THE BLOOD SUPPLY TO THE HORSES FOOT AND THE FARRIER by Tom Ryan A.F.C.L.

Introduction

I hope to show in the following pages the importance of the knowledge of the blood supply in the leg and foot of the horse to the farrier.

In everyday work the farrier only sees blood when a disaster has happened, he dreads the sight of blood, it means he has either misjudged the foot or been careless in the preparation of the foot or in nailing on the shoe.

Blood is the life support system for all tissues in the horse, every part of the body even the innerparts of the hoof wall are maintained by the blood. It carries oxygen and nutrients to the cells and all the elements that go into making the horny structures. The constituents of blood are used for the healing of damaged tissue by breaking down the cells and helping other cells to divide to replace them.

The immune system in the blood is also very important, without it the slightest infection could severely damage the affected tissues and structures.

If the supply of blood to the foot is disrupted for several hours as in laminitis, the laminae in the toe are damaged to such an extent that it will take many months to repair.

Correct methods of farriery may avoid many problems, this is not realisd by some horse owners. Having horses shod on a regular basis by a competent farrier is expensive and the benefits are not always obvious. Today all farriers are under pressure to shoe more horses than they should at a price which perpetuates this practice, this leads to cutting of corners, bad practices and bad tempers, from which all involved suffer the consequenses, but the horse is the only one to carry the lasting effects in the form of damage to his feet.

The farrier should be aware of every aspect of the horses anatomy and the affect he may have on it, the blood supply is part of the hidden anatomy and is not a structure as such, but poor farriery can damage every part of the leg. Corns are damaged blood vessels and if they persist damage to the third phalanx can result. Damage to the heels from badly fitted shoes can cause low weak heels with flat soles which leave the sole open to bruised or punctured soles and the possibility of navicular disease.

Listed in the following pages are first the background of blood and how it reaches the foot, then the main disorders and diseases that I think are related to the blood supply and the farrier. It must be said that the farrier should obtain veterinary guidence before carring out work which is of a remedial nature.

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Blood

Blood has many constituents, its base is plasma in which cells are suspended. The plasma consists of a complex mixture of inorganic salts in solution and blood proteins. The total salts present represent about one percent by weight of plasma, the main salts present are chloride and bicarbonate of sodium.

The blood proteins are of three types, albumins, globulins and fibrinogen.

Glucose and fats are also found in the blood, Glucose is used with oxygen in cells to give energy eg. in muscles.

Corpuscles are of two types, red and white corpuscles. Corpuscles do not divide or multiply in the blood. Red corpuscles are formed in red bone marrow eg. in the cavity of the large metacarpal bone and are used to carry oxygen. Some white corpuscles are formed in the nodules of the lymphatic system, others called myelocytes are formed in red bone marrow. White cells are used as the defence mechanism, they can pass through the walls of blood vessels and accummulate around sites of infection in the tissues to fight foreign bacteria, this results in the formation of pus.

One of the best best known properties of blood is the ability to clot using fibrinogen when a vessel is cut or damaged, by this method further loss of blood is prevented.

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Blood supply

The vessels which carry blood are of two types, arteries which have thicker muscular walls and carry blood in pulses from the heart which divide and terminate in capillaries of microscopic size, and veins which have thinner walls and converge together returning blood to the heart. Arterial blood is bright red because it carries fresh supplies of oxygen and veinous blood is dark red in colour because the oxygen has been spent.

Arterys

The main artery to the leg and foot is the large metacarpal artery (metatarsal in the hind limb) or common digital, it descends the leg on the medial aspect of the flexor tendons (on the inside of the leg next to the flexor tendon). Above the fetlock joint it divides to form the lateral and medial digital arterys, the lateral digital artery which passes under the flexor tendon to the outside of the leg and the medial digital artery which travels down on the inside of the leg. Both the medial and lateral digital arterys progress down each side of the digital flexor tendon until they reach the inner side branches of the third phalanx where they divide again to form the preplanter and planter arterys. The preplanter artery passes through the notch in the wing of the third phalanx and along the preplanter groove to supply the sensitive laminae of the quarters with blood. The plantar artery passes along a groove on the inner side of the wing of the third phalanx just under the distal sesamoid bone (navicular bone) and enters the third phalanx through the foramen to meet the other plantar artery forming the terminal arch within the third phalanx. From this point numerous small arterys pass through the porous third phalanx (but not through the solar surface) to many of the structures around the bone including the distal sesamoid bone and to fill the circumflex artery. Other arterys which are supplied by these main arterys are those of the first and second phalanx and of the digital cushion. By this network all the structures of the leg and foot are supplied with nutrients and oxygen.

