INDUCED ABNORMAL BONE GROWTH WITH PARTICULAR REFERENCE TO THE GROWTH PLATE.

(in 2 volumes)

VOLUME 1

A THESIS submitted for THE DEGREE

OF DOCTOR OF PHILOSOPHY .

in

The FACULTY OF VETERINARY MEDICINE

of

THE UNIVERSITY OF GLASGOW

by

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August 1979

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Acknowledgements

I would like to express my gratitude to Mr. D. MacFarlan in particular and to Mr. J. MacAulay for their assistance with radiographic sessions and for their diligence in looking after the lambs.

The considerable number of decalcified histological sections produced during this study were prepared by Miss N. Burns and her contribution was indispensable.

Professor J.P. Paul and Dr. N. Berme of the Bioengineering Department, University of Strathclyde, gave encouragement, and kindly made available to me the technical expertise of their audiovisual unit.

The greyhound training machine was loaned to me by the Shawfield Stadium and I would like to thank Mr. McAllister and Mr. Boyce who made this possible.

Both the Anatomy and Photography departments of the University of Glasgow Veterinary School have provided facilities without which, this study could not have been performed.

Professor J.R. Campbell acted as my supervisor for most of the duration of this study and I am pleased to acknowledge his assistance and encouragement.

Professor D.D. Lawson has shown interest in this study from the outset and I am grateful to him for his helpful advice and for the use of the facilities of his department.

Finally, the patience and dexterity of Mrs. A. Nuttall who has typed the manuscript of this study deserves special mention.

Summary

Limb deformities were successfully produced in skeletally immature lambs following contralateral limb hip excision arthroplasty. In order to determine the growth potential of the ovine skeleton prior to induced pelvic limb imbalance a preliminary study of normal epiphyseal fusion in comparable lambs has been performed. A detailed investigation of the angular bone deformities that were produced was then undertaken. In particular, the growth cartilage of deformed bones was studied and both decalcified and undecalcified sections of bone extremities prepared. Undecalcified sections permitted microradiographs to be produced and as sequential bone labels were administered the effect on longitudinal bone growth of limb imbalance could be assessed. In addition, angiography was performed and differences were visualised in the blood supply adjacent to the growth plate following induced limb imbalance. Although pronounced angular deformities were produced, in addition, a dramatic increase in bone torsion was encountered in tibiae. In metatarsi there was normally minimal or no torsion in control animals but those undergoing deformity exhibited marked axial rotation. By means of static load bearing and gait analysis observations, the aetiology of induced bone deformities has been investigated. It is postulated that asymmetrical growth plate loading occursand, as a result, abnormal endochondral bone growth is produced.

LIST OF ABBREVIATIONS

LH = Left hindlimb (= non-operated limb in experimental lambs)

RH = Right hindlimb (= operated limb in experimental lambs)

H/E = Haematoxylin & Eosin
VG = Van Giesson
TB = Toluidine Blue
GT = Green Trichrome
} Histological stains

AC = Alizarine Complexone
OTC = Oxytetracycline
XO = Xylenol Orange
FC = Fluorescein Complexone
} Fluorochrome Bone Labels

DTM = Distal, tibial, metaphyseal, cortical sections

DMM = Distal, metatarsal, metaphyseal, cortical sections

DTE = Distal, tibial, epiphysis/growth plate/metaphysis sections

DME = Distal, metatarsal, epiphysis/growth plate/metaphysis sections.

M = MEDIAL

L = LATERAL

A = ANTERIOR

P = POSTERIOR

The above abbreviations have been most commonly used. Others have been utilised but are fully explained in the text.

General Introduction

"The legs of the lame are not equal" Proverbs, Chap 25, vs7.

Whereas this statement is undoubtedly true, any irregularities that exist between paired limbs must depend on the lesion causing the lameness.

In a study of the effects of unilateral hip excision arthroplasty in sheep, Duff (1975), Duff & Campbell (1978), found that while initially lameness was evident in the operated pelvic limb, ultimately growth deformities which developed in the contralateral limb became the more serious clinical impediment (Plate 1).

The study that follows was in the first instance performed to test the reproducibility of the growth deformities that were previously recorded. In addition to investigating in detail this induced deviation from normal ovine pelvic limb development it has been considered advantageous to initially study the growth potential of some elements of the ovine skeleton.

Longitudinal bone growth - the advancement of knowledge.

"If man could learn from history,
what lessons it might teach us!
But passion and party blind our eyes,
and the light which experience gives
is a lantern on the stern,
which shines only on the waves behind us."

Coleridge (1831)

A number of authors, in particular Sissons (1953; 1956; 1971) and Enlow (1962; 1963) have described the early work that has led to current concepts of longitudinal bone growth. Although such historical surveys provide interesting and colourful reading it is apparent that whereas early scientists made remarkably accurate observations, in most instances it was many years before their work was accepted and the significance of their findings realised. Storey (1972) presents this view more strongly when he states that "the travail of early researchers could be likened to that of Bunyan's pilgrims progressing through the "Valley of Humiliation".

Following the observation by Vesalius that foetal bones are composed initially of cartilage it was apparently widely believed that bone transforms directly from cartilage by the addition of 'earthy substances'. This concept was accepted until the middle of the 18th century and numerous theories were proposed for the manner in which the saline like nutrients ('succus nutritius'), which hardened the cartilage into bone, were drawn from the blood (Enlow 1962).

Fallopius, a pupil of Vesalius, produced the first description

of the growth plate cartilage (Portal 1770 cited by Enlow 1962: 1963) but it was 300 years before the functional significance of this structure was understood.

During the 17th and early 18th centuries early microscopists likened bone growth to that of plants (Grew 1681; Leeuwenhoek 1693; Malpighi 1743 - cited by Enlow 1962; 1963). This concept arose from the observed similarities between bark and periosteum and the earlier observations by Spigelius (1631) on appositional bone growth by periosteum.

During the early 18th century however two experimental techniques added greatly to the understanding of how longitudinal bone growth occurs.

Firstly Hales (1726) drilled holes in the surface of a growing chicken limb bone and as the holes were the same distance apart when observed two months later concluded that longitudinal growth of the bone occurred at its extremities (Bisgard & Bisgard 1935; Sissons 1953; 1956; 1971; Enlow 1962; 1963; and Brookes 1971). This observation was confirmed by Duhamel (1742) and John Hunter (cited by Sissons 1953; 1956; 1971) and also by Humphrey (1861); Ollier (1867); Koelliker (1873); Wegner (1874); Haas (1926); Gatewood & Mullen (1927) and Payton (1931) (cited by Bisgard & Bisgard 1935).

The other important series of experiments resulted from exploitation of the bone staining properties of madder. Enlow (1963) cites Cameron (1930) who reported that madder was known and used as a dye by the early Greeks and Romans and in his earlier paper(Enlow 1962) states that the bone staining properties of

madder was recorded both by Mizaldus (1566) and Lemnius (1567). Belchier (1736) rediscovered the properties of this dye in staining bone but it was the experiments of Duhamel (1739; 1742; 1743) with madder which elucidated the mechanisms of bone growth. By intermittently feeding experimental animals with madder Duhamel observed that the dye only coloured the parts of the skeleton which were being formed at the time of administration. Duhamel demonstrated that bone growth involved both growth in length from the extremities and width from beneath the periosteum (cited by Sissons 1971).

More than a century later Humphrey (1858) correlated the advances that had been made and concluded that a bone grows in length by activity of the growth plate and in thickness by periosteal deposition (Enlow 1963). Humphrey's (1958) amplification of Duhamel's conclusions was the result of the early microscopic studies which prevailed at the beginning of the 19th century.

Between the years 1760 and 1770 John Hunter repeated the work of both Hales and Duhamel and formulated the concept that bone growth necessitates the processes of both new bone deposition and resorption of pre-existing bone. He realised the importance of these two processes in the longitudinal growth of a bone and its marrow cavity (Sissons 1971).

Enlow (1963) states that although Spigelius (1631) and Nesbitt (1736) observed both endochondral and intramembranous growth of bone no significance was ascribed to their findings and their work was forgotten. Howship (1817) apparently

received full recognition for his rediscovery of endochondral and intramembranous bone growth.

As attention focussed on the growth plate microscopy was being established as a technique. Howship (1815) observed the parallel arrangement of the 'particles of cartilage' and Goodsir (1845) demonstrated these to be parallel rows of chondrocytes (Enlow 1963). Tomes (1839) is reported to be the first to realise that the proliferation of daughter cells in linear columns results in longitudinal growth of the bone by a process of interstitial growth within the cartilage (Enlow 1963). Goodsir (1845), Koelliker (1852) and Virchow (1860) as a result of microscopic studies concluded that proliferation of cartilage and the subsequent replacement of the tissue by bone were the cellular processes on which longitudinal growth depends (Sissons 1953; 1956; 1971).

In more recent years studies of longitudinal bone growth have employed similar techniques to those used by Hales, Duhamel and John Hunter. The advent of radiography in conjunction with metallic markers implanted into living bone and the discovery of a number of bone stains have enabled more detailed studies to be made.

In addition to normal bone development the investigation of abnormalities now became a possibility.

PART A

INTRODUCTION

"Structure and architecture or morphological features of animals are not uniform and hence cannot be standardised ... Even in the individual, there are differences between bilateral organs (bones, muscles, joints, viscera, vessels, nerves). It is an anatomic adage that variations are the most "constant" finding. However it is possible to establish an average or normal pattern for any major group of animals and to recognise deviations from the pattern"

Didio (1975)

The development of long bones

The development of the skeletal elements of the limbs begins with the formation of a hyaline cartilage model. In the midregion of this model a diaphyseal, or primary, ossification centre develops and at a later date, separate ossification centres appear at either one or both extremities of the long bone. These separate centres at the extremities are referred to as secondary centres of ossification. A number of authors have described the stages in the development of centres of ossification including Leblond & Greulich (1956) Gardner (1956, 1963, 1971); Bloom & Fawcett (1968); Rang (1969) Brookes (1971); Warwick & Williams (1973) and Kalajian & Cooper (1976).

Smith (1955); Gardner (1971) and Boyd (1974) have described the various techniques employed in studies of the foetal skeleton. Radiography, gross dissection of specimens, histological examination of serial sections and examination of cleared specimens (often with the addition of a bone staining dye such a Alizarin) have been most frequently utilised. As

a result of use of such techniques a great number of reports exist describing the appearance of ossification centres in the appendicular skeletons of a variety of species. Appearance of ossification centres in the mouse have been recorded by Johnson (1953) and Green & Fekete (1933); in the rabbit by Ring (1955) and Heikel (1960); in the cat by Smith (1968a and b) and Boyd (1974); in the dog by Hare (1961a); Chapman (1965); Van Sickle (1965) and Wilsman and Van Sickle (1970); in the pig by Hodges (1953); in the sheep by Harris (1937); Andreeva (1940); Tohara & Mijakawa (1953); Smith (1955, 1956) and Rajtova (1971); in the ox by Lindsay (1969) and Lindsay, Boyd and Hogg (1969) and finally in man by Pryor (1923), Davies & Parsons (1927), Flecker (1942) and Kelly and Reynolds (1947).

It is generally accepted that there is a definite sequence of appearance of ossification centres although variations in chronology exist on the basis of individuality, sex and possibly race.

That individuality occurs in the appearance of ossification centres is well demonstrated in the findings of Smith (1956) who on examining sheep foetuses found on comparison of siblings of the same sex and size, one often had more ossification centres than the other.

Sex differences in skeletal development were described in human material by Pryor (1905;1923) and are discussed in more detail below.

Kelly and Reynolds (1947) found differences in the age of appearance of carpal ossification centres in white and negro children but commented on the difficulties due to socio-economic factors in such studies. Van Sickle (1965) has described differences in the manner of ossification of the anconeal process in two different breeds of dog. Other authors, such as Green & Fekete (1933) have found minor differences in the chronology of ossification between their results and those of previous comparable studies which they have ascribed to breed and strain differences.

Epiphyses

There is growth of the secondary (epiphyseal) ossification centre with the result that it essentially occupies the majority of the articular end of the long bone. A layer of hyaline cartilage persists over the actual joint surface and a thicker zone, the growth plate, remains between the diaphysis and epiphysis.

Parsons (1904; 1905; 1908) distinguished between pressure epiphyses (which transmit weight from one bone to another and thus participate in joints), traction epiphyses (which serve as attachments for muscles) and atavistic epiphyses (which represent parts of the skeleton that have lost their function). Parsons classification has been widely accepted although Salter and Harris (196: further divide pressure epiphyses into two groups depending on whether the nutrient vessels enter the epiphysis directly or indirectly.

Growth Plates

The specialised hyaline cartilage, the growth plate, which is primarily concerned with longitudinal bone growth, has often been referred to by a number of ambiguous terms which has led to a degree of confusion in the literature. The terms 'epiphyseal cartilage plate' (Haas 1917) and in particular the synonyms growth plate and growth cartilage will be used in this study.

The growth plates produce longitudinal growth of long bones with only a very small amount of increased bone length being contributed by endochondral ossification on the surface of the epiphysis (Siegling 1941; Leblond and Greulich 1956; Siffert 1966; Rang 1969; Gardner 1971; Little 1973; and Grandage 1976).

It is the growth plates associated with pressure epiphyses that primarily produce longitudinal bone growth. Just as with the epiphyses the growth plates can be classified according to their location. Lewis (1958, Smith (1962a) and Ogden (1974) however studied the histological character of the tibial tuberosity growth plate in man and described the presence of coarse bundles of collagen fibres. Smith (1962a) suggested the term 'fibrous epiphyseal plate' for this slightly aberrant form of growth plate.

Bechtol and Marmor (1959) produced a classification of

growth plates based on morphology. In mammals, these authors recognised 1) a transverse plate 2) a plate convex towards the joint with a cone shaped projection near the centre and 3) a complicated structure with four epiphyseal indentations into which projections from the metaphysis interlock.

The pattern of growth in length of the skeletal elements differs between man and animals (Sissons 1956; 1971; Morscher 1968). In animals, except for the primates, growth usually follows an S shaped curve. In man however, there is reported to be rapid growth in length of the skeleton during the prepubertal stage and in childhood.

There are also reported differences, between species, in the growth plate itself (Little 1973). The germinal zone of the growth cartilage is narrow in small animals but occupies between half and two thirds of the growth plate in larger animals and man. Both Schaerer (1974) and Kember and Sissons (1976) have stressed the dangers of making generalisations between species with regard to the growth plate.

It was discovered in the 19th century that inequalities exist in the contributions made to longitudinal growth by growth plates at either end of diepiphyseal long bones. Bisgard and Bisgard (1935) describe the clinical observations of Ollier (1867) on cases of knee and elbow joint resection in children. Ollier noted that knee joint resection resulted in much greater limb length deficit than elbow joint resection and calculated the contributions to

longitudinal limb growth of both extremities of each bone involved in the resection. Digby (1916) working with human bone specimens further attempted to quantify the growth contribution of individual growth plates. Bisgard & Bisgard (1935) incorporated metallic markers in each end of goat limb bones and measured growth from radiographs. These authors concluded that the proportion of longitudinal growth from the two ends of goat long bones is unequal and that the disproportion is greater for growth postnatally. This latter observation is in agreement with the findings of both Felts (1954) and Brookes (1963) who discovered no marked disparity in the growth rates at the two extremities of human foetal long bone specimens. Heinze and Lewis (1968) obtained similar findings in the developing equine radius. Both Rang (1969) and Salentijn (1974) have suggested that differential growth rates of bone extremities is genetically determined and thus antedate formation of the growth plates themselves.

In the pig Payton (1932) described growth diminishing more rapidly at the bone end with the lesser contribution to bone growth but Bisgard and Bisgard (1935) in experiments with goats failed to confirm this observation.

A number of authors including Lewin (1929); Gardner (1956); Clark (1971); and Salentijn (1974) have stated that the centres of ossification which appear first are usually those of the bone ends that grow relatively faster. The growth plate at that

bone end is considered to usually close last. Warwick and Williams (1973) have pointed out that there are numerous inconsistencies to this generalisation. Smith (1955) found that the epiphysis which ossified first was the last to fuse in the sheep radius, femur and tibia but not in the humerus and ulna.

Both Payton (1932) and Bisgard and Bisgard (1935) constantly found, in the pig and goat respectively, that the growth plate which produced the majority of growth in a long bone was thicker. In more recent years authors such as Morscher (1968), Sissons (1971) and Salentijn (1974) have agreed with this observation. Rang (1969) cites Tapp (1966) who demonstrated, by means of hormone control of growth plate activity, that the thickness of the growth cartilage bears a constant relationship to the amount of bone produced.

Growth Plate Closure

The growth plate becomes narrowed at the time of cessation of longitudinal growth and prior to its obliteration and replacement by bone (Siffert 1966).Whereas it is accepted that the formation of a terminal plate of bone rather than cartilage is associated with cessation of growth a number of authors have suggested that growth may in fact cease prior to bony union of epiphysis and metaphysis. Stevenson (1924) found growth to have ceased despite the presence of intact growth plates in two human cases. Neither of these cases however can be considered normal and indeed one suffered from a hormonal disturbance. This observation was nevertheless searched for

and found in a number of other species including the rat (Dawson 1925), guinea pig (Zuck 1938), European Bison (Koch 1934); ungulates (Todd and Todd 1938) and in sheep (Todd, Wharton and Todd 1938; Smith 1955). Smith and Allcock (1960) stated that a bone has ceased to grow in length at one end when the epiphysis and diaphysis unite over the smallest area. In the cat, Smith (1968b;1969) found that cartilage remnants can remain unossified at the periphery of a fused growth plate for considerable periods after longitudinal growth has ceased. Cartilage remnants may indeed result in erroneous interpretation of the stage of union of epiphyses on radiographs. Cartilage remnants may thus explain the reports of Moss and Noback (1958) and Lavine,Moss and Noback (1962) (cited by Morscher 1968) who observed that in man the longitudinal growth of fingers ceases before the growth plates disappear in radiographs. Siffert (1966) remarks that "radiographic evidence of a lucent area at the growth plate does not necessarily indicate that active growth is still in progress".

The reported lag between cessation of growth and bony union has led Morscher (1968) to postulate that ossification of growth plates may be a consequence of the cessation of growth activity rather than its cause.

It is relevant to mention that although a lag between growth cessation and union has been reported in the rat this species is peculiar in that it demonstrates a phenomenon that

has been called 'lapsed union'. Not all of the growth plates in the rat close. Most plates fuse at an age of 80 - 120 days (Simpson, Asling and Evans 1950). Some however remain open or close only at a very advanced age (Morscher 1968).

It is currently believed that growth plate closure is largely under hormonal control (McKearn and Stewart 1957 cited by Morscher 1968; Rang 1969; Sissons 1971; Little 1973). The influence of hormones on growth plate closure is described in more detail below. Both McKearn and Stewart (1957) and Rang (1969) suggest that as growth plates do not all close simultaneously but in a certain sequence, there must be some local regulation of growth plate closure. There must also be a distinction between the hyaline cartilage of the growth plate and that of the articular surface as the latter appears to be unaffected by the factors which result in growth plate closure. There are known however to be metabolic differences between growth and articular cartilage (Beckmann, Rodogerdts and Buddecke 1975).

Becks, Asling, Collins, Simpson and Evans (1948) made histological observations on fusion of the third metacarpal epiphysis to the diaphysis in the rat. These authors found that when growth is complete the plate lies dormant for some time gradually becoming thinner and then the following changes occur.

1) Proliferating chondrocytes become scanty
2) Chondrocytes form cones and not palisades
3) Hypertrophic cells become infrequent

4) Trabecular new bone formation becomes reduced

5) Cartilage and bone are removed (i.e. some of the growth plate is absorbed and not replaced by bone)

6) Capillary invasion becomes uneven and in places perforates the cartilage to reach the epiphysis. This breach widens and bone is laid down around the vascular tuft uniting the metaphysis and epiphysis.

7) The central trabeculae are slowly removed.

Haines and Mohiuddin (1959) described the stage of union in a number of different human bone specimens and arrived at a less adequate histological sequence of closure. An investigation which involved studying closure of different growth plates with subsequent amalgamation of results seems unlikely to be valid.

Haines (1975) has more recently reviewed the literature and added histological observations of his own on epiphyseal union in both the dog and man. Haines recognised that union occurred involving single or multiple perforations of the cartilage plate and that species differences exist in this manner of closure. He similarly distinguished between early and late union in the mammal and commented that there was usually single perforation of the growth cartilage with no remaining epiphyseal scar following complete union in the smaller and earlier uniting epiphyses. In the larger and later uniting epiphyses Haines (1975) stated that there were multiple perforations in the growth cartilage prior to destruction of the whole plate and that remnants of epiphyseal bone often persisted as an epiphyseal scar.

Cope (1920) described the 'fusion lines' of human bones

and stated that the most obvious and persistent 'scars' encountered were related to the epiphyses of the greater trochanter, femoral head, distal femur, and proximal tibia. The late fusing epiphyses in the rat's pelvic limb include these sites and Dawson (1925) wondered whether this observation was of any significance. From Haines (1975) studies these sites in the rat might be expected to show 'fusion lines' or epiphyseal scars.

Haines (1975) recorded that both with single and multiple perforation types of closure remnants of the peripheral parts of the plates may be found for a period of time with the cartilage columns set at right angles to the axis of the bone as a whole. At full union all such residual cartilage was destroyed. As reported above, Smith (1968b;1969) found similar cartilage remnants in the cat.

Wide variation exists in the literature in observed epiphyseal closure times. Such variation seems largely to result from the use of different criteria for judging union to have occurred. The persistence of cartilage remnants at the periphery of the growth plate would appear to be the most likely source of confusion. Haines (1975) stated that growth plate union begins with completion of the first mineralised bridge between epiphysis and diaphysis and ends with the complete disappearance of the cartilaginous growth plate. In some cases at least there is considerable lapse of time between the commencement and completion of the union process.

