PLEASE FIND ATTATCHED MY ESSAY ON LAMINITIS.

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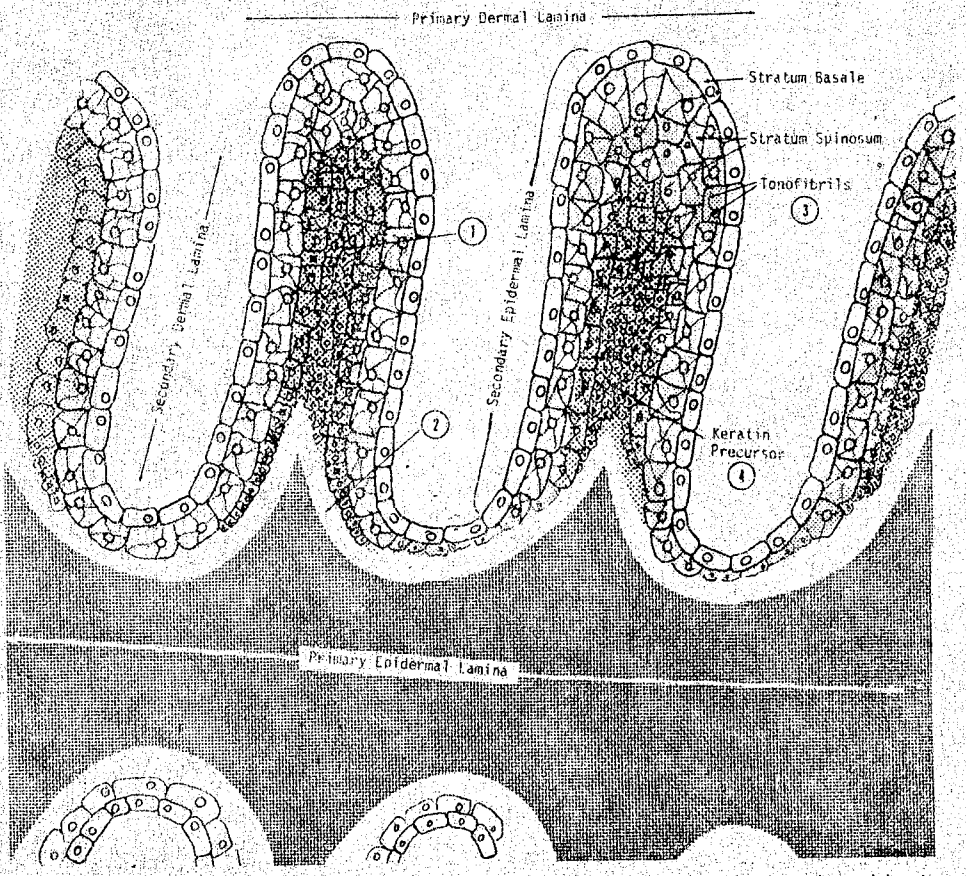
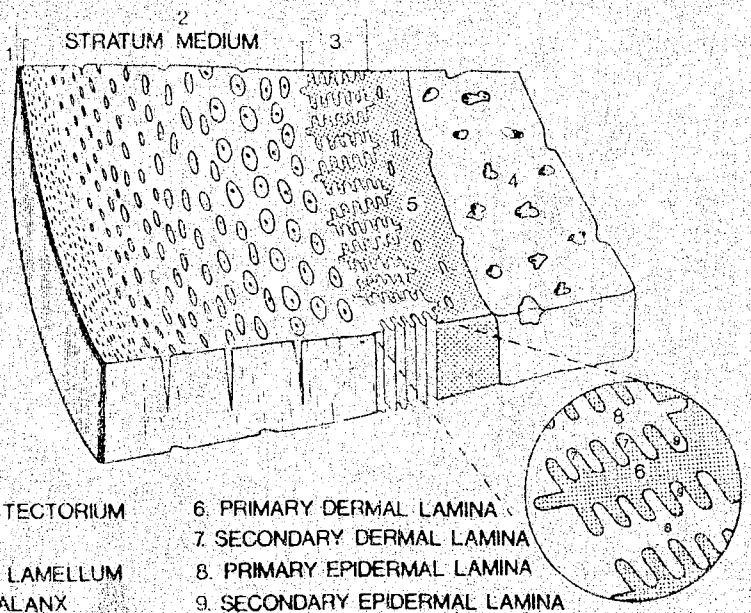


Fig. 1—Diagrammatic arrangement of the primary and secondary dermal and epidermal laminae.