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Veins

Veins are the drainage system of the body they collect used blood and waste and return it to the heart. Veins have non-muscular walls and prevent a backward flow of blood by the use of non-return valves.

There are three main networks of veins to drain the foot. These are the solar, the laminal and the corary veinous plexuses, the three have to collect a vast amount of blood from the capillaries mostly from the horn producing areas, returning it back up the leg to the heart.

The three venous plexuses join to form the lateral and medial veins which follow the same path as the lateral and medial digital arterys. Above the fetlock the lateral and digital veins join together at a point between the deep digital flexor tendon and the suspensory ligament forming a venous arch. From this point three veins go up the leg, the medial following the digital artery, the lateral vein follows the lateral planter nerve, and the deep metacarpal vein follows between suspensory ligament and the medial small metacarpal bone.

At the base of the lateral and medial veins there are no non-return valves, this is because the opening and closing of the heels caused by compression of the digital cusion also compresses the veins stopping backward flow of blood, also as the veins are compressed the blood in them is pumped along the vein back up the leg. The need for this pumping mechanism is because the foot is the lowest part of the body and furthest away from the heart, this movement of blood will also absorb energy from the digital cushion.

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Circulation

Blood circulates in a particular way in the normal foot, when timed it takes approximatly 12 seconds for blood entering the lower leg just above the fetlock to pass down to the foot and return.

Angiographic examinations (X rays using a radiographic marker in the blood) carried out by C. Colles at Equine research station Newmarket show that the terminal arch fills 1.5 seconds after injection of the marker, at the same time the marker shows in the vessels to the coronary band in the front of the foot and the bulbs of the heels.

The circumflex artery which is supplied by the terminal arch fills after 2 seconds, also some small vessels in the bulbs of the heels have filled. As soon as the circumflex artery is completely visible the veins near the artery start to show, the artery to the coronet from the terminal arch is also seen.

Five seconds after the marker was injected all marked blood had left the terminal arch and the veinous plexus of the sole become visible as do the veins draining the bulbs of the heels into the medial and lateral digital veins.

Only at about this time, the supply of arterial blood to the laminae in the toe become evident, moving in an upward direction from the circumflex artery, the laminae at the toe and the coronet are eventually drained by two veins which goto the digital veins.

Arterial supply in the laminae seems to be in an upward direction at the toe supplied through the third phalanx via the circumflex artery, but at the heels flow seems to be downward from the top to the bottom of the laminae, the flow of arterial blood to the sole is from the circumflex artery across the sole to the bulbs of the heels. No blood passes through the solar surface of the third phalanx.

One of the conclusions of this work is that the supply of blood to the toe laminae and to the laminae in the heels is from different local arterys and arterial flow in the laminae is in different directions.

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Horn Growth

All horn growth is dependent on the blood supply, the amount of blood needed is shown by the vast network of vessels in the horn growing areas of the foot (see Blood supply page 3).

Any fluctuation in supply or quality of blood will cause the horn growth to vary, this will produce rings of uneven growth called grass rings.

Horn is produced at the coronary band or cushion which supplies numerous micro-blood vessels to each of the papillae which allow the horn cells to divide. As they grow down they harden (keratinize). Horn is made of three main parts, the fibrous horn tubes, the centre of the tubes called intratubular horn and the intertubular horn which bonds the horn fibres together.

The combination of the tubular and intertubular horn makes a strong yet resiliant structure able to carry the full weight of the horse and protect the structures of the foot. The shape of the wall is crucial to its strength, this is where the farrier plays an important part. If the wall is allowed to grow long, it cannot be supported by the other parts of the foot and may splay out. This is a weak shape and will cause cracks inbetween the horn fibres and parts of the horn will break back past the sole level.

