sterile vacutainers, one plain and one EDTA. For a bacterial culture blood culture tubes are required; these may be obtained from the laboratory.

Send the sample to the laboratory with clear instructions, or examine a stained smear under a microscope (Table 14.2.1).

Property	Normal joint fluid	Abnormal joint fluid
Appearance	Clear, non-turbid, pale yellow	Turbid; may contain lumps/particles; darker yellow; may be blood-stained
Viscosity	Viscous (stringy appearance when stretched between thumb and finger)	Less viscous (negative stringing test)
Total leukocyte count	$< 0.5 \times 10^9$ per L	$> 0.5 \times 10^9$ per L (usually much higher in septic arthritis)
Total protein (use a refractometer)	< 18 g/L	> 18 g/L

Table 14.2.1 Microscopic properties of normal and abnormal joint fluid.

Treatment

Veterinarians and owners should be aware that attempted treatment of a septic joint is a long, slow process requiring many weeks of commitment from both parties.

- Septic arthritis is an **emergency**; treatment should be early and aggressive.
- Ideally for a successful outcome, septic joints should be irrigated with copious fluids, in a sterile theatre, within the first 24 hours. In the working equid context this is not possible and hence often treatment is not possible. If the joint has been septic for over 3 days, success of treatment is poor as irreversible damage occurs during this time.
- Treatment in the field may consist of a standing joint flush under sedation and long-term parenteral antibiotics.
- Antibiotics need to be administered for at least 2 weeks after the resolution of lameness.

Flushing an infected joint

Flushing (lavage) of an infected joint aims to remove dirt, bacteria, fibrin and other inflammatory products which destroy the joint cartilage and permanently damage the joint, such as in septic arthritis. Flushing should begin as soon as joint penetration is suspected; the longer the delay between infection and flushing, the less successful the treatment is likely to be. This through-a-needle technique will not remove solid fibrin or purulent material. Do not attempt this procedure if untrained in the technique.

Required materials

- Sedatives or general anaesthetic
- Local anaesthetic
- 18G to 14G needles or catheters; syringes 2 ml, 5 ml, 20 ml
- Sterile saline bags (at least 1 litre) and intravenous giving set
- Bandages/dressing materials
- Antibiotics (gentamicin or penicillin), NSAIDs, tetanus antitoxin

Flushing the joint

1. Administer prophylactic antibiotics, NSAIDs and tetanus antitoxin.
2. Shave and prepare the area **aseptically** as for intra-articular injection. Several needles may be placed, so prepare the whole joint area plus 3–5 cm in all directions.
3. **Sedate or anaesthetise** the animal as necessary. If not anaesthetised, place a subcutaneous bleb of local anaesthetic at each injection site. If anaesthetised, the joint will need to be scrubbed once the animal is positioned for the procedure.
4. Introduce needles (as for intra-articular injection) into the joint at two distant points, e.g. caudal and cranial pouches of the joint capsule. Synovial fluid should appear if in the correct position; collect this for laboratory evaluation.
5. Using the giving set, attach the fluid bag to one needle ('**ingress**') and gently distend the joint with saline until it flows out of the other needle ('**egress**'). Gently manipulate the joint to encourage flushing. Avoid damaging the cartilage with excessive needle movement.
6. If fluid does not flow, one or both needles may be touching the cartilage or blocked by fibrin or inflamed synovial membrane. **Redirect** needles or withdraw and replace with a new needle. Flush the joint thoroughly, ideally with **10 litres of saline**.
7. Withdraw the 'egress' needle. Aspirate a few ml of fluid from the 'ingress' needle and inject 250–500 mg **gentamicin** or 125 mg **amikacin** before withdrawal.
8. Cover with a **sterile dressing** and bandage the joint. Advise the owner on hygiene of the area, and ensure a 5–7 day course of antibiotics.

Prognosis

Guarded, but early treatment can lead to good results. If the condition has been present for more than 14 days, the prognosis is very poor and **euthanasia should be advised on welfare grounds**.

Prevention and owner communication

Treat skin wounds over joints as a potential risk of a joint infection.

