

DEPARTMENT OF  
OF THE FACULTY OF  
OF THE HORSE'S HOOF (SIDE BONE)

CONTENTS

	Page
SUMMARY .....	1
<b>OSSIFICATION</b>	
1.0 INTRODUCTION .....	1
<b>OF THE CARTILAGES</b>	
2.0 GLOSSARY .....	3
<b>OF THE HORSE'S HOOF (SIDE BONE)</b>	
<b>ASADAT</b>	
1.0 EVOLUTION OF THE HOOF OF THE HORSE .....	5
by	
4.0 MATERIALS AND METHODS .....	9
<b>AZIZ S.S. ASWAD, B.V.M. &amp; S.</b>	
5.0 ANATOMY OF THE HOOF .....	11
6.0 REVIEW OF LITERATURE CONCERNING THE DEVELOPMENT OF PILL .....	13
7.0 DEVELOPMENT OF PILL .....	14
7.1 Distal Palmar Process .....	14
7.2 Hooves .....	17
7.3 Hooves .....	17
7.4 Hooves .....	17
7.5 Hooves .....	17
7.6 Hooves .....	17
7.7 Hooves .....	17
7.8 Hooves .....	17
7.9 Hooves .....	17
10.0 MOMENTS OF THE HOOF .....	20
11.0 YEARS OF THE HOOF .....	24
12.0 REVISION AND CONCLUSION .....	24

THESIS SUBMITTED IN FULFILMENT OF THE CONDITIONS GOVERNING  
THE DEGREE OF DOCTOR OF PHILOSOPHY IN THE FACULTY OF  
VETERINARY MEDICINE, UNIVERSITY OF EDINBURGH.

December, 1971



OSSIFICATION  
OF THE CARTILAGES  
OF THE HORSE'S HOOF (SIDE BONE)

CONTENTS

	<u>Page</u>
SUMMARY .....	vii
1.0 INTRODUCTION .....	1
2.0 GLOSSARY .....	3
<u>ANATOMY</u>	
3.0 EVOLUTION OF THE DIGIT OF THE HORSE .....	5
4.0 MATERIALS AND METHODS .....	5
5.0 ANATOMY OF THE FOOT .....	7
6.0 REVIEW OF LITERATURE CONCERNING THE DEVELOPMENT OF PIII	13
7.0 DEVELOPMENT OF PIII .....	14
7.1 Distal Palmar Process .....	14
7.2 Proximal Palmar Process .....	17
8.0 THE CARTILAGES OF THE HOOF .....	17
8.1 Surfaces .....	18
8.2 Borders .....	18
8.3 Angles .....	19
9.0 TENDONS OF THE FOOT .....	20
10.0 LIGAMENTS OF THE FOOT .....	20
11.0 VEINS OF THE FOOT .....	22
12.0 DISCUSSION AND CONCLUSION .....	24

CLINICAL AND RADIOLOGICAL INVESTIGATION OF SIDE BONE

1.0	REVIEW OF LITERATURE CONCERNING THE CLINICAL AND RADIOLOGICAL EXAMINATION OF SIDE BONE .....	28
1.1	Diagnosis .....	28
1.2	Symptoms .....	30
1.3	Incidence and Distribution .....	33
1.3.1	Breed Incidence.....	34
1.3.2	Age Incidence .....	36
1.3.3	Distribution .....	37
1.4	Aetiology .....	38
1.4.1	Faulty Shoeing .....	39
1.4.2	Traumatism .....	40
1.4.3	Dietary Causes .....	42
1.4.4	Natural Process of Ageing .....	42
1.4.5	Hereditiy .....	43
1.4.6	Structural Factors .....	44
1.4.7	Other Diseases .....	45
1.5	Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate other than from the Proximal Palmar Process of PIII .....	46
2.0	CLINICAL AND RADIOLOGICAL OBSERVATIONS MADE IN THE CURRENT INVESTIGATION .....	47
2.1	Materials and Methods .....	47
2.1.1	Type of Horses .....	48
2.1.2	Radiological Examination.....	49
2.1.3	Positioning .....	49
2.2	Side Bone .....	51
2.2.1	Definition .....	51

	<u>Page</u>
2.2.2 Interpretation of Radiological Examination of Side Bone Formation .....	51
2.2.3 Classification of Side Bone .....	52
2.3 Interpretation of the Various Features seen on the Radiograph of the Foot .....	52
2.3.1 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate other than from the Proximal Palmar Process of PIII .....	52
2.3.2 Location, Origin, Shape and Size of the Bony Islands .....	53
2.3.3 Description of the Radiological Features of the Bony Islands in Relation to Side Bone ...	53
2.3.4 Ossification Centres (Bony Islands) that arise at the Level of the Distal Palmar Process of PIII .....	54
2.3.5 Interpretation of the Dense Area Found in the Region of the Palmar Process .....	55
2.3.6 Description of the Relationship of the Dense Area to the Bruised Angle of the Bar .....	57
2.4 Clinical and Radiological Observations .....	58
3.0 DISCUSSION AND CONCLUSION .....	65

#### HISTOPATHOLOGY

1.0 MATERIALS AND METHODS .....	70
1.1 Fixation .....	70
1.2 Defatting .....	70
1.3 Decalcification .....	71
1.4 Neutralisation .....	73
1.5 Dehydration .....	73
1.6 Clearing .....	73
1.7 Impregnation .....	73

	<u>Page</u>
1.8 Section Cutting .....	74
1.9 Staining .....	75
1.10 Clearing .....	76
2.0 GENERAL REVIEW OF THE HISTOLOGICAL LITERATURE .....	76
2.1 Classification of the Skeletal Cartilages .....	76
2.2 Growth of Cartilage .....	77
2.3 Classification of the Cartilage of the hoof .....	77
2.4 The Cartilage Canals .....	78
2.5 Ossification Fundamentals .....	79
2.5.1 Intramembranous Ossification .....	79
2.5.2 Endochondral Ossification .....	79
2.6 Arrangement of the Elements of Bone Tissue .....	82
2.7 Calcification and Ossification .....	83
3.0 REVIEW OF LITERATURE CONCERNING SIDE BONE .....	83
3.1 Side Bone Features .....	83
3.2 Pathology of Side Bone .....	84
3.3 Origin of Side Bone .....	84
4.0 MICROSCOPIC ANATOMY OF THE CARTILAGE OF THE HOOF .....	86
4.1 Cartilage Canals .....	88
5.0 REVIEW OF THE LITERATURE CONCERNING THE HISTOPATHOLOGY OF THE CARTILAGE OF THE HOOF .....	89
5.1 Ossification Starting from the Proximal Palmar Process of PIII .....	89
5.2 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate other than from the Proximal Palmar Process of PIII .....	

	<u>Page</u>
6.0 HISTOPATHOLOGY OF THE CARTILAGE OF THE HOOF .....	91
6.1 Ossification Starting from the Proximal Palmar Process of PIII .....	92
6.2 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate other than from the Proximal Palmar Process of PIII .....	96
6.2.1 Location and Origin of the Bony Islands .....	97
6.3 Secondary Cartilage Growth Resulting in the Formation of Ossification Centres (Bony Islands) at the Level of the Distal Palmar Process of PIII .....	98
7.0 REVIEW OF THE LITERATURE CONCERNING CALCIFICATION "NON PROVISIONAL" AND CALCIFIED NECROTIC FOCI OF CARTILAGE ....	100
7.1 Aseptic Calcified Necrotic Foci .....	101
7.2 Detection of the Calcified Cartilage .....	101
8.0 CALCIFICATION "NON PROVISIONAL" AND CALCIFIED NECROTIC FOCI OF THE CARTILAGE OF THE HOOF .....	102
8.1 Macroscopic Examination .....	102
8.2 Microscopic Examination .....	103
9.0 DISCUSSION AND CONCLUSION .....	105
 <u>CHEMICAL COMPOSITION (ASH, CALCIUM, PHOSPHORUS AND MAGNESIUM) OF THE PROXIMAL PART OF PIII AND THE ADJACENT SIDE BONE</u> 	
1.0 MATERIALS AND METHODS .....	112
1.1 Estimation of Magnesium .....	115
1.2 Estimation of Calcium .....	115
1.3 Estimation of Phosphorus .....	116
2.0 REVIEW OF LITERATURE .....	117
3.0 RESULTS .....	119

	<u>Page</u>
4.0 DISCUSSION AND CONCLUSION .....	120
5.0 GENERAL CONCLUSIONS .....	121
ACKNOWLEDGEMENTS .....	125
REFERENCES .....	126
TABLES AND ILLUSTRATIONS	

SUMMARY

The anatomy, development and histopathology of the distal phalanx (PIII) and the cartilages of the equine hoof were studied by radiological and histological techniques. Chemical analyses of ash, calcium, magnesium and phosphorus were also included.

A well defined distal palmar process was present in a 5 month fetus. At this stage, the proximal articular surface of PIII was still cartilaginous; its development was completed 5 months post-natally. Side bone was detectable a month later. The notch of the palmar process may be converted into a foramen any time after 2 years of age. Throughout development, the cartilages of the hoof were fibro-elastic and although directly continuous with the proximal palmar process of PIII, they were attached to the distal palmar process by a short ligament.

The side bone developed consistently in all cartilages and was invariably present from 6 months onwards. A means of classification as short, medium, long and marked has been suggested. The short type was the most frequent whereas the others were seldom seen in animals under 3 years of age. The longer types were more commonly observed in the fore than in the hind feet and some bilateral symmetry was present. Although the presence of this bone has been claimed to be a factor in causing lameness, this was not verified.

Bony islands were seen at different sites in the cartilaginous plate and in the short ligament which attached the distal palmar



process to the solar border of the cartilage of the hoof; those of the cartilaginous plate originated near to cartilage canals. A radiological dense area was sometimes seen in the region of the palmar process in animals older than 2 years. This was due to the presence of the bony bridge which converted the palmar notch into a foramen.

Side bone was formed by ossification and not calcification. Bone growth from the proximal palmar process of PIII is one of the factors involved. Its osteogenesis was a normal process of ossification extending from the proximal palmar process of PIII. Bony islands also occurred in the cartilage and at the level of the distal palmar process. There was no evidence of inflammatory reaction but necrosis and "non-provisional" calcification of the cartilage plate occurred and became more marked with age.

There was a significant difference between the mineral composition of the side bone and that of the adjacent part of PIII. This was related to differences in the stages of developments of the two regions.

## 1.0 INTRODUCTION

In Iraq and most of the other middle-east countries the horse is still of great economic importance. Horses and mules are used for various domestic purposes in daily life and are still used extensively in agriculture. These animals are also important in the armed forces as cavalry mounts, for communication and in the commissariat. Exportation of horses, in particular the Arab Horses, is a source of revenue to the country.

Diseases of the horse foot have been the subject of much thought and investigation since the earliest days of veterinary science. When the movement of armies, the cultivation of the land and the transport systems of countries depended on the horse, the detection and treatment of foot lameness was of commanding importance. With the advent of the internal combustion engine the economic importance of the horse, in developed countries, rapidly declined. However, at the end of World War II a resurgence of interest in the riding horse occurred and about this time the horse also became the subject of considerable veterinary research.

The term side bone refers to a condition of the cartilages of the hoof whereby these structures become ossified. Although ossification of these cartilages was known to the French writers Solleysel (1664) and La Fosse (1754), Osmer (1759) was the first English writer to draw attention to the condition. Later Spooner (1840) described the condition as "false ringbone" and Coleman's lectures (Coleman, 1793) refer to it as "quitter bone".

Lack of the precise knowledge, of the basic anatomical development of PIII; of the histo-anatomy and pathology of the cartilage and of the clinical significance of side bones, is the reason for the present investigation. Therefore, in addition to clinical and radiological examinations, anatomical, histopathological and chemical analyses of side bone have been carried out.

2.0 GLOSSARY

The terms used in this work conform with the Nomina Anatomica Veterinaria (1968). In the interests of brevity certain traditional terms have been used (enclosed in parentheses).

Cartilages of the hoof = Lateral cartilages, Cartilages of PIII

Caudal = Posterior.

Central groove of the frog = Central sulcus, Intersulcus groove.

Collateral ligaments of the distal sesamoid bone = Suspensory ligaments of navicular bone.

Collateral ligaments of the coffin joint = Collateral ligaments of the distal interphalangeal joint.

Corium - Coria = Matrix - Matrices.

Coronary border of PIII = Proximal border of PIII.

Cranial = Anterior.

Digital cushion = Digital torus.

Distal interphalangeal joint = Coffin joint.

Distal palmar process of PIII = Retrossal process of PIII.

Distal sesamoid bone = Navicular bone.

Distal sesamoidean cavity = Navicular bursa.

Dorsal border of PIII = Volar border of PIII.

Fetus = Foetus.

(L) = Lateral.

(Lg.) = Long.

(M) = Medial

(Md.) = Medium.

(Mk.) = Marked

(NF) = Near fore.

(NH) = Near hind.

(OF) = Off fore.

(OH) = Off hind.

(PI) = First phalanx, Proximal phalanx.

(PII) = Second phalanx, Middle phalanx.

(PIII) = Third phalanx, Distal phalanx, Coffin bone, Pedal bone.

Palmar foramen (or notch) of PIII = A foramen (or notch) of the angle of PIII.

Palmar processes of PIII = Angle of the fore PIII, Wing of the fore PIII.

Parietal groove of PIII = Dorsal groove of PIII.

Parietal surface of PIII = Dorsal surface of PIII.

Plantar process of PIII = Angle of the hind PIII, Wing of the hind PIII.

Proximal palmar process of PIII = The basilar process of PIII.

(Sh.) = Short.

Single phalang osesamoidean ligament = Distal navicular ligament.

Solar border of PIII = Distal border of PIII.

Solar foramen of PIII = Volar foramen of PIII.

Solar surface of PIII = Volar surface of PIII.

Solar venous plexus = Volar venous plexus.

ANATOMY3.0 EVOLUTION OF THE DIGIT OF THE HORSE

In the zoological classification, the horse is a member of the genus "equus", of the family "equidae", in the order "perissodactyla", sub-order "hippomorpha" (Rothschild, 1965; Sidney, 1965).

In prehistoric days the horse was a small, pentadactylic plantigrade animal with five functional digits. Now, with one, it is a monodactyl (Bradley, 1920). The change from a pentadactyl to a monodactyl was by very slow stages, as are all evolutionary modifications. The horse became a tetradactyl in the Protohippus stage of development; a tetradactyl - tridactyl in the Epihippus stage (three digits touching the ground with one slightly raised). Tridactyl in the Meshippus stage and monodactyl in the modern horse (Hayes, 1904; Axe, 1906; Langley and Rooney, 1967). As the functional digits were reduced the persisting third digit became larger and stronger, so that progressively it bore more weight. The phalanges and joints of this digit likewise became longer, thicker and stronger. The single digit is the third, and the splint bones represent respectively the second and fourth digits.

4.0 MATERIALS AND METHODS

The distal phalanx (PIII) consists of an osseous portion and two plates of cartilage and as approximately one half of each cartilage lies within and the other outside the hoof, its removal was necessary to complete the macroscopical examination.

The techniques used were:

Separating the cartilages. Each hoof was marked for identification on the quarters by means of a drawing knife. The skin was incised and removed up to one centimetre above the coronet. Then, after freezing the foot, a vertical section was made posteroanteriorly through the central groove of the frog by an electrical band saw. Removal of the skin and freezing are essential in preventing choking of the saw by hair, skin and the soft tissues of the foot. The sections were then left in a cold room to be partially thawed. With a scalpel, the remaining skin was then carefully separated downwards from the underlying connective tissue, to the coronary groove. A strong, twenty centimetre long knife with a stout handle was then inserted in the groove and with the sharp edge facing outwards, to avoid cutting the cartilage, a deep incision was made down through the coria, to separate the cartilage from the wall of the hoof leaving the caudal end of the cartilage still attached at the heel. A further incision was then made through the solar corium, continuing below the deep flexor tendon and through the digital cushion (Fig. 1). The half hoof is cut free from the heel then the cartilage separated from the soft tissue attachments so that PIII with the cartilage remains, leaving one centimetre of the common extensor tendon for label attachment. Three hundred and fourteen feet were dissected in this manner (Table 1).

Four feet were cut in horizontal-transverse sections at approximately two centimetres thick, in order to demonstrate the relative position of the cartilages.

One hundred and sixty four halves of PIII, of different breeds and ages, were boiled, degreased and bleached so that the radiographic findings could be confirmed by macroscopic examination.

The ligaments of the cartilage of the hoof were isolated by dissection and demonstrated in specimens partially boiled to remove the corium and digital cushion.

In an attempt to show the vessels in the cartilage canals the blood vessels in the fresh foot were washed out with a solution of normal saline, under pressure of 200 mm. Hg. until the escaping fluid was clear. A 75% solution of Barium sulphate was then injected into the artery and 50% solution injected into the homologous vein (Damancy and Co. Ltd., London). The blood vessels were then ligated and the entire foot X-rayed. It was then frozen for 12 hours, sectioned vertically through the centre and again X-rayed. Although the method was useful in revealing the vascular pattern of the foot (Fig. 2) the technique was not of significant value in revealing the vascularity of the cartilage canals, in the separated cartilage.

## 5.0 ANATOMY OF THE FOOT

The gross structure of the hoof, its coria, the vessels, nerves and tissues other than the skeleton, the cartilages of the hoof and the ligaments of the foot, were studied and were found to conform with the descriptions in standard text books (Bradley, 1920; Sisson and Grossman, 1953; Stump, 1967; Rooney, Sack and Habel, 1969).



When the hoof coria and other tissues are removed, the skeleton of the foot remains. This consists of PIII, the cartilages, the distal sesamoid bone and approximately the lower half of PII. These three bones articulate together at the distal interphalangeal joint.

#### The distal articular surface of PII

This surface presents two condyles separated by a shallow groove. The medial condyle is slightly the larger. The more anterior part of the articular surface contacts the articular surface of PIII while the more posterior area contacts the distal sesamoid bone.

#### Distal sesamoid bone

It has two surfaces, two borders and two extremities. The bone is shuttle shaped. It articulates with both PII and PIII. The surface that articulates with PII faces upwards and forwards, it consists of a central eminence, flanked on each side by concave areas. The flexor surface is directed downwards and backwards. The proximal border is wide and grooved in its middle, narrower and rounded on either side. The distal border presents a narrow facet for articulation with PIII.

#### The articular surface of PIII

The articular surface of PIII faces upwards and backwards. Its central part presents a low ridge which is flanked on each side by shallow concave areas to conform with the corresponding articular surface on PII. There is a narrow flattened area along the dorsal border for articulation with the distal sesamoid bone.

### Joint capsule

The joint capsule is attached around the margins of the articular surfaces of the three bones which form the distal interphalangeal joint (Fig. 1). In front and on the sides it is tight and bound to the extensor tendon and the collateral ligaments. Behind it forms a large pouch which extends upwards to about the middle of PII, where it comes into close contact with the digital sheath. On each side, between the collateral ligaments and the collateral ligaments of the distal sesamoid bone, smaller pouches contact the axial surface of the cartilages. These pouches form especially during flexion. When separating or removing the cartilages these pouches are difficult to avoid (Bradley, 1920; Sisson and Grossman, 1953; Calislav and St. Clair, 1969).

### The distal sesamoidean 'bursa'

Hickman (1964) stated that "it is generally supposed that a bursa is interposed between the tendinous surface of the bone and the deep flexor tendon. This is not so; the area bounded by the joint capsule of the corono-pedal joint, the tendinous surface of the navicular bone, the interosseous ligament and the deep flexor tendon is a potential cavity lubricated with synovia, and as it is not lined by any separate and distinct endothelial membrane it therefore constitutes a joint".

The joint cavity (Fig. 1) extends from the flexor surface of the distal sesamoid bone to the connective tissue which bridges the region from the deep flexor tendon to PII proximally, and to the attachment of the deep flexor tendon on PIII distally. The cavity also contacts the

digital vessels and nerves on the axial surface of the cartilages (Calislav and St. Clair, 1969) and it separates the deep flexor tendon from the flexor surface of the distal sesamoid bone. It is separated from the distal interphalangeal joint by the latter bone and its ligaments (Stump, 1967), it extends proximally and distally beyond the edges of the distal sesamoid bone (Chauveau, 1891) and it contacts the axial surface of the cartilages (Ellenberger and Baum, 1943).

### PIII

This bone is crescentic in shape and lies entirely within the hoof, which normally, conforms to its shape. It is a very dense but porous bone, described as having three surfaces, three borders and two palmar processes (Fig. 3A).

### Surfaces

The parietal surface is convex from side to side and slopes downwards and forwards. The angle of inclination is between 45 - 50 degrees in the fore feet and 50 - 55 degrees in the hind feet. The presence of numerous foramina gives it a characteristic rough porous appearance, like pumice stone. Its declivity increases as it proceeds backwards and the surface becomes narrower. On each side is the parietal groove (lateral and medial) which begins and passes forward from the palmar notch or foramen to end at one of the larger foramina. It carries the dorsal artery of PIII. In the fresh state this surface is covered by the corium of the wall of the hoof which is attached to the periosteum.

The solar surface (Fig. 3B), is half moon shaped, concave and is relatively smooth. Behind, the uneven semilunar crest is a roughened triangular central depression (the flexor surface) for the insertion of the deep flexor tendon. On each side of this surface is a groove in which runs the digital artery to pass through the solar foramen into the interior of the bone. The dorsal margin of the solar surface is roughened for attachment of the single phalango sesamoidean ligament.

The articular surface has been described with the joint.

#### Borders

The coronary border presents the extensor process, to which the common extensor tendon is attached, and on either side of which there is a depression for the distal attachments of the collateral ligaments of the distal interphalangeal joint.

The solar border is thin, sharp and irregularly serrated or notched, the notches opening into various foramina. There is, commonly, a wider notch in front.

The dorsal border is fairly straight and joins the two proximal palmar processes. Along this border is the narrow, flattened area which articulates with the distal sesamoidean bone.

#### Palmar processes

Each process is described as presenting two protuberances which extend back beyond the limits of the articular surface and give attachment to the cartilage of the hoof. Percivall (1832) described it as

a backward projecting process, consisting of two parts separated by a notch through which passes the dorsal digital artery. The proximal part being tubular, with a smooth surface to which is attached the collateral ligament of the distal interphalangeal joint. The distal part being larger, irregular and pumice like and to this he stated that the cartilage of the hoof is attached. According to Chauveau (1891), a deep notch (the origin of the parietal groove) separates each palmar process into two eminences. The proximal eminence is named by Bouley (n.d.), "the basilar process" and the distal, prolonged behind and is designated by Clark (n.d.), "the retrorsal process". Chauveau (1891) and Vaughan (1895) agree with the naming of the processes and with the statement by Percivall (1832) that the distal process is the larger. Vaughan (1895), Sisson and Grossman (1953), Cameron (1907), Muller (1937), Hickman (1964), Smythe (1967) and Morgan (1968) all agree that the cartilage of the hoof is attached to the proximal eminence, not to the distal eminence as stated by Percivall (1832) and Gilruth (1892).