The main indicator of the blood supply to the horn producing areas is the grass rings, these give a "history" of growth (Fig. 7 page 22) and will show changes in season, exercise, wear, trimming and injury or disease. In the spring and early summer, horn is grown at a faster rate, this is probably because of the extra nutrients in the lush grass. Exercise or work will increase the circulation to compensate the demands for oxygen and also repair wear on the structures within the foot. Wear and trimming place pressures on different areas of the foot which may increase or decrease blood supply. In some diseases there are marked growth patterns such as that found in laminitis (see laminitis page 9). Damage caused by treads and quittors (Fig. 12 page 27) will stop horn growth for a time until the coronary band is repaired. When growth is resumed a horizontal crack will show the point of injury.

Horses and ponies which grow horn quicker than it is worn may not need to be shod at all but will required to be trimmed and balanced up from time to time.

If the feet grow too long, strain will be placed on the laminae, this will show its self in the form of redness in the white line. This damage must cause pain or discomfort because as the hoof grows, less of the horses weight is carried by the frog and digital cusion, this causes more weight to pass to the laminae, strain will increase as the feet grow longer and the the foot tips back on its heels, this condition will also cause strain on tendons and ligaments of the foot and leg, this should of course be avoided.

Flat feet

Flat feet or feet without concave soles seem to be prone to many problems, although not directly related to the blood supply they are refered to in several places in this text and so the subject is briefly covered here.

Symptoms

In the normal foot the sole is concave, this is a mechanically strong shape. The arch of the sole is like the arch of a bridge. In the flat foot the sole is not concave and although it is the same thickness and size it cannot support the same weight or give the same protection. A flat foot has low weak heels, a well developed frog, a flat sole and the wall is usually splayed out. The structural strength of the foot is lost, this is a considerable weakness and leads to many problems.

Causes of flat feet

Some horses may inherit flat feet but many more develop them through one of two reasons, from shoes that are left on too long, giving the same effect as the second cause, which is from badly fitting shoes. In either case the foot overgrows the shoe and pressure is placed on the "seat of corn" (see corns page 8) and on the bars of the foot. Every time the foot expands with frog pressure the foot opens over the shoe, but because the shoe is on the inside of the wall the foot cannot contract again, this will damage the wall and bars and cause flat feet.

Treatment

Prevention is of course the best action. Once the foot is in this condition the only thing the farrier can do is to not dress the heels too low and try to improve the foot axis, also the wall should be dressed to make the wall straight. The owner should be told not to leave the horse longer than a month between shoeing and care should be taken when fitting the shoe at the heels to allow for the expansion of the foot and horn growth, shoes with a wide web and good cover at the heels will help (Fig. 3 page 18).

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Corns

A corn is a bruise situated in the angle between the bars and the horny wall. A bruise in any other position is not called a corn (a bruise is escaped blood from damaged vessels). Corns are usually found on the inside heel of a foot with low weak heels, the redness that can be seen is from blood which has escaped from blood vessels damaged by pressure between the retrossal process and the horny sole which damages the blood vessels in the sensitive sole (Fig. 2 Page 17). Corns are usually found in horses that are shod but it could also be said that unshod horses do not work hard. I think that the main cause of corns is a poor standard of shoeing.

It may be that the farrier is not to blame, in some cases the shoes may be left too long before being removed and the feet dressed (Fig 1 page 16) or the horse is worked too hard or too long on the road but in most other cases the farrier is at fault. If the shoes are fitted narrower or shorter than the foot, or if the bearing surface is too narrow this will weaken the heels and cause the foot angle to differ from the pastern angle (the foot becomes flatter), if pressure is put on the "seat of corn" then corns will result. If a horse has had a corn for some time, the retrossal process of the third phalanx (Pedal bone) may be damaged giving signs of pedal ostitis, in these cases it is very difficult to cure the corn.

In all cases pressure must be removed from the corn by thinning the sole at the place of the corn and by using a shoe that will distribute the pressure (ie. broad webbed shoe (Fig 3 page 18), set heel shoe for corn or a bar shoe). The shoes should be fitted longer and wider to support and allow the heels to grow improving the foot axis. The shoes should be on the horse no longer than a month to ensure that the feet do not over grow the shoes. Hard or fast work should be avoided because of the jarring or resulting extra pressure.