Most reports of the chronology of epiphyseal union have been the result of either radiographical studies or observations on bone specimens. As Smith (1956) pointed out, the great disadvantage of anatomical studies of epiphyseal union in experimental animals is that each result necessitates one death. Paterson (1929) considered that the radiographical method of studying human epiphyseal union had distinct advantages as both the normality and exact age of the living subject can be ascertained.

Those authors who have used anatomical studies either alone (Stevenson 1924; Dawson 1925; Koch 1935; Todd and Todd 1938) or in conjunction with radiography (Zuck 1938) have described the process of union as occurring in a number of observable stages. Similarly those studies involving radiography have involved descriptions of a sequence to growth plate closure (Davies and Parsons 1927; Paterson 1929; Todd 1930a; Sidhom and Derry 1931).

The persistence of cartilage remnants proved a problem with radiographic studies and as Sidhom and Derry (1931) pointed out, the appraisal of cap like epiphyses, with no clear radiolucent line corresponding to the growth plate, is very difficult. Largely as a consequence of such difficulties the criteria that have frequently been used in assessing epiphyseal fusion on radiographs are

1) Complete disappearance of the growth plate and thus absence of any radiolucent line with as a consequence

2) The presence of uninterrupted bone cortices at the extremities of the long bones under consideration.

There have been numerous studies of epiphyseal union involving either radiographical or anatomical observations.

In man reports exist by Stevenson (1924); Davies and Parsons (1927); Paterson (1929); Todd (1930a); Sidhom and Derry (1931); Flecker (1942); in the dog by Schlotthauer and James (1952); Smith and Allcock (1960); Hare (1961b) Hanlon (1962); Chapman (1965) and Sumner-Smith (1966). In the cat by Smith (1968b, 1969). In the guinea pig by Zuck (1938). In the rat, Dawson (1925); in the pig, Weiss (1972) in the horse, Todd and Todd (1938), Myers and Emmerston (1966) and Monfort (1968). In the goat, Nowicki (1972) and Rajtova (1974). In the European Bison, Koch (1935) and in ungulates and Sirenia, Todd and Todd (1938).

Stevenson (1924) considered the sequence of epiphyseal union to be the same in both man and the higher primates and stated there was a common sequence in all mammals. Other authors have found variations from the sequence in man in their studies of various species but the union patterns are considered to be similar (Dawson 1925; Zuck 1938; Koch 1935; Todd and Todd 1938). The time relationships of epiphyseal union however show marked species differences (Zuck 1938; Todd and Todd 1938; Rajtova 1974).

Differences also occur in the chronology of closure between individuals of the same species (Todd 1931; Sumner-Smith

1966; Smith 1969) and thus just as with the appearance of ossification centres epiphyseal closure is subject to individuality.

Growth Plate Closure in Sheep

Smith (1955) has reviewed the early literature pertaining to epiphyseal closure in sheep. Smith cites the work of Sanson (1872) who he states was the first to report the order of epiphyseal fusion in sheep. Sanson however only studied the pelvic limb and recorded the order of fusion as

1) Distal epiphysis of tibia with shaft
2) Lesser trochanter of femur with shaft
3) Head of femur with shaft
4) Greater trochanter of femur with shaft
5) Condyle of femur with shaft
6) Trochlea of femur with shaft
7) Proximal epiphysis of tibia with shaft
8) Tubercle of tibia with shaft.

According to Smith (1955) Tschirwinsky (1889) continued Sanson's work and provided an order of fusion for the ovine pectoral limb. The sequence of union given by Tschirwinsky (1889, 1910) was

1) Distal epiphysis of humerus, proximal epiphysis of radius
2) Distal epiphysis of tibia
3) Lesser trochanter of femur
4) Epiphysis of metatarsus
5) Epiphysis of metacarpus
6) Epiphysis of head of femur
7) Epiphysis of greater trochanter
8) Proximal epiphysis of ulna

10) Distal epiphysis of femur, proximal epiphysis of humerus, proximal epiphysis of tibia

11) Distal epiphysis of ulna

Some indication of the time of union of epiphyses in the sheep was given by Tschirwinsky (1910) in his later publication and his results which were obtained by boiling the bones for up to 8½ hours have been recorded alongside those of other workers in Table 1.

In 1897 Lesbre collated data on epiphyseal fusion of the domestic animals and presented the following sequence of fusion of sheep limb epiphyses

1) Distal epiphysis of humerus, proximal epiphysis of radius
2) Scapular tuberosity
3) Epiphysis of phalanx II
4) Epiphysis of phalanx I
5) Distal epiphysis of tibia
6) Epiphysis of metacarpals and metatarsals
7) Fibular tarsal epiphysis
8) Proximal and distal femoral epiphyses and proximal epiphysis of ulna
9) Proximal epiphysis of humerus and tibia, distal epiphysis of radius and ulna

The times of closure given by Lesbre (1897) are presented in Table 1.

Todd and Todd (1938) studied epiphyseal fusion, assessing the stage of union by visual inspection in a small number of crossbred sheep. Of the eight skeletons examined by these authors only five were of known age and thus an estimate

was made in many cases to provide the times given in Table 1.

With the advent of radiography a number of studies have been made on the time and sequence of epiphyseal union in sheep. Tohara and Miyakawa (1953) reported on the radiographical completion of union in sheep limb bones. There is a degree of confusion resulting from translation of their work but it appears that they claim completion of fusion at the times indicated in Table 1.

Smith (1955) radiographed the appendicular skeletons of Clun Forest sheep at approximately two monthly intervals and summarised his findings in a publication a year later (Smith 1956) (see Table 1). The criteria of fusion used by Smith in his study were loss of a radiolucent line on radiographs in the region of the growth plate and continuity of outline of the bone cortices. Since the extensive work of Smith, two other reports have appeared in the literature. A Polish worker (Nowicki 1974) studied the postnatal development of the limb skeleton in sheep from the day of birth. Rajtova (1974) made a similar study in both the sheep and goat, although the criteria on which a judgement of union was made are not clearly stated. The chronology of union given by these authors is given in Table 1.

TABLE 1

	LESBRE (1897)	TSCHIRWINSKY (1910)	TODD & TODD (1938)
PECTORAL LIMB			
Scapular tuberosity	5 mths	-	8 mths
Proximal humeral epiphysis	3½ yrs	16-21 mths	36 mths
Greater tubercle - humerus	-	-	-
Distal humeral epiphysis	3-4 mths	3 mths	8 mths
Lateral humeral epicondyle	-	-	-
Medial humeral epicondyle	-	-	-
Proximal radial epiphysis	3-4 mths	3 mths	6 mths
Distal radial epiphysis	3½ yrs	16-21 mths	fused at 33 mths
Proximal ulnar epiphysis	3-3½ yrs	-	-
Distal ulnar epiphysis	3½ yrs	-	complete at 33 mths
Antebrachial synostosis	-	-	-
Synostosis carpals II & III	-	-	-
Synostosis metacarpals III & IV	-	-	-
Metacarpal epiphysis III & IV	20-24 mths	15½-16 mths	12 mths
Phalanx 1 epiphysis III & IV	7-10 mths	-	-
Phalanx 2 epiphysis III & IV	5-7 mths	-	-
PELVIC LIMB			
Acetabular synostosis	-		
Proximal femoral epiphysis (femoral head)	3-3½ yrs	toward end of 16th mth	complete - 33 mths
Greater trochanter - femur	-	toward end of 16th mth	complete - 33 mths
Lesser trochanter - femur	-	10 mths	-
Distal femoral epiphysis	3-3½ yrs	16-21 mths	33 mths
Proximal tibial epiphysis	3½ yrs	16-21 mths	33 mths
Tibial tuberosity	-	-	-
Distal tibial epiphysis	15-20 mths	10 mths	12 mths
Synostosis carpal & IV tarsal	-	-	-
Fibular tarsal epiphysis	3 yrs	-	-
Synostosis metatarsals III & IV	-	-	-
Metatarsal epiphysis III & IV	20-24 mths	15½-16 mths	12 mths
Phalanx 1 epiphysis III & IV	7-10 mths	-	-
Phalanx 2 epiphysis III & IV	5-7 mths	-	-

TABLE 1 (Cont)

TOHARA & MIYAKAWI (1953)	SMITH (1955) (1956)	NOWICKI (1974)	RAJTOVA (1974)	
			begins	ends
-	4-5 mths	270 days (9½ mths)	5-6 mths	11 mths
-	17 mths	480 days (17 mths)	11 mths	2½-3½ yrs
-	27-28 mths	600 days (21½ mths)	-	-
complete at 1 yr	4 mths	320 days (11½ mths)	3-4 mths	9 mths
-	4 mths	370 days (13¼ mths)	-	-
-	6 mths	370 days (13¼ mths)	-	-
complete at 1 yr	4 mths	400 days (14¼ mths)	3½ mths	11 mths
complete at 4 yrs	20-21 mths	580 days (20¾ mths)	11 mths	3½ yrs
complete at 3 yrs	20-21 mths	540 days (19¼ mths)	10 mths	1½ yrs
complete at 4 yrs	26 mths	580 days (20¾ mths)	-	3⅛-3½ yrs
-	-	500 days (18 mths)	-	-
-	-	200 days (7 mths)	-	2-10 wks
complete at 3 yr	15½-16 mths	450 days (16 mths)	6 mths	2½-3 yrs
complete at 1 yr	-	35 days (1¼ mths)	-	4-14 days
complete at 2 yrs	10 mths	400 days (14¼ mths)	100 days	11 mths
complete at 2 yrs	8 mths	320 days (11½ mths)	100 days	18 mths
	-	280 days (10 mths)	-	-
-	16-18 mths	420 days (15 mths)	5 mths	2-2½ yrs
-	17-19 mths	450 days (16 mths)	-	3½ yrs
-	13-14 mths	320 days (11½ mths)	-	-
complete at 3 yrs	18-20 mths	500 days (18 mths)	10 mths	3½ yrs
-	20-25 mths	560 days (20 mths)	100 days	4½ yrs
-	30 mths	over 2 yrs	128 days	4-5 yrs
-	14-15 mths	480 days (17 mths)	100 days	2½-3 yrs
complete at 1 yr	-	60 days (2 mths)	-	3-10 wks
complete at 3 yrs	-	400 days (14¼ mths)	-	-
-	-	200 days (7 mths)	-	12 wks
complete at 3 yrs	15 mths	450 days (16 mths)	6 mths	2 yrs
complete at 2 yrs	8-9 mths	400 days (14¼ mths)	? 100 days	? 11 mths
complete after 2 yrs	6 mths	320 days (11½ mths)	? 100 days	? 18 mths

X-Irradiation and the skeleton

Myers and Emmerston (1966) in the course of their study on the age and manner of closure of equine pectoral limb epiphyses took care not to use the same forelimb for two consecutive radiographic examinations. Their logic was that by so doing, the effect of x-irradiation on the growing limbs was minimised.

Considerable interest has centred around the effects of ionising radiation on growing bone since the original observations by Perthes (1903), Tribondeau and Recamier (1905) and Forsterlung (1906). Perthes obtained retardment of wing development of one day old chicks following administration of therapeutic doses of external radiation. Tribondeau and Recamier and Forsterlung noted impairment of growth in the cat and rabbit respectively following irradiation. The chondroblasts of the growth plate cartilage are especially susceptible to damage by ionising radiation (Gall, Lingley and Hilcken 1940; Barr, Lingley and Gall 1943; Dawson 1968) whereas the bones of adults are reported to be considerably more resistant (McLean and Urist 1968). The articular cartilage shows no primary injury following irradiation in doses sufficient to result in growth cartilage change (Gall, Lingley and Hilcken 1940). Some authors, such as Aub, Evans, Hempelman and Martland (1952) and McLean and Urist (1968) have suggested that damage to the blood vessels supplying bone is primary to bone cell injury but this may only be true for higher doses

of x-irradiation (Dawson 1968).

The effects of x-irradiation on the growth plate are dose related (Brooks and Hillstrom 1933; Bisgard and Hunt 1936; Hinkel 1942; 1943a; Barr, Lingley & Gall 1943; Barnard, Davies and Kamp 1963; Leatherman 1970) until a maximal response is obtained after which no additional increase in the amount of irradiation will produce any greater changes. (Gall, Lingley and Hilcken 1940; Barr, Lingley and Gall 1943).

The changes in the growth cartilage following x-irradiation range from minimal disruption after which growth function recovers to complete growth arrest. A review of the literature reveals that in general relatively high doses of the type used therapeutically in man, are required to produce growth arrest. The effects of x-irradiation doses of such magnitudes have been described in laboratory animals by Baunach (1935); Bisgard and Hunt (1936); Regen and Wilkens (1936); Gall, Lingley and Hilcken (1940) and Barr, Lingley and Gall (1943). Bisgard and Hunt (1936) demonstrated that although gross retardment of limb development in rabbits following doses of 400r * half this dose produced histological changes in the growth plate and a quarter of the dose produced a reduction in haematopoietic tissue in the medullary cavity. Simon, Reuss and Spear (1947) stated that several workers had shown that there is a reduction in the number of mitotic figures in chick fibroblasts in tissue culture following exposure to

* r = roentgens

doses of the order of 50^{r} and 100^{r}

Fractionation of the dose administered markedly reduces the degree of resulting change in the growth plate (Brooks and Hillstrom 1933; Bisgard and Hunt 1936; Gall, Lingley and Hilcken 1940). Similarly protraction of the interval between fractionated units greatly lessens the effect upon growth (Bisgard and Hunt 1936).

Growth impairment is usually greater in young animals and the younger and more active the growth plate the more sensitive it is to the deleterious effects of irradiation. Early histological changes follow small doses in younger but not older animals. The effect of age on changes induced by irradiation are well demonstrated by the results of Baunach (1935) (cited by Hinkel 1942) who demonstrated that 4 week old rabbits' limbs were stunted by 220r whereas 275-330r were required to produce demonstrable effects in 8 week old rabbits.

Garland (1957) stated that the late effects of properly employed diagnostic roentgen radiation on human bone are negligible. There does not appear however to be any record of the cumulative x-irradiation administered during the course of a study involving frequent radiographical examination on the same individual. Carrig, Morgan and Pool (1975) made the criticism that "information gained from studies on laboratory animals of the effects of irradiation on growth cartilage cannot be strictly applied to other species".

Inanition and general illness

Normal growth of the skeleton can only proceed under conditions of adequate nutrition. Harris (1933) after starving puppies for only three days observed transverse lines in the metaphyses visible on radiographs. Harris discovered these lines were due to alterations in chondrocytic proliferation and the production of an increased zone of provisional calcification. Collins (1966) cites Park (1954) as noting similar changes in the metaphyses of children although he considered nutritional disturbance of at least ten days duration was required to produce a radiologically visible growth arrest line.

Follis (1956) has also described the histological changes in growth cartilage following dietary restriction. Both Kelly and Reynolds (1947) and Schneider and Adas (1964) have reviewed the literature on the growth plate changes resulting from different forms of nutritional deficit.

Stress and generalised illness can also have a deleterious effect on the growth plate and can result in growth arrest lines in the metaphyses. Schneider and Adas (1964) as a result of experiments done by them in rabbits, concluded that metabolic disturbance due to systemic disease was more important than reduced dietary intake in producing growth cartilage change.

Little (1973) stated that stress produces the same effect on the growth plate as the administration of cortisone.

Mannhart (1970) and Shaw and Lacey (1975) amongst others have studied the effect of cortisone on the growth plate in the rat and rabbit respectively. Both studies demonstrated that administration of cortisone produces narrowing of the growth plate and cellular changes as described by Little (1973). Shaw and Lacey (1975) comment that even after small doses of cortisone these changes develop very rapidly.

The Sex Hormones

Pryor (1923) refers to the original statment he made (1905) that - "the bones of the female ossify in advance of the male" and that "this is measured at first by days, then months, then years". This conclusion was based on studies of the time of development and union of secondary centres of ossification in man. A number of other investigators have confirmed Pryor's findings in human material, Davies & Parsons (1927); Peterson (1929); Todd (1930a); Flecker (1942); Bagnall, Harris & Jones (1978); although Todd (1931) sheds some doubt on its truth in late adolescence. Investigations in animals have proved more equivocal.

Although reports exist(Morscher 1968) claiming that female rats are more advanced than males throughout the growth period, Park (1970) found that differences in growth rates were within the variability of genetic and environmental sources. Green & Fekete (1933) found no significant growth rate differences between male and female mice and Zuck (1938) reported that sex differences in the pattern of epiphyseal closure in the guinea pig are negligible. Todd & Todd (1938) failed to find sex differences of real significance in ungulates. Smith (1956)

however, in his study of sheep foetuses, noted that when the size of two foetuses was the same, the sex opposite and there was a difference in the number of ossification centres then it was often the female that had more centres. Rea, Carpenter, Smith & Hoke (1970) noted earlier skeletal maturation in female lambs.

Rajtova (1974) on studying ossification in the goat and sheep considered sexual dimorphism to be more marked in the goat than in the sheep. The males usually had longer and wider bones and some growth plates appeared to close later in the males than in the females. In the dog Smith & Allsock (1960) noted variations in closure times due to 'sex, breeding, and management' which however was not pursued further. Smith (1978) stated that in the dog there was possibly an indication of slightly earlier union in the female. In the cat Smith (1969) noted no obvious sex differences in epiphyseal fusion but commented on a distinctly greater longitudinal growth rate in the male cat.

The hormonal environment appears to be the principal physiological regulator of the growth plate (Rang 1969). Both Growth Hormone and Thyroid Hormone are known to be important. It has been suggested that during growth plate closure hormone action probably produces an arrest of cartilage proliferation with the result that bone formation overtakes it to seal off the growth plate. Invading bone however will not enter uncalcified cartilage and thus Rang (1969) reasoned there must be some transformation

in the cartilage matrix. Both Rang (1969) and Sissons (1971) consider that adrenal androgens are concerned with epiphyseal closure in both the male and the female.

Simpson, Marx, Becks and Evans (1944) have shown that testosterone has a stimulating effect on the growth cartilage. Gardner & Pfeiffer (1943) found that administration of large amounts of oestrogen retards bone growth. It is currently believed that oestrogens result in a decline in growth plate activity.

Silberberg & Silberberg (1971) in reviewing the literature comment that epiphyseal closure follows essentially a genetic pattern but that in adolescent boys, epiphyseal closure and skeletal age in general are more closely related to sexual development and to the levels of urinary 17 ketosteroids than to chronological age. According to these authors the sex differences in bone length have been attributed to the inhibition of growth by oestrogen rather than to growth stimulation by testosterone.

Morscher (1968) stated that there are differences between the growth plate in males and females. Whereas the zone of hypertrophic cells is broader in males and the vacuolisation of these cells is more marked, calcification of cartilaginous ground substance is more pronounced in females. During puberty Morscher reported a qualitative and quantitative difference in the calcification of cartilaginous matrix in female animals.

Methods and Materials

Ten crossbreed lambs were purchased at the age of 10 - 11 weeks and were used to provide information on both the manner and age of closure of the epiphyseal cartilage plates.

All the lambs were housed on straw bedding and fed a ration of concentrates (Ewbol Pencils 302*), adlib hay and water.

All lambs were from Finn Dorset ewes and at the farm of origin a number of different tups were running simultaneously with the ewes. Five tups were used - a Suffolk, a Dorset Down, a Texel, a Clun Forest and a Dorset Horn.

Soon after purchase two lambs (Nos 8 and 10) developed respiratory disease and died. These two lambs were replaced by numbers 23 and 28. Number 28 was from a batch of lambs purchased two months later.

The sex, dates of birth, birth weights and further details of these lambs is given in table 2.

TABLE 2

Experimental Number	Ear Tag Number	Sex	Date of Birth	Birth weight	From Ewe Number	Number of Lambs born to Ewe in that pregnancy	Twins
1	TG 035	F	16.3.76	6lbs	12	3	Twins
2	TG 036	M	16.3.76	6lbs	12	3	
3	TG 040	F	18.3.76	6lbs	17	2	Twins
4	TG 041	M	18.3.76	6lbs	17	2	
5	TG 042	F	20.3.76	3lbs	52	3	-
6	TG 045	F	20.3.76	6lbs	18	2	Twins
7	TG 046	F	20.3.76	8lbs	18	2	
9	TG 078	M	24.3.76	7lbs	47	2	-
23	TG 076	F	24.3.76	8lbs	50	1	-
28	164-24	F	30.5.76	8.3lbs	58	2	-

*BOCM - Silcock.

Lambs 4 and 9 were entire males even though castration was reported to have been performed at the farm of origin. Lamb 2 was a monorchid male.

Seven to fourteen days were allowed for acclimatisation following purchase. The first radiographs were taken at the age of twelve weeks. Thereafter radiographs were taken at monthly intervals until the lambs were twenty months of age. From twenty to thirty months of age radiographs were taken at two monthly intervals. The radiographs that were taken were:-

1) A ventro-dorsal projection of hips and pelvis.

2) A lateral projection of one stifle joint.

3) A lateral projection of one hock joint.

4) An antero-posterior (dorso-plantar) projection of both metatarsae.

5) A lateral projection of one elbow joint.

6) A lateral projection of one carpal joint.

Such radiographs provided information on all the growth plates of the pelvic limb except for the phalanges and those of the pectoral limb between the distal humerus and carpus.

By necessity the hips and pelvis received an exposure of X-irradiation at monthly intervals. The other projections however were performed on a random basis with no preference being given to radiographing either the right or left limb. Thus in the case of growth plates of the pelvic limb distal to the proximal femur, and those of the pectoral limb it was unlikely that an individual growth plate received exposures every month.

The exposures used both initially and at the termination of this study are given in table 3 . The exposures had to be increased gradually at approximately two monthly intervals.