Many brushing or knee lesions are encountered on a daily basis in working equine examinations; any one of these could become a septic joint.

Encourage owners to keep wounds clean and identify/address the underlying causes.

Osteochondrosis

This is a developmental disturbance to the cellular differentiation of cartilage in growing horses (osteochondrosis has not been reported in donkeys). There is a failure of ossification that starts at the growth plates and results in cartilage defects such as a separation from underlying bone, cracking (fissures) and weak spots. Flaps of cartilage develop and can break off to form loose fragments which are referred to as **osteochondrosis dessicans (OCD)**. Subchondral bone cysts can also form.

Causes (Ross and Dyson 2003)

Despite extensive investigations, the cause remains unclear but it is thought to be the result of excessive force on weak bone or cartilage. It is most commonly seen in large-breed fast-growing horses and is mainly seen in yearlings up to 2-year-olds. Diets that are high in energy and have a calcium phosphate imbalance have been commonly associated with the condition. Some

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manifestations have a hereditary component. An inappropriately high workload at a young age may also contribute to OCD development.

Diagnosis

- Joint effusion is the most common clinical sign. Several joints may be involved including phalangeal, fetlock, carpal, shoulder, tarsal and stifle joints.
- Joint lameness and pain can be present but not always.
- Subtle early changes may be visible on radiographs, although these changes are usually not definitive until the disease has reached a severe stage.
- If the cervical spine is affected this can lead to instability resulting in a neurological condition known as **Wobbler Syndrome** (Cervical Stenotic Myelopathy).

Treatment

- Ideally this should be treated as early as possible, so early diagnosis will improve the disease progression and prognosis.
- Horses should be rested and dietary changes made; reduce the dietary energy but ensure ample roughage remains in the diet.
- If not detected early, extensive areas of damaged cartilage can develop which will trigger the development of arthritis, and the prognosis for soundness in adult life is poor.

Sprains – sub-luxations and luxations

A sprain is the stretching or tearing of the support ligaments of a joint. In a mild sprain only a few fibres are torn and the integrity of the support is not lost.

Sub-luxation/luxation is a severe sprain where the integrity of one or more support ligaments for a joint is lost.

A sub-luxation is the partial dislocation of a joint, and a luxation is a complete dislocation of a joint. These can be complicated by fractures such as avulsion fractures, when the stress of the ligament breaking pulls off the piece of bone it attaches to.

Clinical signs

Sprain

- Mild peri-articular swelling, and possibly some laxity of the joint when manipulated
- Lameness and weakness of joint

Subluxation/luxation

- Extensive swelling around joint and adjacent structures
- Severe lameness and weakness
- Sub-luxation (abnormal extension or flexion on manipulation)
- Luxation (abnormal posture)

Treatment

Sprain

- Cold therapy – Apply cold water and ice as soon as possible after trauma.
- Stable the equid and immobilise the limb using a support bandage and confined space. Rest until the lameness has resolved, the length of time depends on severity of sprain. Ensure the bandage is applied correctly and changed regularly to avoid pressure sores and rubs.
- Administer NSAIDs for analgesic and anti-inflammatory effects.
- The prognosis is good.

Sub-luxation/luxation

- These injuries require full immobilisation and, in the working equid context, this is rarely possible.
- Radiography is useful to assess for concomitant fractures and determine prognosis. Although, at best, these images serve as further evidence to euthanase an injured equid rather than to determine a treatment protocol.
- Treatment is by splinting or casting for a minimum of 6 weeks followed by a convalescence of up to 1 year.
- Even with full treatment and no concomitant fractures the prognosis for return to work is extremely guarded.
- Given the limitations of treatment in the working equid context, and the risk to animal welfare if treatment fails, euthanasia should be strongly advised for this condition.

Upward fixation of the patella (locking stifle)

This occurs when the medial patella ligament hooks over the medial trochlea of the femur, locking the hind limb in **permanent extension**.