In some cases (as in laminitis where there is a discharge of fluid and heat) the corn becomes infected and pus is discharged. In cases when the pus is enclosed and cannot discharge, pressure builds up and the pus tries to find an exit, it usually travels up to the coronary cushion causing a quittor (exit of pus through the coronet). Usually this type of corn is called a pipe corn because the pus moves up inbetween the laminae forming a type of pipe.

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Laminitis

I am begining to feel that the name of laminitis is misleading as it has been shown the laminae do not get inflamed, but scar tissue forms where the horny and sensitive laminae have been torn apart by forces exerted on the laminae by the weight of the horse. In the normal foot the laminae are an interlocking bond between the horny wall and sensitive tissues. Where the horny wall is next to the third phalanx, the weight of the horse is passed from bone to hoof wall. The sensitive laminae have many blood vessels and nerve endings, injury to the laminae gives much pain.

Causes of laminitis

Laminitis is caused by a disorder of the cardiovascular system in the horse. Although it shows itself mostly in the feet, it affects the whole body. It probibly shows mostly in the feet because this is the lowest part of the body and the furthest away from the heart and the circulation to the laminae in the toe is easiest disturbed.

If the supply of blood to the toe is restricted and if it is not treated very quickly the cells in the laminae can be damaged by a shortage of oxygen. This leads to breaking down of the bond between the horny laminae and the sensetive laminae (which normally carries most of the horses weight) and rotation of the third phalanx (Fig 6 page 21). The rotation happens because the third phalanx is supported at the back by the digital cusion but at the toe the bond of the laminae is weakened perhaps because of the uneven horn growth. Any circumstance which causes constriction of the blood vessels could cause an attack of laminitis. The main causes are protein poisioning (the horse that gets in to the feed bin and gorges himself) or from being overweight and eating too much spring grass or by poisioning of any kind.

Symptoms

There are two stages of laminitis, acute (sharp or sudden attack Fig. 10 page 25) and chronic (long established Fig. 5 page 20).

The horse may have a crest on its neck and will be leaning onto his hind feet with the front feet outstretched. His hind feet will be forward to take the weight off its front feet onto its less painful hind feet.There may be no distortion of the feet in acute cases, but in the coming weeks if it is not treated chronic laminitis will develop. Scar tissue will show in the gap between the horny and sensitive laminae (Fig. 5 page 20). The toe part of the wall will be pushed forward by the faster growing wall at the quarters because it receives a normal or better supply of blood than the slower growing toe (Fig 7 page 22). This is because blood which should go to the laminae via the circumflex artery "shunts" directly to the

circumflex vein, so there may seem to be a good blood supply but it is not reaching the small arterys to the laminae in the toe. Because horn growth at the coronet is slow it cannot keep pace with growth at the quarters, the coronet may be pulled down so that it dips at the front. The third phalanx will no longer be held in place at the weight carrying toe and will move downward and backward in a rotational movement. This is probably because there is support to the back part of the third phalanx by the shock absorbing digital cusion. The sole will lose its concave shape and at the least flatten or become convex, this is because the weight which was carried by the laminae in the toe through to the third phalanx is now passes direcly to the sole. A bruise will form just in front of the point of the frog this is from vessels damaged by the rotating third phalanx (Fig 8 page 23). Flow of blood across the sole may be restricted by the rotating third phalanx. The bond between the sole and wall will show signs of scar tissue in the toe. When this happens blood and a clear fluid is discharged through the scar tissue. The discharge is often accompanied by abcesses which are caused by invading organisms which thrive in the warm discharge. In these cases lameness shows more in one foot than the other, this can be mistaken for laminitis in one foot but usually it is an abcess.

In horses with strong walls, weak soles and nearly always long feet there is a risk that before the hoof wall deforms all movement of the third phalanx is downward through the sole. Because the sole is not strong enough to support all the weight which was carried by the laminae it now passes to the unsupported sole, which collapses giving a dropped sole and in some cases the bone protrudes through. The bone will have the corium of the sole (soft tissue also known as the sensitive sole) covering it and will emerge just in front of the point of the frog. When this happens abcesses will be found as well. In the two cases that I have experienced the owners have felt the horse was well on the way to recovery when this condition was found.