The International Committee on Veterinary Anatomical Nomenclature in their *Nomina Anatomica Veterinaria* (1968) use, the "lateral and medial palmar processes", to the whole structure of the angle of the fore PIII and plantar to the hind one. Schebitz and Wilkens (1968) give the most recent anatomical description of the angle of PIII, they divide it into two parts, "the proximal palmar process" and "the distal palmar process" and these terms are adopted throughout this thesis.

Regarding the formation of the palmar foramen, Gilruth (1892) stated that before an animal is 4 years old there is a notch between

the basilar and retrorsal processes of PIII. He stated that after this period the notch becomes gradually converted into a foramen. Smythe (1967) stated that this notch is converted into a foramen in aged horses.

#### 6.0 REVIEW OF LITERATURE CONCERNING THE DEVELOPMENT OF PIII

Sisson and Grossman (1953) described the development of PIII as unusual, stating that, while the proximal articular part is still cartilaginous, a perichondral cap of bone is formed in relation to the hoof and later the process extends into the upper part. The angle, in the new-born foal, is <sup>a</sup> small, pointed projection and later the lower part of the articular cartilage becomes ossified to a varying degree .

Tohara (1950) studied the ossification of leg bones of horses, radiologically, and stated that, it was generally accepted that ossification points appear during the fetal stage at the proximal end of PIII. During his studies he was unable to detect any sign of such ossification point during pregnancy but asserts that this is complete in the 4th to 5th month of postnatal life. Zietzschmann and Krolling (1955) and Rooney (1963) stated that the ossification of the proximal part of PIII was completed by the last part of the pregnancy.

The aforementioned anatomists, and those who carried out a wide study of side bone formation, merely refer to the palmar process as being the site of attachment of the cartilage, they did not study the origin of the palmar process or its relationship to side bone formation. Witte (1907) examined, histologically, 130 specimens from well developed side bone cases. Muller (1937) in his histological description of the

normal palmar process examined specimens from 37 heavy horses and 3 working mules with overall ages ranging from 2 - 20 years old. He stated that in 12 horses, 2 of 2 years, 1 of 3 years, 2 of 4 years, 1 of 6 years, 1 of 12 years, 1 of 13 years, 1 of 15 years <sup>1 of 14 years</sup> and 2 of 20 years old, showed a normal palmar process with no trace of ossification of the attached cartilages. Ullrich (1934) and Lowe (1935) carried out a radiological examination of side bone cases without referring to the palmar process. Ullrich (1934) examined the fore feet of 40 slaughtered working horses (7 - 20 years old) and stated that 5 horses (8, 13, 14, 15 and 16 years old), showed no ossification of the cartilages at their attachment to the palmar process.

In the present investigation the structure, development and growth of the palmar process was studied in fetuses, in the newly born and in young foals (Table 1). The palmar process was examined macroscopically, radiologically and histologically.

#### Macroscopic structure and growth of the palmar processes

This study was based on the examination of specimens in which the soft tissues were removed by boiling (for approximately eight hours).

### 7.0 DEVELOPMENT OF PIII

#### 7.1 Distal palmar process

In the prenatal stage, while the proximal articular part of PIII (with its features such as the depressions for the distal attachment of the collateral ligaments and caudal to these the bony elevations of the

proximal palmar process) is still cartilaginous (Fig. 4), the distal palmar process is developed as a small, sharp pointed projection formed by the meeting of the ossified parts of the coronary and solar borders of PIII (Figs. 5A and B). This process presents three borders and three surfaces. The coronary border, is concave and is a direct continuation of the coronary border of PIII (Figs. 5A and B). After birth, this continuation of the border shows a depression in the form of a shallow line which later becomes the parietal groove (Fig. 5C) and as PIII develops this groove becomes deeper (Figs. 3A, 5D and 6A).

The solar border is continuous with that of PIII (Fig. 5A).

The dorsal border is semilunar, leading to the solar foramen (Figs. 6B and C). After birth a bony deposition, the semilunar crest, develops between this border and the foramen. The semilunar crest is a rough ridge formed on and between the under surface of the distal palmar process and the solar surface of the proximal palmar process, anterior to its solar border (Figs. 3B, 6D and 7A). It begins to appear at the age of 2 months and is fully developed by 6 months of age (Figs. 3B, 6D and 7A). The three borders of distal palmar process meet caudally to form the sharp bony projection of the process (Figs. 5A, 6C and 7B). As PIII develops there is a reduction in the sharpness of the process as a result of the development of the semilunar crest (Figs. 3B, 6D, 7A, 7C and 7D).

The parietal surface, [Fig. 5A (between b and c)], is a continuation of the parietal surface of PIII. It is rough and porous resembling



pumice stone but it does not contain any large foramina as is seen on the parietal surface of PIII. The roughness of the surface increases as PIII develops (Figs. 5A - D and 6A).

The dorsal surface, [Fig. 5A (between a and b)], in the pre and postnatal stages, shows that the surface is slightly rough to the touch, which roughness disappears as the bony deposition of the semilunar crest begins. The latter fills the space between the dorsal surface of the distal palmar process and the solar surface of the proximal palmar process but it leaves a notch, "the notch of the palmar process", leading to the parietal groove. At any age over 2 years (Table 2) this notch may or may not develop to become a foramen, "the foramen of the palmar process". Figures 8 and 9 show a well defined foramen of OH foot of a 2 year old thoroughbred horse. Figures 10 and 11 show the parietal aspects of PIII of a 20 year fell pony and a 1 year thoroughbred horse, respectively. In Fig. 10 the foramen is not formed, Fig. 11 the notch is almost closed to form a foramen. The foramen is completed by the junction of the two bony spurs (forming a bony bridge), one spur extending from the proximal palmar process (as in side bone) and the other from the adjacent part of the semilunar crest, not from the distal palmar process. This bony bridge like growth is shown in Figs. 8, 9 and 11. Fig. 8b shows ossification of some of the fibres of the ligament which connect the cartilage to the distal palmar process (bony band).

The solar surface of the distal palmar process (Fig. 6B), is directly continuous with the rest of the solar surface of PIII. It is

smooth in the prenatal stage, in the postnatal stage it becomes rough resembling pumice stone (Figs. 3B, 6D and 7A - D).

## 7.2 Proximal palmar process

This process develops with the proximal articular part of PIII as a smooth, blunt bony elevation, caudal to the depressions on either sides of PIII. Its direction is outward and backward (Fig. 3A), and with it the cartilage of the hoof is united. At the age of 2 months the proximal articular part of PIII is not fully ossified and there is little indication of the appearance of the parietal depressions. At this age also there is no bony elevation on either side of PIII to indicate the formation of the proximal palmar process (Fig. 5D). As the animal grows the proximal articular part of PIII and its adjacent features develop to assume their typical shape. Fig. 3A is OFL aspect of PIII at 5 months old, it shows the proximal articular part of PIII with its features at almost the final stage of development. At 6 months of age, the proximal articular part of PIII is completely ossified and shows traces of bony invasion into the cartilage of the hoof (Fig. 6A) to form side bone.

## 8.0 THE CARTILAGES OF THE HOOF

On each foot two thin curved plates of cartilage rise almost vertically from the proximal palmar process of PIII (Fig. 12). Each cartilage forms an irregular oblique angled parallelogram with a convex abaxial and a concave axial surface. It has four angles and four borders. The acute angles being the cranio-proximal and the caudo-solar.

The cartilages extend from the caudal aspect of the proximal palmar process of PIII covering the dorsal half of the proximal articular surface of this bone, and they extend above the coronet so that almost half of each lies subcutaneously.

### 8.1 Surfaces

The abaxial surface, (Figs. 12 and 13), is smooth, convex and slightly overlaps that of PIII. This surface is covered by the coronary venous plexus and it is pierced by numerous openings for the passage of these veins, particularly in its lower part. Proximally, it lies under the skin and distally under the coronary and laminar coria.

The axial surface, (Fig. 14), is concave and adapted to the structures with which it is in contact. Cranially it is related to PII and the distal interphalangeal joint. Many fibrous bands spring from the axial surface and pass into the tissue of the digital cushion. These fibres are especially numerous in the distal part, where the cartilage is more intimately connected with the digital cushion. The palmar venous plexus, is related to the axial surface of the cartilage and these vessels communicate freely through foramina with the coronary venous plexus on the abaxial aspect.

### 8.2 Borders

The proximal border is thin and slightly convex, and usually there is a notch through which the digital artery and vein pass. The whole of this border is subcutaneous.

The solar border is thick. Its cranial part is blended with the proximal palmar process of PIII and attached to the distal palmar process of this bone by short bands of fibrous tissue which also cover the parietal groove, (there is no cartilaginous attachment between the distal palmar process and the cartilage). The caudal portion of this border turns inwards towards the sole (Figs. 14 and 15).

The cranial border is irregular and is closely attached to the collateral ligament of the distal interphalangeal joint. From the cranio-proximal angle, the cranial border slopes obliquely downwards and backwards.

The caudal border is oblique in the same direction of the cranial border. It is slightly convex at its proximal part and thickens gradually as it descends. The whole of the border is subcutaneous.

### 8.3 Angles

The caudo-solar angle is the thickest and lies just under the perioplic and coronary coria at the bulbs of the heel.

The caudo-proximal angle varies considerably in shape. In some it is sharp, almost a right angle, while in others it is curved, so that, the proximal and caudal borders are continuous.

The cranio-proximal angle is acute, thin and related to PII.

The cranio-solar angle is obtuse and is blended with the proximal palmar process of PIII (Fig. 12).

## 9.0 TENDONS OF THE FOOT

The two tendons which terminate within the hoof are the common extensor tendon and the deep flexor tendon.

The common extensor tendon runs down the dorsal surface of PII over the cranial part of the distal interphalangeal joint to be inserted into the extensor process of PIII. It is attached on either side by a short ligament to the cranial border of the cartilage (Fig. 15).

The deep flexor tendon lies on the caudal aspect of PII, it passes over the flexor surface of the distal sesamoid bone, and inserts on to the semilunar crest and the flexor surface of PIII. The tendon is separated from the distal sesamoid bone by the distal sesamoidean joint cavity.

## 10.0 LIGAMENTS OF THE FOOT

Bradley (1920), Sisson and Grossman (1953), Stump (1967) and Rooney, Sack and Habel (1969) describe the following ligaments of the foot:

The single phalangosesamoidean ligament; the collateral ligaments of the distal interphalangeal joint; a band from the cranial part of each cartilage to PIII; the collateral ligaments of the distal sesamoid bone; a band from the middle part of each border of PI to the proximal part of the cartilage (with a branch to the digital cushion) and short fibres which attach the solar border of the cartilage to the palmar process of PIII. The ligaments of the ergot are also described by most of these authors.

In addition to these ligaments, Bradley (1920), describes a band which connects the cranial part of each cartilage with the common extensor tendon.

The single phalangosesamoidean ligament is a short, but strong structure extending between the distal border of the distal sesamoid bone and the flexor surface of PIII (Fig. 16).

The collateral ligaments of the distal interphalangeal joint are short, strong bands, which are attached proximally to the depressions on either side of the distal end of PII, they widen distally to end in the depressions on either side of PIII (Figs. 12 and 15). They blend with the cranial border of the cartilages.

Another short, strong band which covers the upper part of the above ligament, connects the cranio-proximal angle of each cartilage with the rough eminence in front of the depression at the distal end of PII.

A narrow fibrous band also leaves the abaxial surface of the cartilage, near to its cranio-proximal angle, and passes forward to be attached to the common extensor tendon. Some fibres pass straight across to join the ligament from the other side, so uniting the cartilages, cranially (Fig. 15).

A ligament, arising from the mid part of the border of PI runs downwards and backwards, to be inserted into the proximal border of the cartilage, near the caudo-proximal angle of its axial surface (Fig. 17). It detaches a branch to the digital cushion.

The collateral ligaments of the distal sesamoid bone arise from the depressions on each side of the distal end of PI. They pass disto-caudally, medial to the cartilage, to be attached to the extremities and proximal border of the distal sesamoid bone where the fibres from each side blend with each other (Fig. 16). The ligaments also detach two branches to the axial surface of the cartilage (Fig. 18).

From the cranial part of the solar border of the cartilages short fibres extend to the parietal surface of PIII, covering the parietal grooves (Figs. 19 and 20). These fibres, occasionally become ossified, forming a bony covering (Fig. 8b), and from the solar border of the cartilage to the solar surface of the distal palmar process of PIII (Fig. 14).

The ligaments of the ergot are two thin narrow fibrous bands which arise in the fibrous basis of the ergot, they cross the digital artery and nerve obliquely where they widen and blend below with the digital fascia and the digital cushion on the axial side of the cartilages.

#### 11.0 VEINS OF THE FOOT

A very extensive venous network is present in the corium of the foot. This is described as consisting of three venous plexuses based on the topographic region in which they are located (Stump, 1967; Rooney, Sack and Habel, 1969). The plexuses are the coronary in the coronary corium, the dorsal venous plexus in the laminar corium, and the palmar venous plexus which is related to the solar corium and the

axial surfaces of the cartilages. Numerous veins pass through canals in the cartilage and establish communicating anastomoses between the plexuses (Figs. 4 and 21).

The coronary venous plexus encircles the coronary region of the foot. It covers the distal end of the common extensor tendon, the abaxial surface of the cartilages and the bulbs of the digital cushion (Fig. 13).

The dorsal venous plexus covers the parietal surface of PIII in the deep layer of the lamina corium. It is also attached to the lower part of the cartilages by connective tissue which contains the lower part of the coronary venous plexus.

The palmar venous plexus lies on the deep face of the solar corium and on the axial surface of the cartilages. Around the solar border of PIII it communicates with the dorsal plexus and, through the cartilages, with the coronary plexus.

The deep vein of PIII accompanies the terminal part of the digital artery, which drains the intraosseous plexus within this bone (Sisson and Grossman, 1953).

All the venous plexuses converge at the proximal border of each cartilage to form the medial and lateral digital veins which run upwards in front of the corresponding artery.



## 12.0 DISCUSSION AND CONCLUSION

The skeleton of the foot is the essential anatomical structure involved in this research and the relationship between PIII with the cartilage of the hoof is of paramount importance.

The information gleaned from the standard text books and the literature regarding the normal gross anatomy and the radiological appearance of PIII and its palmar processes, although not substantially inaccurate it falls short of the details required to conduct this study. These comments apply particularly to PIII, its palmar processes, the cartilage of the hoof and its ossification.

Review of the publications of Witte (1907), Ullrich (1934), Muller (1937), Tohara (1950), Sisson and Grossman (1953), Zietzschmann and Krolling (1955) and Rooney (1963) gives very little accurate information relating to the ossification of PIII and to the development of the palmar process.

In this investigation it was found that PIII continues to develop as the fetus increases in age so that at 5 months old a well defined distal palmar process is formed by the meeting of the proximal and distal borders of PIII, but at this stage the proximal articular surface (with its features) is still cartilaginous. The fetus at the age of 10 months shows a noticeable increase in bone precipitate in the proximal part of PIII without giving a definite picture to this part and its surrounding. This appearance of PIII during the first 6 weeks, after birth, resembles more or less that of the fetus at the end of the

gestation period, except that the distal palmar process grows larger, a depression in a form of a shallow line representing the parietal groove is apparent, the semilunar crest develops, the parietal and solar surfaces of the distal palmar process become rougher and porous, and there is a reduction in the sharpness of the distal palmar process.

At the age of 2 months the proximal articular part of PIII still shows little indication of the appearance of the depressions; the parietal groove takes its final definition; the semilunar crest shows more bony deposition; the parietal and solar surfaces of the distal palmar process are more rough and there is more reduction in the sharpness of the distal process.

As is shown in Table 1, no foals were available between the ages of 2 and 5 months old. However, at 5 months of age, PIII is almost at its final stage of development although the semilunar crest is not fully developed. At 6 months of age traces of side bone were detected.

The notch, which lies between the proximal and distal palmar processes, is seen as a wide bifurcation at a time where the proximal process is not yet ossified. As its ossification progresses there is reduction in the size of the notch.

The pumice stone like roughness of the parietal and solar surfaces of the distal palmar process is normally found in the postnatal life of the animal, it increases as PIII develops.

Of the above findings, the development of the proximal part of PIII agrees, to a great extent, with Sisson's and Grossman's (1953)

general statement and Tohara's (1950) radiological findings. These findings conflict with those of Zietzschmann and Krolling (1955) and Rooney (1963).

The present study has shown that evidence of bony invasion into the cartilage of the hoof, side bone, is always detectable in foals of 8 months old and over, a fact which conflicts with the report of Ullrich (1934) and Muller (1937).

The present study has shown that the notch of the palmar process may or may not be converted into a foramen (when bony spurs from the proximal palmar process and the semilunar crest meet) at any age over 2 years old, which does not agree with the findings of Gilruth (1892) and Smythe (1967) who stated that it only occurs when the animal is over 4 years old, where the notch becomes gradually converted into a foramen.

Literature concerning the anatomy of the cartilage of the hoof and its ossification described the cartilage as being attached to the palmar process (Percivall, 1832; Gilruth, 1892; Vaughan, 1895; Cameron, 1907; Muller, 1937; Sisson and Grossman, 1953; Hickman, 1964; Smythe, 1967; Morgan, 1968). While it is generally believed that the cartilage is attached to the proximal palmar process, Percivall (1832) and Vaughan (1895) stated that it is attached to the distal palmar process.

The present investigation found that the cartilage is continuous with the proximal palmar process of PIII and attached to the distal palmar process by a short ligament. This investigation has also shown



## CLINICAL AND RADIOLOGICAL INVESTIGATION OF SIDE BONE

### 1.0 REVIEW OF LITERATURE CONCERNING THE CLINICAL AND RADIOLOGICAL EXAMINATION OF SIDE BONE

#### 1.1 Diagnosis

For more than a hundred years before radiological facilities became commonly available, the clinical diagnosis of side bone, depended on physical examination and observation of the animal's symptoms, at rest, and in motion (Williams, 1872; Fitzwygram, 1894; Lupton, Lond.n.d.; Biddell, Douglas, Thomas, Fleming, MacNeilage, Murray and Trotter, 1910; Miller, 1928; Hungerford, 1953; Smythe, 1967). Consequently, the degree of ossification of the cartilages was a matter of opinion and frequently "amongst experts" disputes resulted in litigation.

With regard to the position of the foot for digital examination, two opinions are supported. Pritchard (1888) considered that the examination should be carried out with the horse being allowed to stand with the weight evenly on his feet, so that the same amount of tension is imparted to all the cartilages. Pressure was then applied with the finger and thumb over the region of the cartilage to compare whether or not normal elasticity was present. Pritchard (1888) does not agree with those who consider that the foot should be lifted and the cartilage tested by moving it inward and outward. He considered that this method was very unreliable, pointing out that in many instances the upper part of the cartilage alone was ossified and in such a case it would readily move inwards and outwards and consequently be misleading.

The second opinion, that both methods should be used to determine the degree of calcification present in the cartilages is supported by Smythe (1959).

Many authors state that the condition can only be diagnosed with certainty when the upper part of the cartilage has ossified. If ossification affects only the lower portion, that is within the hoof, it is difficult or impossible to detect the change (without radiological examination) (Lungwitz, 1898; Axe, 1906; Moller and Dollar, 1906; Cameron, 1907; Reeks, 1918; Hutton, 1933; O'Connor, 1934; Hayes, 1950; Cresswell and Smythe, 1963; Hickman, 1964; Adams, 1966).

#### Radiological Diagnosis

Since Pryer (1929) laid the foundation of the Radiological Department at the Royal Army Veterinary School, great attention has been given to the diseases of the horse's foot. Oxspring's (1935) report on 1345 radiographs made in the same department, mostly dealt with examination of the distal sesamoid bone. He stated that, lack of literature in connection with the interpretation of radiographs of the horse's foot made it necessary for him to make a special study of this subject in order to understand and interpret the significance of the various shadows seen on the film. In Germany, at about the same time Ullrich (1934) was examining side bone in slaughtered horses and Lowe (1935) examined 44 horses' feet radiologically for side bone. Since then great progress in this field has been made. Now it is accepted that radiological examination is essential to determine the degree of ossification that has occurred in the cartilages (Brennan, 1958;

Banks, 1961; Carlson, 1967; Cresswell and Smythe, 1963; Hickman, 1964; Adams, 1966).

## 1.2 Symptoms

Descriptions of the occurrence of clinical symptoms, the degree and the character of lameness shown by horses which have side bone, can be grouped into a number of categories, most of which have had brief support for a few years.

Axe (1906) and Cameron (1907) considered that, in horses lame from side bone, the foot is hot, the action short, and the tread wanting in firmness and it has an inclination to the sound side of the affected foot. In the stable the animal stands with the heel slightly raised from the ground, and if both feet are affected the weight is shifted from one limb to the other at intervals. Hayes (1950) reported that if the cartilage of only one side was affected the animal would generally "dish" the leg either outwards or inwards, away from the affected cartilage to give relief. Cresswell and Smythe (1963) found that when lameness was due to side bone and the horse was moved in a circle, the greatest pain was shown when the affected leg was on the inside. On inspecting the shoe of such a horse, they noted that wear was greater at the outside of the toe. Furthermore, when the horse put the affected foot down, the outside wall struck the ground first and the foot then rocked to the inside wall. Adams (1966) states that lameness may be evident when the horse turns, but it is seldom acute, and that if side bones are the cause of lameness, there will be heat and pain over one or both of the cartilages.

With regard to the character of lameness shown by horses with side bone Cameron (1907) and Hayes (1950) state that the animal moves with a short stilty step and has a tendency to stumble in an attempt to avoid shock on the heels. Wearing of the toe is consequently marked. Miller (1928) states that lameness is usually characterised by taking a short step by the affected limb, and the tendency to do this may result in peculiar short and long steps. Youatt (1874) and Fitzwygram (1894) consider that there can hardly be said to be any special peculiarity about the lameness from side bones, except a certain degree of stiffness of action.

Miller (1928), Leeney (1952), Hickman (1964) and Adams (1966) consider that the onset of lameness is related to the process of ossification where there is inflammation present. Lameness is also attributed to the completion of ossification and the considerable consequent enlargement of the tissue (Spooner, 1840; Hutton, 1933; Hayes, 1950; Cresswell and Smythe, 1963).

With regard to differential diagnosis of side bone lameness, Hayes (1950) states that a horse with side bone goes short, on the toe, and in a style of action which somewhat resembles the gait of navicular disease, but the animal is not so quick in removing the foot from the ground. Cresswell and Smythe (1963) state that, differentiating side bone from other foot lameness occasionally can be difficult. Adams (1966) postulates that side bone accompanies other lameness such as navicular disease, and in many cases the cause of lameness may be wrongly attributed to side bone.



One of the earliest theories was that replacement of cartilage by bone interferes with the physiological movements of the foot and this may be the cause of lameness (White, 1825; Spooner, 1840; Lungwitz, 1898; MacCallum, 1889; Cameron, 1907; Hutton, 1933; Prentice, 1946; Hungerford, 1953; Smith and Jones, 1957; Hickman, 1964).