1. STRATUM TECTORIUM
2. WALL
3. STRATUM LAMELLUM
4. THIRD PHALANX
5. LAMINAR CORIUM
6. PRIMARY DERMAL LAMINA
7. SECONDARY DERMAL LAMINA
8. PRIMARY EPIDERMAL LAMINA
9. SECONDARY EPIDERMAL LAMINA

Fig. 2—Anatomy of the normal hoof laminae.

The subject which I have chosen for my essay is Laminitis. I intend to discuss some of the known causes, and the effect the disease has upon the horse; also the treatment which concerns myself, as a farrier. I do not propose to deal with any of the treatments concerning drugs. I believe that the treatment of this disease especially, Veterinary Surgeons and Farriers must work together, in order to alleviate suffering and discomfort to the horse, due to laminitis.

Statistics show that male and female horses run an equal risk of contacting laminitis. Between four and seven years were found the high risk years for females and seven to ten years were found the high risk years for males. Ponies were found to have a greater risk as far as the case numbers were concerned, but horses had the greatest potential for the severe crippling effect of laminitis. A ratio of total body mass to weight bearing surface, is related to potential severity. This put ponies on the opposite end of the scale from thoroughbreds, in terms of laminitis risk. Highest risk period for ponies is, during the lush growing seasons of spring and autumn. It is very apparent that management, dietary, genetic and stress factors may be responsible for acute laminitis.

Laminitis is the disease which is caused by inflamation of the laminae of the foot, chararterised with the congestion of the laminae with blood. Severe pain results from pressure on the sensitive laminae. It has two degrees of severity namely, Acute and Chronic Laminitis. Whilst it can involve all four feet, the front are most commonly affected. Pedal Rotation and Ostitis are often a sequel to laminitis.

To understnd the changes that occur during the disease we must first look at the laminar region of a normal hoof. (See drawing on opposite page)

There are two types of laminae, Dermal or sensitive laminae and Epidermal or non sensitive laminae.