TABLE 3

	HIP		STIFLE		HOCK		METATARSAE		ELBOW		CARPUS	
	KV	mAS	KV	mAS	KV	mAS	KV	mAS	KV	mAS	KV	mAS
Initially	76	20	61	16	61	12	52	8	52	12	52	8
Finally	76	37	73	20	73	17	67	14	67	17	67	14

A siemens Rubin High Definition Cassette (24 - 30 cms) was used with films taken of both the hock and stifle joints. For other radiographs standard cassettes were used, but with the hip radiographs a Lucidex Radial Grid was interposed between the sheep and the cassette. Kodak XH-1, X-Omat film was used for all the radiographs taken. A Philips Mobile DX-10 unit was utilised and the films were processed by means of a Williamson RP 90 sec. film processor.

In particular two problems were encountered in obtaining adequate radiographs of the pelvic skeleton. Firstly the density of wool in the region resulted in loss of detail in radiographs. Secondly, towards the end of this study, the long exposure times required to obtain radiographs of the pelvis resulted in an increased risk of obtaining a blurred film from movement of the subject. To avoid these difficulties the lambs were clipped free of wool on three occasions during the course of this study, thus improving the quality of radiographs directly and also by removing foreign material trapped in the fleece.

The literature reveals that in any study of the chronology of epiphyseal closure it is essential to define accurately the criteria that have been used in assessing union. On examination of the radiographs obtained in this study it was decided to attempt to record both the initial and final stage of union. The initial stage of union was regarded as loss of the radiolucent line, which

represents the growth cartilage, over at least one third of its width with apparent confluence of epiphyseal and metaphyseal trabeculae. The stage of final or complete union was considered to be when there was complete continuity of the bone cortex outline with no radiolucent defect at the site of the original growth plate.

An attempt was made to estimate the total radiation absorbed by the lambs in the course of this investigation. Radiographic monitor badge films were exposed to a number of different exposure settings of the Mobile DX-10 unit. The films were then tested by the National Radiological Protection Board for the dose equivalent in mrems.

	KV	mAS	
Monitor Film 1)	70	..... 25)	
	+70	..... 20)	i.e. film exposed to the cumulative effect of three exposure settings
	+70	..... 15)	
Monitor Film 2)	70	..... 17)	i.e. film exposed to the cumulative effect of two exposure settings
	+70	..... 14)	
Monitor Film 3)	70	 100	
Monitor Film 4)	65	 100	
Monitor Film 5)	70	 25	

In order to make some assessment of the growth pattern of the lambs used in this study, measurements were made of the interacetabular distance on pelvic radiographs. The measurements were made between the articular surfaces of the acetabuli. In cases where poor positioning of the subject resulted in a slightly oblique

pelvic radiograph, no measurement was made. Some of the pelvic limb radiographs taken of a different group of lambs (see part B of this study) included the pelvis and permitted further interacetabular measurements to be made. These lambs were of the same farm of origin and parentage as those used for studying normal epiphyseal closure.

For legal reasons, it was required to immerse the lambs in an antiparasitic sheep dip on two occasions during the course of this study.

A collection was made of sheep femora (30 specimens) and tibiae (50 specimens) as they became available. The bones were dissected free of soft tissue and the bones 'boiled out' in a routine manner. These sheep were however of unknown breed and age. In a large percentage of these bones skeletal maturation was not complete and thus observations could be made on the partially closed growth plates.

Results

All lambs adjusted well to the husbandry regime employed. It was noted however that the lambs were generally reluctant to eat the concentrate portion of their ration when a new batch of feed was introduced. Although the concentrates were the same throughout the experimental period (i.e. same manufacturer and same product) the lambs were obviously able to discriminate between a fresh batch and the one they had become accustomed to. New batches of concentrates were purchased at approximately two weekly intervals. As the lambs were trough fed it is impossible to state whether some more than others failed to eat their concentrate ration. The reluctance to eat concentrates was of approximately three days duration after which normal consumption was resumed. As hay and water were available on an ad libitum basis it is unlikely that any lamb subjected itself to inanition of any significant degree.

There was no gross indication of systemic disease in any of the lambs in which monthly radiographs were taken. Occasionally some lambs were noted to have bouts of coughing which may have been attributable to dusty hay or some other environmental factor. In no particular lamb was coughing noted over a protracted period, and no other clinical evidence of respiratory disease, a known problem in housed sheep, was found nor was there evidence of pulmonary lesions in comparable lambs (see part B of this study) at slaughter.

Estimates made of the chronology of epiphyseal union by

TABLE 4

I = Incomplete closure at 30 months

Sex	Experimental Lamb Number	Distal Humeral Epiphysis B	Distal Humeral Epiphysis C	Lateral Epicondylar Epiphysis (Humerus) B	Lateral Epicondylar Epiphysis (Humerus) C	Medial Epicondylar Epiphysis (Humerus) B	Medial Epicondylar Epiphysis (Humerus) C	Proximal Radial Epiphysis B	Proximal Radial Epiphysis C	Proximal Ulnar Epiphysis B	Proximal Ulnar Epiphysis C	Distal Radial Epiphysis B	Distal Radial Epiphysis C	Distal Ulnar Epiphysis B	Distal Ulnar Epiphysis C	Proximal Femoral (Femoral Head) Epiphysis B	Proximal Femoral (Femoral Head) Epiphysis C	Femoral Greater Trochanteric Epiphysis B	Femoral Greater Trochanteric Epiphysis C	Femoral Lesser Trochanteric Epiphysis B	Femoral Lesser Trochanteric Epiphysis C	Distal Femoral Epiphysis B	Distal Femoral Epiphysis C	Proximal Tibial Epiphysis B	Proximal Tibial Epiphysis C	Tibial Tuberosity Union to Proximal Tibial Epiphysis B	Tibial Tuberosity Union to Proximal Tibial Epiphysis C	Complete Union of Tibial Tuberosity to Tibial Crest B	Complete Union of Tibial Tuberosity to Tibial Crest C	Distal Tibial Epiphysis B	Distal Tibial Epiphysis C	Fibular Tarsal Epiphysis (os calcis) B	Fibular Tarsal Epiphysis (os calcis) C	Metatarsal Epiphysis B	Metatarsal Epiphysis C
Female	1	-	3M	5M	7M	5M	9M	-	3M	13M	22-24M	19M	20-22M	20-22M	24-26M	16M	23M	17M	18M	13M	18M	20M	20-22M	20-22M	24-26M	14M	20M	24-26M	I	16M	17M	15M	16M	16M	17M
Male	2	-	3M	6M	9M	5M	9M	-	3M	14M	22-24M	20M	24-26M	20M	I	18M	20M	20-22M	19M	14M	17M	16M	20M	22-24M	28-30M	16M	20M	28-30M	I	16M	19M	16M	18M	13M	19M
Female	3	3M	4M	5M	7M	5M	8M	-	3M	12M	20-22M	19M	20-22M	20M	24-26M	18M	20M	20-22M	17M	14M	16M	15M	18M	20-22M	22-24M	20M	24-26M	24-26M	30M	14M	17M	13M	14M	14M	17M
Male	4	4M	5M	7M	7M	7M	10M	3M	4M	13M	22-24M	20M	I	18M	I	18M	20-22M	18M	18M	14M	18M	15M	20M	20-22M	24-26M	18M	20M	20-22M	I	19M	20M	15M	17M	15M	20M
Female	5	3M	4M	5M	7M	5M	7M	-	3M	13M	20-22M	20M	20-22M	19M	24-26M	18M	19M	17M	18M	14M	17M	14M	20M	20-22M	24-26M	18M	24-26M	24-26M	I	13M	17M	13M	16M	16M	17M
Female	6	3M	4M	5M	8M	5M	8M	-	3M	12M	20-22M	20M	20-22M	20-22M	22-24M	23M	20-22M	17M	18M	13M	19M	13M	20M	20-22M	22-24M	12M	24-26M	24-26M	I	13M	14M	13M	14M	13M	15M
Female	7	3M	4M	4M	6M	4M	7M	-	3M	12M	19M	20M	20-22M	20-22M	24-26M	18M	20M	18M	18M	13M	18M	15M	20M	20-22M	22-24M	12M	24-26M	24-26M	I	13M	16M	13M	14M	14M	15M
Male	9	3M	4M	5M	9M	5M	10M	-	3M	13M	20-22M	19M	20-22M	20-22M	24-26M	18M	20M	17M	18M	13M	18M	14M	20M	20-22M	24-26M	12M	24-26M	24-26M	28-30M	14M	18M	13M	14M	14M	17M
Female	23	3M	5M	5M	7M	5M	10M	-	3M	13M	22-24M	19M	22-24M	20-22M	24-26M	18M	20M	17M	18M	13M	16M	15M	20M	20-22M	24-26M	12M	24-26M	24-26M	I	14M	17M	15M	17M	16M	17M
Female	28	3M	6M	5M	10M	7M	10M	-	3M	13M	22-24M	20M	22-24M	20-22M	24-26M	20M	20M	17M	18M	13M	18M	18M	20M	20-22M	22-24M	12M	22-24M	24-26M	I	15M	17M	15M	17M	16M	17M

means of the above mentioned criteria are presented in Table 4.

Estimation of cumulative irradiation

The results for dose equivalents of the exposed monitor films are presented in table 5.

TABLE 5

Monitor Film Number	Exposure		Dose Equivalent (mrems)
	KV	mAS	
Monitor Film 1)	70	25	150 mrem
	+70	20	
	+70	15	
Monitor Film 2)	70	17	110 mrem
	+70	14	
Monitor Film 3)	70	100	350 mrem
Monitor Film 4)	65	100	220 mrem
Monitor Film 5)	70	25	90 mrem

No linear relationship exists between the exposures used and the dose equivalents measured on the radiographic monitor films (table 5). A total possible absorption by each subject for the duration of this study can however be estimated. It can be inferred that the maximum possible dose equivalent for each lamb at each radiographical examination is of the order of 300 - 500 mrems. Each joint however is unlikely to have absorbed even one half of this amount but assuming that the maximum possible absorption occurred the lambs would only have acquired between

6000 and 10000 mrems over the entire duration of this investigation.

Growth Pattern

A 'growth curve' has been plotted (fig 1) from measurements of the interacetabular distances at different postnatal ages. No differences were found between growth curves obtained for male and female lambs.

Manner of Epiphyseal Fusion

For the purposes of description it is convenient to distinguish between those epiphyses subjected principally to traction forces and those subjected to pressure forces.

1) TRACTION EPIPHYSES

A) Pectoral Limb Three traction epiphyses were observable on the radiographic plates. These were the proximal ulna or olecranon process epiphysis and the epiphyses of the medial and lateral epicondyles of the distal humerus.

i) Proximal ulnar epiphysis - Initially the proximal ulnar growth plate was of approximately equal thickness across its width. In a few cases however there was an indication that the growth plate was slightly thicker anteriorly (Plate 2A) Prior to the commencement of closure marked asymmetry of the growth cartilage became apparent (Plate 2B) with marked narrowing anteriorly. This resulted in a wedge or triangular appearance to the proximal ulnar growth plate on lateral radiographs with the widest part of the wedge situated posteriorly. Closure occurred first anteriorly. As closure progressed the apex of

the triangle became attenuated and further removed from the anterior cortex and the base became correspondingly less wide (Plate 2B). The majority of the growth plate had closed by 16-18 months but a cartilage remnant persisted on the posterior aspect till 22-24 months when an intact posterior cortex denoted complete closure (Table 4).

ii) Medial and Lateral Epicondylar Epiphyses - Both of these epiphyses closed in a similar manner to each other and to that described for the proximal ulnar epiphysis. Superimposition of images on lateral radiographs made interpretation slightly difficult.

In both epiphyses, a wedge shaped growth plate was observed with the base directed posteriorly. Closure occurred in an anteroposterior direction, with the posterior cortex possessing a cartilage remnant for a short time after closure of the majority of the epiphysis. Closure was however not as protracted as with the proximal ulnar growth plate.

B) Pelvic Limb Four traction epiphyses were examined, the epiphyses of the greater and lesser trochanter of the femur, the tibial tuberosity and the epiphysis of the os calcis (or fibular tarsal bone).

i) The Greater and Lesser Trochanteric Epiphyses - On ventrodorsal radiographs of the pelvis in young lambs it was relatively easy to

discern both these growth plates (Plate 3) but as closure commenced increasing difficulty was encountered. The nature of attachment of the greater trochanteric epiphysis results in difficulty in judging the initial stages of union. The positioning of the hindlimb was critical if the lesser trochanter was to be visualised in profile. The times given (Table 3) for commencement of closure of these epiphyses can only be considered approximations. In the case of the greater trochanteric growth plate a radiolucent line was usually evident superimposed on the proximal femoral cortex (Plate 3) when closure was incomplete. Disappearance of this line was considered to indicate complete closure. During closure of the lesser trochanter a mottled density was seen at the site of the growth plate. At complete union the lesser trochanter was of fairly even density.

Examination of bone specimens however demonstrated that initially the greater trochanteric epiphysis fuses with the adjacent dorsum of the femoral capital epiphysis. Shortly afterwards the greater trochanteric epiphysis fuses posteromedially, in the region of the trochanteric fossa, and to a lesser extent anteriorly (Plate 4). A defect however along the distal, lateral, line of attachment persists for some time after fusion of the body of the epiphysis (Plate 4). In many bone specimens a marked depression was noted at this site.

The lesser trochanter was incompletely fused in only one

bone specimen. In this case although the anterior aspect was fused there was a pronounced defect posteriorly.

ii) Tibial Tuberosity - Two phases were noted in the fusion of the tibial tuberosity to the adjacent proximal tibia. Initially the epiphysis fused with the proximal tibial epiphysis and then a more protracted union occurred with the tibial crest. On lateral radiographs of the stifle joint both stages of union involved the appearance initially of a wedge-shaped growth cartilage. (Plates 5,A,B & C). The protracted final stage of union may account for the relatively large number of collected bone specimens which exhibited a cortical defect at this site (Plate 6). Sectioning of bone specimens revealed that this is not a surface defect but represents as yet incomplete union of the epiphysis (Plate 7). Following complete union a fairly obvious striated appearance was seen to be present at this site and extending distally over the anteromedial and anterolateral aspects of the tibial crest (Plate 8).

III) Fibular Tarsal Epiphysis - Radiographic examination of this epiphysis was made difficult by its 'cap-like' form. It was found to be relatively easy to judge when the growth plate was completely open and fused, but more difficult to assess the commencement of fusion. In some radiographs there was an indication that the growth plate was wider posteriorly but this finding was not consistent.

2/ PRESSURE EPIPHYSES

A) Pectoral Limb

i) Distal Humeral Epiphysis - In all but one lamb fusion of the epiphysis had started or was starting at the commencement of this study. In those cases which were starting to fuse the most anterior aspect of the growth plate was found to be still open above the condyles. Superimposition of images made it difficult to discern whether the growth plate above either the medial or lateral humeral condyle closed first.

Complete fusion occurred within a short period of time (Table 4)

ii) Proximal Radial Epiphysis - In all lambs except one the proximal radial epiphysis had fused at the commencement of this study. In lamb no. 4 however the epiphysis was in the process of closing at the first radiographic examination. In this case there was noted to be a pronounced cleft, or radiolucent defect on the anterior aspect of the radius although the remainder of the growth plate had fused.

iii) Distal Radial Epiphysis - A slight posterior peaking of the distal radial epiphysis and metaphysis was noted (Plate 9) In some lambs at the time of closure the anterior aspect of the growth plate appeared to close first but in others no clear distinction between fusion of the anterior and posterior aspects became apparent.

iv) Distal Ulnar Epiphysis - The distal ulnar epiphysis fused slightly later than the distal radial epiphysis (Table 4) for a considerable time prior to closure commencing there was

noted to be marked thinning of the growth cartilage of the distal ulnar epiphysis. No apparent asymmetrical manner of closure was detected.

B) Pelvic Limb

i) Capital Femoral Epiphysis - Despite superimposition of the acetabulum on images of this growth plate, radiographs were surprisingly easily interpreted see plate 3. Fusion of the epiphysis was initially with the adjacent greater trochanter (Plate 5)

ii) Distal Femoral Epiphysis - Radiographic examination of this epiphysis suggested that there was a more protracted closure of the supratrochlear aspect of the growth plate. In two lambs the posterolateral aspect of the growth plate was open despite continuity of cortical outline above the medial condyle.

Bone specimens confirmed this observation as in a number of cases a pronounced epiphyseal line existed all around the growth plate except for the posteromedial quadrant (Plate 10). On sectioning such bone specimens however is was found that union had occurred across most of the growth plate width excepting a minor, unfused 'defect' laterally (Plate 11). In younger specimens a lesser defect was also detected medially but the lateral unfused area was always more extensive. The bone specimens confirmed that the supratrochlear region tends to maintain its epiphyseal line longer but even in mature bones a slight depression exists at the junction between the trochlea and anterior metaphysis .

iii) Proximal Tibial Epiphysis - Only a slight asymmetrical pattern of fusion of the proximal tibial epiphysis and metaphysis was noted. The growth plate below the weight bearing area of the tibial condyles appeared to be first to close and in a few radiographs the posterolateral aspect was open although the posteromedial aspect was completely fused.

Bone specimens however revealed that the epiphyseal line initially disappears anteriorly where the proximal tibial epiphysis fused with the tibial tuberosity and metaphysis. Following complete fusion of the proximal tibial epiphysis a 'heaped-up', ridge-like appearance of cortical bone marked the site of the epiphyseal line medially. In some cases a faint depression marked the position of the epiphyseal line laterally.

Bone specimens further revealed that even in cases with an obvious epiphyseal line present the lateral popliteal notch area was usually completely fused.

iv) Distal Tibial Epiphysis - On lateral radiographs the articulation between the lateral malleolus and the distal tibia gives rise to a confusing radiolucent line in this region. Antero-posterior radiographs of the distal tibial epiphysis were present in films taken of the metatarsac. Such radiographs gave an indication of a slightly greater depth of growth cartilage medially in some young individuals.

In some cases a cartilage defect appeared to persist at the lateral periphery for a short time following epiphyseal union.

In all bone specimens examined the distal tibial epiphysis was completely fused. A number of bones however showed a 'heaped-up' ridge-like appearance of cortical bone above the medial malleolus at the site of the distal tibial growth plate.

v) Distal Metatarsal Epiphysis - There can be medial peaking, (of both the metaphysis and epiphysis) of varying degree at the site of this growth plate (Plates 12 A, B & C). Anteroposterior radiographs of the metatarsal growth plate are somewhat obscured by the presence of the volar sesamoids.

In some cases a lateral cartilage defect as noted in some distal tibiae was observed.

Epiphyseal Scars

A) Traction Epiphyses - No obvious scars were seen following epiphyseal union of the medial and lateral epicondyles. During the process of union a certain degree of radiodensity was seen at the proximal ulnar site but no distinct scar was noted.

The traction epiphyses in the pelvic limb also did not show distinct epiphyseal scars. In the case of the greater trochanter little could be concluded from radiographs due to superimposition of its cortices in the projection chosen, (see plates 3 and 13).

Specimens which were known to be fusing or to have recently fused also failed to reveal a distinct scar (plate 13). The lesser trochanter left no obvious scar following union, nor did the tibial tuberosity. The fibular tarsal bone however was more difficult to interpret on radiographs as surface cortical irregularities in the non-articulating extremity resulted in lines of increased bone density that could be confused with scars. It was deduced, on examination of radiographs of bone specimens, that an epiphyseal scar does not normally occur at this site.

B) Pressure Epiphyses - The pressure epiphyses however showed more pronounced and persistent lines of increased density at sites of fusion.

In the pectoral limb both the distal humerus and proximal radius had pronounced scars which however only persisted for a few months following closure. The distal radial epiphysis also possessed an epiphyseal scar which in most but not all cases was less distinct or absent anteriorly (plate 14). A faint scar was considered to be present at the distal ulnar epiphysis.

In the pelvic limb a scar was present following fusion of the capital femoral epiphysis which appeared to disappear after a few months. The distal femur and proximal tibia also possessed scars. Bone specimens were sectioned in a dorsal (or-frontal) plane in such a manner as to leave the central region of the distal femur and proximal tibia intact. Thus

confusing shadows caused by the femoral trochlea and posterior femoral condyles and the tibial tuberosity, tibial crest and posterior tibial condyles were removed. Anteroposterior radiographs revealed that both in the distal femur and proximal tibia the epiphyseal scar tends to be slightly more prominent medially (Plates 15 and 16)

Epiphyseal scars were also observed at the distal tibial and distal metatarsal sites but they were transitory, disappearing a few months after their appearance. There was a tendency for the epiphyseal scar to be less apparent or absent laterally in the distal tibiae. This same feature was seen in a few metatarsi although less distinctly due to the overlying volar sesamoids.

Discussion

Use of interacetabular distance measurements to produce a growth curve in this study is open to some criticism. Sexual dimorphism in the bony pelvis has been reported by Lovell (1965) and Lowrance (1968) in the rabbit and by Bernstein and Crelin (1965) in the rat. Both Lovell and Lowrance found a significantly greater interacetabular distance in female rabbits. There is however no known report of this being true in the sheep. Close scrutiny of the measurements obtained in this study revealed no such sex difference in lambs. Although only three of the ten lambs radiographed were male, the growth curves for these individuals fell within the range of those obtained for females.

Payton (1931) observed that in pigs the rate of growth diminished with advancement of age and Bisgard and Bisgard (1935) confirmed this observation in goats. Bisgard and Bisgard (1935) found that the rate of growth of the goat ulna remained constant during the first four months and then progressed at half its former rate. Growth curves obtained of the feline tibia (Smith 1968) and canine femur (Riser1973) are of a similar shape to fig 1 A similar growth pattern was discovered by Maresh (1943) for growth of a number of long bones in children. After 10 years of age in man however the adolescent growth spurt confuses this pattern.

In the growth curve obtained in this study (fig 1) the most rapid increase in interacetabular distance occurs in the initial period. It seems likely that had it been possible to

conduct this study from birth a similar pattern to that reported by Bisgard and Bisgard (1935) in goats would emerge. It can however be deduced from fig 1 that the period from 3 to 5 months is one of rapid growth in the lambs studied.