Causes

- Poor conformation – straight hindlimb
- Sudden weight loss can predispose to locking. The infra-patella fat pad reduces in size so the patella sits deeper in the trochlear groove.
- Loss of fitness and muscle tone

Clinical signs

The hindlimb is locked in extension, the stifle and hock are in extension, with a flexed fetlock. The foot is dragged behind and the limb cannot bear weight. Locking can be intermittent or can remain locked for long periods. Severe/prolonged upward fixation of the patella is more frequently seen in small equids, particularly donkeys. If there is a history of upward fixation this can be induced by walking in a tight circle or walking up and down a slope. There is stifle effusion as the result of inflammation in the joint.

Treatment

- Unlock patella manually – push the horse backwards and manually push the patella medially and distally; this can be difficult to do.

- NSAIDs – Administer phenylbutazone for a few days.
- Improve the condition of the animal by increasing the muscling to hindlimbs, increase the level of nutrition, administer anthelmintics (if there is evidence of GI parasites) and corrective dentistry as required. This is particularly effective in young animals and the condition can resolve once the hindquarter muscling has increased.
- If repeated locking persists there is a risk of arthritis and joint pain as the joint surface becomes damaged.
- A medial patellar ligament desmotomy should only be considered if attempts at conservative and medical therapy have failed, the patellar continues to lock and the owner will not consider euthanasia. Consult Tribar 2003 for further details on this procedure.

14.8

Conditions affecting the tendons and ligaments

Tendons connect **muscle to bone** in the equine musculoskeletal system. They are fibrous with a limited blood supply and are protected by sheaths containing synovial fluid where they pass over high motion joints such as the carpus.

Think of tendons as rubber bands that can stretch. If the stretching becomes too great the tendon becomes 'strained' and can eventually snap, much like an over-stretched rubber band.

Working equids which are **ridden** have a greater chance of damaging the forelimb tendons due to fetlock hyperextension at high speeds. Those equids which pull **carts or carriages** are more likely to damage the **hindlimb tendons** due to excessive strain exerted when pulling a heavy load (Maranhão et al. 2006), see Figure 14.8.1. Knowledge of work type is therefore important.

Tendon healing is a very slow process due to the low blood supply common to fibrous tissues. Once damaged, tendon fibres are replaced by weaker fibrous tissue which is less flexible. The healed tendon is more prone to further damage in the future, so prevention is always better than cure.



Figure 14.8.1 A mule pulling a cart in a brick kiln in Delhi. Note the hyperextension of the right hind fetlock.

Strains: tendonitis and tenosynovitis

Tendonitis/tenosynovitis = inflammation of the tendon/tendon sheath either through trauma or infection

Tendon injuries in horses are extremely common, with some tendons more prone to injury than others. The superficial digital flexor tendon (SDFT) of the forelimbs is the most commonly affected; damage to this tendon makes up 80–90% of reported tendon injuries (Williams 2001, Pinchbeck et al. 2004). In working horses digital flexor tendonitis was found in at least one limb in 83% of animals examined (Broster et al. 2009). Recent research findings show that the microstructure of the SDFT changes with age, increasing the risk of injury in older horses (Thorpe et al. 2012).

Causes

- Hyperextension (Figure 14.8.2) or other excessive force can cause straining or tearing of tendon fibres.
- Tendon sheaths can become distended with increased synovial fluid (tenosynovitis).
- Infection can also be present, for example with a penetrating injury over a tendon or a tendon sheath.

Clinical signs

Acute tendon strain

- Five signs of acute inflammation (heat, swelling, loss of function, redness, pain) occur over affected tendon. The limb should be palpated both when weight bearing and when raised to permit a thorough examination (Figure 14.8.3).
- Lameness
- Increased synovial fluid in the tendon sheath can make the tendon difficult to palpate so always compare with the opposite leg.

Chronic tendon injury

- Firm thickening of the affected tendon ('bowed tendons'), with or without synovial sheath distension (Figure 14.8.4)
- No heat or pain on palpation

Treatment of acute tendon strain

Treatment is aimed at controlling the inflammatory response rather than being curative.