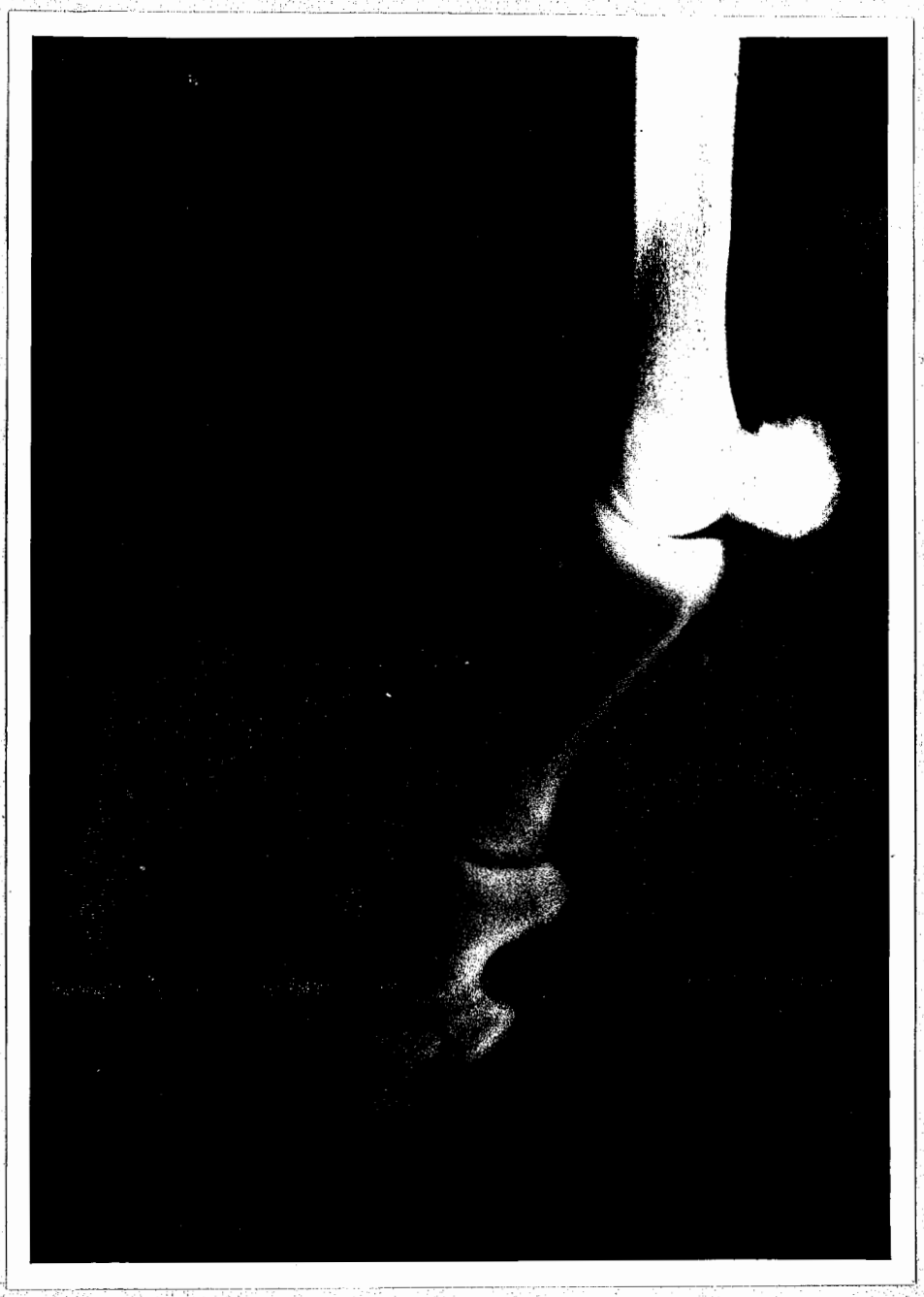
Dermal Laminae

These are formed fromthe corium and are attached to the third Phalanx or pedal bone, (which I shall refer to hereafter a P III.). They contain blood vessels which, nourish both dermal and epidermal laminae, the latter being nourished by capillary diffusion.

Epidermal Laminae

These interlock with the dermal laminae in a form of "Dove-tail" joint which forms an immensly strong bond between the hoof wall and P III. Both the Dermal and Epidermal laminae are further microscopically divided into primary and secondary dermal and epidermal laminae.

Inter digitation of the primary and secondary dermal and epidermal laminae provide mechanical support for the relationship of P III to the hoof wall. The dermal laminae as well as providing blood, they also provide the nerve supply to the stratum basale. At the toe, the dermal laminae are positioned between the hoof wall and P III. Unl[illegible] the corium of the wall has a laminar configuration.



The X Ray is a laminitis case, 12.2hh Welsh pony, three weeks after her first acute attack. The pedal bone has rotated causing the sole to drop. The separation of the laminae is seen as a dark area in front of the pedal bone.

The arterial blood supply to the laminae of the wall consists of anastomatic branches of the terminal arch, the circumlex artery, and the dorsal artery of P III.

There are numerous causes of laminitis, not all of which are fully understood. Namely:-

1. Rations high in carbo-hydrates, including lush growing grasses.
2. The excessive intake of cold water.
3. Retained afterbirth in the womb of a mare.
4. Over-exercise on hard surfaces.
5. Standing still for long periods ie. boat journeys.
6. Can also be sequel to pneumonia or an allergy.