It is perhaps interesting that no noticeable sex differences have been detected in the growth curves. Park (1970) aptly comments however that "within the plethora of variations stemming from genetic and environmental sources it is difficult to regard only one specific change as being due to difference in sex".

Genetic factors undoubtedly are of importance in this study as a number of rams were running with the mothers of the lambs at the time of their conception. With advancement of age an ever increasing phenotypic variation was apparent in the lambs and such variation may well account for individual variation in skeletal growth rate and could equally mask any sex differences that were present. The small number of lambs studied and in particular the few males involved makes it impossible to state categorically that there are no growth differences between sexes.

Other environmental factors that could be interpreted as influencing skeletal growth became apparent but are of doubtful significance.

Although reluctance to feed on new consignments of concentrates was noted it would appear that this has been of little consequence. It seems unlikely that there would have been absolute inanition when the concentrates were refused and the duration of this condition was insufficient to greatly affect body weight. Schneider

and Adas (1964) reported that quite a marked weight loss was required to produce changes in the growth cartilage. Harris (1933) however obtained growth arrestment lines in the metaphyses of puppies after only three days of starvation. No lines of growth arrest were detected in the lambs studied. Park (1954) considers that at least 10 days duration of inanition is required to produce growth arrest lines in children.

With trough feeding it is impossible to know what proportion of its ration each lamb receives. Those consuming the greatest amount of feed would be expected to have growth curves of slightly greater magnitudes than other individuals. The results of such individual variation in the amount of concentrates consumed would be likely to produce greater divergence of the growth curves with advancement of age. There is an indication that this may occur in fig 1 but such divergence could equally be the result of genetic variation. The hierarchial behaviour of lambs in groups is well known.

Although there was no evidence of systemic disease in the ten lambs studied it is known that they were all at least exposed to respiratory disease at the farm of origin and at the commencement of this investigation. Subclinical disease remains an unknown factor which may have influenced

skeletal growth.

Apart from the possibility of subclinical disease, a number of other factors are known to have imposed stress on the lambs during the course of this study. Radiography itself and the necessary restraint imposed on lambs during positioning are likely to have been stressful. Other procedures such as dipping and wool removal were also performed. Although all lambs were subjected to these insults, it is not possible to state that all lambs react equally to such stimuli. Such incidents are unlikely to have influenced skeletal growth.

Both skeletal growth and growth plate closure could have been influenced by the frequent irradiation of the skeleton that occurred. Growth arrest did not occur and would indeed have been an unlikely outcome with the doses of irradiation employed in this study.

As the rem is the unit of dose equivalent which is equal to the absorbed dose multiplied by the quality factor it is possible to calculate the total possible absorbed dose. Vaughan (1971) states that the quality factor for x-irradiation is close to unity and thus a dose equivalent of 6-10 rems is equivalent to an absorbed dose of 6-10 rads. However, as mentioned above each joint is unlikely to have received even one half of the delivered total dose especially as accurate centering over each joint was always employed. Thus the total absorbed dose by individual bone extremities over the duration of this study is likely to have been of the order of 1-2 rads. A slightly

greater absorbed dose possibly occurred in hip joint radiographs due to the higher exposures used, but scatter from increased tissue density would have effectively reduced the dose absorbed by the skeleton.

The vast majority of reports in the literature containing quantitative studies of the effects of x-irradiation on the growth plate have involved use of the units of dose called roentgens.

The roentgen is a unit of exposure and cannot be used to denote the amount of an irradiation dose that is absorbed. Limitations thus exist in conclusions made from the results of quantitative experiments utilizing roentgens as the units of dose.

The advantage of obtaining dose equivalents for different exposure settings, as in this study, is that a better estimate of the actual dose absorption by the skeleton is obtained. Although loss of radiation resulting from scatter within the subject is not accounted for irradiation losses from the source to the subject are not producing a false impression of the absorbed dose.

It can be deduced that the radiographic procedure used in this investigation is unlikely to have caused growth impairment. Even if the dose absorbed was sufficient to cause minimal chondrocytic damage no noticeable impairment of growth would be expected (Kember 1967).

This conclusion is however based on the assumption that the ovine skeleton reacts similarly to that of laboratory animals in which most irradiation studies have been performed. The criticism of Carrig, Morgan and Pool (1975) that"the results of laboratory animal studies on the effects of irradiation on growth cartilage cannot be strictly applied to other species" seems valid. For example, a difference is recorded in the skin erythema dose in man and rabbits (Bisgard and Hunt 1936).

The criteria that have been used to identify complete growth plate closure are identical to those described by Smith (1955; 1956) and the results obtained in this study are similar to those reported by him. It would be improbable that the results of these two studies, albeit using the same criteria would be identical. The husbandry of the sheep in each study is different (not housed in Smith's study) as is their breed, and it is relevent that all Smith's observations above the age of 10 months were in females. The phenotypic differences between individuals in this study has been noted. In addition the individual and genetic variation that has been discussed above would equally apply to the process of growth plate closure and predictably produce variation. Smith (1956) reports a degree of individual variation in skeletal maturation in the Clun Forest flock studied by him. It can be seen however (table 4) that variation in the chronology of closure between the lambs studied is not particularly marked.

There is an indication (table 4) that with a number of epiphyses, fusion in the female antedates that in the male. The small number of lambs studied, the fact that not all epiphyses conform and in particular the small number of males involved, prevents a definite statement being made with regard to sex differences and skeletal maturation.

Todd (1930a) extols the virtues of close scrutiny of anatomical specimens in conjunction with radiographs in studies of epiphyseal union. This author considered data gathered purely from radiographs to be "almost worthless". The limitations of purely radiographic methods of studying growth plate closure have been more recently suggested by the observations of Jelavic (1974). Jelavic found that in rats which showed a radiographically closed growth plate, growth cartilage could be detected histologically.

It certainly seems that the most fruitful study of epiphyseal union would result from a radiographical study together with both gross anatomical and serial histological examinations of the same material. The lack of such studies has hampered progress as has the inadequate definition by authors of the criteria they have used in assessing closure.

When using the presence of an uninterrupted bone cortex as a criterion of complete closure the radiographic projection used is of considerable importance. Smith (1956) comments that in his study of epiphyseal fusion in sheep at least two projections of each region under investigation were attempted but that this was not always possible.

During the course of the investigation it became apparent that it is technically difficult to obtain certain radiographical projections in conscious sheep. Lambs tend to object to forced extension of limbs into either full protraction or full retraction. Thus, although observations on the manner of closure of the distal humeral epiphysis for example would have been augmented by an anteroposterior radiograph, such a projection was difficult to obtain.

It is evident from inspection of bone specimens that the epiphyseal lines, which persist on one aspect of the growth plate only, could easily be missed on radiographs. Thus fusion could be judged to be complete although using total cortical integrity as a criterion this is strictly speaking not true. It seems that radiographical studies of epiphyseal union necessitate adequate descriptions of the projection used. This has rarely been done. Detection of cartilage remnants is thus largely dependant upon the radiographical projection chosen. Of possible significance is the "Mach Effect" which can result in difficulties in radiographical interpretation. From the account of this phenomenon by Grandage (1976) it would seem that in general the Mach Effect would aid in detection of cartilage remnants at the periphery of growth plates but that in cases of bone superimposition confusion may arise.

Generally the transverse plate type of growth plate are relatively easily studied whereas those involving complex indentations and projections are considerably more difficult

to interpret. With broad epiphyses, the multiplicity of cancellous bone shadows in the region of the growth plate makes estimation of the beginning of fusion hazardous. Thus estimates of the commencement of fusion at especially the distal femoral and proximal tibial sites can only be regarded as estimates.

Similarly with caplike epiphyses, such as that of the fibular tarsal bone, estimates of the beginning of fusion are of doubtful accuracy. A different problem was encountered in the case of the distal ulnar growth plate where considerable narrowing occurred over a period of time prior to closure. The two processes of extreme narrowing and apparent closure were so close chronologically that the beginning of fusion was difficult to estimate.

In the case of the proximal ulnar growth plate a posterior cortical defect or cartilage remnant persisted for a considerable period of time gradually diminishing in size. Although the term "cartilage remnant" has been adopted from Smith's descriptions of closure (1968:1969) it has not been confirmed in this study that such defects do in fact contain cartilage.

For the reasons discussed above, in a number of epiphyses accurate estimation of the commencement of fusion from radiographs is difficult. Such epiphyses include the distal ulnar, proximal femoral, greater trochanteric, lesser trochanteric, distal femoral and proximal tibial epiphyses. In this study, observation of

the distal tibial epiphysis was greatly aided by two projections being available for examination. Undoubtedly, more information would have been gained in this study had at least two projections of each bone extremity been utilised.

Smith (1956) provides some comments as to when fusion commenced in different epiphyses of sheep. The individual variation encountered by him however makes interpretation of his results slightly difficult. He considered fusion to commence in the proximal ulnar epiphysis at approximately 7 months. In this study a more conservative approach has perhaps been employed as table 4 demonstrates that the first signs of fusion of the epiphysis was judged to be 12 months. The apparent crossing of the growth plate by a few cancellous trabeculae has not been considered to mark the advent of closure as slight alterations in positioning could easily produce such an appearance. A definite area of confluent cancellous density, between the epiphysis and metaphysis, over approximately one third of the growth plate width was judged as the commencement of closure on radiographs.

There is undoubtedly variation in the confidence with which observations and interpretations are made from radiographs by different investigators. This in itself will result in wide variations in estimates of the process of epiphyseal fusion.

Other epiphyses were judged to be fusing or to have fused at slightly later dates than those given by Smith (1956). Such epiphyses include those of the lateral epicondyle, proximal

femur and trochanters and in most individuals the distal tibia. Despite husbandry, genotypic and interpretation variations between this study and that of Smith (1955, 1956) the results are more comparable with each other than with any other documented study. This perhaps reflects the fact that the same criteria were used in each study for judging closure complete.

Smith (1955;1956) made observations approximately every two months. The inference from this study however is that at least monthly radiographical examinations should be made in order to lessen the experimental errors that are inherent in a study of this nature. Some epiphyses, such as the proximal radial and distal humeral appeared to fuse with marked rapidity.

The traction epiphyses appear to have an almost characteristic manner of closure which is especially evident in the simple transverse growth plates.

The aspect of the growth plate subjected to tensile forces tends to be thicker and have a more protracted closure. Conversely the aspect under relative compression tends to become narrowed and fuse earlier. This feature is especially evident in the olecranon and tibial tuberosity but was also observable in the humeral epicondyles. The greater trochanter also appears to conform and although only one bone specimen aided observations made on the lesser trochanter this epiphysis also appears to

have a tensile aspect posteriorly. In all of these epiphyses the attachment and action of muscles would provide the traction required to produce tension over an aspect of the growth plate.

Parsons (1904) remarked that traction epiphyses 'synostose' at that point where they are pulled most strongly against the neighbouring bone. A similar manner of closure of the proximal ulnar growth plate is reported in the horse by Myers and Emmerson (1966). Smith and Allcock (1960) and Rajtova (1974) produced tracings of radiographs of the olecranon in the dog and sheep respectively. These tracings however do not demonstrate a widened growth plate posteriorly or earlier fusion anteriorly.

Smith and Allcock's (1960) tracings do however demonstrate a more protracted closure to the anterior aspect of the tibial tuberosity in the dog. In the dog tracings they present as in the sheep in this study, the tibial tuberosity fused initially with the proximal tibial epiphysis. Jeffreys (1965) reports that in man also the tibial tuberosity fuses initially with the proximal tibial epiphysis.

In the present study of ovine epiphyseal fusion the only traction epiphysis examined which does not conform to the generalised pattern described is that of the fibular tarsal bone. Unfortunately bone specimens did not aid in the understanding of this epiphysis. Although the cap like nature of this epiphysis made radiographical interpretation difficult an indication of a slight increase in posterior width of the growth plate was observed in a few lambs. A posterior cortical defect can

be seen however in the radiographic tracings of the fusing fibular tarsal epiphysis in the dog presented by Smith and Allcock (1960).

Lewis (1958) found that the tibial tuberosity growth plate in man consists anteriorly not of hyaline cartilage but of coarse bundles of collagen fibres. Smith (1962a) and Ogden (1974) have confirmed this observation and Smith (1962a) suggested the term 'fibrous epiphyseal plate' for the growth plate at this site. In this publication Smith (1962a) cites other investigators who have described the same structure in the tibia of the rat and rabbit. Smith (1962b) found that in the ox also the anterior aspect of the tibial tuberosity growth plate was of a fibrous rather than a cartilaginous nature and discovered similar fibrous epiphyseal plates in the distal greater trochanteric growth plate and between the ilium and ischium in the ox. Recently Badi (1978) performed an autoradiographic study of the fibrous growth plate of the rat tibia.

By means of photoelastic studies on bone models Smith (1962 a and b) estimated the directions of principal compressive and tensile stresses in the regions of the fibrous plates. He demonstrated that each fibrous epiphyseal plate is crossed by strong tensile stresses during activity and that the collagen fibres of each plate and the diaphyseal trabeculae which are formed at it lie parallel to the direction of the prevailing tensile stress. Smith (1962b) further remarked that the new bone added to the diaphysis was of a parallel-fibred, coarsely bundled type and that in each fibrous plate there is a growth zone characterised by proliferation of fibroblasts.

He states that there is no cartilage erosion at the sites of fibrous plates but that it is by the extension of membranous ossification into this zone that the diaphysis at this site increases in length. Salter and Harris (1963) however state that traction epiphyses do not contribute to longitudinal growth of the long bone. This cannot be true in all cases. The fibular tarsal bone for example is equipped with only one growth plate which is associated with a traction epiphysis. Similarly the proximal ulnar growth plate has been ascribed with a contribution to longitudinal growth of the ulna in the goat (Bisgard and Bisgard 1935) and in the dog (Clayton Jones and Vaughan 1970).

In general, the manner of closure of traction epiphyses in this study conforms well with the findings of Smith (1962a and b). The parallel fibred, coarsely bundled bone reputedly produced by fibrous plates is well demonstrated in the sheep tibial crest (Plate 8).

It would be of considerable interest to examine all traction epiphyses histologically to determine whether or not a fibrous component to the growth plate exists. Parsons (1904; 1905) remarked on the similarity between traction epiphyses and sesamoid bones and postulated (1905) that the traction epiphyses were originally sesamoid structures. Parsons (1904) reported that in bats there is no epiphysis at the extremity of the olecranon. Instead the bat is equipped with a sesamoid like bone at this site which is attached to the ulna by fibrous tissue.

If such a fibrous component is present posteriorly at the proximal ulnar site this could account for the protracted closure noted on this aspect. The tibial tuberosity demonstrated a similar protracted closure anteriorly. These two sites however would predictably be subjected to pronounced muscular forces. Other traction epiphyses may be acted upon by muscle forces of lesser magnitude and may not develop a fibrous component. It is interesting, bearing in mind the protracted closure of the anterior aspect of the tibial tuberosity, that May (1964) states that there is generally incomplete ossification at this site in ovine tibiae. It must be assumed that this author has unfortunately examined only tibiae in which the protracted closure was not complete as in a large number of bones examined in this study complete fusion had occurred.

It was noted in this study that the greater trochanteric epiphysis fused initially, along the dorsum of the femoral neck, to the femoral head. Parsons (1904) observed the same to be true in two other mammalian species, the tiger and the hare.

The behaviour of pressure epiphyses is believed to be more constant than that of traction epiphyses (Dawson 1925). It is apparent from this study that many pressure epiphyses fuse asymmetrically and that closure does not simply involve central fusion with gradual replacement of cartilage by bone spreading peripherally. This indication that asymmetry of closure occurs was also suggested by examination of bone

specimens in which epiphyseal lines were seen to be present on some aspects of the bone extremity and not on others.

In the pectoral limb, although only one lamb had an incompletely fused proximal radial growth plate at the commencement of this study, it is interesting that fusion was occurring in a postero-anterior direction. Myers and Emmerson (1966) reported that in the horse also the anterior aspect of the proximal radial growth plate is last to close. In the tracings of radiographs given by Smith and Allcock (1960) of the dog and Rajtova (1974) of the sheep, it can be seen that the anterior aspect of this growth plate is last to fuse.

In the distal radial growth plate however there was no clear indication that asymmetry of fusion occurred although there was noted to be moderate peaking of both the distal radial epiphysis and metaphysis posteriorly.

Examination of the antebrachium of lambs revealed that there is a moderately pronounced anterior bowing of both the radial and ulnar diaphyses (Plate 17). Such bowing persists throughout skeletal growth and is present at maturity with no noticeable increase or decrease in its degree. The aetiology of normal bone curvature is discussed elsewhere in this study but it is relevant to state that the noted bowing results in the presence of compressive and tensile stresses developing on different aspects of the radius (Lanyon & Baggott 1976).

Such forces could account for

asymmetrical closure of the proximal radial growth plate in a manner similar to that described for traction epiphyses. The distal radial growth plate however would seem to be anomalous if such a suggestion is true. It is relevent however that no asymmetry of closure of the distal ulnar growth plate was observed although similar stresses would be applied to the distal regions of both bones. The mechanical environment may well be different at this distal site. Mechanical factors influencing growth plate closure are considered in part D of this study.

The proximal femoral growth plate was difficult to investigate radiographically. An equivocal asymmetry of closure was noted on examination of bone specimens and no definitive statements can be made. Goodfellow (1977) has determined the contact areas of the acetabuli and femoral heads in four mammals, but such a study has not been performed in the sheep.

A constant asymmetrical pattern of fusion was however noted in the distal femoral epiphysis. The posteromedial quadrant was the site of initial complete fusion. It appears that at this site the weight bearing areas and thus those subjected to the most marked compressive forces were the first to fuse. In a similar manner it can be suggested that the medial femoral condyle sustains a greater proportion of load bearing than the lateral condyle. The functional importance of the distal femoral growth plate shape has been discussed by Thomson (1902) and Tidestrom (1974). Tidestrom (1974) stated that the four

prominences appearance is typical of the pressure loaded gingylmus growth plate.

The radiograph tracings of Smith and Allcock (1960) in the dog and Rajtova (1974) in the sheep confirm the observation of relatively later closure of the supratrochlear aspect made in this study.

Smith (1962a) considers that in the distal femur of man body weight will tend to bend the shaft forwards and laterally so that the anterior and lateral aspects are subjected to longitudinal tensile stresses while the posterior and medial aspects experience longitudinal compression. This finding has apparently been confirmed by stresscoat studies of human femora by Pederson, Evans and Lissner (1949) (cited by Smith 1962a). Although factors such as differing femoral curvature and torsion will undoubtedly influence such establishment of tensile and compressive stresses, the increased anterior bowing of the ovine femur when compared with that of man would be likely to accentuate this observation. The habitual floxed knee posture of the sheep in addition to marked anterior curvature would tend to result in anterior tensile stresses in the supratrochlear region.

Similarly the proximal tibial growth plate appears to close initially at the load bearing area. The tracings provided by Smith and Allcock (1960) and Rajtova (1974) demonstrate the same finding in the dog and sheep respectively. Examination of bone specimens revealed that the posterolateral area close to the

popliteal notch frequently showed no epiphyseal line despite other aspects of the tibial plateau being unfused. It is possible that action of the popliteus muscle which traverses this area is responsible for this area of fusion. Frankel (1974) has demonstrated that in man by far the largest component of forces that produce a load on the knee joint are from muscle actions the gastrocnemii providing considerable force across the knee joint when contracting. Any more adequate explanation of this area of fusion awaits further experimentation on the mechanics of the ovine stifle joint.

Similar asymmetry in closure was noted in some of the other epiphyses of the pelvic limb that were studied. Thus in some cases the medial aspects of the distal tibial growth plate appeared to fuse slightly in advance of the lateral aspects. An indication was gained of the same being true for some distal metatarsal growth plates.

The inference therefore is that load bearing tends to provide a greater degree of compressive stress down the medial compartment of the pelvic limb skeleton in sheep at the time of skeletal maturation. The finding of a slightly 'heaped up' appearance of the medial growth plate region in both the proximal and distal tibiae following closure may similarly indicate compression. Clark (1971) states that following fusion of an epiphysis a slight heaping up of bone into a low ridge occurs. No mention is made however of whether certain aspects of a bone tend to have this feature.

It has also been inferred that the pattern of fusion in

traction and pressure epiphyses is comparable. It is suggested that compression can promote closure whereas tension results in a retardment of closure.

It is well known that compression retards bone growth whereas tension can result in acceleration of growth. The influence of such factors on the growth plate is discussed in more detail elsewhere in this study.

A pattern also emerged in the appearance and persistence of epiphyseal scars in the bones studied. It is perhaps interesting that no obvious scars were found associated with traction epiphyses. Cope (1920) described the presence of a 'fusion-line' associated with the greater trochanter in man and similarly the illustration given by Clark (1971) of a human proximal femoral radiograph demonstrates a faint scar in this region.

It is possible that the slight transient condensation of trabeculae noted at the time of fusion of the proximal ulna epiphysis in this study could be interpreted as an epiphyseal scar.

The pressure epiphyses however showed more pronounced scars most of which persisted only a few months following union. Of most interest is the fact that the areas of suggested compression were more likely to have associated with them well defined scars which persisted slightly longer. Examples are represented in radiographs taken of sections of the distal femur and proximal tibia (plates 15 & 16)

It is relevent that Cope (1920) commented that in man scars are best seen and most persistent in the bones of the lower extremity where the stress is greatest.

Conclusions

1) Many pitfalls exist in studying epiphyseal union by radiographs alone. The difficulty in adequately standardising the biological material on which such a study is made prevents definitive statements being made. It is suggested that in order to gain meaningful results it would be necessary to at least include serial histological sectioning of the growth plate to any radiographical study.

The wide diversity of reported chronology of closure that is documented in just one species, the sheep, reflects in particular the individuality that exists between researchers in judging union to be complete. Estimates of commencement of closure on the basis of radiographs are especially hazardous.