14.8.2 An overloaded donkey in Jordan. Note that the hind fetlock is hyperextended.



Figure 14.8.3 Palpation of tendons for the five signs of acute inflammation.

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Cold water hosing twice daily can reduce inflammation but has a limited effect on tendon swelling. Apply a firm pressure bandage along length of tendon, ensuring that the top and bottom of the bandage are not too tight. Administer NSAIDs in decreasing dosage. Continue to rest the animal while under treatment as it may appear sound with analgesia. Rest over a period of 4–6 weeks is the most effective circumstance for tendons to heal. Return to work should be slow with lighter loads to prevent recurrence.

Firing is proven to have no effect on tendon healing. (Firing is the application of heat to burn the skin using a red hot iron; this causes scar tissue to form). Silver and Rosedale (1983) presented the first research study for evidence that firing is not an effective treatment for tendon injuries and reduces the strength of the skin in the burnt areas (Figure 14.8.4).



Figure 14.8.4 Chronic tendonitis left forelimb, with lesser affected right fore; scarring as a result of firing is present on both forelimbs.

Prognosis

Return to normal is unlikely due to the impracticalities of long-term rest in working equids. Permanent bowed tendons do not normally affect the animal's ability to work; however, damage to tendons will predispose to further episodes of tendonitis or rupture.

Tendon rupture

Severe damage to tendons may occur following degeneration on sudden **over-extension** or as the result of a **traumatic laceration** (Table 14.8.1).

Diagnosis

- Severe non-weight bearing lameness
- Transection of the extensor tendon will result in knuckling of the fetlock with attempts to bear weight.
- Transection of both the superficial and deep digital flexor tendon will result in the fetlock dropping significantly and the toe will rock proximally.
- In some cases the transection will be obvious, for example if there is an open wound (Figure 14.8.5). In other cases it may be less clear and diagnosis can be made on fetlock instability.

Treatment

Treat as for open wound management; lavage and debride. Long-term supportive bandages are essential to keep the limb immobilised. This should be a Robert-



Figure 14.8.5 Lacerated flexor tendons in a hindlimb.

Jones as a minimum and will require regular changes and check-ups. The owner should be aware of the long-term management and nursing required before embarking on treatment. In the working equid context this may not be possible or appropriate. Long-term rest and pain management is vital due to the slow healing process and must be discussed with the owner accordingly. Prognosis for return to work is poor and euthanasia should be considered on welfare grounds.

Surgical repair requires a specialist suture technique, general anaesthetic and long-term post-surgical management, all of which are difficult under field conditions.

Tendon	Cause of Rupture	Clinical Signs	Treatment	Prognosis
Extensor tendon	Trauma to dorsal cannon	Knuckling of fetlock Normal weight bearing	Open wound management NSAIDs Surgical repair often unsuccessful	Good if rested and animal learns to flip foot into correct position
Superficial digital flexor tendon (SDFT)	Kicks, wire or sheet metal trauma to back of cannon area	Slightly dropped fetlock	As for open wounds Tetanus prophylaxis Surgical repair only if clean and fresh	Guarded
Deep digital flexor tendon (DDFT)	As for SDFT rupture (usually occurs at the same time)	Severely dropped fetlock Toe raised off ground	Requires surgical intervention Raised heel extensions on shoe	Guarded/poor (Discuss euthanasia in severe cases)
Peroneus Tertius	Wounds Hock hyperextension (trapped or falling animal)	Hock does not flex on forward movement (characteristic) Hock extension whilst stifle flexed Dimpling of gastrocnemius	Rest (9–12 months) NSAIDs Do not suture (extreme load in this tendon)	Fair, although gait will always be abnormal

Table 14.8.1 Features of tendon rupture, including cause, treatment and prognosis.

Desmitis

Desmitis = inflammation of a ligament

Ligaments connect bone to bone. The most common ligaments to be affected by overstrain injuries are the suspensory ligament (interosseous) and the inferior carpal check ligament.

Clinical signs

■ Pain and swelling of:

- proximal lateral metacarpus (inferior carpal check ligament)
- distal metacarpus/tarsus (suspensory ligament).