Those causes most commonly recognised include:-

Grain Founder

This is the ingestion of greater quantities of grain than can be tolerated by the horse's digestive system. Generally occuring when the horse has accidental access to excessive ammounts of grain. The grain most commonly are wheat, corn and barley. Many other types of feeds can cause laminitis. ie. chicken, rabbit, calf or pig food. It is normally twelve to eighteen hours after ingestion before signs of laminitis appear, after causing a delay in treatment.

Water Founder

The ingestion of large ammount of water in an overheated horse, although it is not fully understood, it may be due to gastro enteritis. Therefore horses should be allowed only a small ammount of water after exercise and walked round in order to cool down gradually when overheated.

Road Founder

As the name suggests is due to excessive or fast work on hard ground. This particular type of founder is mainly found on thin walled, and thin soled animals and is very traumatic form of laminitis often resulting in pedal ostitis and severe sole bruising.

Post - Paturient Laminitis

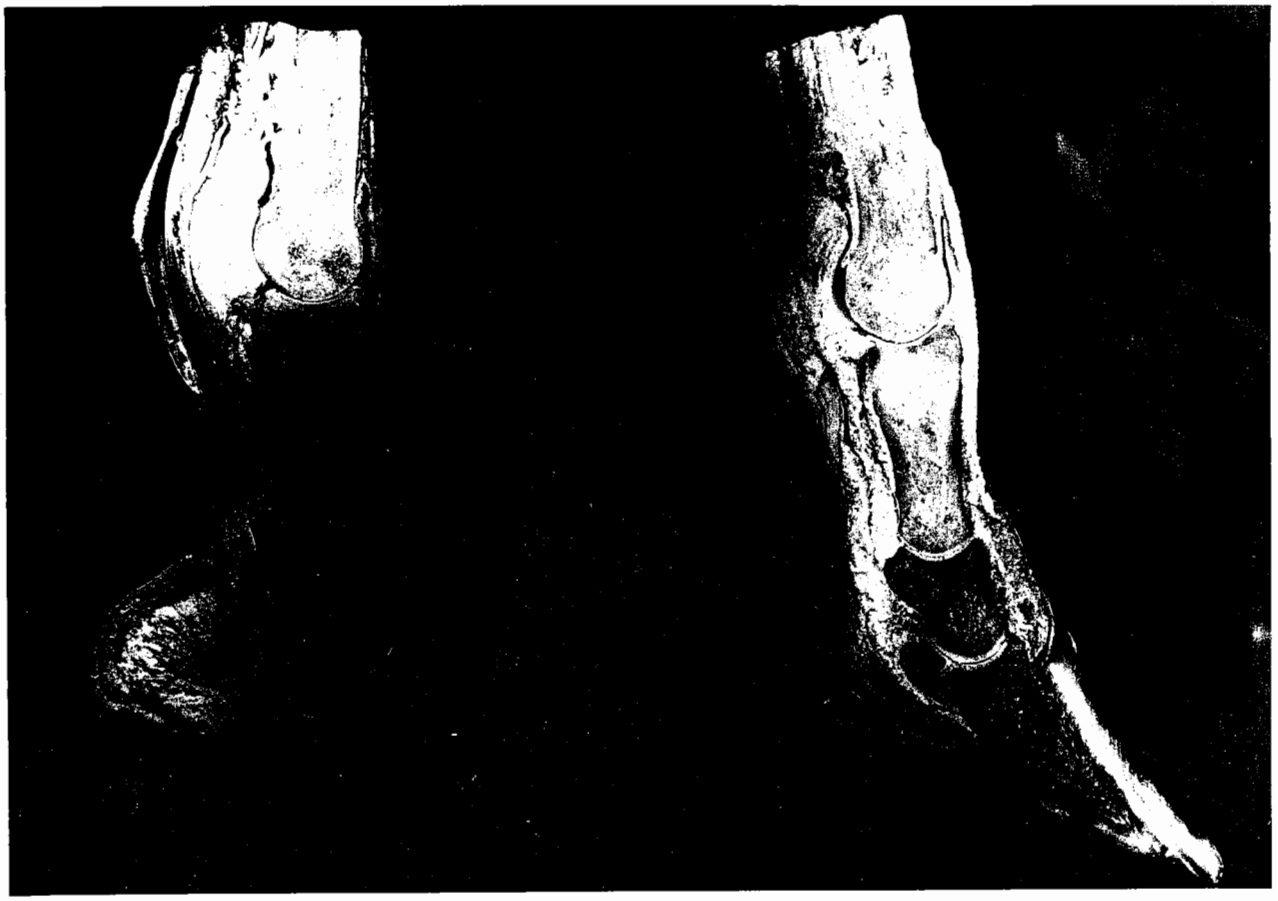
This type usually developes in a mare shortly after foaling, following an infection from retained afterbirth. Often a very serious form of laminitis, it may also follow pneumonia.