The theory that side bone in heavy horses causes no pain or lameness unless it is associated with concurrent foot malconformation, and that lameness from side bone in light horses is due to trotting upon the hard roads, has support from Williams (1872), Youatt (1874), Fitzwygram (1894), Reeks (1918), O'Connor (1934), Hayes (1950) and Leeney (1952).

Lupton (Lond.n.d.) states that side bone frequently causes lameness and when accompanied by ring bone, the ossified cartilages give rise to the most acute and irremediable lameness. Smythe (1959), Cresswell and Smythe (1963) and Smythe (1967) state that it is doubtful if side bone causes lameness unless it is abnormally developed, confined to one side of the foot, or associated as frequently it may be, with ring bone, or some type of coronary exostosis. Waxberg (1953) and Dietz (1968) state that 10% of the horses lame with side bone have shown other pathological processes in the joints, tendons or ligaments of the hoof region.

The following authors state that side bone may or may not be accompanied by lameness:- Axe (1906), Leeney (1911), Ensminger (1951), Wooldridge (1960) and Adams (1966). Fitzwygram (1863) and Miller (1928) think that as soon as the bony deposit is solidified the lameness ceases.

There are other authors who think that ossification of the cartilages may go on without any inconvenience or lameness (Muller, 1937; Thompson, 1946; Brennan, 1958). Muller (1937) examined 38 heavy horses and 3 mules with different degrees of side bone development and he states that none of them showed lameness. Brennan (1958) in support of his view states that an unusual case was observed when a 5 year old mare suddenly went lame while training. The animal was in distress and unable to place its foot on the ground. A fracture of PIII was suspected but radiographs revealed instead a 65 mm lateral side bone that had been fractured. It is of interest to note that the owner who trained and raced the mare since it was 2 years old, said it had never shown any signs of lameness. After the injury the mare was kept in a stall for 2½ months then turned out for 4 additional months. The following year she raced sound.

### 1.3 Incidence and Distribution

A survey of the literature reveals that the condition is relatively common throughout the world. In the United Kingdom, Spooner (1840) has reported that a great proportion of the heavy dray-horses in London have ossified cartilages and throughout the country cart-horses with ossified cartilages are common. Youatt (1874) states that the condition is so prevalent in some districts, especially in the Midland counties, that it is difficult to find aged cart-horses without ossification of the cartilages. Hayes (1950) states that at least 50% of heavy draught-horses in large towns of England and Scotland have side bone.

Townsend (1935) in discussing the condition amongst army horses in the United Kingdom states that during the period 1933-1934, 52 horses were affected with side bone, 11 of which were destroyed because of advanced ossification. In the subsequent 2 years, 40 horses were affected, of which 6 were destroyed for the same reason.

The occurrence of the condition in North America is reported by Lacroix (1916), Holcombe (1923), Liautard (1923) and in South America by Pires (1949) and Cresswell and Smythe (1963). Pires (1949) records that during the years 1938-1945, 76 cases (~~2.50%~~ of 15,012 horses examined at the surgical hospital of the University of Buenos Aires) were affected with side bone.

The condition has been recognised and reported in France, by Solleysel (1664) and La Fosse (1754); in Germany, by Lungwitz (1898), Witte (1907), Ullrich (1934) and Muller (1937); in Australia, by Cameron (1910), Robertson (1932) and Dixon (1963); in Sweden, by Waxberg (1953); Austria, by Zimmermann (1909); in Belgium and Holland, by Lungwitz (1906); in Norway, by Haakenstad (1969), and in India by Stewart (1938).

### 1.3.1 Breed Incidence

Some authors consider that the condition is confined to heavy draught-horses (White, 1825; Spooner, 1842; Youatt, 1874; Thompson, 1895; Elliman, 1904; Biddell, et.al. 1910; Cameron, 1910; Lacroix, 1916; Hayes, 1950); Hungerford, 1953; Smith and Jones, 1957; Frank, 1959; Smythe, 1959; Wooldridge, 1960; Hickman, 1964).

Cameron (1910) in his notes on evidence of side bone, states that

this form of unsoundness is practically confined to draught horses. He has noted also that no thoroughbred horse and no pony has been found affected with side bone and in light horses side bone is so rare that it may be considered negligible.

However, Williams (1872), Fitzwygram (1894), Lungwitz (1898), Axe (1906), Moller and Dollar (1906), Witte (1907), Zimmermann (1909), Leeney (1911), Reeks (1918), Hutton (1933), O'Connor (1934), Pires (1949), Cresswell and Smythe (1963) and Adams (1966), all agree that the condition is not confined to heavy draught animals but it affects the lighter breeds with less frequency. Witte (1907), gives the percentage incidence of side bone as follows:

- a) Heavy horses working on hard roads at quick pace; 94.4%
- b) Light horses working on hard roads at quick pace; 83.0%
- c) Light horses working on hard roads at slow pace; 75.8%
- d) In cavalry troop horses; 7.5%

Zimmermann (1909), reports that the examination of 1,000 horses which were brought to his clinic within two years, revealed that 62 were heavy horses, 44 of which, 70.9% were affected by side bone; 900 were lighter van or coach horses, 279 of which, 31%, were affected, and 38 were saddle horses with 33  $\frac{1}{3}$ % showing side bone. Of the 1,000 animals examined, therefore, 333 (33  $\frac{1}{3}$ %) showed ossification of the cartilages of the hoof.

From the above evidence it is clear that most authorities agree that the incidence of side bone shows a predilection for heavy horses and there is evidence to show that the condition occurs less commonly

in the lighter breeds. It is interesting to note that no mention is made of the condition in thoroughbred animals.

### 1.3.2 Age Incidence

From the early records, up to the present date, most authors relate the development of the condition to animals which are beyond the age of maturity (Williams, 1872; Youatt, 1874; Witte, 1907; Zimmermann, 1909; Hayes, 1950; Hungerford, 1953; Waxberg, 1953; Smythe, 1959; Hickman, 1964).

Witte (1907) records that side bone usually appears between 8 and 12 years of age; it is uncommon among young horses and he has never seen a case in an animal under four years of age.

In the 1,000 animals examined by Zimmermann (1909), 21 were under 5 years old and in this group alterations indicating ossification were detected in only one 4 year old heavy horse.

Waxberg (1953) in his report stated that, in colts and young horses the incidence of the condition was low, but among horses over 4 years old approximately 50% of mares and 25% of stallions were affected.

Smythe (1959) states that, in heavy horses calcification of the cartilages proceeds from 9-10 years onwards as a natural event, but to a lesser extent in light horses unless they are crossbred, as are most heavy weight hunters which frequently develop some degree of calcification of their cartilages after 10 years of age.

Moller and Dollar (1906), Reeks (1918), Hutton (1933), O'Connor (1934) and Woolridge (1960), relate the first development of the condition

to the time the animal put to work.

Cameron's (1910) records show that side bone occurs at 2 years old and the incidence increases each year to maturity. The condition occurring most frequently in aged horses.

### 1.3.3 Distribution

With regard to the distribution of the condition over the 8 cartilages of the horse's feet two opinions have been expressed:

One, held by the majority of authors, is that ossification of the cartilages is more frequently met with in the fore than in the hind feet (Williams, 1872; Youatt, 1874; Lungwitz, 1898; Moller and Dollar, 1906; Witte, 1907; Zimmermann, 1909; Cameron, 1910; Lacroix, 1916; Holcombe, 1923; Miller, 1928; Hutton, 1933; O'Connor, 1942; Pires, 1949; Ensminger, 1951; Leeney, 1952; Hickman, 1964; Adams, 1966).

Cameron (1910) states that the relative frequency was:

254 horses had side bones in fore feet only,

2 horses had side bones in hind feet only,

and 19 horses had side bones in both fore and hind feet

—  
Total 275 of which 273 had side bone in the fore and 21 had side

—  
bone in the hind feet, a proportion of 13 affecting the fore to each 1 hind.

The other opinion is that the condition is confined to the fore feet (Thompson, 1895; MacCallum, 1899; Axe, 1906; Reeks, 1906; Sidney, London.n.d.; Thompson, 1946; Hayes, 1950; Smith and Jones, 1957; Wooldridge, 1960).

With reference to the relative frequency of near-side and off-side affection, Moller and Dollar (1906) state that the cartilages of the near foot suffer more frequently than those of the off, while Cameron's (1910) figures for 254 horses having side bone in the fore feet showed the ratio of incidence between the near and off feet to be 9/10.

With regard to the relative frequency of side bone on the medial and lateral side of the foot, the following authors record that the lateral cartilage is more frequently affected than the medial one (Lungwitz, 1898; Witte, 1907; Zimmermann, 1909; Cameron, 1910; Hutton, 1933; Hayes, 1950). Cameron (1910) found the ratio to be 8/7.

#### 1.4 Aetiology

Theories on the aetiology of side bone can be divided into seven groups.

- 1.4.1 Those who associate the condition with faulty shoeing.
- 1.4.2 Those who consider that trauma is the cause.
- 1.4.3 Those who subscribe to diet being the cause.
- 1.4.4 Those who think that the condition results from the natural process of ageing.
- 1.4.5 Those who blame heredity.
- 1.4.6 Those who consider that structural factors are involved.
- 1.4.7 Those who consider that factors other than those mentioned above are responsible for the condition.

#### 1.4.1 Faulty Shoeing

For over one hundred and fifty years a number of authors have considered that the ossification results from concussion which may be aggravated by the use of calkin shoes. Even recent publications support this idea without factual supporting evidence (Williams, 1872; Youatt, 1874; Gilruth, 1892; Fitzwygram, 1894; Thompson, 1895; Axe, 1906; Sidney, Lond.n.d.; Biddell et. al., 1910; Lacroix, 1916; Holcombe, 1923; Miller, 1928; Hutton, 1933; O'Connor, 1934; Thompson, 1946; Hayes, 1950; Leeney, 1952; Frank, 1959; Wooldridge, 1960; Cresswell and Smythe, 1963; Adams, 1966).

Williams (1872) stated that the cause of the condition was due to shoeing with high calkins and in support of his remarks he comments that high heeled shoes result in:-

- (a) Shock, received by the heels when the foot comes to the ground, being transmitted directly to the cartilages.
- (b) The pressure upon the heels of the wall being unnatural and excessive, the frog being prevented from taking its proper proportion of shock and weight.
- (c) The cartilages are drawn inwards and downwards when the sensitive frog is depressed.

Youatt (1874) stated that the cartilages are designed to preserve the expansion of the upper part of the foot. He considered that when the lower part is limited or destroyed by shoeing, the cartilages became inflamed with the result that cartilage becomes replaced by bone.

Gilruth (1892) stated that in cases where shoeing interfered with normal



frog pressure particularly when the frog was raised from the ground by means of calkins or other methods of shoeing, it is almost invariably found that the cartilages become ossified. He states that even with plain shoes, the foot is far from being in a natural condition, for in most cases no frog pressure is allowed, hence the whole of the weight is transmitted on to the wall which, although far better than on three points, as with calkins, shoeing still limits the use of the frog, the specially adapted portion of the foot for preventing concussion. Consequently, the frog atrophies and becomes predisposed to disease, thereby predisposing other parts of the foot to disease. Biddell et al. (1910) states that the condition is often due to faulty management of the horse's foot in the process of shoeing, one side of the hoof being left higher than the other, so the foot and limb become twisted, outwardly or inwardly when the horse puts his weight on the leg. He, therefore, advises that to maintain a healthy foot and continued integrity of the cartilages, the hoof should be at all times properly levelled when the horse is being shod.

Cameron (1910) who examined 2,636 horses found the incidence of side bone to be above 10% and with regard to side bones being caused by the use of calkins or high-heeled shoes, he records that practically all the draught horses he examined were shod without high-heels.

#### 1.4.2 Traumatism

Those who support trauma as a cause of side bone, fall into three groups. White (1825), Biddell et al. (1910), Holcombe (1923), Liautard (1923), Miller (1928), Stewart (1938), Hayes (1950),

Dixon (1963) and Adams (1966) all support the theory that side bone results from bruises and other injuries to the coronet, such as over-reach, treads, etc.

Lacroix (1916), Robertson (1932), Hutton (1933), Ensminger (1951), and Hickman (1964), support the idea that side bone results from the effect of concussion upon the foot.

Youatt (1874), Gilruth (1892), Fitzwygram (1894), Lungwitz (1898), Elliman (1904), Axe (1906), Sidney (Lond.n.d.), O'Connor (1934), Leeney (1952), Smith and Jones (1957) and Frank (1959) consider that side bone results from either bruises on the coronet or concussion on the foot.

Cameron (1910) in his survey of 2,636 horses states that few of these horses had done any kind of work, particularly road work. The roads travelled by stallions during their season had an earth surface and were frequently cushioned by dust or grass. He doubts whether side bone is ever caused by external violence per se.

However, according to Cameron (1910), Smithcors (1963), Pritchard (1965) and Adams (1966) the exciting causes of many affections in the fore feet is due to concussion and strain of weight bearing. More weight is borne during rest and greater concussion is sustained during movement by the fore limbs than the hind for the following reasons:

- (a) The fore limbs bear more weight than the hind, because of the weight of the head and neck.
- (b) The column of bones of the fore limbs being practically perpendicular from the elbow down is more rigid than that of the hind

limb, in which the angle formed by the tibio-metatarsal bones tends to lessen jarring.

#### 1.4.3 Dietary Causes

Mitchell (1936) in a general consideration of bony disease conditions mentioned in the Horse Breeding Acts considers that defective nutrition in the early stages of development is a predisposing factor which renders the tissues more susceptible to the effects of stresses and strains.

Stewart (1938) working with mules in Rawalpindi where the animals stood in the open all the year round with little or no shade even from trees, considered that an excessive production of Vitamin D from exposure to very strong sun, combined with good supply of calcium, which was believed to be present in the water, might be responsible for an over calcification of cartilages. However, these nutrition theories have not been supported by conclusive evidence.

#### 1.4.4 Natural Process of Ageing

The theory supported by many of the more recent authors on this subject is that the cartilage undergoes natural ossification with advancing age.

O'Connor (1934 and 1942) states that ossification of the cartilages occurs as naturally as ossification in developing bone invades cartilage. Smythe (1959) states that in heavy horses calcification of the cartilages proceeds from 9-10 years onwards as a natural event. Woolridge (1960) and Hickman (1964) consider that ossification may be a natural physiological process because there is always a tendency for cartilage

which is contiguous with bone to undergo ossification with ageing. Cresswell and Smythe (1963) comment that replacement of cartilage by bone is part of the normal ageing process.

Of those who comment on the influence of ageing as the cause and incidence of the condition, all seem to be agreed that the progressive incidence is related to the increasing age of the animal.

#### 1.4.5 Heredity

The idea of an hereditary predisposition being the basic cause of side bone, claimed support from many of the most eminent veterinarians of the last century and this opinion is still widely held. The acceptance of heredity as a causal agent of side bones was doubtless the grounds for precluding the licensing of such animals for breeding purposes (Horse Breeding Act 1918).

The following authors are agreed that heredity is responsible for side bone (Williams, 1872; Youatt, 1874; Fitzwygram, 1894; Thompson, 1895; Elliman, 1904; Axe, 1906; Moller and Dollar, 1906; Reeks, 1906; Biddell et al., 1910; Cameron, 1910; Leeney, 1911; Miller, 1928; Robertson, 1932; Hutton, 1933; O'Connor, 1934; Wilson, 1935; O'Connor, 1945; Thompson, 1946; Hayes, 1950; Ensminger, 1951; Leeney, 1952; Hungerford, 1953; Waxberg, 1953; Wooldridge, 1960; Cresswell and Smythe, 1963; Hickman, 1964; Adams, 1966).

Cameron (1910) who analysed the breeding of certain families of draught horses, states that the condition is of hereditary nature, while other causes (shoeing, work, concussion, etc.) are false factors.

However, Cameron's records are open to criticism on three counts. The first, his animals were not maintained under comparable conditions. Although he stated that, "there is no reason to believe that the hereditary influence of the dam is other than equally potent to that of the sire", the only examination he made was of the male progeny. Secondly, he had no efficient means of detecting the early stages of the development of the condition, there may, therefore, have been many cases passed as free which might have been affected. Thirdly, he had the facilities in his investigation to examine younger animals (less than two years old), but he did not report on them. His records are, therefore, not complete.

Robertson (1932) over approximately eleven years examined 7,894 horses and of these 4,957 were draught animals. His investigation records particularly the hereditary nature of side bone on the sire's side, also the influence of dams of unsound sires on sound families. He concluded that there is clear indication of the influence of the "dam" in introducing unsoundness into sound families and in increasing unsoundness in unsound families.

Like Cameron's (1910) records, Robertson's (1932) although different are still substantially lacking in conclusive evidence.

#### 1.4.6 Structural Factors

Although it is appreciated that heredity has an important influence on conformation of the horse's foot, some authors consider that certain features in the conformation of the foot as distinct from hereditary

tendencies per se, predispose to the condition. Thompson (1895) comments that side bone development is very rarely found in flat footed horses. MacCallum (1899) believes that the condition usually affects horses with long oblique pasterns. Moller and Dollar (1906) state that the condition is often progressive, and is much more serious in animals with upright, narrow heels, where the horn does not yield, than in broader heels with more elasticity. Leeney (1911) and Waxberg (1953) appear to support this view also. O'Connor (1945) considers that faulty conformation and action are transmitted from parent to off-spring. They result in unequal distribution of weight on the limbs which in turn results in side bone. Pires (1949) attributes the condition to bad conformation of the foot. Adams (1966) states that, side bones are most common in horses having poor conformation. He then locates the condition according to the conformation of the horse's foot, by saying that, horses with toe-in are prone to develop lateral side bone while horses with toe-out are prone to develop medial side bone. However, in both these conformations, side bone may eventually develop in both cartilages.

#### 1.4.7 Other Diseases

A number of other conditions are believed to contribute or predispose to side bone. White (1825) finds that the disorder is often met with and sometimes caused by quittor. Reeks (1906) considers that side bones result from other diseases of the foot, they occur as a sequel to subhorny quittor, suppurating corn and to complicated quarter sandcrack.

Liautard (1923) supports quarter cracks and quittor as a cause and adds corns and sprains. Other diseases are mentioned by Ensminger (1951), while Wooldridge (1960) includes inflammatory diseases of the foot, such as quittor, suppurating corn and complicated quarter sand crack.

1.5 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate Other than from the Proximal Palmar Process of PIII

The following authors agree that there are bony islands found in different parts of the cartilage plate of the hoof; not associated with PIII (Spooner, 1840; Williams, 1872; Pritchard, 1888; Gutenacker, 1901; Vogt, 1906; Lungwitz, 1919; Habacher, 1921; Lowe, 1935; Muller, 1937; Bengtsson, 1962; Raker, 1968; Dietz, 1968). Lowe (1935), using X-ray technique, found that in 13 cases there were bony islands in different parts of the cartilage. Muller (1937) during his investigation, noticed that in heavy horses aged (2-20) years in 10 out of 40 cases there were bony islands ranging in size from 3 mm. to 4 cm. in diameter. Those islands had no connection with the ossification at the palmar process of PIII, although in 6 of these cases he also found evidence of ossification starting from the palmar process of PIII. In 4 other cases he recorded bony islands without noticeable changes in the palmar process of PIII.

Witte (1907) has stated that it is not uncommon to find slit-like fractures running in different directions of the ossified part of the cartilage, these slits had rounded and smooth edges. Other authors considered this feature as being different centres of ossification, mainly Pritchard (1888), Gutenacker (1901) and Lungwitz (1919). However,

Witte (1907) did not share this opinion.

Cameron (1910) stated that amongst hundreds of horses examined, "I have never seen any specimen showing evidence of ossification having commenced at the summit or margins of the cartilage. It always commences at the base, where the cartilage is joined to and rests on the palmar process of PIII". However, Lowe's (1935) radiological findings and Muller's (1937) histological description of these bony islands are convincing evidence of their existence.

## 2.0 CLINICAL AND RADIOLOGICAL OBSERVATIONS MADE IN THE CURRENT INVESTIGATION

### 2.1 Materials and Methods

Four hundred and sixty-five equines and two donkeys presented at the large animal clinic of the Royal (Dick) School of Veterinary Studies during the period 1967 - 1970 were examined clinically and radiologically. Also reviewed were the X-ray films of 232 equines presented during the period 1958 - 1962 and the case records of 83 horses presented during the period 1963 - 1966.

The patients were of two categories; one, those admitted to the hospital for surgical or medical treatment and the other category, those presented as out-patients for clinical and radiological examination of the feet. In both categories the history, breed, age and sex of each animal were recorded.

In the first category, animals were weighed on being admitted,



rested and observed in the horse box. Next morning the animal was examined standing squarely with the weight evenly distributed on all four feet. It was next quietly walked then straightaway trotted slowly as required, giving particular attention to the animal while turning. It was turned in a tight circle to the left and right, and backed to detect any lameness. It was then taken to the theatre for manual and radiological examination. The ease with which the manual examination is performed depends largely on the extent to which the animal has been previously handled; all sources of sudden movement or any actions likely to upset or excite the horse were avoided. The manual examination was carried out by having the horse standing with his weight equally distributed on all four limbs. The whole limb was examined by inspection, palpation and manipulation.

The cartilages of the hoof were examined (a) with the animal's foot carrying weight. The procedure was to place the examiner's left foot between the horse's hoofs from the front, with the left knee pressed firmly against the front of the horse's near knee; bending over towards the back of the limbs, with the thumbs of both hands pressing upon the cartilages, or having the horse shift his weight on to the foot being examined, by an assistant lifting the opposite limb; (b) with the foot raised, the upper part of each cartilage is grasped between fingers and thumb and moved from side to side.

#### 2.1.1 Type of Horses

The number and percentage of each type of equine presented over the period 1963 - 1970 is indicated in Table 3.

### 2.1.2 Radiological Examination

The horse's shoes were removed wherever practicable, the hoof trimmed and all foreign material removed. It was not found necessary to pack the collateral and central grooves of the frog since the shadows of these grooves lay away from the areas of side bone and did not interfere with the interpretation of the films.

The horses to be examined were restrained by an assistant controlling the head. It was not found necessary to resort to the use of sedatives. Some difficulties were encountered with unbroken horses, particularly in accurately positioning the hind feet for radiological examination. In most cases radiography of the hind feet took at least twice as long as for the fore feet.

### 2.1.3 Positioning

Tavernor and Vaughan (1962) state that for an anteroposterior view of the foot, the hoof should be tilted so that the wall at the toe is vertical, the cassette is then placed vertically behind the heels and the beam directed horizontally at the centre of the coronet. As it is not possible to direct a beam horizontally at ground level, the hoof is raised on a wooden block.

Carlson (1967) states that for the anteroposterior view the radiograph should be taken with the animal's hoof directly upon a (screened or non-screened) cassette which lies flat on the floor. The central X-ray beam being directed at an angle of 45 degrees to the floor and centred at the distal interphalangeal joint. For better study of

the lateral and medial palmar processes of PIII, he advises that oblique views may be taken with the cassette again flat on the floor.