2) Observations on the manner of closure were possible on the material available in this study. Such observations would have been augmented by including a number of radiographical projections into the experimental design. The availability of bone specimens, albeit of unknown origin, greatly aided this part of the study.

A pattern emmerged in the manner of closure of almost all of the traction epiphyses studied.

A similar though less marked manner of closure has been

suggested in pressure epiphyses.

It is postulated that some indication of the mechanics of the limb can be gained from close scrutiny of the growth plates at the time of their closure. It is possible that epiphyseal scars imprint this information on the bone for a period of time after closure is complete.

PART B

DEFORMITY

"In nature there's no blemish but the mind,
None can be call'd deform'd but the unkind"

Shakespeare - Twelfth Night IV, 403

The antiquity of limb deformities is demonstrated by their depiction in both primitive cave paintings and Egyptian papyri. Swinyard and Pinnet (1969) further report that literature written during the Dark Ages contains numerous records of limb deformities which were frequently explained in terms of divine retribution for transgressions.

Normal bone contour

Before any mention can be made of abnormal bone angulation it is necessary to realise that long bones have characteristic shapes which are relatively constant within a given species. Variation does however exist between species. For example, May (1964) described the variations that exist in femoral shape between the sheep, dog and ox. In particular normal diaphyseal curvature from one extremity to another is of interest in an appraisal of skeletal deformity. Whereas the sheep femur has a gradual diaphyseal anterior curvature, May (1964) reports that in the dog anterior curvature is more pronounced at the junction of the middle and distal thirds of the femoral diaphysis. Walensky (1965) studied anterior femoral curvature in man and found marked racial differences.

A perusal of standard anatomical texts in the sheep (May 1964)

and Man (Warwick and Williams 1973) however reveals a remarkable similarity in the tibial diaphyseal contour in both species. The tibial diaphysis is not straight but follows a slightly sinuous curve in both man and sheep despite the absence of a functional fibula in the ovine skeleton.

The characteristic bowing of the radius and ulna in normal sheep was noted in part A of this study.

It is essential to acknowledge that normal bone curvatures occur before any appraisal of deformity can be performed. The possible aetiologies of such normal curvature will be considered in part D of this study.

Angular Limb Deformity

Limb deformities may be the result of abnormal angulation at the articulations or within the bones that constitute the appendicular skeleton. Angular bone deformity is usually considered to be due to either

I) Fractures
II) Bending within the shaft of the bone.
III) Asymmetrical growth plate activity.

I) Diaphyseal Fractures Diaphyseal fractures are common in both man and animals and a fixed angular deformity may result from malunion. Such deformities are of limited relevence to this investigation but the bone overgrowth that is reported to follow diaphyseal fracture is of some importance. As two lambs sustained femoral fractures during this investigation (one of whom also fractured the tibia of the same limb) an opportunity to assess post fracture overgrowth was presented.

A number of authors including Compere and Adams (1937); Brodin (1955); Wood (1972) and Edvardsen and Syversen (1976) have remarked on the increase in longitudinal bone growth that follows a fracture. Following unilateral tibial fracture in rats Wray and Goodman (1961) observed initially a growth retardment of both the ipselateral and contralateral femora. Following retardation there was noted to be an increased rate of femoral growth which was significantly greater in the fractured limb. Warrell and Taylor (1976) and Taylor and Warrell (1977) similarly reported that following periosteal sectioning of one tibia in the rat an overgrowth of both tibiae occurred. In an investigation of femoral fractures in children, Edvardsen and Syversen (1976) however failed to detect any appreciable overgrowth of the intact femur.

Following proximal ulnar osteotomy in pigs, Beckmann, Rodgerdts and Buddecke (1975) demonstrated increased metabolic activity of the proximal growth cartilage. Unpublished results of one of these authors indicated that osteotomy induced growth stimulation.

It has been suggested that the overgrowth that occurs following long bone fracture is the result of hyperaemia (Bovill 1963; Persson 1967). Yahsley and Harris (1965) however described the vascular effects of periosteal stripping and fracture of rabbit tibiae and suggested that the noted hyperaemia resulted from increased nutritional demands of the thickened growth plate and was not the primary cause of growth stimulation. As a result of experiments in chickens, Crilly (1972) proposed that the sectioning of the periosteum that occurs in displaced fractures

results in 'decompression' of the growth plate. Warrell and Taylor (1976) demonstrate the uncertainty that exists by stating that overgrowth may be the result of decompression or may be due to a vascular effect. Brodin (1955) discovered that following a growth stimulating procedure (periosteal division and elevation) in rabbits there was increased activity of the growth plate furthest away from the operative site with concomitant reduced activity adjacent to the traumatised area. Jenkins, Cherry and Hodgson (1975)amongst others have described the clinical use of periosteal sectioning in limb lengthening procedures.

Diaphyseal fractures can influence bone growth in another manner. Morton and Starr (1964) described premature closure of the anterior portion of the proximal tibial growth plate, with resulting knee deformity, following prolonged immobilisation of tibial shaft fractures in man. A number of reports exist of premature growth plate closure resulting from immobilisation or as a sequel to paralytic conditions such as poliomyelitis. (Bisgard 1933; Sissons 1952; Makin 1965; Bottling and Scarse 1965; Roberts 1970; and Slee 1972). Some authors, including Hulth and Westerborn (1963) have suggested that a relative reduction in metaphyseal capillary circulation produces the growth cartilage changes that follow immobilisation.

I. ii) <u>Has Epiphyseodesis in one end of a long bone a growth stimulating effect on the other end?</u>

Such a question was the title given to an experimental study conducted by Heikel (1961) in rabbits. Proximal tibial

epiphyseodesis produced no appreciable effect on the distal tibial growth plate (Heikel 1961).

A number of years previously Reidy, Lingley, Gall and Barr (1947) found that there was a stimulation of growth at the untreated end of a bone after growth at the other end had been impaired by irradiation. Kritter and Blount (1959) suggested that the trauma which inhibited one growth plate in surgically induced epiphyseodesis probably stimulated the other. This suggestion was based on observations of clinical cases in children.

Experimentally, Hall-craggs (1968;1969) observed growth acceleration at the distal end of rabbit tibiae following epiphyseodesis or stapling of the proximal tibial growth plate. Hall-craggs obtained a similar but less marked response at the proximal tibia following distal epiphyseodesis. Such experiments led Hall-craggs to suggest that there may be a system of growth control intrinsic to a bone which permits a growth plate to compensate for inadequacies of growth by another.

I. iii) Methods of estimating bone growth contribution by growth plates Brief mention has been made in part A of this study of the differing contributions made to longitudinal growth by the growth plates at each end of a long bone. In any investigation of growth plate contribution it is essential to be able to identify a reference point in the bone from which measurements can be made. The most usual method has been to instal metallic markers that can be identified on subsequent radiographs (Bisgard and Bisgard 1935; Sissons 1953; Kritter and Blount 1959; Heikel 1961;

Heinze and Lewis 1968; Hall-craggs 1968;1969). Heikel (1961) cites his earlier work (Heikel 1960b) in which he demonstrated that his technique of incorporating radiopaque markers had no appreciable effect on long bone growth.

An alternative method of estimating the contribution made by growth plates to longitudinal bone growth was described by Digby (1916). Digby reasoned that the site of primary ossification of a long bone diaphysis could be estimated by noting where a line drawn through the nutrient canal meets the long axis of the bone. Objections however can be made to Digby's reasoning. For example, Payton (1934) following observations of the skeleton in madder fed pigs suggested that there was constant remodelling of the nutrient foramen and that it moved independently of diaphyseal growth, thus bearing no relationship to the unequal contribution to growth by each bone end. Also Lutken (1950) described the considerable variation that existed in the position of the nutrient foramen in human femora and humeri that were studied. Hughes (1952) however noted that anomalies of nutrient canal direction, in a large number of mammalian and avian bones examined, occurred particularly in femora and radii. Brookes (1971) has reviewed the theories that have been proposed to explain nutrient artery obliquity and concludes that inequality of rates of longitudinal bone growth is not the only factor involved.

It is interesting that despite the criticms that can be made of Digby's method, Bisgard and Bisgard (1935) confirmed its usefulness in measurements on goat bones. These authors

obtained comparable results following estimates of bone growth by each end of goat long bones by Digby's method and by use of radiopaque markers.

Riser (1973) stated that by identifying the nutrient artery it was possible to measure the growth contribution of each end of a long bone. He used the nutrient foramen as a reference point but details of this method were not provided. Such a technique is perhaps open to even more criticism than that described by Digby (1916).

II. Bending within the shaft of the bone Bending within the shaft of the bone, or bowing, can have a variety of aetiologies and is best documented in the human literature. It is convenient to initially describe the important clinical conditions in man and then consider any comparable conditions that have been described in the domestic species.

II i) Congenital Bowing of the long bones in man In those regions of the skeleton where there are normally two long bones (i.e. shin and antebrachial regions) congenital absence or mal-development of one bone element frequently results in bowing of the remaining bone. A number of reports document such cases including those by Gaenslen (1915), Middleton (1934); Browne (1936); Blockey and Lawrie (1963); Amstutz (1972) and Westin, Sakai and Wood (1967). Coventry and Johnson (1952) and Turek (1967) comment that in man the fibula is the long bone most frequently found absent at birth and a search of the literature certainly reveals that in the neonate bowing is most commonly encountered in the tibia (Caffey 1947; Angle 1954; Durbin 1971; Baldwin and Weiner 1974). Even so, Durbin (1971) states that

congenital angular deformities are rare in the human skeleton.

Not all tibial deformities are the result of fibular lesions, and a number of reports exist of tibial bowing in which the fibula is intact. Congenital bowing of the tibia may result from generalised skeletal disease such as osteogenesis imperfecta congenita or be of unknown aetiology.

Badgley, O'Connor and Kudner (1952) have suggested the term kyphoscoliotic tibia for tibial bowing in which no aetiology is apparent. These authors justify this term by stating that the majority of cases of tibial bowing have a concomitant rotational deformity.

A condition known as congenital pseudarthrosis of the tibia also exists in children and has been described in many reports including those by Beals and Fraser (1971) Wellwood, Bulner and Graff (1971); Hardinge (1972); Andersen (1976); Newell and Durbin (1976) and Pompe van Meerdervoort (1978). A number of authors including Badgley, O'Connor and Kudner (1952), Murray and Lovell (1974) and Rathgeb, Ramsey and Cowell (1974) have described the prefracture appearance of bowed tibiae which proceed to pseudarthrosis. It is thought that congenital pseudarthrosis results from a developmental abnormality of the primary centre of ossification. Bain and Barrett (1971) consider that a generalised cartilage dysplasia is responsible for congenital bowing of long bones. Cases of pseudarthrosis may present with concomitant neurofibromatosis or fibrous dysplasia

but in others no evidence of disease is found. Hardinge (1972) also described bowing and fracture of the tibia in older children in which the tibia had previously been normal. Congenital pseudarthrosis of the fibula has also been described in man (Dooley, Menelaus and Paterson 1974, 1975).

In human cases of tibial bowing, the affected bone diaphysis becomes thickened with marked increase in cortical thickness on the concave aspect of the deformity. Not all cases progress to pseudarthrosis, Caffey (1947) described regression of the degree of bowing in elements of both the pelvic and pectoral limb. Similarly Amstutz (1972) reported that in 38 cases of congenital absence of the fibula 29 cases had tibial bowing which tended to diminish with age. Caffey (1947) remarked on 'metaphyseal spurs' which were especially evident medially in the knee joint metaphyses of two cases of tibial bowing described by him. He remarked on the similarity in radiographic appearance of these metaphyses and those encountered in rachitic bow legs and in cases of tibia vara.

Numerous other congenital defects can result in long bone bowing in human infants. Such conditions are copiously described in a number of texts and will only be named. Such conditions include osteogenesis imperfecta congenita, dyschondroplasia, multiple exostoses, Morquio-Brailsford disease, comptomelic dwarfism and achondroplasia. Ponseti (1970) reported that in children achondroplasia results in more severe tibial shortening and thus the fibula is relatively longer and undergoes bowing.

Deformity is also reported to occur in some cases of epiphyseal dysplasia (Dysplasia Epiphysealis Multiplex, Dysplasia Epiphysealis, Hemimelia and Dysplasia Epiphysealis Punctata)

II. ii) Congenital Bowing of the long bones in animals No reports of a condition similar to tibial pseudarthrosis have been discovered in the veterinary literature. Chesney and Hanlon (1971) however report a condition of proximal femoral pseudarthrosis in an infant rhesus monkey. These authors considered their findings to represent a case of coxofemoral pseudarthrosis but were unable to state whether it was of traumatic or congenital origin. Heinze (1966) however does state that angular bone deformities in the horse are often congenital. The majority of angular deformities that occur in horses limbs would appear to be the result of nutritional inadequacies (Adams 1974).

Gruneberg (1963) has described a number of conditions that result in true bone deformities in chickens and a variety of laboratory animals. Such conditions include congenital absence of the ulna with consequent radial bowing. Both Grüneberg (1963) and Hutt (1964) describe Ancon sheep which are endowed with short, crooked limbs due to an inherited chondrodystrophy.

Of considerable interest is a condition of dwarfism that occurs in the Alaskan Malamute. Sande, Alexander and Padgett (1974) described the radiographic features of this disease and reviewed the documented literature. This condition is known to be genetically transferred and results in lateral bowing of the forelimbs and a valgus foot deformity. Longitudinal growth of the ulna is retarded

relative to that of the radius which results in the abnormal bone curvature.

II. iii) Nutritional factors related to long bone bowing

Deformities of bone resulting from nutritional imbalances are of considerable importance and are commonly encountered in both man and animals. Their recognition however has resulted in a reduction in both the incidence and severity of such conditions.

Rickets classically produces limb deformity and the pathogenesis of disease is similar in man and animals. In infantile rickets a failure of provisional calcification occurs resulting in an increased depth of the growth plate. The hypertrophic cells form irregular clumps rather than columns and some of these clumps eventually become distorted due to weight bearing. Osteoid is deposited subperiosteally in the bone diaphysis but calcification is incomplete and bending of such diaphyses may occur (Jubb and Kennedy 1970). In man, if tibial bowing occurs, both limbs are symmetrically affected (Turek 1967; Apsley 1968; Fourman and Royer 1968; Paterson 1974). With bending of bones, the cortex on the concave side becomes thickened and metaphyseal spurs or flares are normally found. Similar metaphyseal changes are reported in animals by authors such as Riser (1964) and Campbell (1968). The lack of metaphyseal vascular invasion that occurs in rickets has been described in experimental rats by Trueta and Buhr (1963) and Harris (1968).

Jubb and Kennedy (1970) consider that rickets is rare in horses although some cases probably do occur. Adams (1974)

has suggested that rickets may be important in the production of both medial and anterior deviations of the carpus in foals. In piglets and young dogs and cats however severe rickets is more frequently seen. Rickets in calves is reported to result from phosphorus and vitamin D deficiency and bowing at the carpus and pastern may be evident (Jubb and Kennedy 1970; Greenough MacCallum and Weaver 1972). In lambs rickets may result from vitamin D deficiency although on phosphorus deficient terrain sheep are seldom considered to develop clinical osteodystrophy (Jubb and Kennedy 1970).

The most severe deformities in man occur in cases of refractory rickets (vitamin resistant rickets).

Other mineral imbalances are known to be important sources of skeletal abnormalities in the domestic species. Manganese deficiency can result in bone deformities in the bovine skeleton (Greenough, MacCallum and Weaver 1972). In some cases zinc deficiency results in bowing of the bovine hindlimb (Blackman, Miller and Morton 1967).

Jubb and Kennedy (1970) describe other osteodystrophic diseases of undetermined cause in sheep and goats which are reported to result in angular bone deformity. In New Zealand a condition called 'Bowie' affects suckling lambs and is endemic in certain areas. Limb deformity is especially apparent in the forelimbs and can result in either a valgus or varus foot. The latter is more common. The deformity is due to changes in the distal radius and proximal metacarpus. Provisional calcification is reported to be normal and osteoid is not present in excess, bones

being of normal density and strength. The defect appears to be in the process of invasion and erosion of the calcified cartilage which results in an increased growth cartilage depth. In some ways this condition is therefore similar to rickets.

Metaphyseal collapse and impaction occurs either medially or laterally probably within the zone of calcified cartilage. Such collapse is thought to produce the gross deformity. Phosphate fertiliser applied to pastures apparently reduces the incidence of this condition (Jubb and Kennedy 1970).

II iv) Other factors Other factors can produce bowing or bending of bones. Borden (1974) has recognised a condition of traumatic bowing of previously normal forearm bones in children. Such bowing is considered to result from plastic deformation of the bone following trauma. Tschantz and Rutishauser (1967) amongst others have demonstrated, experimentally, the elastic and plastic responses of the normal dog ulna in vivo. No clinical report of traumatic bowing has been discovered in the veterinary literature.

In the more mature human skeleton apart from osteomalacia, diseases such as osteogenesis imperfecta tarda, fibrous dysplasia and Paget's disease can result in bowing of the long bones.

III Asymmetric growth plate activity

i) Fractures involving the growth plate - Some physical characteristics of the growth plate The possible influences of diaphyseal fractures on bone growth have been described above. By far the most common cause of uneven or asymmetrical bone

growth is that of fractures involving the growth cartilage.

As early as 1919 Haas noted that trauma was the most frequent cause of disturbance of growth of bones. The growth cartilage is regarded as being the weakest structure in a long bone. Harsha (1957) cites Hutchinson (1894) and Fairbanks (1951) who stated that the strength of the fibrous capsule is from 2-5 times that of the growth plate. Aitken and Ingersoll (1956); Morscher (1968) and Chung, Batterman and Brighton (1976) have remarked on the importance of soft tissues in maintaining the integrity of the growth plate. Ollier (1867) is cited (by Morscher 1968) as having stated that the periosteum increases the mechanical resistance of the growth cartilage. More recently Huller and Nathan (1970) however obtained inconclusive findings on the contribution of the periosteum to bone strength. It is stated that many authors believe that it is possible to disrupt the epiphysis completely from its metaphysis and yet not displace it due to the stabilising effect on the growth plate of the surrounding soft tissues (Harsha 1957). Both Morscher (1968) in rats and Chung, Batterman and Brighton (1976) in children, report an increase in the mechanical strength of the growth plate with increase in age. Morscher (1968) further demonstrated that immediately before and after the beginning of sexual maturation in rats the increase in tensile strength of the growth plate slowed down and in males even a decrease in qualitative strength occurred. Only after the completion of sexual maturation did the tensile strength increase again.

At sexual maturation the growth plates of female rats were considerably stronger than those of males. The influence

of hormones on the mechanical strength of growth cartilage is further suggested by castration studies in male rats which prevented the observed decrease in mechanical strength (Morscher, Desaulles and Schenk 1965). Bright, Burstein and Elmore (1974) and Bright, Elmore and Burstein (1974) found a similar sex difference in the mechanical strength of rat growth plates. Salter and Harris (1963) remark that growth plate injuries are more common in boys than girls and ascribe this difference to physical activity. These authors also comment that in children growth plate injuries are slightly more frequent in periods of rapid growth.

The mechanical weakness of the growth plate is further suggested by the statement of Rogers (1970) that "the epiphyseal separation of the young has been properly identified as the analogue of the dislocation or ligamentous injury of the adult".

b) The fracture line Certain diseases such as scurvy, rickets, osteomyelitis and endocrine imbalance make the growth cartilage more vulnerable and predispose to fracture.

Haas (1917) observed that with experimental epiphyseal separations the fracture line invariably passed through the hypertrophied cartilage zone. A number of authors consider this to be the usual manner of fracture in clinical cases (Abbott and Gill 1942; Salter and Harris 1963; Laud and Babulkar 1970). It has however been suggested that the fracture line normally involves a number of the zones of the growth cartilage (Morscher 1968; Johnston 1975) and indeed Bright and Elmore (1968) found that certain zones of the cartilage

were more susceptible to certain types of force than others. Brashear (1959) similarly suggested that the line of cleavage through the growth cartilage is dependent on the forces imposed.

Not all growth plate injuries are associated with growth disturbance (Gregory 1976). The important sequelae to fracture involve damage to the resting or germinative zone of the growth cartilage with the establishment of areas of fusion between the epiphysis and metaphysis (Ford and Key 1956; Cassidy 1958; Harris 1972; Oh, Craig and Banks 1974). Such premature fusion may result in either deficiency of bone length or if confined to one aspect of the growth plate, asymmetrical growth and deformity. In man premature closure of the distal femoral growth plate in particular can result in severe leg length discrepency and produce a cosmetic defect in addition to disability (Stephens and Louis 1974; Lombardo 1975; Lombardo and Harvey 1977). In animals however, long bone shortening is of lesser importance as provided the deficiency is not too great, extension of the appropriate joint conceals the defect.

c) Fractures involving the growth plate in animals In the dog in particular, complete or partial premature closure resulting from trauma to one of the antebrachial growth plates produces a not uncommon clinical deformity of the unaffected bone. The magnitude of the deformity is dependent upon the growth potential remaining in that bone undergoing abnormal curvature.

In the pectoral limb of the dog premature closure of the distal radial growth plate over its entire area is rare and produces marked shortening although angular deformity may or

may not develop (O'Brien 1971). Newton, Nunamaker and Dickenson (1975) report quite marked valgus deformity developing in such cases. O'Brien, Morgan and Suter (1971) noted that following premature closure of the distal radial growth plate, the proximal growth plate remained open but the trabecular pattern and width of the spongiosa suggested that diminished bone growth was occurring. These authors also described a loss of the normal anterior radial curvature in these cases.

Partial closure of the distal radial growth plate produces angular deformity which is most commonly valgus (O'Brien 1971; Newton, Nunamaker and Dickinson 1975).