(Compare reaction with that in the opposite leg.)

■ Lameness is variable, often mild.

■ A loss of the sharp outline of the fetlock occurs with suspensory ligament damage, 'rounding of the fetlock'. This is often associated with sesamoiditis.

Many animals will have signs of suspensory ligament desmitis, with or without lameness.

Treatment

As for tendonitis, with cold hosing, pressure-bandaging, NSAIDs and rest in acute cases

Prognosis

Good for mild ligament strains, but poor for severe injury to the suspensory ligament

Hygroma

See Section 14.10.

14.9 Conditions affecting the muscles

When examining the musculature of an equid, note any **tremors** or **fasciculations** before palpating the muscles to detect heat, pain or swelling. Ancillary diagnostic tests, such as measuring the muscle enzymes, serum creatinine kinase and aspartate aminotransferase, may be helpful when diagnosing muscle damage or necrosis. However, the clinical signs alone are likely to be supportive of a diagnosis.

Exertional rhabdomyolysis ('tying up', 'Monday morning disease')

Exertional rhabdomyolysis (ER) is defined as **acute muscle cell damage** following exercise. In severe cases, equids can die from this condition as the result of necrosis of the muscle and kidney failure following filtration of high levels of myoglobin. ER is a complex syndrome with multiple causes.

This condition frequently occurs following exercise preceded by a long period of rest, hence the term 'Monday morning disease'. An increase in work can have the same effect. An increased risk has been associated with carbohydrate overload and selenium deficiency. The mechanism of this is not completely understood. It is thought that high carbohydrate diets result in increased glycogen storage. During exercise there is a sudden breakdown of glycogen in muscles causing a local acidosis and muscular vasoconstriction which leads to muscle cramping and damage. Electrolyte imbalances may also predispose to this condition.

Clinical signs

- Generalised stiffness and reluctance to move shortly after a period of exercise
- Muscle tremors or spasms, particularly over the back and hindquarters
- Muscles, particularly of the hindquarter, feel tense and solid. Affected muscles are painful on palpation.
- Elevated heart rate and respiratory rate
- Sweating
- Anxiety and a tucked-up appearance
- Red-brown urine (myoglobinuria)

Treatment

- Administer NSAIDs to reduce inflammation and alleviate pain.
- Acepromazine (see Section 7.1) is used for muscle relaxation but ensure that the animal is not dehydrated prior to treatment with this medication as acepromazine has profound hypotensive properties.
- Administer IV fluids (at least 10–15 litres) to address dehydration but also to improve glomerular filtration rates in the face of myoglobin filtration by the kidneys.
- In severe cases corticosteroids may be administered.
- Vitamin B, Vitamin E and selenium may help muscle recovery.
- Avoid moving the animal if possible. Do not force the animal to walk if it is reluctant.
- Reduce grain intake and replace with abundant high fibre diet, such as hay.
- When restarting work do so gradually and slowly. Reintroduce grains once the animal is working again.

Prognosis

This is good in most cases, although recovery may take days to weeks. Advise the owner to return the animal to work slowly, especially after rest periods. Some cases may show a number of consecutive episodes, particularly if the animal has not been adequately rested before restarting work.

Exhaustion and muscular cramping

In some circumstances, working equids are given little opportunity to rest. This is detrimental to the health and welfare of the animal and will shorten its working life.

High workloads combined with energy, water and electrolyte deficiencies can easily overcome the coping strategies of a stoic working equid (Figure 14.9.1).



Figure 14.9.1 Collapsed working horses in Pakistan.

Clinical signs

- Depression, lethargy, collapse
- Little interest in food/water
- Muscular cramping
- Mild colic signs
- **Synchronous diaphragmatic flutter** – Diaphragm contractions occur due to electrolyte deficiencies. It may appear as though the animal is hiccuping.
- Reduced sweating; sweat sticky rather than watery
- Increased heart rate and respiratory rate

Treatment

- Offer water. If the equid will not drink, administer fluids through a stomach tube or give at least 10–15 litres of sterile fluids intravenously (see Chapter 6 Dehydration and fluid therapy).
- Rest
- To prevent reoccurrence add half a tablespoon of salt and half a tablespoon of potassium chloride (KCl) to feed when equids are undergoing long or strenuous exercise.