Anteroposterior oblique views of the palmar processes of PIII are taken with the central X-ray beam directed at an angle of 45 degrees to the floor and at a 45 degree angle to the median plane, the beam being centered at the lateral or medial palmar process of PIII. The X-ray cassette may be divided by a lead cover so both processes of PIII can be demonstrated on one 20 x 25 centimetre cassette.

In the present investigation both methods were used, that is with the cassette behind the heels (with the hoof raised) and with the cassette on the floor. In the first method, a wooden block was used with a gutter at an angle of 45 degrees to the floor to secure the toe so that the anterior wall of the hoof at the toe was vertical. This position is comfortable to most animals. Using a mobile X-ray unit, the cassette was placed close behind the heels using a special holder (Fig. 22), and the exposure made with the X-ray beam horizontal to the floor and directed at the centre of the coronet. The exposure varied from 60-80 K.v. x 100 ma. x 0.04 second at a 76-90 centimetre focalfilm distance and the K.v. varied in accordance with the size of the specimen. *Using a Kodak Royal Blue Medical Film and a High Speed Intensifying Screen.* According to Carlson (1967) radiographs taken between 76-90 centimetres provided sufficient distance to minimise distortion.

Anteroposterior oblique views of the palmar processes of PIII were taken with the foot in the same position in the wooden block, the cassette being placed vertical to the respective heel and the beam directed

horizontally at a 45 degree angle to the median plane of the foot. Using a film, divided by a lead cover, both palmar processes of PIII were shown by two exposures.

The second method, with the foot directly upon a cassette flat on the floor, was used occasionally when horses would not take the weight on the other foot, but this method was not favoured because most animals objected to standing on the cassette and an assistant was required to keep the foot in position.

## 2.2 Side Bone

### 2.2.1 Definition

During the present investigation the term "side bone" is used to express only any degree of bony invasion from the proximal palmar process of PIII into the cartilage of the hoof.

### 2.2.2 Interpretation of Radiological Examination of Side Bone Formation

It has already been stated (section 7.2) that the proximal part of PIII completes its ossification at the age of 5 months. At this period of the animal's life the anteroposterior radiograph of PIII (Fig. 23A) shows the contour of the proximal palmar process as a smooth rounded elevation on either side of PIII. Such radiographic view has been used as reference for comparison with radiographs of older animals (Fig. 23B and C) which show irregularity in the outline of the proximal palmar process, due to bony invasion of the cartilage (side bone) from the proximal palmar process of PIII.



Such changes in the outline of the proximal palmar processes were better seen with the aid of a magnifying glass and a spotlight, in X-ray films of foals aged 6 and 7 months. At the age of 8 months and over the irregularity of the bony invasion disappears and the outline looks clear and a well-defined projection (side bone) is seen (Fig. 23D).

### 2.2.3 Classification of Side Bone

It was decided to describe the extent to which side bone was present as "short", "medium", "long" and "marked" (Figs. 24-27). This classification is based on the anatomical relationship of the side bone to other bony landmarks of the foot (see 2.1.3).

In short side bone the ossification does not extend beyond a line level with the proximal border of the distal sesamoid (navicular) bone (Fig. 24). Medium side bone extends beyond the level of the proximal border of the distal sesamoid, but not beyond a line running through the middle of PII (Fig. 25). Long side bone extends from the middle of PII to its proximal articular surface (Fig. 26). Marked side bone extends beyond this line, that is, the proximal articular surface of PII (Fig. 27). The term "high" degree of side bone was used to include "medium", "long" and "marked" side bones.

## 2.3 Interpretation of the Various Features Seen on the Radiograph of the Foot

### 2.3.1 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate other than the Proximal Palmar Process of PIII

In the present study of 465 clinical cases, radiological investigation revealed that 46 cartilages (31 horses) showed well defined bony

islands in different parts of the cartilage of the hoof (Table 4 and Figs. 24B, 27P and 28).

From 80 post-mortem cases, in 17 horses radiological examination revealed that 66 cartilages showed bony islands. These 17 horses represented different breeds and their ages ranged from 4 - 23 years (Table 5). Radiological diagnosis of very small bony islands was not practical in the undissected cartilages. Clinical detection of the bony islands was impossible unless they were of considerable size in the subcutaneous half of the cartilage.

#### 2.3.2 Location, Origin, Shape and Size of the Bony Islands

The radiological findings have shown that these bony islands are scattered at random in the cartilage with no fixed site of origin (Figs. 29-35). They range in size from a tiny spot or spots to a well defined bony island measuring up to 38.0 x 39.0 mm. (Table 5).

The shape of the bony island is not constant, whether in the same cartilage, foot or animal (Figs. 29-35).

#### 2.3.3 Description of the Radiological Features of the Bony Islands in Relation to Side Bone

During the radiological examination difficulty was encountered in classifying side bone of certain radiographs which showed a bony island in the vicinity or in direct contact with side bone (Figs. 27N, 27P, 28D and 31). Figs. 27N and 31 give the appearance of fractured side bone and Fig. 28D gives the appearance of bony island joined with side

bone. Of these feet, the foot shown in Fig. 31 was dissected and the lateral radiograph (Fig. 30) showed that the bony island is separate from side bone. In those cases where a bony island was completely apart from the side bone (Figs. 24B, 27P (NFL) and 28), only the side bone was used in the classification. In other cases where the side bone appeared to be fractured, or where the bony island and side bone appeared continuous, the whole extent of the bony change was used in classification (Fig. 27N).

It was not possible to examine horses feet, with bony islands, at different intervals of their lives; therefore, it was difficult to decide whether or not these bony islands were going to blend into the side bone to form one bony unit and give the appearance of a high degree of side bone. In one case, a 16 year old British percheron which showed a bony island in the 4 fore cartilages, radiological examination at an interval of 5 months, showed no gross changes in the size or distance of any of these islands from their specific side bone.

#### 2.3.4 Ossification Centres (Bony Islands) that arise at the Level of the Distal Palmar Process of PIII

During the radiological examination of the feet in the antero-posterior position, incidental rotation of the foot sometimes occurs due to difficulty in controlling the animal. On such occasions X-ray films revealed that there were sometimes bony islands in unexpected places of the foot, particularly, at the level of the distal palmar process of PIII (Figs. 36 and 37). Such X-ray exposures were frequently

used with advantage to show full details of the palmar processes of PIII.

In the present study 24 post-mortem cases have had 34 bony islands at the level of the distal palmar process of PIII. Although their location is fixed the distances between the bony island and the distal process is variable. The bony islands may be only slightly separated from the distal palmar process (Fig. 34), or at varying distances from it. However, it was never more than 3.0 mm. distant (Table 6). The shape and size is variable (Table 5 and Figs. 29, 32, 33, 39, 40, 41 and 42) and they may occur as a single island or multiple islands (Fig. 41).

It is not unusual for a bony island to blend smoothly into the distal plantar process of PIII, forming one bony unit showing no evidence of any reaction surrounding the union (Fig. 38).

#### 2.3.5 Interpretation of the Dense Area Found in the Region of the Palmar Process of PIII

Interpretation of the various features seen on the radiograph of the foot, other than a dense area in the region of the palmar process (Fig. 43), were studied and were found to conform with the descriptions in the standard orthopaedic and radiology books (Carlson, 1961; Hickman, 1964; Schebitz and Wilkens, 1968; Douglas and Williamson, 1970).

Study of the 1586 radiographs of horses aged 2 years and over, made in the large animal clinic of the Royal (Dick) School of Veterinary



Studies during the period 1958 - 1970, revealed that 1138 had a dense shadow at a constant part of the palmar process and it was thought that this dense area was somewhere in the distal palmar process.

Specimens showing the above feature were boiled out; X-rayed; examined macroscopically and were sectioned transversally at various thicknesses. Figure 44 is the medial half of OH third phalanx cut into 5 transverse sections; section A1 is cut at 6 mm. distant from the solar surface, section A2 is 4 mm. thick, sections A3 and A4 are each 6 mm. thick and the 5th section contains the proximal part of PIII and most of the side bone. Section 3 is the only one which showed two different shadows at its caudal end. The first (short arrow) indicates the dark area of the foramen of the plantar process and the second (long arrow) indicates the dense area of the bony bridge which converts the palmar notch into a foramen (section 7.1).

To show that the dense area is not in the solar surface of the distal plantar process but is due to the presence of the bony bridge, is confirmed by radiographs 44B and C. Radiograph B consists of sections A1, 2, 4 and 5, omitting section 3 and it shows no dense area. In radiograph C section 1 was not included, but the shape and size of the dense area (section 3) is shown. Figure 45A-D is a series of radiographs of the boiled PIII, shown in Fig. 43. Fig. 45A shows a dense area on each side. The lateral one is well defined and the medial, less well defined, has no depression on the outer aspect of posterior part of PIII (arrow) as seen on the lateral one. If the bony band (section 7.2) which sometimes covers the parietal groove is absent

the dense area becomes more evident. This is illustrated by Fig. 45 radiographs A to D. In A the band is present on the medial side; in B, C and D it is absent where a distinct notch is shown. In radiograph C the lateral bony bridge was removed and in D both bridges were removed, consequently the medial dense area in C is more obvious and no dense areas are shown in D.

Figures 46 and 47 are radiographs of PIII of a 4½ years Clydesdale in which a foramen was formed in the lateral palmar process only. Figure 46 shows a dense area on the lateral side.

To locate the shape and size of the dense area, PIII was placed on its solar surface, then radiographs taken at angles of inclination of 60, 70, 80 and 90 degrees with the horizontal and the X-ray beam directed at the centre of the extensor process. Figure 46 shows that the location, size and shape of the dense area differs on the radiograph with the degree of inclination. After removing the bony bridge (Fig. 47) the dense area disappeared completely.

#### 2.3.6 Description of the Relationship of the Dense Area to the Bruised Angle of the Bar

It was noticed that in the radiographs of the feet of most horses showing bruises at the angle of the bar, such dense areas were seen. It was decided to investigate this relationship. Using a paste of Barium sulphate, horse's feet showing such features were marked at the point of the bruises (angle of the bar) and X-rayed to show the relationship. It was found that the distal palmar process overlies the angle

of the bar (Fig. 48) and that the site of the bruise lies below the bony bridge.

In the dissected cartilages of the feet which were examined by manipulation (Table 1) it was found that in some cases the distal palmar process was, to a certain extent, flexible while others were more rigid. When the soft tissues were more completely removed further manipulation revealed that the rigidity was due to the bony bridge across the palmar notch, furthermore the removal of this bridge restored once more the flexibility to the distal palmar process.

The presence of this bony bridge which reduces the flexibility of the palmar process may therefore have some clinical significance in relation to bruises at the angles of the bars.

#### 2.4 Clinical and Radiological Observations

In the present investigation, radiological examination of 2892 cartilages from 527 equines and 2 donkeys ranging in age from 6 months to 26 years, all showed some side bone development.

Of these animals, 246 (46.7%) showed no sign of lameness while the remaining 281 (53.3%) were lame to varying degrees.

Of the 246 'sound' animals 199 showed a short degree of side bone, 31 showed a medium degree, 1 a long degree and 4 showed a marked degree of side bone. Of these 246 animals 231 showed no other foot conditions while in the remaining 15 animals concurrent conditions were evident, namely: 12 had bruised foot, 2 dropped sole (1 of which showed a ring

bone of PI in three feet) the fifteenth had sand cracks in all four feet. With regard to the degree of side bone in these 15 animals, 13 showed a short degree and the remaining 2 showed a medium degree of side bone.

Of the 281 lame animals in only 5 of them was the lameness not determined, in the remaining 276 animals the lameness was diagnosed as due to other recognisable pathological changes.

Of the total 281 lame animals, 213 showed a short degree of side bone, 45 showed a medium degree, 14 showed a long degree and the remaining 9 animals showed a marked degree of side bone.

With regard to the degree of side bone in the 5 animals without concurrent attributable causes of lameness, one a 4 years lightweight pony and one a 12 years heavyweight pony showed a short degree; 2 lightweight hunters aged 7 and 9 years showed a long degree and the remaining one a 9 years heavyweight hunter showed a marked degree of side bone.

The incidence of the degree of side bone in the different breeds and crossbred equines was as follows:

Type of Equine	Number Examined	Short Side Bone	Medium Side Bone	Long Side Bone	Marked Side Bone
Shetland pony	22	22			
Exmoor pony	13	13			
Welsh pony	14	10	4		
Welsh cob pony	3	2		1	
Welsh X	13	10	3		
Iceland pony	1	1			

Type of Equine	Number Examined	Short Side Bone	Medium Side Bone	Long Side Bone	Marked Side Bone
Fell pony	2	2			
Highland pony	8	6	1	1	
Riding pony (lightweight)	21	21			
Riding pony (heavyweight)	24	11	7	3	3
Trotter	16	16			
Thoroughbred X	13	13			
Thoroughbred X riding pony	26	21	5		
Thoroughbred	208	179	26	2	1
Hunter (lightweight)	111	79	23	9	
Hunter (heavyweight)	19	8	5		6
British Percheron	2	1	1		
Clydesdale	5	5			
Heavy draught	5	2	1		2
Irish draught	1				1
Overall	527	422	76	16	13

The incidence of the degree of side bone in relation to age was as follows:

Age (Y)	Short	Medium	Long	Marked	Total
Under 1	32				32
2	17	2			19
3	19	7		1	27
4	34	5		1	40
5	34	6	1	1	42
6	61	9			70
7	41	10	2	2	55
8	32	6	1		39
9	37	11	5	2	55
10	27	3	2	1	33
11	13	2	1		16
12	18	2	1	2	23
13	8				8
14	5	2	1	1	9
15	5	1	1		7
16	3	1			4
17	8	3	1	1	13
18	4				4
19	2				2
20	16	1		1	18
21					
22					
23	5	4			9
24					
25		1			1
26	1				1
Totals	422	76	16	13	527

The breed and age incidence in relation to side bone degree was as one might expect, the highest incidence was the short degree side bone and this was particularly so in young and very young animals. Whereas, with one exception, a 5 year thoroughbred, the 13 equines showing a marked degree of side bone were heavy horses and 10 of these occurred in animals over 6 years old.

Of the 1437 feet from the 527 equines which were examined, (443 NF, 440 OF, 277 NH and 277 OH) a short degree of side bone was shown by 1247 feet and the remaining 190 showed a high degree, that is Md. = medium, Lg. = long or Mk. = marked. The proportions were 85 NF (65 Md. + 9 Lg. + 11 Mk.), 85 OF (63 Md. + 13 Lg. + 9 Mk.), 9 NH (md.) and 11 OH (8 Md. + 3 Lg.).

These 190 feet (105 equines) with high degree side bone, in 29 a single foot was examined, in 32 both fore feet were examined, in 1 three feet were examined and in the remaining 43 all four feet were examined.

In the 43 in which all feet were examined the distribution of side bone was as follows: 6 showed a high degree in OF foot only, in 27 it was present in both fore feet, 1 showed a high degree in three feet (both fore and OH foot) and the remaining 9 equines showed the high degree in all four feet.

In comparing these 43 animals for the relative frequency of incidence to the near side and off side; and the medial to lateral side of the four feet, the incidence was as follows:

In 17 equines the cartilages of the NF foot had side bone development to virtually the same extent as in the OF (16 Md. and 1 Lg.). In 10 equines the side bones of NF foot was more advanced (>) than in the OF (5 Md.>Md., 2 Lg.>Lg., 1 Mk.>Md., 1 Mk.>Lg. and 1 Mk.>Mk.). In the remaining 16 equines the side bones of OF foot was more advanced than in the NF (5 Md.>Sh., 9 Md.>Md., 1 Mk.>Sh. and 1 Mk.>Mk.).

Of the 10 equines which showed a high degree of side bone in the fore and hind feet, in 4 a medium degree of side bone in NH foot was virtually the same as in the OH. In 2 the medium degree of side bone in the NH foot was more advanced than the medium degree of OH. In the remaining 4 equines the OH foot showed more advanced side bone than the NH (1 Md.>Sh., 2 Md.>Md. and 1 Lg.>Md.).

The comparisons between medial and lateral cartilages of each foot were as follows:

Of the 37 NF feet, in 19 equines the medial cartilage had side bone development to virtually the same extent as in the lateral (17 Md. and 2 Lg.). In 9 equines the medial side bone was more advanced than the lateral (5 Md.>Sh., 2 Md.>Md. and 2 Mk.>Lg.). In the remaining 9 equines the lateral side bone was more advanced than the medial (6 Md.>Md., 1 Lg.>Md., 1 Mk.>Md. and 1 Mk.>Mk.).

Of the 43 OF feet, in 23 equines the medial cartilage had side bone development to virtually the same extent as in the lateral (22 Md. and 1 Lg.). In 6 equines the medial side bone was more advanced than the lateral (2 Md.>Sh., 1 Md.>Md., 1 Lg.>Md., 1 Mk.>Lg. and 1 Mk.>Mk.).



In the remaining 14 equines the lateral side bone was more advanced than the medial (4 Md.>Sh., 7 Md.>Md., 1 Lg.>Md., 1 Lg.>Lg. and 1 Mk.>Sh.).

Of the 9 NH feet in 5 equines the medial cartilage had side bone development to virtually the same extent as in the lateral (5 Md.). In the remaining 4 equines the lateral side bone was more advanced than the medial (4 Md.>Sh.).

Of the 10 OH feet in 5 equines the medial cartilage had side bone development to virtually the same extent as in the lateral (5 Md.). In the remaining 5 equines the lateral side bone was more advanced than the medial (4 Md.>Sh. and 1 Lg.>Sh.).

The incidence of the high degree side bone in the fore feet compared to the hind feet was 4.3/1. This ratio was what one would expect from the published literature (section 1.3.3).

The incidence of high degree in both fore feet compared to one foot was 6.2/1.

The incidence of high degree between the near and off feet was as follows:

In the fore feet : 8 OF/5 NF

In the hind feet : 4 OH/2 NH

Although the ratio of the extent to which side bone developed on the lateral cartilage in relation to the medial cartilage of NF foot showed no difference, a difference was shown in the ratio between the cartilages on the different sides of the other three feet:

	<u>Foot</u>	<u>Lateral/Medial</u>
	NF	1/1
	OF	7/3
	NH	4/0
	OH	5/0

### 3.0 DISCUSSION AND CONCLUSION

In the light of the comments made regarding the anatomy of PIII and its palmar processes, and considering the clinical and radiological investigation of side bone, the various opinions and theories of other authors may be brought into perspective. The following points merit particular mention.

Side bone development and other changes in the cartilage and the palmar process of PIII are best seen by anteroposterior and anteroposterior oblique radiological examination.

Side bone is described, as any degree of bony invasion from the proximal palmar process of PIII into the cartilage of the hoof. It can be classified as short, medium, long and marked.

The bony islands and the most common degrees of side bone (short and medium) cannot usually be diagnosed without radiological examination. Diagnosis by digital palpation is quite unreliable unless the side bone is advanced to the stage of being long or marked and unless the bony islands are large.

Although, Cameron (1910) believes that there are no bony islands in the cartilage of the hoof, and Witte (1907) considers that islands are

fractured side bone, the majority of authors found that quite apart from side bone, there were bony islands in different parts of the cartilage plate (Spooner, 1840; Williams, 1872; Pritchard, 1888; Gutenacker, 1901; Vogt, 1906; Lungwitz, 1919; Habacher, 1921; Lowe, 1935; Muller, 1937; Bengtsson, 1962; Raker, 1968; Dietz, 1968).

In this research the high incidence of bony islands recorded in Tables 4 and 5 gives further confirmation of their existence, their shape, size and variable location. Of these 48 horses (Tables 4 and 5), lameness was detected in 9 animals which were also showing other pathological changes of the foot.

Although most authors agree that the bony islands form at random sites throughout the cartilage plate and that they occur commonly from 2 years old onwards, no mention is made in the literature regarding their formation at the level of the distal palmar process of PIII (Table 6).

The dense area sometimes found in the region of the palmar process in animals of 2 years old and over, is due to the presence of the bony bridge which converts the palmar notch into a foramen. A possible relationship between the occurrence of this bridge and the presence of bruises at the angle of the bar is noted.

With regard to lameness and its relationship to bony islands and side bone, there is little evidence from this investigation to support the opinions of those who consider side bone can cause lameness

(White, 1825; Spooner, 1840; MacCallum, 1889; Fitzwygram, 1863; Williams, 1872; Axe, 1906; Cameron, 1907; Lupton, Lond.n.d.; Miller, 1928; Hutton, 1933; Hayes, 1950; Leeney, 1952; Smith and Jones, 1957; Cresswell and Smythe, 1963; Hickman, 1964; Adams, 1966). The weight of evidence supports that when lameness is present the cause is not due to side bone but some other concurrent condition. Of 527 animals examined 281 were lame and 246 were sound. In the lame group a concurrent and obvious cause was present in all but 5 animals, in other words in only 5 animals could side bone be suspected as the sole cause of lameness, whereas in the 246 sound animals side bones were present to the same extent as the 281 lame animals.

In the 5 cases which were lame and in which no other cause of lameness was detected it may be considered that side bones were the cause, however, it is possible that lameness was due to some other undetected cause. The opinion of Waxberg (1953), Smythe (1959), Cresswell and Smythe (1963), Smythe (1967) and Dietz (1968) that side bone does not cause lameness, unless it is associated with concurrent condition in the foot, is therefore largely supported by the evidence of this investigation. The opinion of Muller (1937), Thompson (1946) and Brennan (1958) that ossification of the cartilages progresses without inconvenience or lameness are also supported. However, as it was not practicable to examine side bone development throughout the animal's life one cannot say that some degree of lameness due to side bone or bony island formation could not occur.

The present investigation reveals that side bone is present to some extent in every cartilage of every equine over 6 months old. The short degree of side bone is most common in all types of equines. However, a high degree of development has been revealed as occurring in 11 types and breeds including ponies and thoroughbred horses.

The opinion of Cameron (1910) and many other authors who found that side bone was confined to the heavy draught horses, and Witte (1907), Zimmermann (1908), Pires (1949) and others who agreed with Cameron (1910) but found that the condition could also occur less frequently in light breeds, cannot now be supported. Likewise, the opinion that the condition is limited to mature animals or that it occurred only or more frequently in the fore feet cannot now be supported. Evidence was, however, found to support that a higher degree of ossification was more frequently found in the fore than the hind feet, the ratio being 4.3/1 and there was evidence that the incidence of a high degree of side bone was significantly higher in old animals than in young ones.

Obviously when ossification of the cartilage is present to some degree in all animals over 6 months of age there can be little support to the opinions of those who consider that shoeing can cause or contribute to the condition or that work or trauma are aetiological factors. Likewise, it is difficult to reconcile the cause of side bone to disease or malformation when it seems manifest that the process of ossification of the cartilage is natural and progressive. This evidence that the ossification of the cartilage is or may be a natural

physiological process was certainly noted by O'Connor (1934 and 1942), Wooldridge (1960) and Hickman (1964).

This research has not included a detailed study of the possible influence of heredity or diet on this condition. In the light of the evidence which supports that side bones are a natural process of ossification of the cartilage, it is difficult to support that this normal development is an hereditary disease or that diet has any special significance in its cause.