In the canine pectoral limb the distal ulnar growth plate is the most commonly injured (Clayton Jones and Vaughan 1970, O'Brien, Morgan and Suter 1971). The results of premature closure of the distal ulnar growth plate have been frequently reported (Hanlon 1962; Riser and Shirer 1965; O'Brien, Morgan and Suter 1971; O'Brien 1971; Rudy 1971; Lau 1977) and in summary produce varying degrees of ulnar shortening and valgus deformity of the distal limb due to continued radial growth. Bowing of the radius can be marked. The cortical bone of the distal radius is of increased thickness on the concave aspect. Distal radial epiphyseal separation as a result of severe radial bowing has been reported (O'Brien, Morgan and Suter 1971). Secondary changes occur in both the elbow and carpal joints.

Carrig and Morgan (1975) produced growth retardation of the distal ulnar growth plate experimentally in dogs and observed the radiographic changes. Although premature closure of the

distal ulnar growth plate in the dog is invariably of traumatic origin, it is of interest that Lau (1977) reports this condition in 23 related Skye Terriers. Other aetiological factors apart from trauma have been suggested (Rudy 1971).

In the dog, premature closure of both the distal radial and distal ulnar growth plates results in antebrachial shortening and lateral bowing of both diaphyses with resulting valgus (O'Brien, Morgan and Suter 1971).

Premature closure of the proximal radial growth plate in the dog has been reported by Clayton Jones and Vaughan (1970) and by O'Brien, Morgan and Suter (1971). Minimal change in radial shape occurs.

That irreparable damage can occur without gross displacement of the epiphysis being apparent has been demonstrated by a number of authors (Riser and Shirer 1965; O'Brien, Morgan and Suter 1971). Epiphyseodesis and deformity can occur despite a normal radiographic appearance of the bone extremity.

Premature closure of the distal fibular growth plate in the dog is also documented (O'Brien 1971) Asymmetrical premature closure of either the medial or lateral aspects of the distal tibial growth plate can occur in the dog with subsequent deformity developing (O'Brien 1971; Rudy 1971;). Similarly asymmetrical premature closure of the proximal tibial growth plate produces deformity (O'Brien 1971). A compression injury of the distal femoral growth plate has been suggested as possibly being the cause of some stifle valgus deformities in large breeds of dog (O'Brien 1971).

III d) Fractures involving the growth plate in man Whereas the distal ulnar growth plate is most frequently affected in the dog, Eleason and Ferguson (1934) (cited by O'Brien, Morgan and Suter 1971) report that in children the distal radial growth plate is most commonly injured. In man trauma can similarly result in either complete or partial growth plate closure and thus produce bone length deficiency or deformity (Siegling 1937; Abbot and Gill 1942; Hohl and Luck 1956; Cassidy 1958; Jackson and Cozen 1971; Salter and Best 1972; Ryoppy 1972; Marti 1974; Langenskiold 1975; Hamada, Smith and Stryker 1976; Lombardo and Harvey 1977; Barrett, Nohme and Riseborough 1978).

The seriousness of the outcome of some growth cartilage fractures has led to classifications being made according to the direction of the fracture line and the likely prognosis. Perhaps the most frequently quoted classification is that of Salter and Harris (1963) which has also been adapted for use in veterinary orthopaedics (O'Brien 1971). According to this classification two types of fracture in particular are considered to produce growth disturbances. Recently however Lombardo and Harvey (1977) have questioned the usefulness of such classifications and Ireland (1933) suggested that the prognosis is more dependent on the growth plate involved. Wegner (1974) has shown that the tensile strength of the growth plate cartilage in pigs varies with its anatomical site. Matthieson (1957) noted that in man, following growth plate injury, bony bridges tended to be formed at certain favoured sites.

III e) Experimental studies of growth plate injury As early as 1877, Vogt (cited by Haas 1919) investigated bone growth following injury to growth plates in lambs and goats and Haas (1919) performed extensive studies of a similar nature in dogs and cats. More recently similar experiments on sequelae of traumata to growth plates in a number of species have been performed (Imbert 1951; Ford and Key 1956; Key and Ford 1958; Campbell, Grisolia and Zancorab 1959; Bright, Burstein & Elmore 1974). As a result of Ford and Key's investigations it was discovered marked deformity followed damage to the periphery of the growth plate and Campbell and co-workers noted that the degree of bone retardation was roughly proportional to the size of the cancellous bone bridge that formed. Campbell, Grisolia and Zancorab (1959) also commented that if the bone bridge was relatively small normal longitudinal bone growth occurred.

III f) Other factors resulting in growth plate injury Apart from trauma similar deformities can result from other conditions affecting the growth plate. For example in man Makin (1965) demonstrated that in the majority of poliomyelitis cases shortening of the fibula exceeds that of the tibia. Valgus tibial deformity results with associated knee and ankle valgus. Similarly Ansell (1972) reports that in Stills disease (juvenile rheumatoid arthritis) premature growth plate closure of the shin or antebrachial bones can result in deformities due to different growth rates between the paired bones. Stills disease can also produce stimulation of growth cartilage (Turek 1967).

Infection adjacent to the growth plate can influence its activity. Roberts (1970) described deformity and shortening of a long bone in man due to osteomyelitis and Turek (1967) reported similar changes in metaphyseal tuberculosis infection and congenital syphilis. Neoplasms may also result in premature growth plate closure (Burdelow, Cracco and Book 1975) and limb deformities have been induced following incomplete surgical epiphyseodesis in clinical cases (Regan and Chatterton 1946).

III ii) Asymmetrical growth without premature closure Bone deformity can also occur due to asymmetrical growth plate activity where no bone bridges have developed. Such asymmetrical growth is a source of a number of important clinical conditions in both man and animals.

a) Ankle valgus in man Just as congenital absence of the fibula in man results in tibial growth deformity, so with fibular fractures, valgus deformity at the ankle may result. Similarly deformity may follow surgical resection of the fibula either for removal of a localised lesion or for use as a bone graft in for example subtalar arthrodesis. It is reported that in man the lower third of the fibula is less likely to regenerate than the middle third and that the potential for fibular regeneration decreases with age (Hsu, Yau, O'Brien and Hodgson 1972).

Reports of such ankle valgus in man have been made by Langenskiold (1967); Wiltse (1968; 1972;) Hsu, Yau, O'Brien and Hodgson (1972) and Hsu, O'Brien, Yau and Hodgson (1974).

The distal tibial growth plate becomes wedge shaped with narrowing of the plate laterally. In addition there is a

progressive elevation of the lateral malleolus. Most authors agree that the lack of normal thrust supplied by the growing fibula laterally probably impedes growth on that aspect. Arrestment, but not correction, of the deformity can be achieved by fusing the distal metaphysis of the fibula and tibia (Langenskiold 1967). Lack of lateral support resulting from proximal migration of the lateral malleolus may cause an abnormal stress to be placed on the lateral aspect of the distal tibial growth plate with a resulting growth retardment (Hsu, Yau, O'Brien and Hodgson 1972). In some cases hypertrophy of the lateral distal tibial cortex is present (Hsu, Yau, O'Brien and Hodgson 1972).

Makin (1965) described that in man, initially, the distal tibia and fibula make the major contribution to bone length but that after a period of time there is a reversal with the proximal growth plates providing the major portion of longitudinal growth. Wiltse (1972) and Hsu, Yau, O'Brien and Hodgson (1972) have suggested that such reversal of growth plate activity may account for their noting that valgus deformity is particularly likely to occur following fibular defects in the young child. The proximal fibular growth plate produces the majority of fibular growth after the bone has reached approximately one third of its adult length. Thus for much of its growth the fibula is dependent upon an intact proximal growth plate which maintains the position of the lateral malleolus by means of an intact fibular diaphysis. These authors reason that loss of fibular integrity results in loss of this stabilising force on the lateral malleolus.

b) Genu valgum (knock knees) In man genu valgum is a disorder which usually starts during childhood and may be the result of rickets, fractures or rarely other conditions (MacEwen and Dunbar 1958). In addition genu valgum is commonly the result of unequal growth of the femoral condyles. In affected children the medial condyles grow more rapidly than their lateral counterparts. A number of authors have described an initial varoid phase, followed by a valgoid phase which may occur during normal pelvic limb development in children (Bohm 1933; Blount 1941; Price 1949; Knight 1954; Sherman 1960). The valgoid phase usually begins at about 2 years of age and reaches a maximum when the child is $4\frac{1}{2}$ years old. It is between these ages that genu valgum is most commonly seen (Price 1949; Morley 1957). Because of this observation cases with mild genu valgum have been considered to exhibit physiological knock knee (MacEwan and Dunbar 1958; Bateson 1966). In the majority of cases spontaneous correction of deformity occurs with growth and advancement of age (Sharrard 1974; Pistevos and Duckworth 1977). MacEwan and Dunbar (1958) in a limited series of children found that genu valgum was always bilateral but sometimes more marked in one limb than in the other.

Some cases do not spontaneously correct and are referred to as cases of true genu valgum (Bateson 1966). In such cases there is peaking of the lateral femoral and tibial cortices. True genu valgum deformities occur despite integrity of the growth plates and are considered to be exaggerated cases of physiological knock knee. There may be a racial factor

involved, as severe genu valgum is reported as being uncommon in British children (Bateson 1966). In severe cases the distal femur and proximal tibia are rotated outwards, by the action of biceps femoris and tensor fascia lata, and the distal tibia undergoes inward or medial torsion. There is a general contraction of soft tissue elements of the lateral compartment of the limb and elongation of those of the medial compartment. The medial collateral ligament becomes stretched and joint instability results. In severe cases pes valgus is frequently present (Price 1949; Wiles and Sweetham 1965; Turek 1967). It has been suggested that valgus deformity is secondary to ligament laxity (Salter 1970).

Riser, Parkes, Rhodes and Shirer (1969) have described genu valgum in giant breeds of dog. These authors stress that the majority of cases in the dog are only mild and barely detectable clinically or on radiographs. The salient clinical signs in the dog are described in detail by Riser et al (1969). Hypodevelopment of the lateral femoral metaphysis and epiphysis was detected and the deformity was especially apparent in the distal femur and proximal tibia. Cavities were described on the medial and lateral aspects of the femoral condyles and an abnormal degree of coxa valga was considered to be present. In histological sections the growth plate was found to be thicker on the lateral side of the proximal tibia. Rudy (1971) reports that slight femoral head anteversion occurs in such cases which may result from or cause the valgus deformity at the stifle

joint. Lateral patellar luxation and stifle joint instability are also described (Rudy 1971).

c) Genu Varum (Bow Legs) It has already been stated that the human tibia has a sinuous curve to its diaphysis. Wiles and Sweetham (1965) remark on the slightly curved contour of the medial aspect of the human tibia prior to explaining how apparent bow legs may occur. These authors state that both internal rotation of the limb (from femoral retroversion) and obliquity of the distal tibial epiphysis can result in apparent bow legs.

As with genu valgum, rickets was a common cause of bowleg but in recent times the incidence of rachitic deformities is very much reduced. Fractures and other conditions can result in genu varum.

Genu varum can also occur as a developmental abnormality associated with the varoid phase of limb growth. The varoid phase that has been identified precedes the valgoid phase and predictably cases of bow legs tend to present slightly earlier than those with knock knees. As with genu valgum, bowing of the legs is considered to be physiological in many instances with spontaneous correction being the usual outcome (Bateson 1966).

Severe bow legs involves both the tibia and the femur and there is an associated internal or medial tibial torsional deformity. Some cases are reported to only involve the tibia (Turek 1967). The torsional deformity that occurs results in an intoed posture of the feet. Cases of severe bow legs are uncommon in British children.

Cases of genu varum have been described by Blount (1941); Forrest (1949); Holt, Latourette and Watson (1954) and Christie and Stempfel (1955). On radiographs there is seen to be metaphyseal peaking of the distal femur and proximal tibia and increased cortical thickness of both bones medially.

d) Tibia Vara (synonyms - Blounts disease, Osteochondrosis, Deformans Tibiae)

This condition was first described by Erlacher (1922) and although uncommon in Britain has been encountered in a number of other countries. The primary lesion is in the proximal tibia. Some confusion exists in the literature due to authors (e.g. Barber 1939, 1942; Leonard, Exeter and Cohen 1946) referring to cases of severe physiological bow legs as examples of tibia vara.

Tibia vara was studied extensively by Blount (1937, 1941) who introduced the term 'osteochondrosis deformans tibiae' and distinguished between infantile and adolescent forms of the disease. The infantile group developed severe bowleg after normal development at about two years of age. There was a preponderance of female cases in the survey performed by Blount (1937) and frequently the children were overweight. In the infantile form of Blounts disease a marked varus deformity of the proximal tibial metaphysis immediately below the epiphysis occurs. In addition widening and irregularity of the proximal tibial growth plate medially, and hypodevelopment of the medial tibial condyle are recorded. Most importantly, the medial projection or peak of the proximal tibial metaphysis

is seen to contain areas of radiolucency on radiographs. Some lateral subluxation of the distal femoral condyles relative to the tibial condyles occurs. It is always the posteromedial aspect of the proximal tibia that is affected (Golding and McNeil-Smith 1963) and in 50% of infantile cases the lesions are bilateral.

The adolescent form is less common than the infantile disease (Kessel 1970). In the adolescent group reported by Blount (1937) the age of onset was between 6 and 12 years in previously normal children. The adolescent type is characterised by irregular narrowing of the medial aspect of the proximal tibial growth plate with marked varus deformity occurring immediately below the proximal tibial epiphysis. Some authors state that in the adolescent form of tibia vara the epiphysis is not deformed and spontaneous regression may occur (Langenskiold 1952; Langenskiold and Riska 1964). Blount (1937) however reported that individuals can demonstrate the infantile appearance and subsequently the adolescent type of the disease.

The radiographic findings in adolescent tibia vara suggest retarded growth plate activity medially. Sometimes obliteration of the medial growth plate is seen on radiographs (Turek 1967). Adolescent tibia vara is reported to be a unilateral affection (Turek 1967). Internal or medial tibial torsion also occurs in the adolescent condition.

It is concluded that tibia vara is a progression of severe physiological bow leg (Blount 1942; Bateson 1966; 1968) although

it is characterised by a local disturbance in growth of the proximal-medial portion of the tibia (Gailey 1956). Langenskiold and Riska (1964) described the development of tibia vara in three cases that previously had radiographically typical changes of physiological bow leg.

Lateral form of Blounts Disease Bateson (1966) reports both early and advanced cases of lateral Blount's disease in Jamaican children. Bateson, in the same article, explains the progression from true genu valgum deformity through to true tibia valga. Unlike tibia vara, tibia valga does not appear to be associated with subluxation between the distal femur and proximal tibia. In Bateson's early case of tibia valga a small defect was noted in the lateral part of the proximal tibial metaphysis which corresponded exactly with the type of defect seen in the medial proximal tibial metaphysis in cases of tibia vara.

Genu vara in the dog In the toy breeds of dog varus deformity of the distal femur is associated with medial tibial rotation and medial patellar luxation may result. The deformity in the femoral diaphysis is situated near to its distal epiphysis. De Angelis (1971) considers the important anatomical features of cases of medial patellar luxation to include coxa vara, varus deformity of the distal third of the femur and valgus deformity of the proximal tibia. Such changes result in a bowlegged or genuvara appearance. Congenitally bowed femora in association with coxa vara also occurs in children (Amstutz and Freiberger 1962).

Stader (1944) (cited by Kodituwakku 1962) has described

the tendency of some small breeds of dog to be bowlegged in their pelvic limbs.

There is also considered to be medial tibial torsional deformity in cases that suffer congenital medial patellar luxation.

Distal Tibial deformity in the dog Angular limb deformity may result from asymmetrical growth of the distal tibia in the dog. Rudy (1971) stated that angular deformity of the distal tibial shaft is usually unrelated to other deformities of the limb and results from an uneven rate of growth of the distal tibia. This author however considers that eccentric epiphyseodesis is the most likely explanation of such deformities.

The Distal Tibial Epiphysis in Man Wiles and Sweetham (1965) consider that obliquity of the distal tibial epiphysis is an occasional cause of pes valgus in man. These authors state that sometimes rickets is the cause of defective epiphyseal growth but that sometimes no cause can be found. They suggest that in those cases with no apparent aetiology, there is a temporary defect in growth similar to that in genu valgum. Obliquity of the distal tibial epiphysis can result in apparent bowing of the tibia. This defect in the distal epiphysis usually corrects itself spontaneously during growth but on rare occasions obliquity is severe and requires surgical correction (Wiles and Sweetham 1965).

e) Madelung's Deformity in Man This condition was described by Madelung as a spontaneous luxation backwards of the ulna (Perkins 1961). Madelungs deformity is an uncommon condition

and is characterised by a bony prominence, the distal end of the ulna, being present over the back of the carpus (Shands, Raney and Brashear 1967). The distal extremity of the radius is considered to be the primary seat of the disturbance (Kelikian 1974). The ulnar border of the radial shaft is much shorter than its radial margin which is thought to be due to a disturbance in the distal radial growth plate. Kelikian (1974) explains that the distal radial epiphysis is normally thinner on its ulnar than on its radial sector. The volar portion of the epiphysis is also thinner than the dorsal half in normal development . In Madelungs deformity, the ulnar and volar sectors of the distal radial epiphysis are reduced to flattened out slivers and may not even be in evidence. It is this classical description of the epiphyseal change associated with this deformity that has led authors such as Rang (1969) to remark on its similarity to infantile tibia vara.

Madelungs deformity is more common in girls than in boys (Salter 1970; Kelikian 1974) and because it is a deformity associated with growth tends to be seen during the periods of rapid growth in adolescence, (Shands, Raney and Brashear 1967; Aegerter and Kirkpatrick 1975). As a result of asymmetric growth failure at the distal radial growth plate bowing and shortening of the distal radius occurs. The hand and carpus is carried forward with the radius leaving the ulna, in which growth proceeds unimpeded, dorsally.

Madelungs deformity is often bilateral in which cases the deformity is usually symmetrical. A proportion of cases have a hereditary background which appears to be autosomal dominant, but no large surveys are available. There may also be a racial factor, as the condition is more common in some countries than in others (Wynne-Davies 1973). Madelungs deformity may be found in association with other conditions and has been reported in cases of genu varum (Aegerter and Kirkpatrick 1975) and Turners syndrome (Henry and Thorburn 1967). Kosowicz (1960) described medial tibial metaphyseal lesions in a series of cases of Turners syndrome.

Some cases of Madelungs deformity may be associated with trauma or bone dysplasia but in many instances the condition is idiopathic (Henry and Thorburn 1967).

f) Madelungs Deformity in Animals It appears that at least two cases documented in dogs have been erroneously termed Madelungs Deformity. Salerno (1960) (cited by O'Brien, Morgan & Suter 1971) described what appears to be a case of premature closure of the distal radial growth plate in a foxhound. Hanlon (1962) similarly described a case of premature closure of the distal ulnar growth plate in a Weimeraner puppy for which he stated the human counterpart may have been Madelungs deformity.

A condition somewhat similar to Madelungs deformity is however reported in the rabbit pectoral limb. Pearse (1960a and b) (cited by Gruneberg 1963) has described a hereditary deformity that manifests at

between two and three weeks of age in rabbits. The anomaly however is not in the distal radial growth plate but is seen in the distal ulna. The distal ulnar growth plate becomes grossly enlarged and by asymmetrical growth produces diaphyseal curvature and valgus deformity of the distal extremity. The corresponding growth plate of the radius remains normal. Widening and irregularity of the distal ulnar growth plate was observed on histological examination. This condition was found to be the result of a simple recessive gene.

g) Other conditions of the antebrachial growth plates in animals

The ulnae of the Alaskan Malamute dogs which develop deformity grow at a slower rate than the radii and thus endochondral bone growth is defective. Similarly Riser and Shirer (1965) described a condition of retained growth cartilage in the ulnar metaphyses of the giant breeds of dog. These authors considered that in affected dogs abnormal cartilage turnover resulted in extension of the growth cartilage into the metaphysis with associated longitudinal growth retardment of the ulna. Microscopically the retained cartilage in the metaphysis consisted of hypertrophied cartilage cells of questionable mineralisation.

In horses, bone deformities resulting from abnormal growth plate activity have been reported (Delahanty and Gibbens 1953; Rooney 1963; Heinze 1966; Vaughan 1976; Campbell 1977). Much interest has been recently concerned with the condition referred to as 'epiphysitis'. Epiphysitis was reported by Rooney (1963) as normally manifesting as a non painful enlargement of either the distal radius, metacarpus or more rarely metatarsus of

rapidly growing horses. The affected growth plate was found to be narrowed with increased bone density in the adjacent epiphysis and metaphysis. Disorganisation and premature fusion of the growth cartilage was detected histologically.

Paatsama, Jussila and Alitalo (1970) have similarly described the disrupted histological appearance of the distal radial growth plate which they considered to be the aetiology of lameness in the horse affected.

Mason and Burke (1973) described a roughened volar radial metaphyseal aspect as being not uncommon in young racehorses but considered this finding asymptomatic.

Scoliosis

Similarities exist between the growth of vertebrae and long bones, (Gooding and Neuhauser1965; Karaharju 1967). Scoliosis is a clinically important spinal deformity which has received much attention in the human literature. Many authors believe that scoliosis is a penalty of the adoption of bipedalism as a habitual gait.

Naturally occurring scoliosis is rare in quadrupedal animals. Congenital scoliosis has however been reported to occur in association with other deformities in 'contracted foals' (Rooney 1966; Boyd 1976) and in piglets following ingestion of hemlock by the sow (Dyson and Wrathall 1977). James (1970) states that naturally occurring scoliosis is also found in fish and mice and Sawin and Crary (1964) described scoliosis in the rabbit. Birds may also develop congenital scoliosis and reports exist in ducks (Rigdon and Mack 1968) and in chickens

(Taylor 1971; Riggins, Abbott, Ashmore, Rucker and McCarrey 1977). In dogs, Hoerlein (1956;1971), Hare (1961b)and Morgan (1968) have described congenital malformations of the vertebral column some of which result in spinal deformity.