Malignant oedema (Clostridial myonecrosis)

Malignant oedema is an acute, frequently fatal toxæmia affecting all species and ages of animals and is usually caused by the contamination of wounds with soil.

Infection with *Clostridium septicum*, *perfringens* or *chauvoei* via wounds, surgery or contaminated needles

Clinical signs

- Characterised by a rapid clinical course with signs appearing from just a few hours up to 48 hours following an injury or injection
- Extensive pitting oedema with gas formation under the skin (subcutaneous emphysema)
- Pain over affected area
- Rapid progression to massive swelling and muscle necrosis with jelly-like exudate
- Fever, anorexia and depression
- **Myoglobinuria**
- Generalised toxæmia; increased pulse and respiratory rates, injected mucous membranes (dark pink to red colour)
- A stained smear of the exudate, or aspirated fluid, may show large Gram +ve rod-shaped bacteria.



Figure 14.9.2 A case of clostridial myositis that was treated with a myotomy procedure as well as supportive antibiotic therapy. The horse recovered although the iatrogenic wounds took months to heal. (Image provided by Klara Saville)

Treatment

- Very high doses of penicillin for at least 10 days
- At least 10–15 litres intravenous fluids; continue fluid administration over several days if possible. This is particularly important if the animal has myoglobinuria to reduce the risk of renal failure.
- Surgical drainage of the site if localised
- The early use of a myotomy/fasciotomy procedure has been described to expose the anaerobic bacteria to oxygen (Figure 14.9.2). This procedure is associated with an improved prognosis. However, the iatrogenic deep muscle wounds take a long time to heal and require intensive management (Peek, Semrad and Perkins 2003).
- NSAIDs

Prognosis

Clostridial myonecrosis is usually fatal in a few days. Sequelae of this condition include laminitis and colic as the result of the toxæmia.

It is essential to ensure hygiene when injecting animals; encourage colleagues and para-professionals to change needles and follow good clean practice.

Hypocalcaemia

Cause

Calcium depletion, for example in lactating mares (milk fever, eclampsia), or following hard

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work or transport (transit tetany). This is a very satisfying and easy condition to treat. However, the signs are often not recognised in time by owners and veterinarians alike which can result in the death of the animal.

Clinical signs

- Stiff gait
- Muscle fasciculation (twitching, particularly around the jaw and the back of the upper forelimb)
- Dysphagia (difficulty eating) as the muscles of the jaw are affected
- Ataxia (unsteadiness)
- Profuse sweating
- Pyrexia
- Thumping noise in time with breathing, caused by abnormal function of phrenic nerve (synchronous diaphragmatic flutter). The equid can appear to be hiccuping.
- Seizures, coma and death if untreated

Treatment

Calcium borogluconate: 250–500 mls per 500 kg body weight. Dilute the calcium solution in 4 x the volume of saline; administer this mixture by slow IV infusion. Calcium borogluconate should be readily available in most countries as it is the same formula as that used to treat cattle with 'milk fever' (hypocalcaemia).

Prognosis

Good with early treatment. If hypocalcaemia recurs it may be helpful to supplement the diet with calcium.

Muscle atrophy

Muscle atrophy is defined as the decrease in mass of a muscle; this may be the result of poor nutrition, reduced use of a muscle (disuse atrophy) or a denervation of that muscle.

Generalised muscle atrophy is usually symmetrical and is the result of poor nutrition, cachexia, malnutrition, old age.

Localised muscle atrophy can be caused by:

- damage to the nerve innervating the muscle. Severe asymmetrical atrophy results from the loss of nerve stimulus.
- immobilisation of a limb, or chronic lameness
- injury to the muscle attachment.

Treatment

Identify and address the primary cause if possible. If a nerve is compressed it may regain function. However, if a nerve is transected and the neural sheath is not intact then the neurons will not regrow and the atrophy will be permanent.