## 1.2 Fixation

After fixing, the animals were washed in running water for 24 hours, and were then placed in a 10% formalin solution for 48 hours. Approximately 24 hours were allowed to pass of 10%, 10%, 10% and 10% grades. The material was then placed in a mixture of equal parts of absolute alcohol and ether and kept for 24 hours. The material was changed every 24 hours. The purpose of this procedure was to make the material more suitable for sectioning. The following procedure is reported

HISTOPATHOLOGY1.0 MATERIALS AND METHODS1.1 Fixation

Six hundred and twenty eight cartilages of the 314 feet dissected (Table 1) representative specimens were chosen for histological examination (Table 1).

Vertical and transverse sections (Figs. 19 and 21) of the chosen specimens were made by means of a fine fret-saw. These slices 5 - 15 millimetres thick were then placed in a 10% solution of formal saline fixative for at least 7 days. Formalin was chosen because it penetrates well and permits the use of a large variety of staining methods and is an excellent preservative. In the fixation of bone, it is important to ensure that penetration of the fixing fluid is complete because incomplete fixation cannot be remedied at a later stage.

1.2 Defatting

After fixing, the sections were washed in running water for 12 hours, and were then taken through ascending grades to absolute ethanol. Approximately 24 hours were allowed in each of (50%, 75%, 96% and 100%) grades. The material was then placed in a mixture of equal parts of absolute ethanol and ether for approximately 72 hours, the solution being changed every 24 hours. The purpose of this treatment was to defat the marrow spaces of the bone. This defatting procedure is important

because if not carried out the fat protected bone from the subsequent decalcification and remaining bone damaged the cutting edge of the microtome knife, necessitating frequent wearisome and tedious honing to restore the cutting edge. Complete defatting also expedited decalcification.

### 1.3 Decalcification

The methods of decalcification tested include, formal - formic acid and formal - formic acid in the presence of ion exchange resin.

For decalcification by formal - formic acid the following solution was used:

Formalin (40% Formaldehyde)	5 ml.
Formic Acid (S.G. 1.2)	25 ml.
Distilled water	70 ml.

Relatively large volumes of the decalcifying fluid were employed and the solution was changed daily. The specimens were suspended in the fluid by means of a waxed thread. Mallory (1944) recommended that the material should be suspended in the acid solution so that the extracted salts would fall to the bottom of the container, so maintaining contact and access between the solution and the remaining ossified tissue. This process usually took about 20 - 35 days. With this procedure there were no artefacts formed to interfere with the subsequent examination of the sections which were very satisfactory.

The test used to determine that decalcification was complete, was to add concentrated ammonium hydroxide solution (S.G. 0.880) to 5 ml.



of the decalcifying fluid a drop at a time until it was neutralised. 0.5 ml. of a saturated solution of ammonium oxalate was then added. The absence of turbidity after 5 minutes was taken as indicating the absence of calcium ions in the fluid and hence complete decalcification of the bone.

The formal - formic acid solution used with ion exchange resin (Copland, 1967) was:-

Formalin (40% Formaldehyde)	10 ml.
Formic Acid (S.G. 1.2)	15 ml.
Distilled water	75 ml.

The ion exchange resin used was "ZEO-KARB" 225 (Hydrogen form) Cation Exchange Resin, manufactured by the Permutit Company Limited.

The resin was layered over the bottom of a glass dish and covered by the decalcifying fluid. The slices were placed on a disc of filter paper over the resin to prevent the resin granules sticking to the slices during the testing process. The proportion of resin used was about one tenth of the volume of the fluid and twice the volume of the material to be decalcified.

The results obtained by this method were as good as those obtained using no ion exchange resin and complete decalcification was achieved in only 4 - 12 days, using only one batch of fluid and resin. A disadvantage in using the ion exchange resin was that the chemical test for determining the presence of calcium salts could not be done directly on the decalcifying fluid. To get a positive chemical test result,

the slices had to be placed in fresh decalcifying fluid for 3 hours without the ion exchange resin being present. In this investigation the presence of calcium salt was determined by X-ray examination. The chemical test was not used as the standard procedure.

#### 1.4 Neutralisation

After the slices had been decalcified they were neutralised with 5% Sodium carbonate for 12 hours to remove residual acid. They were then washed under running tap water for the same period.

#### 1.5 Dehydration

Following neutralisation, the slices were dehydrated with ascending grades of ethanol as follows: 50% for 24 hours, 75% for 24 hours, 96% for 12 hours and absolute ethanol for 6 - 12 hours.

In the last two grades they usually passed through two changes of each grade. Also, the time was reduced to a minimum in both grades to avoid undesirable hardening of the tissues.

#### 1.6 Clearing

Chloroform was used as a clearing agent. Slices were passed through two changes of this agent with six hours in each. Longer periods of time were tried but hardening occurred. Benzene, when tried, made the cutting process very difficult.

#### 1.7 Impregnation

The slices were impregnated in an oven embedder and "Paraplast" (Biological Research Inc., Bridgeton, MO, USA) was found to give

superior results to the usual paraffin waxes. Although both the manufacturers and Culling (1963) state that "Paraplast" eliminates the necessity to cool, it was found that cooling the "Paraplast" wax blocks with ice made section cutting much easier. In order to lessen the hardening effect of heat, the time spent in the wax oven was reduced to the absolute minimum, usually 8 hours were allowed. Three changes of wax were made during this period.

#### 1.8 Section cutting

The Heavy Duty Rotary Microtome (Baird and Tatlock Ltd., London) was used and sections were cut 5 - 7 micrometres thick. Interrupted serial sections (every tenth section) were mounted on clean slides and left 9 hours in the drying oven at 37 - 40°C. Higher degrees of heat were tried but the sections tended to raise from the slide and were washed off during the staining process.

The preparation of the interrupted serial sections of bone presented two main difficulties.

Firstly the blocks contained decalcified bone, cartilage, ligaments, tendon and other tissues and, since different degrees of density were passing over the knife blade at the same time, folding and distortion of the sections commonly resulted.

It was very difficult to produce good continuous ribbons of sections especially at predetermined section thickness because of folding and fragmentation.

These problems were largely overcome by regular and frequent

sharpening of the microtome knives and also by the application of ice to the surface of the block between every few sections.

### 1.9 Staining

Several stains were selected to show the different tissue elements so that they were readily distinguished.

The following stains were used.

Haematoxylin and eosin was used as a routine stain. Ehrlich's alum haematoxylin was tried and gave satisfactory results. The counter stain used was 1% aqueous eosin. Weigert's iron haematoxylin and Van-Gieson, Mallory's Aniline blue-orange G. method, Alcian blue and Masson's trichrome were used to differentiate the collagen fibres and cartilage matrices. Verhoeff's stain demonstrated elastic fibres.

### 1.10 Clearing

After staining, the sections were dehydrated and cleared with fresh xylol but as this caused shrinkage and made microscopical focusing difficult, other clearing agents were tried and used namely:- ethanolic - xylol (equal parts), carbol - xylol (1 in 3 parts), carbol - xylol (equal parts) and saturated carbol - xylol.

The ethanolic - xylol (equal parts) gave good results, but the saturated carbol - xylol was best. [Made by adding anhydrous crystals of pure phenol to xylol until no more will dissolve (Gatenby and Painter, 1937).]

## 2.0 GENERAL REVIEW OF THE HISTOLOGICAL LITERATURE

### 2.1 Classification of the skeletal cartilages

Skeletal cartilage is classified into three main types: hyaline, elastic and fibrocartilage.

Of these hyaline is the most common. In hyaline cartilage the intercellular substance is a firm gel. Although it appears to be homogeneous both in the gross and in the ordinary microscopic preparations, it contains considerable quantities of both fibrous and amorphous kinds of intercellular substance. The fibrous kind is presented by collagenic microfibrils, fibrils and fibres. The collagen in the intercellular substance is, however, obvious in the electron microscope, where it often appears as fibrillar material (Ham, 1969). It is found in articular cartilages, laryngeal cartilages, cartilages of the trachea and bronchi, cartilages of the nose and in the primordia of most of the bones during development.

In fibrocartilage the matrix is pervaded by collagen fibres. Fibrocartilage often merges gradually into connective tissue or hyaline cartilage. It is found in the menisci of joints, in intervertebral discs, at the margins of articular cartilages, the accessory cartilage of the patella and in some tendons and tendon sheaths.

Elastic-cartilage is characterised by a network of fine or coarse elastic and collagen fibres in the intercellular substance. The network of fibres varies in density in different cartilages. Elastic cartilage is found in the epiglottis, the corniculate portion of the arytenoid, the

cartilage of the third eyelid and part of the cartilage of the Eustachian tube (Schafer, 1946; Trautmann and Fiebiger, 1952; Ham and Leeson, 1965; Bloom and Fawcett, 1968).

## 2.2 Growth of cartilage

According to Maximow and Bloom (1957) and Ham and Leeson (1965) the growth of cartilage takes place in two different ways. The first is interstitially in which the mature cartilage cells divide and give rise to new cartilage cells. These, in turn, multiply to form further cells and cartilage.

The second is by appositional growth in which new layers of cartilage are apposed to one of its surfaces. This depends on the activity of the perichondrium, which is a layer of connective tissue, surrounding all cartilaginous structures except the articular surfaces. Its inner surface usually exhibits a gradual transition from ordinary connective tissue to cartilage, and this process is continuous.

## 2.3 Classification of the cartilage of the hoof

A study of the literature reveals three opinions regarding the classification of the cartilage of the horse's hoof, these are:-

Hughes and Dransfield (1953) and Smythe (1967) state that it is composed of hyaline cartilage. Bradley (1920), Sisson and Grossman (1953), Taylor (1959) and Rooney, Sack and Habel (1969) state that, in young animals the cartilage is hyaline but as age advances it changes to fibrous type. Williams (1872), Lungwitz (1906), Erle (1913), Reeks (1918), Muller (1937), Piers (1949) and Trautmann and Fiebiger (1952)

state that the cartilage of the hoof is fibrocartilage.

Erle (1913), in his thesis on the study of the histological structure of the cartilage of the hoof, examined cartilages of horses ranging in age from 7½ months to 20 years and using different stains including Weigert's haematoxylin and Resorcin - Fuchsin for elastic tissues came to the conclusion that the cartilage plate is surrounded by a dense connective tissue, the perichondrium, which is more dense on the abaxial than on its axial surface. The chondrocytes, which are arranged in the direction of the cartilage fibres, are small and oval in shape but as they approach the centre of the cartilage plate they become larger and more rounded. He found no elastic tissue and concluded that the cartilage of the hoof, in young and old horses, was fibrocartilage. These fibres assume straight and curved courses crossing each other.

#### 2.4 The cartilage canals

Haines (1934) states that the primary function of the cartilage canals is to carry nutriment into the interior of blocks of cartilage too large to be supplied by diffusion through their substance, thereby allowing the cartilage to grow, and preventing its early calcification or death. Hurrell (1934) in his conclusion regarding the function of the vascular canals states that, they amount in effect to inflections of the deeper perichondrial layers. They carry blood vessels and embryonic connective tissue which can supply osteoblasts when required to the interior of cartilage masses. The blood vessels also prevent

the degeneration of the cartilage which would occur if large masses had to depend only on diffusion from their perichondrium.

## 2.5 Ossification fundamentals

The histogenesis of bone, or osteogenesis, is the same wherever it occurs. Two types are described intramembranous and intracartilaginous or endochondral (Schafer, 1946; Weinmann and Sicher, 1947; Ham and Leeson, 1965; Bloom and Fawcett, 1968; Arey, 1968; Cruickshank, Dodds and Gardner, 1968).

### 2.5.1 Intramembranous ossification

This term refers to the bone formed from mesenchymal fibroblast cells which have previously formed collagen fibres. Intramembranous ossification begins when a cluster of these cells differentiate into osteoblasts which so form a centre of ossification. After these osteoblasts appear, it is not very long before in some of them the characteristic organic intercellular substance of bone becomes apparent. Others proliferate to increase the cell numbers in the region. The product of such process is often called membrane bone. It is found in certain flat bones of the skull, for example, the frontal, parietal, occipital and temporal bones and part of the mandible.

### 2.5.2 Endochondral ossification

Endochondral ossification is a more complex process of ossification. Bones at the base of the skull, in the vertebral column, the pelvis and the extremities are called cartilaginous bone because they are first formed of cartilage. The cartilage is then replaced by the



process of ossification. Thus, there is an intermediate stage not encountered in developing membrane bones.

As endochondral ossification spreads toward the ends of the cartilage, the stages are more clearly segregated in a series of transverse zones:-

1. Quiescent (or reserve) zone. Chondrocytes are irregularly and loosely scattered throughout the intercellular substance of the cartilage.

2. Proliferative (or mitotic) zone. A cell divides as do the daughter cells, forming conspicuous rows. These rows are arranged parallel with the long axis of the cartilage, the cells of a row are crowded, flattened and separated by little matrix.

Dodds (1930 and 1932) in considering the types of arrangement of cartilage cells in the endochondral ossification, came to the conclusion that there were three, well recognised, main types of arrangement of chondrocytes. They are as follows:-

- I. The chondrocytes form in longitudinal rows, a state common in diaphyseal ossification.

- II. The chondrocytes occur in nests, as in epiphyseal centres. They form compact groups of nests of four or more cells with no linear arrangement.

- III. The chondrocytes are arranged in a uniform distribution as in the middle of the cartilage model when endochondral ossification is

about to begin, at the time of the first invasion of the marrow tissue from the periosteal region.

3. Maturation zone. Cells lie in continuity with the columns of the previous zone but are much larger, due to the accumulation of phosphatase and glycogen.

4. Calcification zone. Deeply basophilic, calcified matrix is the feature of this relatively narrow zone. The cells at this level have reached the peak in their life cycle.

5. Regressive zone. Here, the cartilage cells are dying or undergoing actual dissolution. The matrix between successive cells is dissolving, thus opening lacunae. These spaces add to the tubular channels already produced in the matrix and the vascular primary marrow extends into the newly opened spaces where the osteoblasts differentiate from the mesenchymal cells of the marrow tissue.

6. Ossification zone. The osteoblasts gather on the exposed plates of calcified cartilage and cloth themselves with the characteristic organic intercellular substance of bone, so they become osteocytes. This addition significantly extends the spongy bone already present.

Weinmann and Sicher (1947), in classifying the development of bone, state that it would be more appropriate to classify bone as (a) those which replace a temporary cartilaginous skeletal part (endochondral ossification); (b) those which are not preceded by a cartilaginous organ, and (c) those without a cartilaginous predecessor where, during their formation and growth, cartilage is differentiated

from connective tissue and then plays an important part in the growth of this type of bone. Cartilage which forms skeletal parts prior to bone development is called primordial or primary cartilage. Cartilage which is differentiated during development and growth of bone should be referred to as secondary cartilage, for example, the clavicle and mandible are membranous bones in which secondary cartilage develops later.

## 2.6 Arrangement of the elements of bone tissue

Mature bone is lamellated, that is, it is laid down in thin layers of ground substance. The shape and arrangement of these lamellae are markedly different in the two types of bone, which can be distinguished macroscopically as spongy or cancellous bone and compact bone (Weinmann and Sicher, 1947).

Spongy bone consists of bars, plates or tubules of bone of varying thickness and length which form a trabecular network through which there are communicating spaces. The single trabecula consists of a few lamellae, generally arranged parallel to each other or in concentric layers. These trabeculae are bathed in tissue fluid which is derived from the capillaries of the spaces between the trabeculae.

In compact bone most lamellae are arranged in a cylindrical system. Each system consists of a varying number of concentric lamellae, grouped around a narrow axial canal which contains blood vessels and a small amount of loose connective tissue. A system of these concentric lamellae is called a Haversian system (Weinmann and

Sicher, 1947). Such systems are present only in compact bone (Arey, 1968).

## 2.7 Calcification and Ossification

Calcification is the process of the deposition of calcium salts in cells or tissues, living or dead, whether or not the structures to be calcified have the characteristics of bony tissue. Ossification is a process of connective-tissue differentiation in which is laid down a strong and compact system of collagen fibres, blended with a non-fibrillar ground substance of mucopolysaccharides and enclosing living cells whose cytoplasmic processes ramify and interconnect with one another, or with the periosteal or endosteal surfaces, through minute canaliculi. This structure is peculiarly favourable to the deposition of calcium salts. Its calcification (provisional) results in bone, but calcified, or after decalcification in vitro, bone tissue is quite distinctive and readily recognised under the microscope (Collins and Curran, 1959).

## 3.0 REVIEW OF LITERATURE CONCERNING SIDE BONE

### 3.1 Sidebone features

There are two opinions expressed namely - side bones are due to calcification or to ossification. Pritchard (1888) described side bone as calcification or ossification of a portion or the whole of the cartilage of the hoof. The following authors are of the opinion that side bone is a calcification of the cartilage - Stewart (1938),

Hungerford (1953), Smythe (1959), Dixon (1963), and Smythe (1967). However, more authors consider that side bone is due to ossification (Johne and Lungwitz, 1889; Gilruth, 1892; Gutenacker, 1901; Witte, 1907; Zimmermann, 1908; Cameron, 1910; Lowe, 1935; Muller, 1937; Hickman, 1964; Douglas and Williamson, 1970).

### 3.2 Pathology of side bone

As regards the pathology of side bone, some consider that it arises from inflammation while others refute this view.

Witte (1907), Zimmermann (1908), Eberlein (1908), Habacher (1921), Volkman (1936), Pires (1949), and Dixon (1963) consider that the primary change is an osteitis arising in the wing of PIII, and that ossification follows as a secondary reaction. Similarly Youatt (1860), Fitzwygram (1863), Axe (1906), Cameron (1907) and Adams (1966) consider that the development of side bone is due to inflammation of the cartilage of the hoof.

Williams (1872), Smith (1890), O'Connor (1942) and Frank (1959) consider that side bone is not a consequence of inflammation. Spooner (1840), John and Lungwitz (1889), Gilruth (1892), Gutenacker (1901) and Muller (1937) agree that side bone development is a normal endochondral ossification.

### 3.3 Origin of side bone

Very little histological investigation has been done in the investigation of this condition. Most writers base their opinion on the macroscopic changes that occur at the palmar process. Those who

made histological investigations were unable to identify the normal boundary between PIII and side bone. Gilruth (1892) states that, in the fetal foot, there is no distinction in structure between the portion that will persist as cartilage and the portion which will be converted into bone. Pires (1949) stated that PIII and the cartilage of the hoof formed a unit in which the histological limit between both tissues was unrecognisable.

With regard to the ossification of the cartilage there are three theories regarding this process.

The first, supported by German authors, is that the ossification begins at the points of attachment of the collateral ligaments of distal interphalangeal joint and the collateral ligaments of the distal sesamoid bone (Johne and Lungwitz, 1889; Gutenacker, 1901; Hugentobler, 1907; Heusser, 1928).

The second theory which is held by a majority of authors, is that the ossification commences at the junction of PIII with the cartilage of the hoof (Spooner, 1840; Williams, 1872; Youatt, 1874; Witte, 1907; Zimmermann, 1908; Cameron, 1910; Holcombe, 1923; Liautard, 1923; O'Connor, 1934; Lowe, 1935; Frank, 1959; Smythe, 1959; Hickman, 1964; Morgan, 1968).

The third theory is that ossification extends from the palmar process of PIII (Pritchard, 1888; Smith, 1890; Gilruth, 1892; Axe, 1906; Muller, 1937; Pires, 1949). The last two authors specify that the ossification extends from the proximal palmar process of PIII.

As stated earlier Muller (1937), examined clinically 37 heavy working horses and 3 mules, he chose a foot from each case for postmortem examination. A histological examination was made of a section of one of the cartilages from each animal and he found that the ossification starts from the proximal palmar process of PIII. Muller (1937) mentions 17 cases, including a case of a mule, of complete ossification of the cartilage of the hoof; 7 cases showed widening of the proximal palmar process of PIII; 6 cases, including 2 cases of mules, showed a slight widening of the proximal palmar process and 12 horses showed no sign of ossification. He did not agree, with his German colleagues, that the origin of side bone was at the areas of insertion of the collateral ligaments of the distal interphalangeal joint and the collateral ligaments of the distal sesamoid bone as he found no evidence to support such statements.

#### 4.0 MICROSCOPIC ANATOMY OF THE CARTILAGE OF THE HOOF

Literature referring to the histological structure of the cartilage deals largely with well developed side bones in horses of over 2 years old. Erle (1913) examined cartilage slices from each border and the middle of the cartilage plate of a 7½ month old foal. With this exception, the normal structure of the cartilage and its replacement by bone do not seem to have been investigated.

The cartilage, which is in direct continuation of the proximal palmar process of PIII (Figs. 4, 49 and 50) forms the basis of the bulb of the heel caudally (Fig. 21). It is completely surrounded by a

dense fibro-elastic and highly vascular perichondrium. The perichondrium send rays of fibres into different parts of the cartilage substance (Fig. 50) and through cartilage canals to meet at the other side of the cartilage surface (Figs. 4, 21 and 51). Its density and quantity is greater on the abaxial than on the axial surface of the cartilage (Fig. 50). The inner layer of the perichondrium is chondrogenic. In examining the serial sections, it was difficult to decide exactly where the inner border of the perichondrium stops and the cartilage begins (Figs. 50 and 51).

The chondrocytes can be grouped into two ill-defined arrangements. One at the inner surface of the perichondrium where the cells are small, flattened and compact, and are disposed with their long axis running parallel with the perichondrium fibres. The other more centrally in the cartilage plate where they are large rounded cells.

The intercellular substance of the cartilage consists of two types of fibres both of which are abundant and intermixed. The fibres are collagenous and elastic. The collagen fibres are disposed in all directions, so forming an irregular texture, in which the chondrocytes are disposed in groups in the direction of the collagenous bundles (Fig. 52). The elastic fibres are similarly arranged to the collagenous fibres throughout the cartilage matrix. These fibres are fine and stain black with Verhoeff's stain. Figs. 53 - 59 are sections from cartilages of different breeds and ages ranging from 9 months fetus to 15 years old horses. All show the presence of the fine elastic fibres, but demonstration of these fibres in calcified cartilages was more difficult to



show due to the deep staining of the differentiated calcified tissue by Verhoeff's stain (Fig. 58).

#### 4.1 Cartilage canals

Like other cartilage matrices in the body, the cartilage of the hoof is non-vascular and contains no nerves; but it is perforated by a number of canals, which exist in the fetal stages and persist in later life. The number and location of these canals differs in different feet of the same animal, and in the two cartilages of the same foot. Progressively these canals increase in number towards the solar border of the cartilage. The canals are particularly numerous at the point where the fibro-elastic cartilage blends with the hyaline cartilage of the proximal palmar process of PIII (Figs. 49 and 60A - D).

These canals contain mesenchymal cells and vessels (vein, artery and lymphatics) which lie in a matrix of loose connective tissue. The cartilage ground substance and the cartilage cells stain deeply with eosin round the region of the canals. Figs. 21 and 49 show the connection of these canals with the perichondrium of the cartilage of the hoof. The structure of the contents and walls of the canals is similar to that of the perichondrium and the cartilage underlying it (Figs. 51 and 61).