Grass Founder

A very common type of laminitis amongst horses, or especially ponies, grazed on summer grass. It is most commonly found in overweight horses. Those affected, usually develop a heavy crest caused by a build up of fatty tissue.

There are many other miscellaneous causes of laminitis which have been suspected by Veterinarians, but never proven. In fact, any number of Psychological stress factors could cause it.

Laminitis has two recognised degrees, Namely:- Acute Laminitis and Chronic Laminitis.



The section on the right is normal with the pedal bone parallel to the hoof wall. The foot on the left shows separation with the pedal bone rotated and pressing down on the sole.

Acute Laminitis

'Obel' characterised the degree of lameness in Acute Laminitis according to the following:-

Grade 1. When standing, the horse incessantly lifts the feet at intervals of a few seconds. It is not lame at the walk, but very short and stilted at the trot.

Grade 2. The horse will move willingly at the walk, but the gait is stilted. A fore foot may be lifted without difficulty.

Grade 3. The horse moves reluctantly and vigorously resists any attempt to lift the forefoot.

Grade 4. At this stage the horse does not move without being forced.

Inside the hoof wall, the cause of the lameness is the pooling of blood within the capillary beds of the Laminar corium. Pedal rotation occurs within seventy two hours and separation of dermal and epidermal laminae at the junction of the primary and secondary epidermal laminae takes place. Severe degeneration of the wall laminae occurs primarily at the toe. That portion of laminar tissue between the wall and P III being damaged more than the quarter or heel. As pedal rotation progresses, pressure necrocis occurs in the corium of the sole dorsal to the point of the frog. In some severe cases where marked laminar changes have occured, serum accumulates providing an ideal place for bacterial growth.

In the normal hoof, microcirculation accepted concept, suggests that only a small ammount of capillaries are perfused at a given time. Capillary dilation occurs by the release of histamine. The resultant perfusion washes histamine away and the capillary closes. Thus capillary perfusion occurs on alternating basis. Capillary walls of sufficient integrity, allow an exchange of ions, nutrients and wastes of cellular metabolism, in addition to maintining a proper fluid balance between the cells interstitium and plasma. Red blood cells traverse capillaries of lesser diameter due to the ability of red blood cells to change shape.

Circulatory Changes In Hoof Affected with Laminitis

Many cases of acute laminitis are initiated by digestive upset or metritis, as suggested earlier. In both instances a common denomonator exists in Bacterial Endotoxin. The constriction caused in both arteriols and venules, results in capillary congestion. Resultant arteriovenous shunts, would then by - pass the capillary beds in the laminar corium. The fact that the digital capillaries are enclosed in the near rigid hoof, compounds the effect of any pooling in the laminar capillaries. In the hoof heat is present over the sole, wall and coronary cushion and if a hoof tester is used it will be found to be tender over the entire sole area.