Hygroma

A hygroma is an acquired subcutaneous synovial bursa.

As hygromas are generally caused by a **repetitive chronic trauma**, there are certain predilection sites: the elbow, the hock, the withers and the carpus. The bursa is a small cavity filled with synovial fluid that acts as a cushion between two tendons or between tendon and bone. Hygromas are considered a cosmetic problem and are not usually associated with lameness.

Causes

- Repeated chronic trauma
- The majority of carpal hygromas communicate with the carpal joint. The carpal hygroma can form from a synovial fistula from the extensor tendon sheath or the carpal joint.
- Infectious causes Brucellosis is a rare cause of fistulous withers and poll evil. In working equids the most likely cause of fistulous withers is a poorly fitted harness and onchocerciasis has also been proposed as a possible cause (Doumbia 2011). However, if an equid has been in contact with infected cattle (*B. abortus*) or pigs (*B. suis*), the lesions should be considered infectious in origin. Brucellosis is a zoonotic disease; wear gloves when treating the animal and enforce strict isolation from animals and humans. Brucellosis is generally a notifiable disease; check local regulations. It is reasonable to keep equids separate from Brucella-infected cattle, and cattle separate from equids with discharging fistulous withers.

Clinical Signs

- Diffuse swelling. (Differentiate from carpal distension in which the joint will be involved.)
- No lameness
- Infected hygromas will present as an obvious wound, often discharging purulent material. There may be an associated lameness, particularly if the hygroma is connected to a joint causing a septic arthritis.

Treatment

- Hygromas are generally only a cosmetic problem.
- In the acute stages, firm bandaging and NSAIDs may reduce the swelling.
- Drainage is ineffective and carries the risk of infection of nearby joints and tendon sheaths.
- Infected hygromas will require more aggressive treatment involving sterile flushing and antibiotic therapy.

■ It is important to prevent these lesions occurring by reducing repetitive trauma to a particular site. (For example, improve the harness design to prevent fistulous withers, or provide soft bedding to prevent capped hock.)

Type of Bursitis	Clinical signs	Cause	Treatment and prevention
Bicipital	Painful swelling at point of shoulder	Penetrating injury at point of shoulder Chronic strain injury (conformational or compensatory)	Puncture wounds: treat aggressively with debridement, antibiotics/joint flushing, NSAIDs and rest.
Calcaneal 'capped hock'	Fluid swelling at point of the hock (between gastrocnemius and SDF tendons)	Kicking the cart Trauma from lying down on hard floors without sufficient bedding/padding	Unnecessary if sterile, otherwise treat aggressively. Pad front of cart/protect hock to prevent further injury. Beating will contribute to problem as animals kick in response. Ensure a soft area is provided for the animal to lie down.
Olecranon 'capped elbow'	Fluid swelling at the elbow	Repeat trauma to point of elbow (e.g. pressure from shoe when animal lying down)	Medical treatment unnecessary if sterile, otherwise treat aggressively. Ensure good hoof care/shoeing technique. Ensure a soft area is provided for the animal to lie down in when resting.
Carpal hygroma	Fluctuant swelling over the carpal joint	Repeated trauma to the knees e.g. falling/stumbling on uneven ground Kicking a stable door	Medical treatment unnecessary if sterile, otherwise treat aggressively. Ensure a soft area is provided for the animal to lie down in when resting. Ensure feet are kept well-trimmed to minimise stumbling and do not work at fast speeds.
Atlantal 'poll evil'	Deep discharging sinus midline behind the ears	Ill-fitting headgear Infected wound Brucellosis	Difficult due to position (requires daily flushing). Sedate and clean. Prolonged systemic antibiotics (continue 5 days after signs disappear). Often recurring – warn owner.

Type of Bursitis	Clinical signs	Cause	Treatment and prevention
Supraspinous fistulous withers	Swelling and pain in wither region, can rupture to discharge pus in severe cases. Secondary osteomyelitis	Ill-fitting harness or saddle Onchocerca Brucellosis	Clean and debride. Administer systemic antibiotics if deep. Advise owner on prevention. Keep harness off wound, use a 'doughnut bandage' until healed. Prognosis is poor if osteomyelitis present.
Navicular	Severe lameness following sole/frog injury Pain response to hoof testers across heels	Deep puncture wound to frog or middle third of sole	Poor response to antibiotics/flushing Complications can include navicular fracture. Prognosis is usually hopeless.