In man, although congenital scoliosis occurs, by far the most frequent clinical entity is idiopathic scoliosis in which no definite cause is apparent (Riddle and Roaf 1955). Scoliosis may however also occur in a number of paralytic and other conditions many of which have been associated with deformities of the appendicular skeleton. Such conditions include neurofibromatosis (Chaglassian, Riseborough and Hall 1976), Stills disease (Rombouts & Rombouts 1974), Osteogenesis imperfecta (Milgram, Flick and Engh 1973; Hoek 1975; Bauze, Smith and Frami 1975), Myelomeningocele (Sharrard 1968; Raycroft and Curtis 1972; Sriram, Bobechko and Hall 1972), Marfans syndrome (Robins, Winter and Moe 1974; Robins, Moe and Winter 1975), multiple epiphyseal dysplasia (Herring 1976), muscular dystrophy (Robin and Brief 1971), cerebral palsy (Sharrard 1968); Tuberculosis (Yau, Hsu, O'Brien and Hodgson 1974); Poliomyelitis (many authors including James 1970), a number of other neurological conditions (Yoslow, Becker, Bartels and Thompson 1971; Clark 1974; Yamada, Yamamoto, Tamura and Tezuka 1974;Hensinger and MacEwan 1976), in association with deformities of the appendicular skeleton (Makley and Heiple 1970) and in association with an osteoid osteoma (Keim and Reina 1975).

It has been suggested that a biochemical basis to idiopathic scoliosis exists (Glauber, Fernbach, Massanyi and

Medgyesi 1962; Ponseti, Pedrini and Dohrman 1972) and a familial tendency to ligamentous laxity has been detected in some cases (Veliskakis 1973).

A number of authors have either suggested or detected a genetic background to cases of idiopathic scoliosis (Esteve 1958; MacEwan and Cowell 1970; 1972; Filmo and Thompson 1971; Wynne-Davies 1968; 1974; 1975; Robin and Cohen 1975) and it is interesting that in identical twins, the severity of deformity di[illegible]red (Fisher and DeGeorge 1967). This latter finding indicates the importance of environmental factors in modifying the deformity and such factors are discussed in part D of this study.

Whatever the origin of the deformity, there is always a growth disturbance producing distortion of vertebrae and intervertebral discs (Roaf 1963).

Some of the structural changes that have been noted can be listed as follows:-

1) The vertebra is deformed and has a convex and concave side, the latter being shorter in length.

2) The bone structure of the concave side is of increased density.

3) The spinous process is turned towards the concave side.

4) There is atrophy and reduced growth cartilage proliferation on the concave side.

5) On the convex side growth cartilage proliferation is accelerated

(adapted and abridged from Karaharju 1957)

The wedging and deformation of the vertebrae are noted only in curvatures which begin before growth is complete which suggests that deformity must be related to the growth plate (Arkin 1949).

Progression of the scoliotic curve does however occur in adults (Coonrod and Feierstein 1976aand b).

The rotation of the vertebrae that occurs in advanced scoliosis is in such a direction that the body turns towards the convex side. Rotation occurs in the apical vertebrae (the most laterally displaced), and becomes accentuated as the scoliotic deformity increases.

Other conditions such as kyphosis, hyperlordosis and vertebra plana which also result in often marked spinal deformity will not be discussed.

There have been many attempts to produce scoliosis experimentally. Some techniques that have been used will be discussed in part D of this study.

Rotational deformities A number of clinical deformities have been described. In many instances axial torsional deformities are apparent in addition to angular deformity. The importance of abnormal rotation has been mentioned briefly in the case of scoliosis. It is necessary however to consider in slightly more detail the normal and abnormal axial torsion that may occur in the important long bones of the pelvic limb.

Tibial Torsion

Hutter and Scott (1949) have defined tibial torsion as any twisting of the tibia on its longitudinal axis which produces a change in alignment of the planes of motion of the proximal and distal articulations. Use of the term tibial torsion however can lead to ambiguity and thus it is necessary to further

refer to such torsion as being in either an internal (medial) or external (lateral) direction. A number of studies (such as those by LeDamany 1909; Strauss 1927; Steindler 1935; Elftman 1945; Hutter and Scott 1949) have established that in the skeletally mature human tibia there is normally up to 20^{o} of external or lateral torsion. Measurements of tibial torsion by radiographical and anatomical methods either by studies on cadavers or bone specimens) have all given similar values for average tibial torsion in man. There is however considerable individual variation about the average figures which are usually quoted. The literature documents a slightly greater degree of lateral torsion in the right human tibia than in the left limb. That the effect of tibial torsion in man is manifested in the distal extremity is demonstrated by the radiographical observations of Goergen, Danzig, Resnick and Owen (1977) on the tibiotalar joint in man. These authors found that in the adult the coronal plane of the ankle is orientated in about 15 - 20^{o} external rotation.

Both Le Damany (1909) and Hutter and Scott (1949) stated that there is minimal tibial torsion at birth but unfortunately only a limited number of observations have been made. Both Nachlas (1934) and Knight (1954) consider that newborn infants commonly have quite marked medial tibial torsion of up to 30^{o}. It is reported that there is a rapid increase in tibial torsion during the first year of life and LeDamany (1909) noted (on limited observations) that it is during the fifth to sixth year of life that the adult amount of tibial torsion is reached.

Much of the concern in man has been with regard to medial tibial torsion with its resultant unsightly postural and gait abnormalities. Medial tibial torsion has frequently been found in combination with other deformities. These include Talipes valgus (Sell 1941), congenital talipes equinovarus (Sell 1941; Barnes 1956), flat foot, metatarsus varus (Sell 1941; Knight 1954; Barnes 1956) and tibia vara.

The effects of extremes of both medial and lateral torsion in infants have been discussed by a number of authors including Kite (1954, 1967). Although in some children the abnormal degree of tibial torsion persists, it is known that in others it disappears. Hutter and Scott (1949) stated however that extremes of torsion do not correct with skeletal development.

It has been suggested that medial tibial torsion is an atavistic condition as it was observed in certain hominid skeletons and in the anthropoid apes by Strauss (1927-cited by Nachlas 1934). LeDamany (1909) however obtained measurements of external torsion similar to that of modern man in a study of hominid tibiae. There are also discrepancies in the reported extent of medial torsion in the anthropoid apes by these two workers.

Le Damany (1909) reported the degree of tibial torsion in a number of different animals but found there to be none in approximately half of those species studied. The sheep was not included in his series.

Some work on tibial torsion in sheep is currently being performed by Lanyon and colleagues who in a preliminary study (Lanyon 1977) found there to be a gradual increase in the amount of tibial torsion (medial torsion) in growing lambs.

Experimentally, Appleton (1934) observed medial and lateral torsion of the tibiofibula in young rabbits in which he induced internal and external rotation of the limbs respectively.

More recently Moreland (1975) produced tibial torsion in young rabbits by applying a continuous torsional load to the bone.

Femoral Torsion

Wolff (1868) is cited (Dunlop, Shands, Hollister, Gaul and Streit 1953) as being the first to recognise a normal axial rotation or torsion of the femur in man. For purposes of measurement the angle of femoral torsion is considered to be the angular difference between the longitudinal axis of the femoral neck proximally and the transcondylar plane distally. To distinguish between the direction of torsion the terms + femoral torsion and - femoral torsion have been adopted by some workers to denote respectively an anteriorly directed or posteriorly directed femoral head and neck relative to the transcondylar plane. Synonyms that have been used include anteversion, antetorsion or anterior twist for + femoral torsion and retroversion, retrotorsion or posterior twist for - femoral torsion. The term angle of declination is also a synonym for femoral torsion but it fails to denote the direction of axial

rotation and as a consequence is rarely used. Although the axis of the femoral neck is used in measurements of anteversion and retroversion, it is believed that femoral torsion occurs in the shaft rather than in the femoral neck.

Numerous studies have been made in attempts to quantify normal femoral torsion in man. Soutter and Bradford 1903; Durham 1915; Elftman 1945 and Kingsley and Olmsted 1948 have made measurements on anatomical specimens and Roberts (1962) on radiographs of dissected bones. Much work has however been concerned with developing methods of accurately assessing femoral torsion 'in vivo' by means of radiography,(Rogers 1931; 1934; Dunn 1952; Dunlap, Shands, Hollister, Gaul and Streit 1953; Ryder and Crane 1953; Magilligan 1956; Shands and Steele 1958; Crane 1959). Many of these radiographical methods involve the use of trigonometry to calculate the angle of femoral torsion. Alvik (1962) considers that the true angle of anteversion can only be measured on bone specimens and a number of authors, such as Dunn (1952) and Ryder and Crane (1953) have discussed the difficulties encountered with radiographic techniques. Indeed Somerville (1957) has stressed that it is difficult to estimate the degree of rotational deformity even when it is known to be present. Even with anatomical specimens however considerable difficulties occur especially with determining the true axis of the femoral neck.

Despite the reported problems, it has been established that at birth the human infant is endowed with a large degree of femoral anteversion which gradually diminishes with the

advance of childhood. In the human foetus anteversion is reported to appear at about the third month and increase steadily through gestation. Roberts (1962) has however shown that there is a diminished rate of increase towards the end of pregnancy.

Wide variation is discovered in the degree of femoral torsion both in foetal and adult human studies. For example, Kingsley and Olmsted (1948) found the variation to be between 20^{0} retroversion and 38^{0} anteversion in adults and 10^{0} retroversion and 64^{0} anteversion in foetuses. Despite such wide variation, average values for the degree of femoral torsion in adult humans are ofter quoted. Durham (1915) found the average value to be 11.90 and Kingsley and Olmsted (1948) found 60% of normal adults had an anteversion angle of between 0 and 15^{0}. In human studies the right femur is reported to have a slightly greater anteversion angle than the left (Kingsley and Olmsted 1948; Roberts 1962) and the female angle was found to be slightly greater than the male (Kingsley and Olmsted 1948) McSweeny (1971) found a higher incidence of abnormally great femoral torsion in female children.

Increased femoral torsion has been associated with a number of diseases in man. These include congenital hip dislocation (Durham 1915; Dunlop et al 1953; Somerville 1953; Shands and Steele 1958; Alvik 1962; Roberts 1962; Harris and Wilson 1970; McSweeny 1971; Mitchell 1972) cerebral palsy (Baker

1962, McEwan, Fabry, and Shands 1971; McSweeny 1971); toeing-in (Somerville 1957; Crane 1959; McSweeney 1971; McEwan, Fabry and Shands 1971; Bailey and Wooley 1976); and toeing out (Crane 1959; McSweeny 1971). Katz (1968) considered that the increased anteversion reported in cases of Legg Calve Perthes disease may be a result of the disease rather than a contributory cause of the condition. Although McSweeney (1971) regarded these to be an association between anteversion and Perthes disease, McEwan, Foley and Shands (1971) have doubted whether such an association exists. Coxa vara and valga have also been associated with abnormalities in anteversion angle (Shands and Steele 1958; Amstutz and Freiberger 1962) but Harris and Wilson (1970) considered that on radiographs apparent valgus may in fact be due to anteversion.

Alvik (1962) has stated that the degree of anteversion depends partly on the angle of inclination of the pelvis. With a pelvis inclination of 65° or less the angle of anteversion is usually greater than 20°.

It has been confirmed (Harris 1972) that lateral torsion deformities of the hips have a relative retroversion and that medial rotation deformities are associated with increased anteversion. Harris (1972) implies an association between femoral and tibial torsion in infants, a finding which has certainly been reported in the classical experimental work of Appleton (1934). Elftman (1945) however has stated that in the adult there appears to be no significant correlation between femoral and tibial torsion.

In the dog, estimates have been made of anteversion angle by radiographic measurements (Nunamaker, Biery and Newton 1973) and by direct measurement on bone specimens (Riser and Shirer 1965; Riser 1973).

There have been several experimental attempts to produce femoral torsion.

Appleton (1934),following induced postural abnormalities in rabbits, obtained increased anteversion in femora of limbs caused to adopt a medially rotated attitude. Appleton (1934) noted increased retroversion in the other limb which adopted a laterally rotated attitude.

Schneider (1962) produced abnormal degrees of internal and external torsion in dog femora following high femoral shaft osteotomy and found marked correction of the induced deformities occurred with growth.

Axer, Karplus, Ryzetelny and Styr (1971) measured femoral torsion in 4-5 weeks old rabbits in which either, or both, the distal femoral and proximal tibial growth plates were damaged. They discovered that if the lateral aspect of the growth plates were damaged genu valgum and external femoral torsion was produced. Following medial growth plate damage genu varum and internal femoral torsion developed. The greatest torsional change was produced if both growth plates were damaged. These authors found the normal anteversion angle to be 9^{o} which gradually decreased with age. Smith, Ireton and Coleman (1950) luxated the hip joint in 3-5 week old dogs and noted that a

degree of coxa vara and a relative reduction in anteversion angle occurred. These authors discovered there to be normally an average anteversion angle of 23^{o} in mature dogs.

PART B

Methods and Materials

Forty two crossbreed lambs aged 10 and 11 weeks were purchased and seven to fourteen days were allowed for acclimatisation. These lambs were obtained from the same farm as those purchased for observations on normal epiphyseal closure and thuswere also Finn Landrace crosses. Details of these lambs are given in Table 1 , Appendix 1 . The experimental period for each lamb commonced when it was exactly three calendar months of age. The lambs were divided into groups of three with two lambs undergoing unilateral excision arthroplasty and the third lamb serving as a control. All were kept under the same conditions and husbandry as employed for the lambs used on the study of normal epiphyseal closure. The maximum postoperative survival time was twelve months and the groups of lambs were slaughtered after a period of weeks or months as shown in Table 6 . The age of any individual lamb at slaughter is equivalent to its survival time within the experimental plan together with the three months prior to its involvement in the experiment. The control lamb for each group was slaughtered after the same interval of time as those which underwent surgery.

The experimental procedure following slaughter was altered slightly soon after the initial sheep were slaughtered. These lambs were thus replaced which accounts for there being more than three lambs in the one and two week survival groups.

Lamb No.37 sustained a fracture of the right tibial diaphysis six weeks postoperatively but as this was the operated limb, was retained in the experimental group. A replacement No.55 was however

TABLE 6

Experimental Series

Induced Deformity lambs

Lamb No	Surgery Date	Slaughter Date	Survival Time
21	23.6.76	30.6.76	1 week
58	25.2.77	4.3.77	1 week
56	25.10.76	1.11.76	1 week
24	24.6.76	8.7.76	2 weeks
33	30.8.76	13.9.76	2 weeks
40	28.10.76	11.11.76	2 weeks
41	25.10.76	8.11.76	2 weeks
42	22.10.76	12.11.76	3 weeks
43	22.10.76	12.11.76	3 weeks
45	22.10.76	19.11.76	4 weeks
46	22.10.76	19.11.76	4 weeks
49	25.10.76	20.12.76	2 months
50	25.10.76	20.12.76	2 months
51	25.10.76	17.1.77	3 months
52	25.10.76	17.1.77	3 months
38	2.9.76	23.12.76	4 months
54	28.10.76	17.2.77	4 months
29	30.8.76	17.1.77	5 months
36	1.9.76	19.1.77	5 months
37	30.8.76	14.2.77	6 months
39	30.8.76	14.2.77	6 months
55	25.10.76	11.4.77	6 months
17	23.6.76	2.2.77	8 months
18	23.6.76	2.2.77	8 months
14	22.6.76	29.3.77	10 months
15	22.6.76	29.3.77	10 months
12	21.6.76	25.5.77	12 months
13	21.6.76	24.5.77	12 months

Control Lambs

Lamb No	Slaughter Date	Survival Time
20	30.6.76	1 week
59	2.3.77	1 week
57	11.3.77	2 weeks
25	8.7.76	2 weeks
44	12.11.76	3 weeks
60	21.3.77	4 weeks
48	19.12.76	2 months
53	17.1.77	3 months
30	23.12.76	4 months
31	17.1.77	5 months
35	14.2.77	6 months
19	2.2.77	8 months
16	29.3.77	10 months
11	24.5.77	12 months

brought into the group in case the fractured limb influenced the results. The tibial fracture of No.37 was reduced and internal fixation by means of an intramedullary steinman pin performed. A healed midshaft right femoral fracture was found post mortem. Lamb No.38 was found post mortem to have a healed, although overriden fracture of the midshaft right femur.

All lambs undergoing surgery were starved the day prior to surgery. A pen was constructed with sawdust bedding to ensure this occurred. Anaesthesia was induced using intravenous pentobarbitone sodium[1] at a dose rate of 20 mg/kg. Endotracheal intubation was facilitated by the use of a laryngoscope. Following intubation anaesthesia was maintained by means of an O2/Halothane mixture in a semiclosed Boyles circuit with absorption. The operating table was tilted in such a manner as to permit drainage of saliva from the buccal cavity and to prevent accidental inhalation of saliva it was found beneficial to ensure that the cuff on the endotracheal tube was adequately inflated.

The surgical site overlying the femoral head and neck to be removed was clipped and prepared utilising Pevidine Iodine Solution.[2] In all cases in this experimental series the right femoral head and neck was removed. By operating on the right limb in every case, subsequent observations were simplified.

The surgical approach was basically antero-dorsal as described by Duff (1975) and as shown in fig 2. Blunt dissection enabled separation of the subcutaneous fat which allowed the superficial gluteal muscle to be identified. Curved mayo scissors were used

1 Sagatal. Pentobarbitone Sodium 60 mg/ml. May & Baker Ltd.

2 Povidone iodine with detergent - Surgical Scrub - Berk. Pharmaceuticals Ltd.

to bluntly dissect beneath the superficial gluteal muscle following which tenotomy of this muscle was performed close to its insertion over the greater trochanter. By incising the proximal few centimetres of the fascia lata and similarly cutting through the gluteal fascia better exposure of the middle gluteal muscle was obtained and also the sciatic nerve could be more easily identified and preserved.

The middle gluteal muscle was identified and again following blunt dissection with the aid of curved mayo scissors tenotomy was performed. Gluteal tenotomy was performed in such a manner as to leave sufficient tendinous insertion to enable subsequent suturing. Usually a small amount of haemorrhage was encountered following middle gluteal tenotomy, due to the muscular as well as the tendinous insertions of this muscle, but in no case was this a problem.

Finally the deep gluteal muscle was identified and sectioned in a similar manner. At this stage, the antero dorsal aspect of the acetabular rim and femoral head was easily identifiable by palpation. Manipulation of the pelvic limb distally was found to be helpful. A scalpel was used to incise the dorsal joint capsule. Escape of synovial fluid confirmed that the hip joint had been entered. Metzenbaum scissors were used to extend this incision in both a caudal and cranio ventral direction. There was normally a degree of haemorrhage on incising the joint capsule but again this was not troublesome. On exposing as much of the

femoral head as possible it was found useful to use straight mayo scissors as a lever to lift the femoral head out of the acetabulum and then the round ligament could be more easily sectioned using metzenbaum scissors. On sectioning the round (teres) ligament the femoral head could be luxated easily. Bone cutting forceps were routinely used for the osteotomy through the femoral neck.

The surgical approach to the hip joint described, with gluteal tenotomy being performed, is similar to that described by Brown (1953) and Piermattei and Greeley (1966) in the dog.

Following removal of the femoral head and neck in the experimental lambs an antibiotic spray[1] was administered to the surgical field. The joint capsule was not sutured.

The gluteal muscles were reapposed and sutured in turn by means of 0 chromic catgut[2] horizontal mattress sutures and the subcutaneous fat similarly brought together. An 0 chromic catgut continuous suture was placed in the subcutis to appose the skin edges. Vertical mattress nylon skin sutures completed the closure.

The control lambs were not subjected to either anaesthesia or any form of surgery.

Postoperatively those lambs which underwent excision arthroplasty were allowed to recover and the endotracheal tubes were removed as soon as voluntary swallowing was apparent. The operated lambs were then placed in a pen separate from the others until they were able to stand and move freely.

1. Framyspray - Polymyxin B, Bacitracin, Neomycin. Fison Ltd. pharmaceutical div.
2. Ethicon Ltd.

In order to gain information on the relative weight bearing of each limb in both normal lambs and those which underwent surgery, a simple platform scale apparatus was designed and assembled. The construction and use of this apparatus is described in detail in Part D of this study.

In order to record any developing deformities the experimental lambs together with their controls, were radiographed on the same occasions as platform scale measurements were made. An antero-posterior projection was chosen as likely to be most informative. A portable X-Ray machine (A.E. Dean & Company (X-ray Apparatus) Croydon) and standard 12" x 15" cassettes with Kodak RP54 and RP14 film were used. The films were processed by means of a Williamson RP, 90 second, film processor. The exposures administered were 70KV and 15mA in every case. The time varied from 0.5 seconds in small lambs to 1.5 seconds in long surviving groups. The machine was mains compensated prior to use on each occasion. The focus film distance was kept constant at 30 inches. In all cases the method of radiographing the limbs was the same and as in Fig 3 By using a table in the manner shown, the lambs were easily restrained and only two operators were required. Whilst one operator held the lamb in a sitting position on the table the other held the hindlimbs in position and at the same time set and delivered the necessary exposure. It became apparent that lambs were much less liable to resist restraint when held in a sitting position than when held with the hindlimbs in full and forced backward extension.

Immediately prior to slaughtering the groups of lambs at the end of their experimental period, photographs were taken so that any gross limb deformities or abnormalities of posture could be recorded (see part D).

Heparinisation (25000 units intravenously[1]) permitted the lambs to be prepared for angiographic studies as is described in part C of this study. Following heparinisation; euthanasia was performed by intravenous administration of an overdose of pentobarbitone sodium[2].

As soon as possible after death, measurements were made on the ranges of joint movement by means of a simple goniometer (plate 18) constructed for the purpose. Measurements were made of full flexion and extension of both the stifle and hock joints (fig 4) and metatarso-phalangeal (fetlock) joint mobility was assessed in subjective terms.

The limbs of those showing deformities together with their controls were then photographed in as near to an antero-posterior view as could be maintained.

The bones of each pelvic limb were dissected free of all soft tissue except for the periosteum. The bone specimens thus obtained were then stored in a deep freeze prior to subsequent study.