Where the rate of hoof growth in the toe is slower than at the heel which has a better supply of blood, growth will be of a an uneven rate (Fig. 7 page 22). As the horse will be reluctant to move, hoof wear will be slow, so in a short period the horn becomes quite long (Fig. 11 page 26), in months the hoof may be curling up if not trimmed.

The horse in itself will be in acute pain, reluctant to move if standing. Some lay down, occassionaly some "sit" on mangers to take the weight off their feet. In most cases the horse will be sweating or blowing (breathing heavily because of the pain).

Treatment

As said before all treatment should only be carried out under veterinary instruction.

If the vet and farrier are called as soon as possible and the owner is willing to put much effort into the management of the horse, then he should recover in months. If the owner is lazy in the care of the horse, then laminitis can last for years so the owners co-operation is the most important part in the horses recovery.

In acute laminitis the farrier should remove the shoes and rasp the wall at the toe away to the laminae (Fig. 9 page 24). The heels should be left at their normal hieght as there is no rotation of the third phalanx at this time.

In chronic laminitis, the genearal rule for the farrier is to maintain the correct position of the third phalanx, which in most cases is rotating, by triming the heels. The wall will be growing out and should be rasped severely to nearly the coronet to maintain the same angle as the pastern. This will reduce the pull on the laminae at the toe and may reduce distortion. The horse is best kept unshod (this will support the third phalanx at the sole) and kept on a peat bed in a covered yard so he can move around (if he is able). This will provide a dry surface which will reduce the risk of infections (abscess). The owner should walk the horse on a soft non-abrasive surface at frequent intervals if possible. This will be painful at the begining of each session, but the horse will feel better after a few minutes as the blood circulation improves. In time (up to and beyond a year) the hoof will recover its correct shape providing a full blood supply is restored to all the tissues of the foot.

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Navicular Disease

The navicular bone is also called the distal sesamoid bone and is found at the rear of third phalanx, it enlarges the surface area of the joint of second and third phalanx, it also acts as a fulcrum for the deep flexor tendon.

The navicular bone is anchored in place by the interosseous and suspensory navicular ligaments. Through the interosseous ligament 8 or 9 arteries pass supplied from the terminal arch in the third phalanx. Proximal supply is from 2 or 3 arteries passing through the suspensory ligament supplied from the palmar artery of the second phalanx. The palmar artery is supplied by the lateral and medial digital arteries.

The deep flexor tendon passes under the distal sesamoid and is cushioned by the navicular synovial bursa.

When born the horse has a basic supply of blood to the navicular bone, within 48 hours of birth the diameter of the arteries has increased and in 7 days the arteries have developed many small vessels within the bone to meet the increasing demands. After 4 to 6 months the supply to the flexor cartilage is reduced, so there is very little trace of it.

In two or three years the supply has developed so the arteries along the distal border penetrate the bone and are responsible for the small foramen found in this area.

The blood supplied to the navicular bone could be said to increase with the demands placed on it.

Causes of navicular disease

The main cause of navicular disease is thrombosis of the vessels supplying the navicular bone. This causes a shortage of blood and this brings about ischaemic changes in the bone which in turn cause pain. This condition may also be aggravated by spasmodic work patterns and a low hoof pastern axis.

Symptoms

Navicular disease starts with intermittent lameness which is most noticeable the day after hard work and at this early stage rest will ease symptoms.

To the farrier the symptoms are several, the first signs to look for are that the horse dislikes frog pressure, that is to say there will be greater wear in the toe region of the shoe than the heels. Pointing of the foot with most of the weight on the toe is another sign, this is most evident when the horse is grazing or feeding from a bucket on the ground, when the same foot is rested repeatedly. In more advanced cases the affected foot will be slightly narrower although it may still be low heeled. In advanced cases it may be quite upright.

To the rider the horse may try to avoid the camber at the side of the road and try to go to the middle or onto the

verge, going down hill also causes discomfort. The horse may stumble, this is usally because he is trying to put his toe down first and not over flex the pedial joint. When jumping the horse may refuse or not reach his normal height because of the pain when landing.

The most reliable method of diagnosis is X ray. The changes are best seen in advanced cases. The changes visible are osteoporosis (small spaces in the bone tissue) and enlarged vascular channels or foramen. These enlarged foramen are a response to navicular disease, not a cause and are formed when vessels in the periostium develop to supply blood to the affected areas.