Table 14.10.1 Common bursal injuries in working equids, including cause, treatment and prevention.



Figure 14.10.1 Illustrations of common bursal injuries in working equids: calcaneal 'capped hock', supraspinous 'fistulous withers', carpal hygroma and olecranon 'capped elbow'.

14.11

Case study – Malignant oedema

Area India

Attending veterinarians Dr Dharmendra and Dr Alok Shukla

Summary Successful management of a Clostridial infection (malignant oedema) in a stallion

History

A 6-year-old skewbald stallion presented with a history of a ventral swelling that had developed over 3 days. The stallion had been treated by a local unqualified healer, with injections in the neck, using an old needle.

Clinical findings

Ventral oedema was prominent on lower abdomen, neck, scrotum and penis (Figures 14.11.1 and 14.11.2). The horse would not eat or drink. On palpation sub-cutaneous emphysema was present. Oral mucous membranes were congested, with a CRT of 3 seconds. The respiration rate was 28 breaths per minute, and the heart rate 64 beats per minute. Rectal temperature 39.1°C.



Figure 14.11.1 Ventral oedema affecting abdomen, prepuce and scrotum.



Figure 14.11.2 Severe ventral oedema.

Diagnosis

Based on history and clinical examination the disease was diagnosed as malignant oedema (Clostridial myonecrosis) which is caused by *Clostridium* spp., a gram positive anaerobic bacteria.

Treatment

1. Procaine penicillin 20,000 IU/kg BID IM in combination with metronidazole 10 mg/kg BID IV for 7 days
2. A fasciotomy/myotomy procedure was used to minimise the anaerobic environment. This involved making incisions in several sites across the oedematous area.
3. Flunixin 1.1 mg/kg SID IV for 7 days
4. Intravenous fluid therapy
5. Palliative care was provided by the owner; feed and water were raised off the ground to reduce the oedema (14.11.3). Soft bedding and good green feed were provided.

Preventive messages discussed with owner:

- Always ask local healers to use a new (sterile) disposable syringe and needle.
- When an animal is unwell it is preferable to seek advice from a veterinarian or a trained local healer.

Outcome

The recovery was uneventful and animal well again after 10 days (Figure 14.11.4).

Discussion

Malignant oedema, also known as Clostridial myonecrosis, is an acute, generally fatal toxæmia of cattle, equids, sheep, goats and pigs usually caused by *Clostridium septicum*. Other *Clostridia* implicated in wound infections include *C. chauvoei*, *C. perfringens*, *C. novyi*, and *C. sordellii*.

In a retrospective case series, malignant oedema (Clostridial myonecrosis) was diagnosed in nine horses with signs of illness that included fever, depression, painful muscular swellings, and toxæmia. The infection followed intramuscular injections in eight horses and developed in a puncture wound in one horse. Treatment consisted of surgical fenestration of the involved muscle, high doses of penicillin, NSAIDs and analgesics, and supportive fluid therapy.

Five of the horses recovered and four died. Those that died had advanced signs of the disease at admission (Rebhun et al. 1985). A more recent study (Peek, Semrad and Perkins 2003) demonstrates a higher survival rate in horses treated for Clostridial myonecrosis at two equine referral centres in the United States.

The early use of a myotomy/fasciotomy procedure can improve the prognosis in cases of Clostridial myonecrosis. However, deep iatrogenic wounds are created when using this technique and it is vital that these wounds can be managed appropriately throughout the lengthy healing process. It is remarkable that the outcome of this case in India was positive considering the fatality rate of this condition and the prolonged clinical course prior to veterinary examination.



Figure 14.11.3 The owner taking good care of his recovering horse.



Figure 14.11.4 Dr Dharmendra examining the recovered case.

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