5.0 REVIEW OF LITERATURE CONCERNING THE HISTOPATHOLOGY OF  
THE CARTILAGE OF THE HOOF

5.1 Ossification Starting from the Proximal Palmar Process of PIII

Johne and Lungwitz (1889) were the first to describe histologically the ossification in the cartilage of the hoof. They state that the cartilage cells in the vicinity of the bone proliferate and later their capsules start to shrink, indentate and slowly turn into bone cells. In their conclusion they consider that this process corresponds to normal endochondral ossification.

Gilruth (1892) examined, histologically, advanced side bone in 5 horses and found that the type of bone is of a normal character and is similar in quality and structure to that of the palmar process of PIII.

Gutenacker (1901) agrees with the histological findings of Johne and Lungwitz (1889).

Witte (1907) who examined histologically a great number of cartilages undergoing early ossification also agreed with Johne and Lungwitz (1889) in distinguishing between a bony region and a proliferating cartilage cell zone. He stated that in the bony zone there is dilatation and widening of the Haversian canals, which is due to the activity of the osteoclasts and thus he considered these changes as characteristic of rarified osteitis or inflammatory osteoporosis.

Zimmermann (1908) agreed with Witte (1907) that there was dilatation of the Haversian canals at the palmar process of PIII and the cartilage cells near this bony zone showed degenerated capsules

and nuclei and that in this zone the ground substance of the cartilage shows an undulating regimentation. He believed therefore that the beginning of the complaint is a rarified osteitis of the palmar process of PIII.

Muller (1937) describes the normal transition from the proximal palmar process of PIII to the cartilage of the hoof in a 15 years old light carriage horse (which showed no macroscopical evidence of side bone). He described three different zones:- (a) A zone of fibrocartilage. In this zone there are irregularly distributed typical cartilage cells, embedded in a matrix which is rich in fibrous connective tissues. (b) A zone of proliferation in which the chondrocytes are much larger and more closely packed than in the previous zone. In some cells the nuclei disappeared, while in others signs of mitotic division ceased. Still other cells were seen indicating that multiplication was active, these were invaded by blood vessels and abundant of giant cells were evident in the nearby bony zone. These giant cells were only found in the newly formed bone with abundant mesenchyme and blood vessels. (c) Bony zone. This is a spongy bone in which the trabeculae were extremely thin and Haversian canals were absent. The marrow cavities were mainly filled with fatty tissue, the bone lamellae next to the cartilage were associated with undifferentiated mesenchyme, blood vessels and abundant multinucleated giant cells which were adjacent to the wall of the cavities. Muller (1937) went on to describe cases of different stages of side bone, which show the same features mentioned above. He concluded that this process is almost a normal endochondral

ossification with one peculiarity namely, the absence of columns of cartilage cells (or row arrangement). He concluded that the older ossified parts of the cartilage showed features of compact bone, and the process of ossification may extend to involve the whole cartilage or it may stop at any stage when the undifferentiated mesenchymal cells of the bone lamellae next to the cartilage loses its ability to form osteoblasts by differentiating into a fatty tissue.

## 5.2 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate Other than from the Proximal Palmar Process of PIII

Muller (1937) describing the bony islands as areas developing from the cartilage canals, stated that the blood capillaries and mesenchymal cells invade the cartilage substance to form bony islands. He commented that the osteogenesis of these islands is the same as that originating from the palmar process of PIII, that is, both go through the same stages of normal endochondral ossification but again no column or row arrangement of the chondrocytes is evident.

## 6.0 HISTO-PATHOLOGY OF THE CARTILAGE OF THE HOOF

The following changes have been observed as a result of the present investigation:-

Ossification starting from the proximal palmar Process of PIII.

Ossification centres arising in various parts of the cartilage plate other than from the proximal palmar process of PIII.

Secondary cartilage growth, resulting in the formation of ossification centres at the level of the distal palmar process of PIII.

Calcification "non provisional" and calcified necrotic foci of the cartilage of the hoof.

The first two changes have been described by many authors, but none mentioned the latter two changes.

#### 6.1 Ossification Starting from the Proximal Palmar Process of PIII (Side Bone)

In this research the histological investigation was directed to study of the early development of side bone and its osteogenesis, and to confirm these changes by radiological examination. Serial sections of cases of different fetal ages and newly born, young horses and horses up to 23 years old were studied (Table 1).

The pattern of change which was established is illustrated by describing a few selected cases to show the features that characterise the ossification that generally occurs.

Only in foals of 6 months and older have traces of ossification been found. Fig. 62 is a transverse section, of a 6 months old thoroughbred foal, taken from the upper most part of the proximal palmar process of PIII. It shows the bony invasion of this process into the fibro-elastic cartilage at the right side, while the proximal hyaline articular part of PIII, left side, is still not yet fully ossified. Both sides show the same ossification features, the only difference is that the proliferating chondrocytes of the cartilage of the hoof are of no special orientation, their arrangement corresponding with the different direction of the cartilage fibres; Fig. 63 shows this arrangement which is taken

from a seven months Exmoor foal; Fig. 64 is a transverse section taken from a 6 months old thoroughbred X connemara foal, from the lowest part of the proximal palmar process of PIII where it meets the cartilage of the hoof. This again shows the same features of the invasion into the cartilage of the hoof. Fig. 65 shows a field of the same section in which the osteoblasts lining the newly formed bone cavities as well as the osteoclasts in the Howship's lacunae. In studying other sections of these cases it was found that the direction of the ossification is caudally, that is from the proximal palmar process of PIII into the cartilage of the hoof, the bony invasion into the fibro-elastic cartilage is not regular throughout. This fact was confirmed radiologically (Fig. 66), and as the ossification in these cases was just beginning, it was difficult to decide to what other direction it might extend, but in studying older age groups, a well defined bony invasion was revealed. Figs. 67A - D are transverse sections of an 8 months old Exmoor pony, they illustrate parts of PIII and the cartilage of the hoof, and they reveal that the direction of this ossification process is backward and upward, which corresponds to the direction in all other cases in older groups. Fig. 68 which corresponds to the whole dense field of Fig. 67A in which the chondrocytes show the active stage of mitotic proliferation with at the lower end a zone of resting chondrocytes. The proliferating chondrocytes have no definite arrangement other than that determined by the direction of the cartilage fibres. This feature is the same in all other cases. Fig. 69 which corresponds to Fig. 67B shows the whole field of the bony invasion with other features of the

ossification which can be studied more closely in Fig. 70 which is part of the previous field. In this illustration one can see that most of the mature chondrocytes with their calcified matrix are undergoing dissolution, thus opening up lacunae which contain mesenchymal cells and blood capillaries travelling from the bone marrow of the proximal palmar process of PIII.

The developing ossification process is illustrated by Fig. 71 which shows part of the field shown in Fig. 67D. Fig. 71 shows the following features changing from cartilage to bone:-

- (a) Quiescent (or resting zone). The chondrocytes show slight, slow growth and are irregularly scattered through the cartilage matrix.
- (b) Proliferative zone. Adjacent to the resting zone is an active, mitotic zone of chondrocytes. In this zone the chondrocytes are irregular or of no definite arrangement, the chondrocytes of this zone count no more than 2, 4 and 8 arising from one mother cell.
- (c) Zone of maturing cartilage cells. The chondrocytes are relatively larger and stain deeply blue with haematoxylin.
- (d) Zone of provisional calcification. This is a narrow zone of calcification in which the cartilage cells and the intercellular matrix shows deep basophilic reaction.
- (e) Regressive zone. The chondrocytes in this area show degenerative signs. The deepest areas exhibit erosion of capsular matrix related to vascular invasion by capillaries accompanied by mesenchymal cells, which differentiate into osteoblasts, derived from the marrow cavity of the bone adjacent to them.

(f) Ossification zone. The exposed calcified cartilage plates are clothed with the osteoblasts, accompanied by osteoclasts. These giant cells are only found in areas where there is active bone formation or newly formed bone which is spongy in nature. Its trabeculae are thin and contain calcified cartilage cores which escape destruction during the rapid reconstruction process from cartilage to bone. The spaces between these trabeculae are filled with mesenchymal cells, blood vessels, osteoblasts differentiated from the mesenchymal cells and osteoclasts which are often found in the more or less shallow grooves called Howship's lacunae (Figs. 62 - 65 and 69 - 74).

Growth of this bone continues and the trabeculae anastomose with one another so that they enclose spaces (Figs. 75 and 76). Each space contains fatty cells and blood vessel. This type of bone, which is called tubular spongiosa (Weinmann and Sicher, 1947) was found in all cases examined with advanced side bone. In some cases of long standing side bone, to this tubular spongiosa several layers of new bone were added to the trabeculae. Consequently the spaces between the trabeculae were diminished in size so that the spongy bone became more compact as shown in Fig. 76.

It has been noted that, in areas where the bone marrow cavities still contain mesenchymal cells, there is always new bony invasion into the neighbouring cartilage (Fig. 74). No such invasion can be traced in cases where the marrow cavities are filled with fatty cells (Fig. 75).

In some areas where the ossification process is complete or in



other words there is no new bony invasion, the cartilage fibres enter the nearby bone surface at right angles to these surfaces, similar to Sharpey's fibres. (Fig. 77).

Fig. 78 is a vertical section of an 8 months old thoroughbred foal showing the extent of bony penetration into the cartilage of the hoof.

Fig. 74 shows a vertical section at the points where new bony invasion is taking place into the cartilage of the hoof of a 3 years old thoroughbred horse. It shows the same features as those described previously.

## 6.2 Ossification Centres (Bony Islands) that arise in Various Parts of the Cartilage Plate Other than from the Proximal Palmar Process of PIII

Bony islands in 8 cartilages of 6 horses, (1 thoroughbred, 1 Exmoor pony, 1 fell pony and 3 heavy horses), were examined histologically, and all were found to show the same osteogenic features as those illustrated by Figs. 67-74.

Fig. 79 which is a vertical section of a bony island in a 5 years old thoroughbred horse, shows a resting zone, a proliferative zone and a maturing zone where the chondrocytes are deeply stained. In the last zone the intercellular matrix shows a narrow zone of calcification. In the same figure, the mature chondrocytes with their calcified matrix are undergoing degenerative changes, that is, erosion of the capsular matrix and opening of the lacunae due to vascular invasion. These vascular capillaries are accompanied by mesenchymal cells which

differentiate into osteoblasts, derived from the cartilage canals at different locations. Fig. 80 shows the exposed calcified cartilage plates clothed by osteoblasts and osteoclasts. These giant cells Fig. 81 are found in areas where there is new bone formation. The type of bone formed by these islands is spongy in nature, the trabeculae are thin and contain calcified cartilage cores which escape destruction during the rapid reconstruction process from cartilage to bone. The spaces between the trabeculae are filled with mesenchymal cells, blood capillaries, osteoblasts and osteoclasts.

Growth of this bone continues and the trabeculae anastomose with one another forming a tubular spongiosa (Figs. 82 and 83), in which several layers of new bone are added so that the spaces are diminished and the spongy bone is converted into compact bone (Fig. 83). In areas where the bone marrow cavities still contain mesenchymal cells there is always new invasion of the neighbouring cartilage (Fig. 84). Fig. 85 shows the bony island at the right side of the figure and side bone at the left side. The bone at both sites shows the same osteogenic features, which appeared as fractured side bone in the X-ray film (Fig. 86), no such invasion can be traced in areas where the marrow cavities are filled with fatty cells (Fig. 87).

#### 6.2.1 Location and Origin of the Bony Islands Table 5

As stated earlier (section 2.3.1) the radiological findings show that these bony islands are scattered randomly in the cartilage of the hoof at no fixed site. Histological serial sections revealed that these

Islands always arise in areas, whether in the middle or at the periphery of the cartilage plate, where there is a cartilage canal. Figs. 87 and 88 show the cartilage canals in the middle of the cartilage plate where the bony islands are formed. Figs. 89 and 90 show invasion of the cartilage by mesenchymal cells and blood capillaries, at the perichondrium, from a cartilage canal.

### 6.3 Secondary Cartilage Growth, Resulting in the Formation of Ossification Centres (Bony Islands) at the Level of the Distal Palmar Process of PIII

To understand the formation of these bony islands, it is necessary to understand the relevant osteogenic behaviour of the distal palmar process of PIII.

The distal palmar process of PIII develops as a membranous bone distal to, and some distance from, the cartilage of the hoof, it grows along the previously defined surface of membranous connective tissue (in the pre and postnatal stages) (Figs. 91 and 92). As explained earlier, the development and growth of membranous bone is simpler than endochondral bone. The distal palmar process is not preformed in cartilage but develops from a condensation of the embryonic mesenchyme. However this becomes complicated by differentiation of cartilage which is not part of the primordial cartilagenous skeleton, but develops secondarily. This cartilaginous cap plays an important part in the growth of the distal palmar process, which develops by simultaneous growth of the cartilage and its replacement by bone. Fig. 93 is a vertical section of a 7 years old highland pony showing the constitution of the distal palmar process cap, and it shows the differentiation of

the fibroblasts into cartilage cells. This development is rapid in the fetal and young stages (Figs. 91 and 92), but as the animal grows up the process becomes slower and less active (Figs. 94 and 95). In areas where the bone marrow cavities still contain mesenchymal cells there is always new cartilage cell formation and bone invasion, while in areas where the marrow cavities are filled with fatty cells no such invasion was found (Fig. 96). This figure also shows the tubular spongiosa with its tendency to develop compact bony characters. The osteogenesis is like that in the endochondral ossification.

Five cartilages, with their attachments, in 5 horses, (7 years old highland pony, 15 years old thoroughbred, 16 years old Exmoor pony, 16 years old hunter and 17 years old Shetland pony), were examined histologically and it was found that all of them had the same osteogenic features as those of the distal palmar process of PIII. Mainly the fibroblasts differentiate into chondroblasts and then into chondrocytes. Figs. 42, 97 and 98 are transverse sections in the NF M distal palmar process of a 15 year old thoroughbred horse which shows the location of the bony island within the connective tissue. The bony island is quite clear from the distal palmar process and the cartilage of the hoof. The illustrations also show the differentiation of the secondary cartilage cap which gives the same picture as that of the distal palmar process of PIII (Fig. 47). Fig. 99 is a transverse section of a 17 years old Shetland pony showing the distal plantar process at the right side which is represented by Fig. 95 and the bony island formation on the left side which is represented by Fig. 100, both sides show the same histological features.

In areas where there is new bone formation there are always bone forming cells and osteoclasts while in old bone the marrow cavities are filled with fatty cells (Fig. 101). The process of development is similar to that of the distal plantar process. The type of bone formed by these islands is again tubular spongiosa (Fig. 101) with new layers of bone being added during the growth process, the marrow spaces become diminished and converted into a compact bone (Fig. 102).

#### 7.0 REVIEW OF LITERATURE CONCERNING CALCIFICATION "NON PROVISIONAL" AND CALCIFIED NECROTIC FOCI OF CARTILAGE

During the present study the writer could not find any publication which described or mentioned the "non provisional calcification" or "the calcified necrotic foci" of the cartilage of the hoof. Pathologists and histologists mention calcification of the cartilages of the larynx, ear, bronchi and nasal septum associated with old age in man.

The deposition of calcium, in situations other than developing or regenerating bone, is explained by two main theories. The first is that it is a senile process, the second that it is pathological.

Harris (1933) states that, in old age, calcification may be seen in all tissues that have a relatively poor blood supply and relatively show rate of exchange of tissue fluid. Arey (1968) considers that in old age the calcification of some cartilages is a characteristic phenomenon, the matrix becoming opaque, hard and brittle through lime deposition.

The following authors support that calcification is a pathological

process. Florey (1962) states that calcium salts are only precipitated in the normal body in the formation of bone and all deposits in soft tissues are pathological. Muir (1964) remarked that, in senile calcification there occurs a deposit of lime salts, first of all in fibres or matrix between the cells. It differs from ossification in that the deposits occur irregularly and without any definite relation to the organic matrix. He considers that the process is closely related to that seen in pathological conditions. Montgomery (1965) states that the deposition of calcium in situations other than developing or regenerating bone is a relatively common occurrence known generally as pathological calcification.

#### 7.1 Aseptic calcified necrotic foci

Necrosis, is generally defined as death of a cell or groups of cells, when they still form part of the living body (Gaiger and Davies, 1960; Muir, 1964). Haines (1934) and Hurrell (1934) in studying the cartilage canals, consider that, these help in supplying nutrient to large blocks of cartilage, thereby allowing the cartilage to grow and preventing early calcification or death. Arey (1968) states that the deposition of calcium in cartilage matrix interferes with the diffusion of nutrients with the consequent death and dissolution of cartilage cells.

#### 7.2 Detection of the calcified cartilage

Muller (1858) first detected calcified cartilage areas by using material decalcified with chromic acid and stained with haematoxylin.

He reported that the calcified areas in the cartilage matrix were as a rule still recognisable by their staining reaction and he showed that the calcium salt was deposited in the matrix, not in the cartilage cells. Cameron (1930) found that, with complete decalcification, it was possible using haematoxylin to distinguish calcified from non-calcified areas. He came to the conclusion that although haematoxylin does not stain calcium salts it often identifies the areas in which changes favourable to the deposition of calcium salts are taking place. Bloom and Bloom (1940) agree with these statements and they added that, the location of calcified areas in decalcified material, can be detected by its reaction with haematoxylin in giving a deep purple colour.

In the present study Ehrlich's haematoxylin was used to determine the presence of calcium salts but other methods and agents also gave satisfactory results. Using Van Gieson stain, the calcified areas reacted with the picric acid and gave yellowish-green colour (Fig. 103). After using Masson's trichrome (light green) technique the non-calcified fibro-elastic fibres were prominent and they ran in different directions; the fibres were later hidden by calcium deposited in the matrix so that a homogeneous appearance was obtained (Fig. 104).

## 8.0 CALCIFICATION "NON PROVISIONAL" AND CALCIFIED NECROTIC FOCI OF THE CARTILAGE OF THE HOOF

### 8.1 Macroscopic Examination of the Calcified Cartilages

By simple digital manipulation of the cartilages, it was difficult to decide whether or not the cartilages were calcified but cartilages

with calcium deposition were usually hard to cut and the cut surfaces were gritty to touch.

## 8.2 Microscopic Examination

As stated earlier serial sections cut from blocks taken from 63 cartilages of the 50 horses shown in Table 1 were studied. The number of blocks selected was decided from the macroscopic appearance and the radiological findings, with the object of discovering the nature of side bone, particularly the nature and distribution of the islands and the relationships of the proximal and the distal palmar process of PIII.

Every tenth section of the serial sections made and approximately 100 sections of each block were mounted, stained and examined. By such examination, parts of the cartilages other than those concerned with the radiological findings were therefore examined.

This study revealed areas of different degrees of non provisional calcified cartilage which might otherwise have been missed (Table 1).

The staining technique used for determining the deposition of calcium salt was the haematoxylin reaction described by Muller (1858), Cameron (1930) and Bloom and Bloom (1940), in which a deep purple colour reaction indicated calcium deposition. It was noticed that the quality of the purple colour reaction seemed to change with the quantity of the calcium deposition in the calcified matrix. The colour changes ranged from faint purple in cases of mild calcification, to purple in cases of heavy deposition and to very deep purple in cases which showed areas of very heavy calcification. It was not unusual to meet two or three colour reactions in the same section (Figs. 105 and 106).



The calcium salt deposits invade the fibro-elastic matrix of the cartilage (Fig. 104), the chondrocytes look as though imprisoned by an eosinophilic halo like capsule which contrasts them from the matrix. The dimensions of this capsule vary with the degree of the calcium salt deposition. It is either enclosed with the open face continuing with the uncalcified matrix, or closed (completely separated from the non calcified matrix) with the isolated chondrocytes surrounded by the eosinophilic halo like capsule, or it may be closed with the isolated chondrocytes surrounded by very narrow or non eosinophilic halo like capsule. Conforming with the colour reactions it is not uncommon to meet two or three forms of calcification in the same section (Figs. 105, 106 and 107). Cases of the third form of calcification, which are with non eosinophilic halo like capsule, show the necrotic foci in which the chondrocytes have no nuclei and the cellular capsules are broken down leaving empty, dead, calcified cavities (Fig. 108). In other places there are heavier calcified necrotic areas where the calcified cartilage matrix with their dead chondrocytes tear off leaving an empty space (Fig. 107).

Although these cartilages changes were detected in all the breeds of the animals examined, from 3 years and older, because there were not sufficient numbers of animals in the different breed and age groups, no conclusions have been made regarding the age and breed incidence.

## 9.0 DISCUSSION AND CONCLUSION

A review of the literature failed to reveal any original paper giving significant details of the histological structure of the cartilage of the hoof.

Erle (1913) studied the nature of the cartilage fibres and its cells but not the cartilage canals.

Bradley (1920), Hughes and Dransfield (1953), Sisson and Grossman (1953), Taylor (1959), Smythe (1967) and Rooney, Sack and Habel (1969) found that the cartilage in young horses was hyaline, but with the exception of Hughes and Dransfield (1953) and Smythe (1967) they agree that as age advances it changes to fibrocartilage. Lungwitz (1906), Erle (1913) and Trautman and Fiebiger (1957), Williams (1891), Reeks (1918), Muller (1937) and Pires (1949) also support that it is fibrocartilage.

In the present investigation it is established that from the fetus to old age the cartilages of the hoof are fibro-elastic.

Gilruth (1892) and Pires (1949) who studied the histology of PIII and its relationship to the cartilage found no sharp histological distinction between the cartilage and PIII, either in the fetal or the postnatal stages.

The present investigation reveals a distinct histological difference between the two structures namely in the fetus and young foal the proximal palmar process of PIII is hyaline cartilage while the

cartilage of the hoof is fibro-elastic. Although both tissues blend with each other the cartilage of the hoof is distinguished by the presence of the collagenous and elastic fibres.

The process by which side bone is formed has been reported by a large number of authors. There are those who agree with Johnes and Lungwitz (1889) that side bone is ossification of the cartilage, those like Smythe (1967) who consider that side bone is calcification and Pritchard (1888) who supports both opinions.

The opinion that the primary cause of side bone is an osteitis; that it is caused by inflammation; that it is not a consequence of inflammation; and that it is a normal process of ossification have all been supported.

The present histological investigation established that the ossification process invades the cartilage at an early stage in the animal's life. Traces of such invasion were detected in cartilages of 6 months old foals, and foals of 8 months show well defined invasion. This invasion progresses from the proximal palmar process of PIII extending into the fibro-elastic cartilage in a backward and upward direction. This process of ossification was found in all the cartilages examined from horses of 6 months old and over. The histological features are similar to that of normal bone development in cartilage (endochondral ossification) showing all its feature except that the chondrocytes are arranged in a uniform distribution. This arrangement conforms with that described by Dodds (1930 and 1932). These chondrocytes follow the direction of the collagen fibres. The end result of

this ossification is a tubular spongiosa which becomes compact bone in old long-standing cases. During the development of the tubular spongiosa to compact bone, the spaces of the spongiosa diminish in size till the wide canal like spaces change to form the Haversian system characteristic of the compact bone. This process may halt at any stage when the mesenchymal cells differentiate to fatty cells.