Treatment

First of all it should be said that in a sound horse having low week heels, all effort must be used to correct the hoof pastern axis. The same can be said in navicular cases, this is best done by rasping the wall to match the angle of the pastern (by no means as much as in laminitis) and by not rasping the heels. The horse should be shod using a wide webbed shoe with a rolled toe and fitted wider and longer than the foot to support the weak heels and prevent the heels being damaged when they grow. The shoes should not be left on longer than one month.

Treatment using warfarin developed by Chris Colles M.R.C.V.S. at the Equine Research Station, Newmarket, has proved very successful by allowing the blood vessels in the periostium time to replace those lost through thrombosis. These areas of thrombosis will be reabsorbed by the new blood supply in time.

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Punctured soles

In all puncture wounds the white blood cells are called into action to protect the body from invading organisms. These corpuscles can pass through walls of vessels and accumulate at the sites of infection to surround and digest foreign micro-organisms. White cells and debris are ejected from the body as pus.

The sole protects the third phalanx and sensitive tissues from damage, so when the sole is damaged it has already taken much of the force of impact so although the sole is punctured it has given protection, but the body is now open to infection.

The next line of protection is the white blood corpusles, which attack any foreign bodies which may be found and neutralise them. As the volume of white cells builds up it will wash the foreign bodies out providing there is an exit. If the sole closes up after the puncture happens the pus can not be discharged and is trapped inside the sole. The pressure of the pus inside the sole builds up as more white cells are produced forming a cavity. The foot becomes warm and the leg may swell just above the fetlock around the flexor tendons.

If the extra pressure does not reopen the puncture hole then the next stage is for the pus to travel along the sole following the path of least resistance, usually towards the heels. If it still cannot find an exit then the pus will follow the laminae to the coronet and burst out between the coronary cushion and the horny wall to form a "quittor" (Fig. 12 page 27).

The quittor will disrupt horn growth at the coronet for a while as the pus is discharged, leaving a horizontal line in the horn which will grow down with the hoof. In time, when the infection is defeated, nomore pus is produced and normal wall and sole growth can resume.

In a few months time when the foot is being dressed for shoeing, the cavity which held the pus will be become visible. This in some cases is the first time it may be realised that the horse had a punctured sole.

What has just been described is the course that an undetected puncture wound usually takes.

If a puncture wound is suspected then the shoe should be removed and feet dressed so as to clean the foot. Then the foot should be then examined for marks which may show the site of puncture. In established cases these may be black marks which should be cut back to determine if they are the cause of the trouble. Once the abscess has been found, the cavity should be opened to allow drainage of pus, this will lessen the risk of a quittor. The drainage of pus will reduce pressure and pain.

The horse should be kept on a clean dry surface and poulticed to encourage drainage, tetanus cover should be checked.

Conclusion

The subjects in the preceding pages have shown that the blood supply is fundamental to the well being of the foot, and any event which disturbs the circulation of blood can have far reaching consequences.

If the farrier is unfortunate to make a foot bleed he should remember that the bleeding is the first stage in the repair of the foot.

Knowledge of the position of the blood vessels shows the farrier that he should do all in his power to reduce the risk of risen clenches, as shown in the section on arterys and veins, they are positioned at the point on the fetlock where many horses strike themselves.

The position where the nails are placed in the shoe could affect the opening and closing of the heels, if the nails are placed in the heel part of the shoe, also this will restrict the shock absorbing action of the foot and the pumping effect.

With a wider understanding of navicular disease it may be possible to avoid many of the conditions which precede it. Knowledge of the circulation within the foot can only help the farrier in conditions such as laminitis.

I believe the farrier should do all in his power to inform his customers of the need for regular shoeing and he should make every effort to shoe the horse correctly. The effect of bad shoeing may not be obvious on the first or second shoeing but in time the feet will alter and be very difficult to correct.

The need for remedial shoes in most cases should be seen as a failure in the past to shoe the horse correctly.