Chronic Laminitis

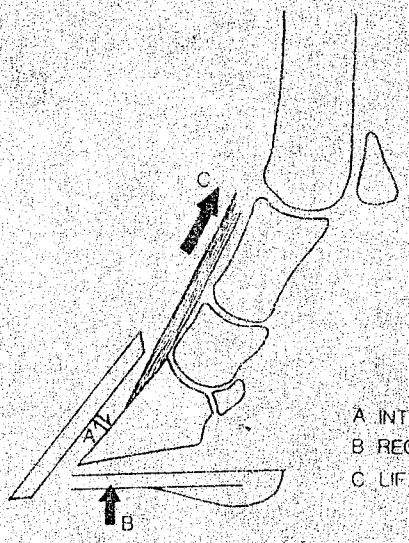
This is a sequel to acute laminitis, and is characterised by the change of shape of the hoof and type of lameness. Horses suffering from chronic laminitis have a tendancy to land on the heel, in an exaggerated motion. The sole is dropped and flat (often convex), due to pressure from the rotated pedal bone. The horn at the junction of wall and sole in the toe region especially, is very soft and pumice-like. Chronic laminitis causes very heavy ring formation on the hoof wall, due to an inflamation of the hoof wall. Another feature commonly found is seedy-toe, resulting from the separation of the laminae. An infection similar to thrush, may invade the sole and destroy all protection of the pedal bone.

Morphologic change in the perfect hypothetical hoof and various therapies Suggested:-

Pedal bone rotation invariably happens as an aftermath of laminitis occuring, sometimes as early as eighteen hours. This is caused by the pull of the deep flexor tendon on the posterior surface of P III, and inflamation of the toe. Considering this, the logical therapy would seem to be, elevation of the heels to ease the pressure on the tendon.

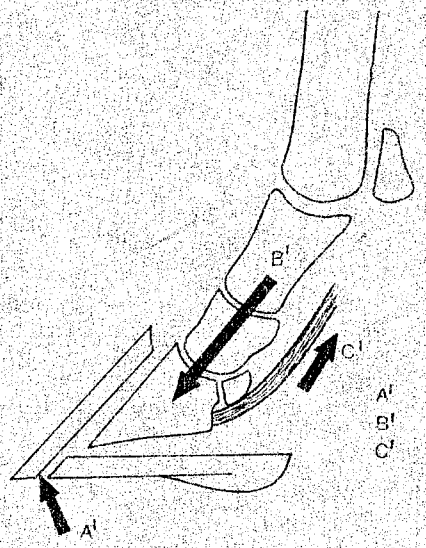
In exact contrast to this, it is suggested by most leading authorities, that we lower the heels drastically of those horses affected with laminitis. The excessive body weight of the animal being a major contributing factor to pedal rotation, shoeing with leather pads packed with tar or plastic compounds to support the sole, have also been recomended. In order to correct severe cases of Pedal rotation, it has been suggested that the wall at the toe, be drastically thinned in conjuction with lowering the heels.

In order to determine the various methods of therapy, we must first examine the structures supporting the normal hoof and the forces favouring displacement of the pedal bone in laminitis. Supporting forces are shown in Fig. 1. The interlocking force represents the interdigitation of the primary and secondary dermal and epidermal laminae (Fig. 3). This surface composed of approximately six hundred primary and thousands of secondary laminae, holds the pedal bone in suspension from the hoof wall. The bearing surface of the hoof forms a reciprocal force against body weight. In fact, the ground pushes up on the solar surface of the pedal bone with a force equal to the body weight of the involved quadrant. The common extensor lifts the toe of the pedal bone through a variable tension on the extensor process. The forces deviating the pedal bone are shown (Fig. 2). As the hoof breaks over mid-stride, a tearing force is exerted against the toe and contributes to a separation from the dorsal wall of the pedal bone. The longer the toe and greater the bearing surface, the greater will be the tearing force. The effect of body weight is exerted through the driving force. In the normal hoof, the driving force is directed in a straight line, through P I, P II and P III. A pulling force is exerted posteriorily and upward on the volar surface of P III by the deep flexor tendon.