This process of side bone formation largely confirms the statements of Johne and Lungwitz (1889), Gilruth (1892), Gutenacker (1901), Witte (1907), Zimmermann (1908), Muller (1937) and others. It does not agree with the statements of Stewart (1938), Hungerford (1953), Smythe (1959), Dixon (1963) and Smythe (1967), namely that the side bone is calcification of the cartilage of the hoof.

The opinions expressed by Fitzwygram (1863), Youatt (1874), Axe (1906), Cameron (1907), Witte (1907), Zimmermann (1908), Volkman (1936), Dixon (1963), Adams (1966) and others that side bone formation results from osteitis or as a result of an inflammatory process of the cartilage of the hoof is not supported. The present evidence supports the opinions of Williams (1872), Smith (1890), O'Connor (1942) and Frank (1959) that side bone is not a consequence of inflammation, also the opinion of Spooner (1840), Johne and Lungwitz (1889), Gilruth (1892), Gutenacker (1901) and Muller (1937) who consider that side bone formation is a normal process of ossification.

With regard to the point from which side bone takes origin the three opinions that have been expressed are:-

That the ossification commences at the points of attachment of the collateral ligaments of the distal interphalangeal joint and the collateral ligaments of the distal sesamoid bone (Johne and Lungwitz, 1889; Gutenacker, 1901; Hugentobler, 1907; Heusser, 1928). That ossification of the cartilage commences at its attachment to PIII (Spooner, 1840; Youatt, 1874; Witte, 1907; Zimmermann, 1908; Cameron, 1910; Liautard, 1923; Holcombe, 1923; O'Connor, 1934; Lowe, 1935; Frank, 1959; Smythe, 1959; Hickman, 1964; Morgan, 1968). That the ossification extends from the palmar process of PIII (Pritchard, 1888; Smith, 1890; Gilruth, 1892; Axe, 1906; Muller, 1937; Pires, 1949). The last two authors found that the ossification extends from the proximal palmar process of PIII into the cartilage of the hoof.

The present research investigation revealed that all cartilages examined of horses over 6 months (with the exception of the fibres which cover the parietal groove), showed no trace of ossification of any ligament of the foot, but showed side bone formation as a continuance of the ossification process of the proximal palmar process.

The present findings therefore accord with those of Muller (1937) and Pires (1949).

This investigation revealed that the ossification to form bony islands began at points where the osteogenic cells are found, that is, in the cartilage canals. From these canals, capillaries and undifferentiated mesenchymal cells invade the cartilage. Osteoblasts develop from

the mesenchymal cells and as the invaded tissue becomes calcified, the dead cartilage is replaced by bone. This process may halt at any stage when the mesenchymal cells differentiate into fatty cells.

The osteogenesis and type of bone formed in side bone development or the bony islands is the same, except that in side bone the osteogenic cells originate from the ossified proximal palmar process of PIII, while in the bony islands their origin is from the cartilage canals, which confirms the histological findings of Muller (1937).

In the short band of ligamentous fibres which passes between the cartilage and the distal palmar process well defined bony islands were found (Table 6).

Particular attention was given to the development and growth of the distal palmar process, during its pre and postnatal periods. And it was found that, while this part of PIII develops as a membranous bone in the prenatal period, it continues to grow slowly by a process, called by Weinmann and Sicher (1947) as secondary cartilage development. In its process of ossification the fibroblasts differentiate into cartilage cells which in turn undergo the usual stages of endochondral ossification. The bony islands all of which are formed from the same type of connective tissue, pass through similar stages of ossification. In some cases, the ossification process continues forward to meet and unite with the distal palmar process. In such circumstances this ossification process and union with the distal palmar process has shown no evidence of surrounding inflammatory reaction. In other words there

is as yet no evidence to support that an inflammatory reaction occurs at this site and therefore no support for the use of the term pedal osteitis in this connection.

The type of bone formed in both sites is tubular spongiosa which in time becomes compact bone. There are always bone forming cells in areas where new bone is being formed while in other places fatty cells are a characteristic feature indicating that ossification has stopped.

The character of the bony islands that are formed in relation to the distal palmar process, conforms with the islands that form at other sites in the cartilage.

In man "non provisional" calcification in the cartilages of the larynx, ear, bronchi and nasal septum is recognised (Harris, 1933; Florey, 1962; Muir, 1964; Montgomery, 1965; Arey, 1968). In veterinary literature Sisson and Grossman (1953) have mentioned the calcification of the thyroid and cricoid cartilages of the horse, Pierard (1968) has described the calcification of the laryngeal cartilages in the dog and Fraser, Withers and Spreull (1961) have mentioned that necrosis occurs in the conchal cartilage of the ear of the dog.

In the cartilages of the horse's hoof, although bony islands have been described by other authors calcification and necrosis have not been previously described.

In this investigation detection of decalcified areas where the calcium salt deposition took place were clearly established by using the





CHEMICAL COMPOSITION (ASH, CALCIUM, PHOSPHORUS AND MAGNESIUM)OF THEPROXIMAL PART OF PIII AND THE ADJACENT SIDE BONE

The chemical composition of bone varies with dietary alterations (Sobel, Rockenmacher and Kramer, 1945), with physiological changes and from pathological causes (Ushijima, 1958; Armstrong and Singer, 1965).

The ossified part of the fibro-elastic cartilage of the hoof and the adjacent part of PIII were both analysed and the values compared.

#### 1.0 MATERIALS AND METHODS

Source of materials used for the present study were from horses from various districts of Edinburgh, presented to the Department of Pathology, Royal (Dick) School of Veterinary Studies (Field Station), for post-mortem investigation.

From each of the 16 horses listed in Table 7, two bone specimens were taken and analysed chemically (B1 = side bone and B2 = the part of PIII adjacent to B1). However, in cases not showing advanced side bone, it was difficult to decide whether B1 was entirely side bone or if it included a part of PIII. Furthermore it was impractical to ensure that B1 was entirely bone and not part bone and part fibro-elastic cartilage.

The specimens were dried at 110°C for at least 12 hours, extracted with petroleum ether 60 - 80° for 2 - 3 days, powdered and weighed. The

powder was ashed in a muffle furnace at 500°C for 18 hours, cooled in a dessicator, again weighed and the percentage ash of the dried fat free bone was determined.

The ash was digested by adding 50% HCl acid and heating on a hot water bath for 20 minutes. The digest was cooled and transferred to a 100 ml. volumetric flask, diluted to the appropriate mark and well mixed. (Solution A.)

This solution was the material used for the estimation of total calcium, magnesium and phosphorus. Three samples were taken from each solution for each estimation.

Calcium and magnesium were determined by SP.90 Atomic Absorption Spectrophotometer - Unicam Instruments Ltd., Cambridge, England.

Phosphorus was determined by Vitration Precision Colourimeter - Vitration Dieren, Holland, distributed by Fisons Scientific Apparatus Ltd., London.

#### METHODS OF PREPARING THE SOLUTIONS FOR:

##### The $\text{LaCl}_3$ (6500 p.p.m.) solution

To prepare the stock solution (65.000 p.p.m.), 87.0 gms. of  $\text{LaCl}_3 \cdot 7\text{H}_2\text{O}$  was dissolved in water, 100 ml. of  $\text{N.HNO}_3$  was added and the solution was made up to 500 mls. with distilled water. To get diluent solution of 6500 p.p.m., a 100 mls. of the stock were diluted to 1 litre with distilled water.

Calcium stock solution (1mgm./ ml.)

0.250 gms. of  $\text{CaCO}_3$  (Specpure) was dissolved in a few mls. of nitric acid and the solution was made up to 100 mls. with distilled water.

Magnesium stock solution (1 gm./ ml.)

1.014 gms. of  $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$  (Analar) was dissolved in water and made up to 100 mls. with distilled water.

Mixed prediluted Ca/Mg standards

1.	(4 + 1)	4 ml. Ca stock) 1 ml. Mg stock)	Made up to 100 ml. with distilled water
2.	(8 + 2)	8 ml. Ca stock) 2 ml. Mg stock)	Made up to 100 ml. with distilled water
3.	(12 + 3)	12 ml. Ca stock) 3 ml. Mg stock)	Made up to 100 ml. with distilled water
4.	(16 + 4)	16 ml. Ca stock) 4 ml. Mg stock)	Made up to 100 ml. with distilled water

From each of these standards, (1, 2, 3 and 4), 40 ml. was pipetted into a separate volumetric flask and made up to 1 litre with  $\text{LaCl}_3$  (6500 p.p.m.) solution. Therefore, these standards represent the following concentrations:-

1.	0.4 p.p.m. Mg	3.	1.2 p.p.m. Mg.
	1.6 p.p.m. Ca		4.8 p.p.m. Ca.
2.	0.8 p.p.m. Mg	4.	1.6 p.p.m. Mg.
	3.2 p.p.m. Ca		6.4 p.p.m. Ca.

Standard phosphate

0.880 gm. of potassium dihydrogen phosphate was dissolved in water.

This was transferred quantitatively to a 1 litre flask, 10 ml. of 10 N  $H_2SO_4$  was added and diluted to the mark with distilled water and mixed. This is 0.2 mg. P./ml.

#### Working phosphorus standard solution

Before use, the standard phosphate solution was diluted 10:100 with distilled water, therefore, the standards were prepared with a solution containing 0.02 mg. P/ml. Using a grade "A" pipette, the following quantities, 0.5 ml., 1 ml., 1.5 ml., and 2 ml. were pipetted from the working solution to represent the four standards used which are of the following strength:-

10  $\mu$  gms., 20  $\mu$  gms., 30  $\mu$  gms. and 40  $\mu$  gms.

#### 1.1 Estimation of Magnesium

0.2 ml. of solution A was pipetted in 10 ml. tube and 3.8 ml. of  $LaCl_3$  (6500 p.p.m.) solution was added and mixed by shaking. This is 1:20 dilution of solution A.

After saving 0.2 ml. of this dilution (for calcium estimation) the four mixed Ca/Mg standards and the remaining diluent were atomised in the Unicam SP.90 set at a wavelength of 285.2  $\mu$ m. and a recorder trace was obtained.

#### 1.2 Estimation of Calcium

To the retained diluent 3.8 ml. of  $LaCl_3$  (6500 p.p.m.) solution was added. This is 1:400 dilution of solution A.

The four mixed Ca/Mg standards and the diluent were atomised in

the Unicam SP.90 set at a wavelength of 422.7  $\mu\text{m}$  and a recorder trace was obtained.

### 1.3 Estimation of Phosphorus

0.1 ml. of solution A was pipetted in graduated tube and 4.9 ml. of distilled water was added. This is 1:50 dilution of solution A. To 1 ml. of this dilution 5 ml. of molybdate working solution, (25 gms. of ammonium molybdate in about 200 ml. distilled water with 300 ml. 10 N  $\text{H}_2\text{SO}_4$  added, and made up to 1 litre with distilled water), was added and this was followed by the further addition of 2 ml. of amino naphthol sulphonic acid working solution, (292.5 gms. of sodium metabisulphite dissolved in approximately 1600 ml. distilled water to which 5 gms. of 1 amino 2 naphthol 4 sulphonic acid was added, shaken until dissolved, then 10 gms. of sodium sulphite was added and left in the dark for 18 hours when the mixture was made up to 2 litres with distilled water, shaken and filtered into a brown bottle).

The final mixture was made up to 10 ml. with distilled water, mixed and allowed to stand for 15 minutes, for the colour to be fully developed.

The standards and the sample were compared in the colourimeter set at a wavelength of 682  $\mu\text{m}$ .

## 2.0 REVIEW OF LITERATURE

Review of literature revealed that in nutritional and physiological studies of bone composition, the common practice is to take a single bone from an experimental animal as representative of the skeleton as a whole (Bogert and Hastings, 1931; Booher and Hansmann, 1931; Hess, Berliner and Weinstock, 1931; Armstrong and Singer, 1965).

In spite of the individual difference in the chemical composition of the bones in the same skeleton (Hammett, 1925; Taylor and Moore, 1956; Taylor, Moore and Hertelendy, 1960). Strobino and Farr (1948), Pullar (1961) and Woodward (1962) are agreed that regional variations in ash content within a single bone related to the differences in age of the different regions.

Strobino and Farr (1948) made an extensive study to determine some of the causes of variations noted in the nitrogen and ash content of bone specimens from humans of various ages who had no disease of the skeletal system. In bovine bone samples they found that the older region of the bone showed a higher ash content. They stated that for the scapula, the ash content is lowest near its cartilage. It increases as one progresses from this point towards the glenoid cavity (63.4% - 70.1%). In the ribs and long bones they found that the ash content decreases progressively toward the ends. They stated that in the ribs, the region of maximum ash content was found at about the angle of the rib which corresponded with the first region of fetal ossification.

Significant alterations have been attributed to age and sex in

different animals (Hess et. al., 1931; Strobino and Farr, 1948; Taylor et. al., 1960; Pullar, 1961; Woodward, 1962; Jowsey, 1968). Booher and Hansmann (1931) who analysed the tibias of 8 new born human infants stated that the percentage of ash and of calcium in the dry fat free bone of male and female were virtually identical, Blitz and Pellegrino (1969) who carried out extensive studies on composition of adult human bone found negligible sex differences in specimens from patients of the same age.

References on the chemical composition of the bone of horses are few, however Morgulis (1922), Cenni and Frateschi (1953), Ushijima (1958) and Blitz and Pellegrino (1969) have studied certain bones namely nasal, metacarpus and femur, but no mention of PIII or other parts of the skeleton were found in the literature.

Cenni and Frateschi's (1953) study of the composition of the metacarpal bone in young and adult horses revealed that the quantity of ash in young horses is below that of old ones. Ushijima (1958) studied the relationship between the nasal and metacarpal bones in the varying ages of 57 horses. He classified the horses into four age groups; up to 3 years in which the growth of bone seems to have been completed, those 4 - 8, 9 - 14 years old and over 15 years old. Although, he found that there is an increase in the bone ash and a slight increase of phosphorus, calcium, calcium/nitrogen ratio and specific gravity of the dried bones in the four age groups; he concluded that the comparison between ages 4 - 8 years old and ages 9 - 14 years old did not show





#### 4.0 DISCUSSION AND CONCLUSION

The literature has revealed that regional variations in ash content in a specific bone is related to differences in age of the different regions of the bone (Strobino and Farr, 1948; Pullar, 1961; Woodard, 1962). It has already been stated (section 7.2) that the proximal part of PIII (B2) completes its ossification at the age of 5 months.

After this age, ossification of the fibro-elastic cartilage of the hoof (B1) begins and progresses to a variable extent. The present analytical study has revealed a considerable difference between the mineral composition of the two regions. It is not unreasonable to attribute this difference to the age during which ossification develops in the two regions (B1 and B2).

Although Ushijima (1958) did not recognise composition differences in the groups of horses he examined, it is generally accepted by most authors, that bone ash composition increases progressively with the age of the animal. In this investigation the analysis of side bone compared with the adjacent part of PIII showed significant difference, however no progressive change related to age was found in these specimens.

## 5.0 GENERAL CONCLUSIONS

### Anatomy

In the prenatal stage the distal phalanx (PIII) continues to develop as the fetus increases in age, so that at 5 months old a well defined distal palmar process is formed but its proximal articular surface is still cartilaginous. At 2 months after birth, the proximal part of PIII and its features are still incompletely developed. At 5 months of age, PIII development is complete and at the age of 6 months side bones were detected.

The pumice stone like roughness of the parietal and solar surfaces of the distal palmar process is normally present at birth and throughout the animal's life, this roughness increases as PIII develops.

The notch of the palmar process may or may not be converted into a foramen at any age over 2 years old.

The cartilages of the hoof are continuous with the proximal palmar process of PIII and attached to the distal palmar process by a short ligament.

### Clinical and Radiological

Side bone development and other changes in the cartilage and the palmar process of PIII are best seen by anteroposterior and antero-posterior oblique radiological examination.

Side bone is described, as any degree of bony invasion from the proximal palmar process of PIII into the cartilage of the hoof and is

classified as short, medium, long and marked.

The bony islands arise in various parts of the cartilage plate and these and the most common degrees of side bone (short and medium) cannot usually be diagnosed without radiological examination. Diagnosis by digital palpation is quite unreliable unless the side bone is advanced to the stage of being long or marked and unless the bony islands are large.

Bony islands may also be found in the short ligament which attaches the distal palmar process of PIII to the solar border of the cartilage.

The radiological dense area sometimes found in the region of the palmar process in animals of 2 years old and over, is due to the presence of the bony bridge which converts the palmar notch into a foramen. A possible relationship between the occurrence of this bridge and the presence of bruises at the angle of the bar was noted.

In the circumstances of this investigation there was no clear cut evidence to support that side bone and/or bony islands caused lameness; but no opportunity was available to examine these animals regularly throughout the formation of the side bone or the bony islands, therefore one cannot exclude the possibility of formation of lameness occurring as a result of side bone formation.

Side bone consistently developed in all eight cartilages of all equines and it was invariably present from 6 months onwards. The degree of development varied in equines of the same type and age group. The short degree of side bone was most common in all types of equines.

The high degree (medium, long and marked) seldom occurred in animals under 3 years of age. This degree was more frequently met with in both fore feet than in one and more frequently in the fore than in the hind feet.

### Histopathology

The cartilages of the hoof are fibro-elastic at all stages of their development, in fetuses, in young and in adult horses. Elastic fibres were clearly demonstrated in all specimens examined.

The cartilage of the hoof, although different in structure is continuous with the hyaline cartilage of the proximal palmar process of PIII.

Side bone formation results from ossification, not by calcification of the cartilage.

One of the processes responsible for ossification in the cartilage is bone growth from the proximal palmar process of PIII. This development can be detected in animals of 6 months old and over.

The osteogenesis of side bone is a normal process of ossification (endochondral ossification), which has shown no evidence of inflammatory changes.

The bony islands that are found scattered in the cartilage plate, originate from areas where there are cartilage canals. Their osteogenesis and the type of bone which is formed is similar to that of side bone.

Bony islands which occur at the level of the distal palmar process, develop by secondary cartilage growth which undergoes ossification. Their osteogenesis and the type of bone formed is similar to that of the growth of the distal palmar process.

Necrosis and non provisional calcification of the cartilage plate occurs in all breeds of horses. Calcification was not found in horses under 3 years old and necrotic areas in horses under 4 years old. Both these changes become progressively more common with increase in age.

#### Chemical Analysis

The chemical analysis of side bone compared with the adjacent part of PIII has revealed a significant difference between the mineral composition of the two regions. This was related to differences in the development stage of the two bony regions. Increase in the animal age showed no significant increase in the ash composition of both regions.

ACKNOWLEDGEMENTS

This research was carried out in the Department of Veterinary Surgery under the direction of PROFESSOR J.S.A. SPREULL to whom I feel deeply indebted for his constant encouragement, guidance and helpful criticism. I wish also to thank him sincerely for his hospitality and kind care.

I welcome the opportunity to thank MR. J.A. FRASER for his continued interest, instructive criticism, unfailing patience and the facilities he provided that made the completion of this work possible.

I wish to express my sincere gratitude to PROFESSOR A.R. MUIR (Department of Veterinary Anatomy) for permitting me to use the laboratory facilities and in evaluating the histological preparations.

To DR H. PAVER, I owe my grateful thanks for his willing assistance and guidance during the statistical analysis of the results.

My special thanks are due to the Department of Biochemistry, Animal Diseases Research Association - Moredun Institute, for granting me the use of their Atomic Absorption Spectrophotometer, Vitration Precision Colourimeter and the Olivetti Computer Programa 101, and to the Department of Veterinary Pathology for providing facilities in the post-mortem room.

It is a pleasure to record my thanks for the understanding shown by the technical staff of the above departments and to MR R.T. THOMSON for his assistance in the photographic work incorporated in the thesis.

This study was partly supported by a grant from the Ministry of Higher Education of the Republic of Iraq and by a financial contribution from the author's father.

I would like to take this opportunity to express my grateful thanks to my father, SALMAN ASWAD and to my mother, KEYFIYAH ABRAHAM for her very generous agreement to guarantee my study by signing £6,720.48 notary act with the Ministry of Higher Education of the Republic of Iraq.

Adams, O.R.(1966) "Lameness in Horses" 2nd Edition, p.240.  
Lea and Febiger, Philadelphia, Pa.

Armstrong, W.D. and Singer, L.(1965) "Composition and  
Constitution of the Mineral Phase of Bone" Clin.Orthop.38.179.

Arey, L.B.(1968) "Human Histology" 3rd Edition, p.87. W.B.  
Saunders Company, Philadelphia, London, Toronto.

Axe, J.W.(1906) "The Horse its Treatment in Health and  
Diseases" 1st Edition, Vol.6, p.387. London: The Gresham Publishing  
Company.

Banks, W.C. (1961) "The Value of Radiographs in Certain Equine  
Lameness" Sthwest.Vet.14.108.

Bengtsson, G.(1962) "Systematic Radiological Examination  
Concerning the Cartilage of the Hooves of North-Swedish Stallions"  
Proc.9th Nordic Vet. Congr., Copenhagen.1.121.

Biddell, H., Douglas, C.I., Thomas, D., Fleming, G., MacNeilage, A.  
Murray, G. and Trotter, W.R.(1910) "Heavy Horses - Breeds and  
Management" 5th Edition, p.205. London: Vinton and Company Ltd., 8,  
Bream's Buildings, Chancery Lane, London, W.C.2

Biltz, R.M. and Pellegrino, E.D.(1969) "The Chemical Anatomy  
of Bone" J. Bone Jt.Surg.51-A,No.3.456.

Bloom,W. and Bloom, M.A.(1940) "Calcification and Ossification -  
Calcification of Developing Bones in Embryonic and Newborn Rats" Anat.  
Rec.78.497.

Bloom, W. and Fawcett, D.W.(1968) "A Textbook of Histology"  
9th Edition, p.212. W.B. Saunders Company, Philadelphia, London, Toronto.

Bogert, L.J. and Hastings, A.B.(1931) "The Calcium Salts of  
Bone" J. Biol.Chem.94.473.

Booher, L.E. and Hansmann, G.H.(1931) "Studies on the Chemical  
Composition of the Human Skeleton I - Calcification of the Tibia of  
the Normal Newborn Infant" J.Biol.Chem.94.204.

Bouley, H.(n.d.). Cited from Chauveau (1891).

Bradley, O.C.(1920) "The Topographical Anatomy of the Limbs of  
the Horse" pp.21,87. Edinburgh, W. Green and Son Limited.

Brennan, B.F.(1958) "The Veterinarian and Racetrack" Veterinary  
Scope. 3(2).2.

Calislav, T. and St. Clair, L.E. (1969) "Observation on the Navicular Bursa and the Distal Interphalangeal Joint Cavity of the Horse" *J. Am. vet. med. Ass.* 154 (4), 410.

Cameron, S.S. (1907) "Side Bone" *J. Dep. Agric. Vict.* 5. 376.

Cameron, S.S. (1910) "Hereditary Unsoundness in Horses" *J. Comp. Path. Ther.* 23. 115.

Cameron, G.R. (1930) "The Staining of Calcium" *J. Path. and Bact.* 33(4). 929.