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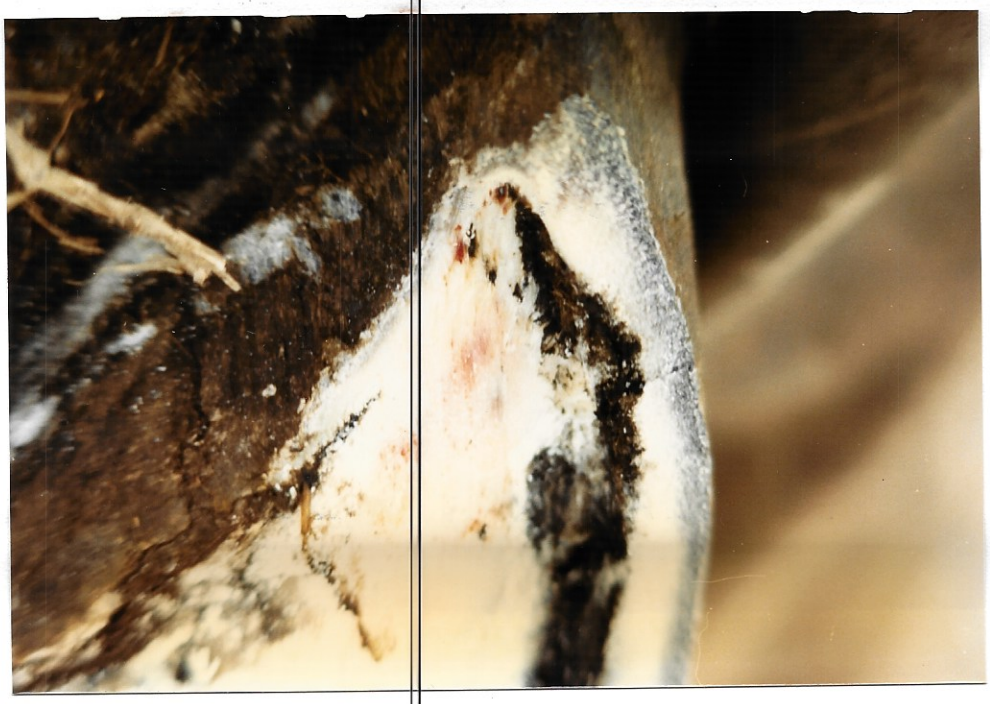
Fig. (1)

Corns
This shoe has been left on too long and the shoe has been over grown, pressure is now placed on the "seat of corn".

Fig. (2)



The shoe has been removed to show bruising of the sole from pressure on the sensitive sole between the third phalanx and horny sole.
Note also the damage to the bar from the shoe.

Fig. (3)

A flat shoe suitable for corns and low weak heels, made from 1" x 1/4" (Mordax nail requested by owner).

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Fig. (4)

Normal laminae, the width of laminae should be the same from heel around the toe to the other heel.

Fig. (5)

Foot surface of a horse with chronic laminitis, note the damage to the laminae and bruising of the sole at the point of frog.

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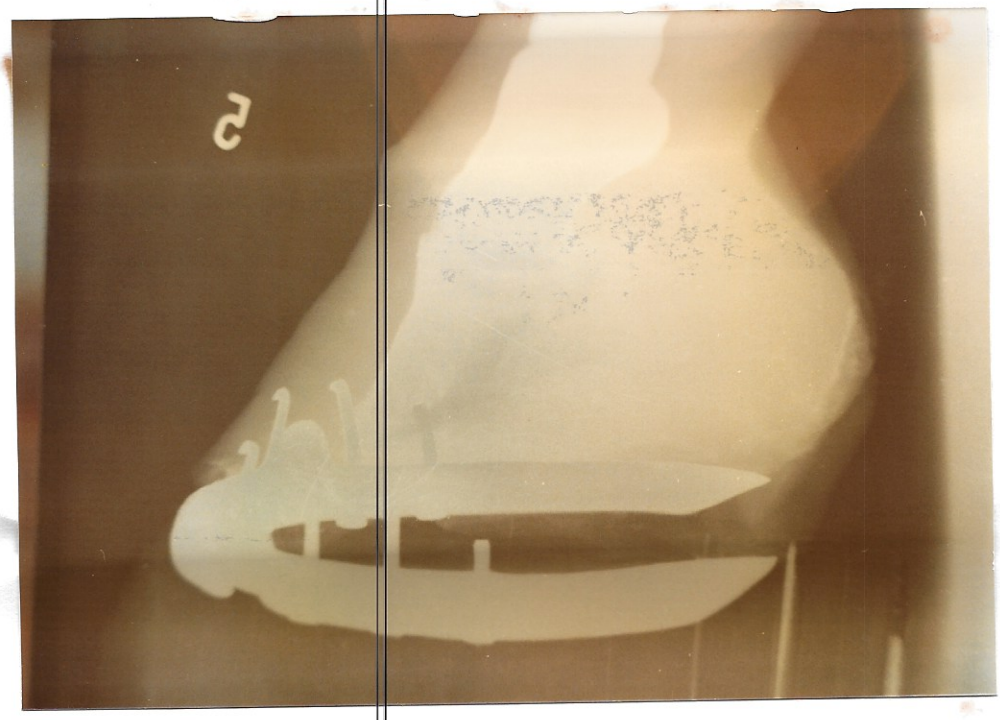
Fig. (6)