A INTERLOCKING FORCE
B RECIPROCAL FORCE
C LIFTING FORCE

Fig. 1—Forces supporting the normal hoof that are primarily involved in pedal rotation in laminitis.



A' TEARING F
B' DRIVING FO
C' PULLING F

Fig. 2—Forces that are primarily involved in alter ing the position of the *os pedis* in laminitis.

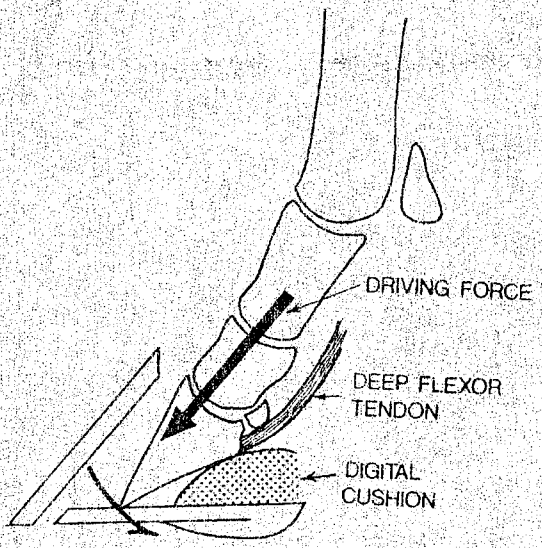


Fig. 4—Pedal rotation in process. The driving force, exerted by the body weight, appears to be exerted high on the extensor process of PIII rather than through the normal straight line relationship. The deep flexor tendon and the digital cushion passively act as a fulcrum.

Supporting forces and deviating forces are finely balanced in the normal hoof, allowing the pedal bone to maintain its proper position. Once this balance is lost, the tearing, pulling and driving forces all contribute to pedal rotation.

Once rotation begins, the driving force has an exaggerated effect (Fig 4). As the pedal bone begins to rotate, straight line relationship no longer exists between the short pastern and the pedal bone. With the digital cushion and the attachment of the deep flexor acting as a fulorum, the toe of the pedal bone is driven down.

The digital cushion is virtually unaffected by the disease. On the basis of this, it is difficult to accept the therapy of raising the heels of the affected hooves. The reason being, that whilst the pulling force may be lessened by decreasing the tension on the deep flexor, the support of the reciprocal force is virtually eliminated due to increased dead space beneath the sole. When the deviating effect of the driving force,(body weight through P I and P II) is exaggerated.

The therapy of lowering the heels as the pedal bone rotates is more understandable. Thought this increases the pulling force of the deep flexor, the support of the reciprocal force is enhanced and the exaggerated stress of the driving force is alleviated (Fig 4). Thinning the wall at the toe is a reasonable measure, because shortening the bearing surface decreases the tearing force exerted against the to as the hoof breaks over midstride. This is further aided by a shoe with rolled toe. For the same reason a horse with neglected hooves should have them trimmed with a short rounded toe at the very onset of laminitis being diagnosed. The application of a leather pad with tar beneath, or an acrylic covering of the hoof is also of benifit, because the elimination of dead space beneath the sole increases support for the reciprocal force. Standing the horse in sand, or peat has virtually the same effect, as both the former conform, easily to the shape of the sole and thus decrease dead space.

Ideally, it must be concluded that the clinically sound treatment for pedal rotation in laminitis is:- To lower the heels, thin the toe and roll the toe of the shoe. It must also be remembered that the bearing surface of the shoe be well seated out, with the nails in the back two thirds of the shoe. If clips are to be used, side clips at the toe quarters are prefered. The shoe should also be thinned, not narrowed, at the heels to conform to the gait of the affected animal. The shoes should be removed and replaced every four weeks to achieve maximum results.

In chronic laminitis the hoof wall may be grooved at the quarters, toe and again across the sole to alleviate pressure in the hoof, and provide expansion at the quarters. This treatment is of great value espicially when combined with corrective shoeing.