Carlson, W.D. (1967) "Veterinary Radiology" 2nd Edition, Revised Part 3 with the assistance of Gillette, E.L. pp. 538, 595. London: Bailliere Tindall and Cassell.

Cenni, B. and Frateschi, T.L. (1953) "Study of the Composition and Strength of the Metacarpal Bone in Young and Adult Horses" *Ann. Fac. Med. vet. Pisa.* 6. 38.

Chauveau, A.P. (1891) "The Comparative Anatomy of the Domestic Animals" Revised and enlarged with the co-operation of Arloing, S. 2nd English Edition, Translated and edited by Fleming G., pp. 116, 211: London: J. and A. Churchill II, New Burlington Street.

Clark, B. (n.d.) Cited from Chauveau (1891).

Coleman, E. (1793) Cited from "The Early History of Veterinary Literature and Its British Development" by Smith, F. (1924) Vol. 3. p. 24. London: Bailliere, Tindal and Cox, 8 Henrietta Street, Covent Garden.

Collins, D.H. and Curran, R.C. (1959) "Modern Trends in Pathology" Edited by Collins, D.H. p. 300. London: Butterworth and Co.

Copland, A.N. (1967) "An Investigation of the Blood Supply to the Long Bones in the Pig with Particular Reference to the Arterial Supply of the Epiphysis and the Epiphyseal Plate" Thesis, University of Edinburgh.

Cresswell, H. and Smythe, R.H. (1963) "Equine Medicine and Surgery - A Text and Reference Work" 1st Edition, p. 434. American Veterinary Publications, Inc., 114 North West Street, Wheaton, Illinois, Drawer KK, Santa Barbara, California.

Cruickshank, B., Dodds, T.C. and Gardner, D.L. (1968) "Human Histology" 2nd Edition, p. 296. E. and S. Livingstone Ltd., Edinburgh and London.

Culling, C.F.A. (1963) "Handbook of Histopathological Techniques" 2nd Edition, p. 72 London: Butterworth and Co. (Publisher) Ltd.



Dietz, O.(1968) "Veterinary Encyclopedia. (3) Diagnosis and Treatment" Edited by Wamberg, K. and McPherson, E.A. p.1577. Medical Book Company, Copenhagen, Denmark.

Dixon, R.T.(1963) "The Nature of Injuries Causing Foot Lameness in Fast Gaited Horses" Aust.vet.J.39.177.

Dodds, G.S. (1930) "Row Formation and Other Types of Arrangement of Cartilage Cells in Endochondral Ossification" Anat.Rec.46.385.

Dodds, G.S.(1932) "Osteoclasts and Cartilage Removal in Endochondral Ossification of Certain Mammals" Am.J.Anat.50.97.

Douglas, S.W. and Williamson, H.D.(1970) "Veterinary Radiological Interpretation" p.130. Published by William Heinemann Medical Books Ltd., London.

Eberlein, (1908) "Die Hufknorpelverknöcherung" Handbuch der Thierärztlichen Chirurgie und Geburtshilfe. Band 4, p.450. Herausgegeben: Bayer, J. und Frohner, E. Wien and Leipzig.

Ellenberger, W. and Baum, H.(1943) "Handbuch der Vergleichenden Anatomie der Haustiere" p.328. Springer-Verlag, Berlin.

Elliman (1904) "Elliman's Embrocation for Horses, Dogs, Birds, Cattle" 4th Edition, p.60. Published by Elliman, Sons and Co., Slough, England.

Ensminger, M.E.(1951) "Animal Science" 2nd Edition, p.992. The Interstate Printers and Publishers, Danville, Illinois.

Erle, H.(1913) "Untersuchungen über die Hufknorpel des Pferdes" Inaugural-Dissertation zur Erlangung der Veterinar-Medizinischen Doktorwürde der Universität Leipzig.

Fitzwygram, F.W.(1863) "Notes on Shoeing Horses" 2nd Edition, p.112. London: Smith, Elder and Co., 65 Cornhill.

Fitawygram, F.W.(1894) "Horses and Stables" 4th Edition, p.324 London: Longmans, Green and Co.

Florey, H.(1962) "General Pathology" Based on Lectures Delivered at Sir William Dunn School of Pathology, University of Oxford. Edited by Flory, H. 3rd Edition, p.402 London: Lloyd-Luke Medical Books Ltd., 49 Newman Street.

Frank, E.R.(1959) "Veterinary Surgery" 6th Edition, p.201. Burgess Publishing Co., Minneapolis, Minnesota.

Fraser, G., Withers, A.R. and Spreull, J.S.A(1961) "Otitis Externa in the Dog" J. Small anim.Pract.2.32.

Gaiger, S.H. and Davies, G.O.(1960) "Gaiger and Davies' Veterinary Pathology and Bacteriology" 4th Edition. Reprinted by Gaiger, S.H. and Davies, G.O. p.16 London: Bailliere, Tindall and Cox, 7 and 8 Henrietta Street, Covent Garden, W.C.2.

Gatenby, J.B. and Painter, T.S.(1937) "The Microtometist's Vade" - Mecum (Bolles Lee) - A handbook of the Methods of Animal and Plant Microscopic Anatomy. 10th Edition, p.71. London: J. and A. Churchill Ltd., 104 Gloucester Place, Portman Square.

Gilruth, J.A.(1892) "The Functions of the Lateral Cartilages" Vet.Rec.5.358.

Gutenacker, F.(1901) "Die Hufkrankheiten Des Pferdes" Ihre Brekennung, Verhutung and Heilung Fur Thierarzte and Studirende der Thiermedicin. p.337. Stuttgart: Verlag Von Ferdinand Enke.

Haakenstad, L.H.(1969) "Chronic Bone and Joint Diseases in Relation to Conformation in the Horse"Eq.vet.J.1(6) 248.

Habacher, F.(1921) "Uber die and Behandlung des Vollhufes mit Berücksichtigung der Hufknorpelverknöcherung und Schnenspannung" Winer Tierarztl. Monatsschr.8.307.

Haines, R.W.(1934) "Cartilage Canals" J.Anat.68.45.

Ham, A.W. and Leeson, T.S.(1965) "Histology" 5th Edition, pp.377, 380. London: Pitman Medical Publishing Co. Ltd., J.B. Lippincott Company.

Ham, A.W.(1969) "Histology" 6th Edition, p.377. London: Pitman Medical Publishing Co., Ltd., J.B. Lippincott Company.

Hammett, F.S.(1925) "A Biochemical Study of Bone Growth - I. Changes in the Ash, Organic Matter, and Water During Growth" (Mus Norvegicus Albinus). J.Biol.Chem.64.409.

Harris, H.A.(1933) "Bone Growth in Health and Disease" pp.49, 213. Oxford University Press, London: Humphrey Milford.

Hayes, M.H.(1904) "Points of the Horse - A Treatise on the Movements, Breed and Evolution of the Horse" 3rd Edition p.674. London: Hurst and Blackett Limited, 13, Marlborough Street, W.

Hayes, M.H.(1950) "Veterinary Notes for Horse Owners" 14th Edition. Revised by Tutt, J.F.D., p.227. London: Hurst and Blackett Ltd.

Hess, A.F. Berliner, F.S. and Weinstock, M. (1931) "An Investigation of the Comparative Ash Content of the Metaphyses and Shafts of Bones" *J.Biol.Chem.*94.9.

Heusser, H.(1928) "Über den Bau und die Funktionen des Pferdehufes und ihre Beziehungen zur Hufknorpelverknöcherung und zum Humechanismus" *Schweizer Arch.Tierheilk.*70.169.

Hickman, J.(1964) "Veterinary Orthopaedics" pp.105, 363, 382. Printed in Great Britain by Oliver and Boyd Ltd., Tweeddale Court, Edinburgh, 1. and 39A Welbeck Street, London W.1.

Holcombe, A.A.(1923) "Special Report on Diseases of the Horse" p.438. U.S.D.A. Govt. Printing Office, Washington.

Hugentobler, J.(1907) "Die Veränderungen des Hufknorpelfesselbeinbandes und der Zehenbinde, Sowie ihre Beziehungen zur Schalenbildung und Verknöcherung der Hufknorpel" *Schweizer Arch. Tierheilk.*49.159.

Hughes, H.V. and Dransfield, J.W.(1953) "McFadyeans Osteology and Arthrology of the Domesticated Animals" 4th Edition, p.155. London: Bailliere, Tindall and Cox.

Hungerford, T.G.(1953) "Diseases of Livestock" p.411. Grahame Book Company: Martin Place and Elizabeth Street, Sydney.

Hurrell, D.J.(1934) "The Vascularisation of Cartilage" *J.Anat.*69.47.

Hutton, J.P.(1933) "Common Diseases of the Foot in Horse" *Vet.Med.*28.324.

Johne and Lungwitz, A.(1889) Cited from Muller (1937).

Jowsey, J.(1968) "Age and Species Differences in Bone" *Cornell Vet. (Supplement)* 58.74.

Lacroix, J.V.(1916) "Lameness of the Horse" *Chicago-American Journal of Veterinary Medicine. Veterinary Practitioners Series No.1.* p.155.

LaFosse, E.G.(1754) Cited from "The Early History of Veterinary Literature and its British Development" by Smith, F.(1924) Vol.2, p.91. London: Bailliere, Tindall and Cox, 8, Henrietta Street, Covent Garden.

Langley, J.A. and Rooney, J.(1967) "The Horse's Foot" *The British Racehorse*, December, p.548.

Leeney, H.(1911) "The Standard Cyclopedia of Modern Agriculture and Rural Economy" Vol.2,p.6. The Gresham Publishing Company, 34 and 35 Southampton Street, Strand, London.

Leeney, H.(1952) "Home Doctoring of Animals" 5th Edition, Revised by Sanders, C.G., p.267. London: Farmer and Stock-breeder Publications Ltd.

Liautard, A.(1923) "Special Report on Diseases of the Horse" Revised by Mohler, J.R.p.316. U.S.A. Govt. Printing Office, Washington.

Lowe, C.W. (1935) "Ein Beitrag zur Kenntnis der Hufknorpelverknöcherung" Tierärztl. Rdsch.41.121,137.

Lungwitz, A.(1898) "A Text Book of Horse Showing" Translated from the 8th German Edition by Adams, J.W. p.154. Philadelphia, J.B. Lippincott Company, London: 6 Henrietta Street, Covent Garden.

Lungwitz, M.(1906) "Handbuch der vergleichenden mikroskopischen Anatomie der Haustiere" p.22. Berlin, Verlagsbuchhandlung Paul Parey, S.W., Hedemannstrasse 10.

Lungwitz, M.(1919) "Einige bemerkswerte Beobachtungen an kranken Hufen, Knocheninseln innerhalb der Hufknorpel" Der Hufschmid. 37.96.

Lupton, J.I.(London n.d.) "Mayhew's Illustrated Horse Doctor" p.388 London: Griffith Farran Okeden and Welsh, Newberry House, Charing Cross Road and Sydney.

MacCallum, A.I. (1899) "Common Ailments and Diseases of Farm Stock" p.16. Edinburgh: E. and S. Livingstone.

Mallory, F.B.(1944) "Pathological Technique" p.48. W.B. Saunders Company, Philadelphia and London.

Maximow, A.A. and Bloom, W.(1957) "A Textbook of Histology" 7th Edition pp.117,126 Philadelphia: Saunders.

Miller, W.C.(1928) "Black's Veterinary Dictionary" p.877. A and C. Black Ltd., London, W.1.

Mitchell, W.M.(1936) "A General Consideration of the Disease Conditions Mentioned in the Horse Breeding Acts" Vet.Rec.48.1365.

Moller, H. and Dollar, J.A.W.(1906) "The Practice of Veterinary Surgery" Vol.III,p.628. Gay and Bird, London.

Montgomery, G.L.(1965) "Textbook of Pathology" Vol. I, p.92  
E. and S. Livingstone Ltd., Edinburgh and London.

Morgan, J.P.(1968) "Radiographic Diagnosis of Bone and Joint  
Diseases in the Horse" Cornell Vet.(Supplement) 58.28.

Morgulis, S.(1922) "A Comparative Study of the Composition  
of the Femur" Proc.Am.Soc.Biol.,J.Biol.Chem.50.11.

Muir, Sir R.(1964) "Muir's Textbook of Pathology" 8th Edition.  
Revised by Cappell, D.F., p.116. London: Edward Arnold (Publisher)  
Ltd.

Muller, E.(1937) "Uber den Beginn der Hufknorpelverknöcherung"  
Dissertation, Wein.

Muller, H.(1858) "Uber die Entwicklung der Knochenstanz nebst  
Bemerkungen über den Bau rachitischer Knochen" Z. wiss.zool. 9.147.

Nomina Anatomica Veterinaria (1968) "Submitted by the  
International Committee on Veterinary Anatomical Nomenclature and  
Adopted by the General Assembly of the World Association of Veterinary  
Anatomists Paris 1967" Printed in Austria by Adolf Holzhausen's  
Successors, Vienna.

O'Connor, J.J.(1934) "Encyclopedia of Veterinary Medicine,  
Surgery and Obstetrics" 2nd Edition, Edited by Wooldridge, G.H.,  
pp.920,986, 1038. Oxford University Press, London.

O'Connor, J.J.(1942) "Dollor's Veterinary Surgery" 3rd Edition,  
Edited by O'Connor, J.J., p.943. London: Bailliere, Tindall and Cox,  
7 and 8 Henrietta Street, Covent Garden, W.C.2.

O'Connor, J.J.(1945) "Lameness in Horses" Vet. J.101.204.

Osmer, W.(1759) Cited from "The Early History of Veterinary  
Literature and its British Development" by Smith, F.(1924) Vol.II,  
p.91. London: Bailliere, Tindall and Cox, 8, Henrietta Street,  
Covent Garden.

Oxspring, G.E.(1935) "The Radiology of Navicular Disease, with  
Observations on its Pathology" Vet. Rec.15.1433.

Percivall, W.(1832) "The Anatomy of the Horse, Embracing the  
Structure of the Foot" p.58. London: Longman, Rees, Orme, Brown,  
Green and Longman, Paternoster Row.

Pierard, J.(1968) "La Calcification des Cartilages Larynges du Chien" Econ.Med.anim.9.46.

Pires, A.(1949) "Tratado de las enfermedades del pie del Caballo" p.257 Editorial: Guillermo Kraft Limitada, Buenos Aires.

Prentice, A.(1946) "Your Horse and His Care" 2nd Edition p.60. Printed by Alex. Pettigrew Ltd., At the Atlas Works, Coatbridge.

Pritchard, Prof. (1888) "Side Bone" Veterinarian.61.119.

Pritchard, C.C.(1965) "Relationship between Conformation and Lameness in the Equine Foot" Auburn Vet.22.11,29.

Pryer, A.A.(1929) Cited from Oxspring (1935).

Pullar, E.M.(1961) "Regional Variation in the Ash Content of Limb Bones of the Pig" Aust. Vet.J.37.252.

Raker, C.W.(1968) "Clinical Observations of Bone and Joint Diseases in Horses" Cornell Vet.(Supplement) 58.15.

Reeks, H.C.(1918) "Diseases of the Horse's Foot" 2nd Edition, p.437. Alexander, Eger, Chicago.

Robertson, W.A.N(1932) "The Hereditary Character of Side Bone" Vet.Rec. 12.83,119.

Rooney, J.R.(1963) "Equine Medicine and Surgery" 1st Edition, p.408. American Veterinary Publications, Inc., 114 North West Street, Wheaton, Illinois, Drawer KK, Santa Barbara, California.

Rooney, J.R., Sack, W.O. and Habel, R.E.(1969) "Guide to the Dissection of the Horse" Corrected and reprinted (1969). Published by Sack, W.O.(1967). Distributed by Edwards Brothers, Inc. Ann Arbor, Michigan.

Rothschild, L.(1965) "A Classification of Living Animals" 2nd Edition, p.47. Longmans, Green and Co. Ltd., 48 Grosvenor Street, London, W.1.

Schafer, S.E.(1946) "Schafer's Essentials of Histology" 14th Edition, p.131. Edited by Carleton, H.M. Longmans, Green and Co. London, New York, Toronto.

Schebitz, H. and Wilkens, H.(1968) "Atlas of Radiographic Anatomy of Dog and Horse" p.179. Paul Parey In Berlin and Hamburg, 1 Berlin 61, Lindenstrasse 44-47.

Sidney, J.(1965) "Transactions of the Zoological Society of London" Vol.30. London: Published by the Society.

Sidney, S,(Lond.n.d.) "The Book of the Horse" p.670. Cassell and Co.Ltd., London.

Sisson, S. and Grossman, J.D.(1953) "The Anatomy of the Domestic Animals" 4th Edition. Revised. W.B. Saunders Company, Philadelphia, Pa.

Smith, F.(1890) "The Operation for Sidebones" J.Comp.Path. Ther. 3.308.

Smith, H.A. and Jones, T.C.(1957) "Veterinary Pathology" 1st Edition, p.52. London: Henry Kimpton, 134 Great Portland Street, W.1

Smithcors, J.F.(1963) "Equine Medicine and Surgery" 1st Edition, p.492. American Veterinary Publications, Inc., 114 North West Street, Wheaton, Illinois, Drawer KK, Santa Barbara, California.

Smythe, R.H.(1959) "The Examination of Animals for Soundness" p.51. London: Crosby Lockwood and Son Ltd., 26 Old Brompton Road, S.W.7.

Smythe, R.H.(1967) "The Horse - Structure and Movement" 1st Edition, p.42. London: J.A. Allen and Co.

Sobel, A.E., Rockenmacher, M. and Kramer, B.(1945) "Composition of Bone in Relation to Blood and Diet" J.Biol.Chem.159.159.

Solleysel, J.L.(1664) Cited from "The Early History of Veterinary Literature and its British Development" by Smith, F.(1924) Vol.I pp.350,364. London: Bailliere, Tindall and Cox, 8 Henrietta Street, Covent Garden.

Spooner, W.C.(1840) "A Treatise on the Structure, Functions and Diseases of the Horse" p.249. London: Longman, Orme, Brown, Green and Longmans, Paternoster Row.

Spooner, W.C.(1842) "A Compendium of the Veterinary Art" p.279. London: Longman, Brown, Green and Longmans; Houlston and Stoneman; and C. Dolman.

Stewart, C.M.(1938) "Prevalence of Side Bones in Mules" J.R.Army vet. Cps. 9.132.

Strobino, L.J. and Farr, L.E.(1949) "The Relation to Age and Function of Regional Variations in Nitrogen and Ash Content of Bovine Bones" J.Biol.Chem. 178.599.

Stump, J.E.(1967) "Anatomy of the Normal Equine Foot, Including Microscopic Features of the Lamellar Region" J.Am.vet.med.Ass. 151.1588.

Tavernor, W.D. and Vaughan, L.C.(1962) "Radiography of Horses and Cows" Br.vet.J. 118.359.

Taylor, T.G. and Moore, J.H.(1956) "The Effect of Calcium Deposition on the Chemical Composition of Bone Minerals in Laying Hens". Br.J.Nutr. 10.250.

Taylor, T.G., Moore, J.E. and Hertelendy, F.(1960) "Variations in the Mineral Composition of Individual Bones of the Skeleton of the Domestic Fowl" Br.J.Nutr. 14.49.

Taylor, J.A.(1959) "Regional and Applied Anatomy of the Domestic Animals" Part II, Thoracic Limb, p.179. Oliver and Boyd. Edinburgh: Tweeddale Court. London: 39A Welbeck Street, W.1.

Thompson, H.(1895) "Elementary Lectures on Veterinary Science" pp.32,59. Whitehaven: T. Brakenridge and Co. Limited.

Thompson, H. (1946) "Elementary Veterinary Science" 5th Edition. Revised by Duncan, A.C., p.119. London: Bailliere, Tindall and Cox.

Tohara, S.(1950) "Radiographical Studies on the Ossification of Leg Bones of Horses" Jap.J.vet.Sci. 12.11.

Townsend, C.H.S.(1935) "The Modern School of Thought on the Practical Effect of Certain Unsoundness on the Serviceability of Riding Horses" Vet.Rec. 15.1112.

Trautmann, A. and Fiebiger, J. (1952) "Fundamentals of the Histology of Domestic Animals" Translated and Revised from the 8th and 9th German Edition 1949 by Habel, R.E. and Biberstein, E.L., Comstock Publishing Associates, Itaca, New York.

Ullrich, H.(1934) "Rontgenologische Untersuchungen an den Zehenknochen der Vordergliedmaßen von Schlachtpferden im Hinblick auf die Prognose der periartikulären Schale und der Hufknorpelverknöcherung" Inaugural - Dissertation. Hanover.

Ushijima, J.(1958) "Biochemical Studies on So-called Osteomalacia (Osteodystrophia Fibrosa) in Horses, III. Some Factors Relating to Changes in the Composition of the Bone" Jap.J.vet.Res. 6.11.

Vaughan, I.(1895) "Strangeway's Veterinary Anatomy" 5th Edition. Edinburgh: Bell and Bradfute, 12 Bank Street.



Vogt, S.(1906) "Hufknorpelverknöcherung" Deutsche Tierärztliche Wochenschrift. No. 4, 37.

Volkman, D.(1936) "Die Ursachen der Verknöcherung der Hufknorpel" Tierärztl. Rdsch. 20.380.

Waxberg, G.(1953) "Report on Investigations Concerning Side Bone etc. in Swedish Breeds of Ardennes Horses" Collected papers from the Royal Veterinary College of Sweden, Stockholm. 27.35.

Weinmann, J.P. and Sicher, H.(1947) "Bone and Bones - Fundamentals of Bone Biology" pp.27, 55. London, Henry Kimpton, 25 Bloomsbury Way, W.C.1.

White, J.(1925) "A Compendium of the Veterinary Art" 14th Edition, p.282. London: Sherwood, Jones and Co.

Williams, W.(1872) "The Principles and Practice of Veterinary Surgery" p.309. Edinburgh: Maclachlan and Stewart, 64 South Bridge, London: Simpkin, Marshal and Co.

Wilson, T.(1935) "Examination of the Unsound Horse - the Significance of Certain Defects and Ailments which Constitute Unsoundness" Vet.Rec.15. 1369.

Witte, K.(1907) "Die Hufknorpelverknöcherung der Pferde" Monatshefte für Praktische Tierheilkunde. 18.241.

Woodard, H.Q.(1962) "The Elementary Composition of Human Cortical Bone" Hlth.Phys. 8.513.

Wooldridge, W.R.(1960) "Farm Animals in Health and Disease" 2nd Edition, Revised, p.418. London: Crosby Lockwood and Son, Ltd., 26 Old Brompton Road, S.W.7.

Youatt, W.(1874) "The Horse with A treatise on Draught" 4th Edition, p.426. Revised and Enlarged by Watson, W. London: Longmans, Green and Co.

Zietzschmann, O. and Krolling, O.(1955) "Lehrbuch der Entwicklungsgeschichte der Haustiere" p.363. Paul Parey, Berlin, Germany.

Zimmermann, A.(1908) "Beitrag zur Verknöcherung der Hufknorpel beim Pferde" Osterr Monatshefte f. Tierheilk. 32.529.