X ray of a foot showing rotation of third phalanx. Note the anterior line of the third phalanx is not parrallel with the wall and the third phalanx is not in line with the second phalanx.

Fig. (7)

This horses feet have been neglected for some time as well as having laminitis.
The rings in the horn give the "history" of the horse. At the end of the toe the rings are paralell, higher up the rings are closely spaced but at the heel are more widely spaced, this shows the uneven growth found in laminitis.

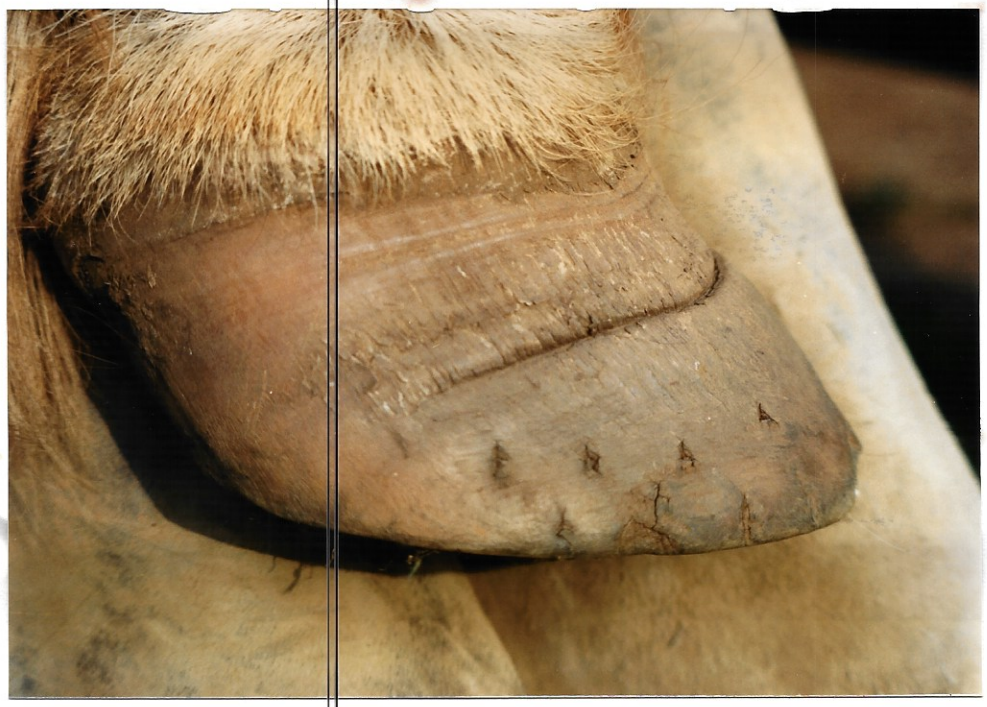
Fig. (8)

Brusing of the sole from the rotating third phalanx in laminitis.

Fig. (9)

Treatment for acute laminitis.
The wall of the hoof is rasped away at the toe but tapering to the normal thickness at the coronet.

Fig. (10)



This shows a very acute attack of laminitis several months ago, it also shows the heel part of wall was not afected.

Fig. (11)

Long laminitic feet (the same feet are pictured in Figs. 5 and 7)

Fig. (12)

A quittor which has finished discharge and was associated with laminitis (fig. 10).

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