The frozen bones were removed in turn from the deep freeze and radiographs of both tibiae and femora made on a high definition cassette (24 x 30 cms Siemens Rubin) in both lateral and anteroposterior projections. The metatarsae were radiographed in an anteroposterior projection only.

1. Heparin Injection B.P. 1000 units/ml. The Boots Company Ltd.
2. Euthatal - Pentobarbitone sodium - 200 mg. per ml. May & Baker Ltd.

In all cases the middle and distal rows of tarsal bones were left articulated with the metatarsae. The bones were returned to the deep freeze as soon as possible after radiography and other procedures described below in an attempt to prevent complete thawing of the specimen. Repeated thawing and refreezing was found to have a deleterious effect on the articular cartilage .

The radiographs of the dissected bones permitted a number of measurements and observations to be made.

1) Tibial, Femoral and Metatarsal Length - the tibial length was measured on an anteroposterior radiograph as shown in fig 5B The anatomical landmarks chosen, between which the measurements were made were the medial intercondyloid eminence and the anterior lip of the distal tibial epiphysis.

Two measurements however were made on anteroposterior radiographs of the femora. Firstly the distance between the dorsum of the femoral head and the medial femoral condylar articular surface was measured in those femora which had not suffered excision arthroplasty. Secondly the distance from the summit of the greater trochanter to the lateral, distal femoral articular surface was recorded (fig 5A).

The metatarsal length measurement was similarly made on an anteroposterior radiograph. The junction of the proximal metatarsus and distal row of tarsal bones was easily identifiable and served as the proximal landmark. Distally, a line was drawn joining the medial and lateral condylar ridges and the midpoint of this line taken (fig 5c)

2) Tibial Plateau Angle - The tibial plateau angle relative to the long axis of the bone in an anteroposterior view was estimated as shown in fig 6. Two measurements were made on radiographs in this projection. The long axis was determined by bisecting the diaphyseal width at the two points shown. The angle made by both a line through the metaphyseal/ growth plate junction and a line across the tibial condyles to the long axis was measured.

An estimate was also made of the tibial plateau angle on lateral radiographs (fig 7.) Because of the presence of the tibial crest in a lateral radiograph however, simple bisection of diaphyseal widths failed to adequately represent the long axis of the tibia. Bisection of a line joining the most anterior and most posterior regions of the proximal tibial epiphyseal plate, and mid-diaphyseal width bisection was carried out. A line drawn connecting these two points produced a good approximation of the tibial long axis. A line was drawn from anterior to posterior along the tibial condyle and the angle this made with the tibial long axis measured. In many cases both the lateral and medial condyles could be identified on the radiographs and in these cases a line was drawn as an average of the two condylar angles. Difficulties were however encountered. The slope of the lateral tibial condyle was greater than that of the medial condyle. Thus the two radiodense contours that were visible on radiographs were at divergent angles. It was difficult, in all cases, to know exactly where to draw a tangent to the tibial condyles which were curved in a convex manner. In addition, in young lambs general lack of bone density resulted in no definite radiodensity identifying the tibial condyles and the proximal tibial epiphysis did not have the characteristic

adult contour.

Femoral Condylar Measurement - In addition to examining the proximal tibial plateau for observable and measurable abnormality, it was considered desirable to gain some information on any relative changes between the medial and lateral femoral condyles. It was also of interest to estimate whether or not any deformity was occuring in the distal femoral metaphysis. The femoral condyles were observable on anteroposterior radiographs. To achieve the second objective, measurements were made as shown in fig 8.

A base line was obtained by connecting the two femoral condylar surfaces and a perpendicular was drawn midway along its length in such a manner that it passed through the centre of the intercondyloid fossa (fig 8). A line was then drawn connecting the centre of the fovea capitis and the point of intersection of the perpendicular and the baseline. The angle made by the perpendicular and the line passing through the fovea capitis was measured.

Estimation of contribution to longitudinal growth of the proximal and distal tibial growth plates - The site and direction of the tibial nutrient foramen was observed on antero-posterior tibial radiographs. Measurements were made in the manner described by Digby (1916) and as shown in fig 9 . A line was drawn through the nutrient canal and projected across the medullary cavity. The point of intersection of this line with the long axis of the tibia was noted. and served as a reference point from which measurements could be made in proximal and distal directions. The tibial long axis was determined in the same manner as described above.

Femoral Neck Angle - The angle to the long axis of the femur made by a line drawn joining the dorsum of the fovea capitis and the base of the greater trochanteric epiphysis was considered to be the closest approximation to the femoral neck angle that could be made. The femora were radiographed with the posterior aspects of both the femoral head and the greater trochanter in contact with the cassette. Thus, any effect of anteversion on the projected femoral neck angle was eliminated. The long axis of the femur was determined by bisecting the diaphyseal width at two sites, halfway down and one quarter of the way down the length of the femur (fig 10B). An estimate of femoral neck angle could not be made in those femora which had undergone excision of the femoral head and neck.

Greater Trochanteric Base Angle - In addition the angle made by a line drawn through the most medial and lateral regions of the greater trochanteric growth plate with the long axis of the femur was measured (fig 10A). This measurement was made on the same femoral radiograph as used for estimating the femoral neck angle.

Angular bone deformities - A note was made of the radiographical changes seen in those bones which underwent deformity. In addition, information was available on the developing deformities from the radiographs taken at weekly intervals throughout the experimental period.

a) Tibial Deformity - from anteroposterior radiographs of those dissected tibiae with macroscopic deformities, estimates were made of the angular deviations of the distal tibiae. To reduce errors in measurement resulting from the three dimensional nature

of the deformities, the tibiae were radiographed with the distal region of the bone in contact with the cassette.

The long axis of the tibia was determined by bisecting the diaphyseal widths at two points, one being at halfway down the length of the bone and the second at the point one quarter of the way down the bone (fig 11). A line drawn through these points adequately represented the tibial long axis. Two measurements were then made. A line drawn at the distal tibial growth plate/metaphysis junction was bisected and a perpendicular drawn as shown in fig 11. The angle made by this perpendicular and the tibial long axis was taken as an estimate of the deformity occurring at the metaphysis.

In addition a line was drawn across the articular surface of the distal tibial epiphysis (fig 11). This line was similarly bisected and a perpendicular drawn to meet the tibial long axis. The angle between these two lines was measured and was taken as an estimate of the angular deformity of the distal tibial articular surface relative to the long axis of the tibia (fig 11).

A further measurement was made in those cases in which tibial deformity was evident on lateral radiographs. As with the measurements of tibial plateau angle, the long axis was obtained by bisecting the most anterior and posterior regions of the proximal tibial epiphyseal plate and the mid-diaphyseal width (fig 12). Because of torsional deformities, in some cases a true lateral radiograph of the distal tibial articular surface was not obtained. An approximate measurement was made by drawing a line parallel to the

apparent distal articular surface in these cases. Positioning of the bone for radiographs in this projection was extremely difficult and thus measurements of deformity in this plane were considered to be more subject to error. In those cases in which a line could be drawn across the articular surface the method of estimating the angular deformity was as for measurements on antero-posterior radiographs and as shown in fig 12.

b) Metatarsal Deformity - The deformities observed on anteroposterior radiographs of metatarsi were measured in a similar manner to that employed with the tibiae. The long axis of the metatarsus was determined by bisecting the diaphyseal width at the two sites shown (fig 13) and drawing a line to pass through the two points obtained. Perpendiculars were drawn from midway between the medial and lateral condylar ridges. The angles made by these perpendiculars with the metatarsal long axis were measured and provided estimates of the angular deformity at the metaphysis and articular surface respectively. As with the tibial radiographs the metatarsi were positioned with their distal regions in as close contact as possible with the cassette.

As well as these measurements on radiographs, a number of other procedures were carried out on the frozen bone specimens.

1) Tibial, Femoral and Metatarsal Length - The lengths of the bone specimens were measured, but because of attached fibrous tissue, especially over malleoli on tibiae and the greater trochanter on femoral specimens, the results were deemed too inaccurate.

2) Tibial Torsion - An estimate was made of tibial torsion both in control and experimental lambs. The method of measurement was based on a technique used by Lanyon (1977) for similar observations on sheep tibiae. The bone specimen was placed on the stand as shown (Plate 19 and fig 14) and the pointer on the protractor mount positioned so as to cross transversely the proximal joint surface (fig 14). The angular reading was recorded. The bone specimen was left untouched while the same procedure was carried out at the distal joint surface. Again the angular reading was taken and the difference between the two readings calculated. The resulting angular measurement was considered to be an estimate of the degree of torsion of that tibia. The method was repeated three times in a random fashion for each tibia. In most cases there was agreement of the results within 2^{o}. In the few cases where discrepancies existed repeat readings were performed until three readings with values within 2^{o} or less were obtained.

3) Metatarsal Torsion - Metatarsal torsion was estimated in the same manner and using the same apparatus as described for measuring tibial torsion. The angular difference between the transverse planes of the proximal and distal articular surfaces of the bone specimens being recorded. As the tarsal bones (except for the fibular and tibial tarsal bones) were left articulated on metatarsal specimens the proximal intertarsal joint surface of the middle tarsal bones served as the proximal articular surface.

Measurements were again made on three random occasions.

4) Femoral Anteversion Angle - A method of measuring the angle of anteversion of the femoral head and neck was devised. The apparatus used was as shown diagrammatically in fig.15. By placing the bone on its posterior surface the horizontal corresponded to a line joining the posterior aspects of both femoral condyles. The estimated angle of anteversion was measured relative to this horizontal. A 0.064" Kirschner wire was held in a position representing the long axis of the femoral neck by means of two pieces of soft paraffin wax[1] One piece was placed on the dorsum of the femoral head and the other on the summit of the greater trochanter (fig 15) The wire was positioned to lie alongside the trochanteric crest/trochanteric fossa margin and over the midpoint of the fovea capitis (fig 15). The Kirschner wire was further positioned so that its extremity was in contact with the horizontal and could thus be placed at the centre of the 180° protractor. Observations were made with the horizontal (equivalent to the posterior aspects of the condyles) at eye level and the angle made by the Kirschner wire read by observation through the protractor.

Readings were repeated three times in a random fashion and further readings taken if a discrepancy of greater than 2° was present. In most cases this was not necessary. The wax was easily removed prior to returning the specimens to the deep freeze.

1 Soft Paraffin White B.P. - Evans Medical Limited.

5) Angular bone deformity - Finally those bones with observable gross deformities were photographed in an antero-posterior view.

During the course of this study the pelvic limbs of a number of lambs and sheep not included in this experimental or control series, became available. The bones of these limbs were dissected free of soft tissue and used to provide boiled out bone specimens. These sheep were of various unknown breeds and breed crosses and of unknown age at slaughter.

These specimens were used for observations on tibial torsion (58 specimens) and estimates of femoral anteversion angle (30 specimens).

RESULTS B

11. Deformity Examples of post-mortem limb deformities are given in plates 20 A to C and of dissected bones in plates 21 A to D.

It is convenient to describe initially the deformities that were noted on anteroposterior radiographs taken of the dissected bones postmortem prior to considering the sequence of changes that culminated in the deformities described.

As a generalisation gross deformity was not seen before 2 months postoperatively. All but one of the lambs slaughtered after this time had grossly demonstrable deformity at either the distal tibial or distal metatarsal sites. In a number of cases deformity was present in both of these bones but normally was more severe at one or other site.

The final deformity in each lamb slaughtered 2 months postoperatively is given in table 7. Only the LH unoperated limb is represented in this table.

In the vast majority of instances, the angular deformity of the unoperated LH limb resulted in the distal articular surface being directed medially and thus varus deformity normally occurred. Exceptions to this generalisation were found in metatarsae, of longrunning lambs numbers 39, 55, 18, 14 and 12, in which a degree of valgus deformity occurred. All of these cases had severe distal tibial varus.

Radiographic appearance of deformed bones postmortem

A) Tibiae Examination of radiographs of deformed bones revealed a variable appearance of the distal tibial metaphysis and growth

TABLE 7

Lamb Number	Postoperative Survival Time	Tibial Deformity	Metatarsal Deformity
49	2 months	Minimal	-
50		Minimal	Slight
51	3 months	-	Minimal
52		Slight	Minimal
38	4 months	-	Moderate
54		Minimal	Minimal
29	5 months	Minimal	Slight
36		Marked	Moderate
37		-	Moderate
39	6 months	Marked	Minimal*
55		Moderate	Minimal*
17	8 months	Minimal	Moderate
18		Extreme	Minimal*
14	10 months	Extreme	Slight*
15		Minimal	-
12	12 months	Marked	Minimal Slight*
13		-	Minimal

Approximate angular deformity at distal articulating surface

Minimal = $0 - 5^{o}$

Slight = $5 - 10^{o}$

Moderate = $10 - 20^{o}$

Marked = $20 - 30^{o}$

Extreme = 30^{o}

* = Valgus deformity

plate which was dependant upon the degree of deformity present.

For simplicity, typical changes noted for each stage of severity will be considered. The described lesions were detected on postmortem anteroposterior radiographs. See plates 22 A to D

a) Minimal Tibial angular deformity The relevant changes seen in the distal tibia of such cases varied from changes purely in the metaphyseal trabeculae to detectable abnormalities in the growth plate. In all cases however an increase in both the density and extent of metaphyseal trabecular pattern was noted medially in the distal tibia. In some cases increased density was also noted subchondrally in the medial sector of the epiphysis.

In more advanced cases which had a slightly greater degree of angular deformity widening of the distal tibial growth plate was apparent. In some cases a radiolucent area adjacent to the growth plate laterally was observed which was sometimes associated with a mottled appearance of the lateral, distal metaphyseal trabeculae. In most cases with growth plate changes widening was greater laterally than medially.

b) Slight Tibial angular deformity Changes noted were essentially as described for cases of minimal deformity although more obvious. Thus the metaphyseal trabecular pattern was denser and coarser medially, and there was more obvious increased epiphyseal density medially. In addition the medial distal metaphyseal cortex was of greater thickness than normal and increased radiodensity of the medial malleolus was

noted. Slight metaphyseal and epiphyseal peaking medially was observed. The widening of the growth plate was more noticeable and a definite lucent area in the metaphysis adjacent to the lateral growth plate occurred. The medial sector of the growth plate was also widened and a mottled density in the lateral metaphysis adjacent to the lucent area was apparent.

c) Moderate Tibial angular deformity Changes noted were essentially a progression of those noted above. The metaphyseal changes consisted of a thickened distal metaphyseal cortex and increased metaphyseal density, medially. Medial peaking of the metaphysis and epiphysis was slightly more marked, as was increased medial malleolar density. The medial sector of the growth plate was more obviously thickened than before. Laterally, growth plate widening was apparent and an irregular contour to the adjacent lateral metaphyseal cortex was present. Radiolucent areas were present in both the metaphysis and epiphysis laterally. Lucency of the metaphysis adjacent to the lateral sector of the growth plate was again in evidence.

d) Marked Tibial angular deformity At this stage the thickened medial cortex was not confined to the distal metaphyseal region but was also apparent at mid-diaphyseal level. The medial distal metaphyseal trabeculae had become so dense that a trabecular pattern was barely recognisable. Medial peaking of the metaphysis and epiphysis was marked and there was marked density of the medial sector of the epiphysis. Both malleoli appeared enlarged and the medial malleolus was of

increased density. The medial aspect of the distal tibial epiphysis had a deformed contour below the level of peaking. The medial sector of the growth plate was thickened, especially between the metaphyseal and epiphyseal peaks, and areas of mottled density were present adjacent to the growth plate. Laterally more marked areas of lucency and density were present both in the epiphysis and metaphysis close to the growth plate. Patchy lucency was apparent in the metaphysis immediately adjacent to the lateral growth plate which was thickened. The lateral metaphyseal cortex adjacent to the growth plate had an irregular contour. The lateral distal metaphyseal cortex was slightly thickened.

e) Extreme Tibial angular deformity The cortical changes in such cases were as described in the marked deformity bones. There was however if anything slightly less marked metaphyseal and epiphyseal peaking medially. There were seen to be increased metaphyseal density, adjacent to the distal tibial growth plate both medially and to a lesser extent laterally, although mottled areas of lucency were apparent in the most lateral sector. Laterally, a lucent defect was situated in the metaphysis immediately adjacent to the growth plate. The lateral sector of the growth plate was not obviously thickened. Although the medial aspect of the growth plate was thickened in one case (no 18) in another (no 14) thinning and areas of apparent fusion were observed. The medial malleolus was noticeably more dense and enlarged whereas the lateral malleolus although enlarged was relatively radiolucent.

B) Metatarsi

Anteroposterior radiographs of those metatarsi with grossly observable angular deformity revealed an almost identical sequence of change (see plates 23 A to G).

In a similar manner lucent defects appeared in association with the lateral sector of the growth plate and eventually became separated from the growth cartilage by new endochondral bone. Defects so produced came to lie adjacent to the cortex and became disrupted by remodelling of the metaphysis. In at least two lambs numbers 29 and 17 it appeared that two separate defects developed from the lateral growth cartilage.

One prominent feature in metatarsi however was a pronounced thickening of the medial growth plate sector. Lambs number 18, 14, and 12 however all of whom had predominantly tibial deformity and only minimal or slight metatarsal involvement showed marked narrowing of both the medial and lateral aspects of the growth plate at slaughter. In lambs nos 17 and 13 with pronounced metatarsal deformity the lateral growth plate was grossly narrowed at the time of slaughter although the medial sector was still grossly thickened in both cases.

The changes noted in those lambs with valgus metatarsal deformity are of particular interest. In lamb no 39 valgus was minimal. Metaphyseal and epiphyseal peaking was observed medially. The growth plate was thicker laterally than medially. Lamb no 18 possessed metaphyseal and epiphyseal

peaking both medially and to a lesser extent laterally. Although both the medial and lateral distal metatarsal cortices were slightly thickened, this feature was more marked laterally. In contrast, the medial cortex was thicker at mid-diaphyseal level. The growth plate was slightly thicker laterally.

In case no 14 peaking was also present medially and laterally and the lateral distal metaphyseal cortex was slightly thicker. The lateral sector of the growth plate was thickened.

Lamb no 12 showed slightly greater thickening of the medial diaphyseal cortex with moderate peaking and trabecular increase medially. The distal metaphyseal cortex was thicker laterally however. Trabecular density was also slightly increased laterally.

Changes noted in the distal tibiae and metatarsi of cases showing no gross deformity Despite no gross deformity being present changes were present in both tibiae and metatarsi of lambs slaughtered prior to 2 months postoperatively. Such lesions are summarised in Table 8.

In all of these cases, minimal change was judged to be present when growth plate changes in conjunction with metaphyseal trabecular increase was observed. In all other cases trabecular increase appeared to be present in the LH limb but comparison with the operated RH limb could simply reflect loss of trabeculae as a result of disuse in the latter. Metaphyseal trabecular change was not sufficiently marked in these cases however to state that there was a noticeable and definite increase relative to the metaphysis of control lambs.

TABLE 8

Lamb Number	Postoperative Survival Time	Distal Tibial Change	Distal Metatarsal Change
21		-	-
56	1 week	-	-
58		-	-
24		-	-
33	2 weeks	minimal	-
40		-	minimal
41		-	-
42	3 weeks	-	-
43		minimal	-
45	1 month	minimal	minimal
46		minimal	minimal

Other bone changes The greatest emphasis has been placed on the appearance of lesions on anteroposterior radiographs as this was the projection used when monitoring the developing deformity. Radiographs in lateral projections including femoral radiographs, also revealed changes concomitant with distal angular deformity. In addition some abnormalities were detected in the proximal regions of the bones considered.

Briefly observed abnormalities were:

1) Femora: In a number of lambs, the distal femoral growth plate of the non-operated limb was slightly thickened medially. Associated with such change was a degree of metaphyseal and

epiphyseal peaking of the posteromedial aspect. These femoral changes were usually most apparent in the long surviving individuals, but was detectable as early as 2 months post-operatively. This feature was not soon in lambs that only underwent gross metatarsal deformity.

2) Tibiae: Similar changes occurred in the proximal tibiae of a few lambs all of which were in long surviving groups. As with femora, a slight thickening of the medial proximal tibial growth plate was observed, sometimes in association with mild medial peaking and occasionally with concomitant increased density of the proximal tibial epiphysis medially (plate 24).

Distally, in those lambs with severe distal tibial deformity, the posterior cortex was noticeably thickened. The anterior distal cortex in such cases had a less well defined contour with irregularity being apparent in some cases. The distal metaphyseal trabeculae tended to be more profuse and of a coarser nature posteriorly in these cases. Perhaps the most interesting changes occurred in the tibial tuberosity growth plate of the experimental lambs. In many of the lambs slaughtered after one month postoperatively the tibial tuberosity growth plate of the non-operated left hind limb was noticeably thicker than in comparably aged control animals (Plate 25 A to D). The increased thickness was in the direction of longitudinal tibial growth. The tibial tuberosity growth cartilage of the operated limbs was in some instances narrower but in most equal to that of comparably aged control lambs (Plates 25 A & B).

In longer surviving lambs the tibial tuberosity growth plate was almost symmetrical in both the operated and non-operated limbs.

3) Metatarsi: The tarsal bones (distal and middle rows) were left articulated with the metatarsi. In many cases an increased density of the medially situated tarsal bones was apparent. Such change was especially marked in cases of metatarsal deformity. In addition to tarsal bone change, the proximal metatarsal metaphysis normally exhibited a more profuse and coarser trabecular pattern medially.

Similar observations were made in those lambs with valgus metatarsal deformity. In such cases trabecular increase and greater bone density was medial.

Weekly monitoring of distal tibial growth plates and metaphyses

The sequence of abnormalities noted from weekly radiographs was essentially as described above with progression through the stages of deformity being largely dependent on the postoperative survival time. However, other changes, not observed in postmortem tibial radiographs, were detected on scrutiny of the sequences of radiographs that were available.

Although the observed changes were apparent in all cases, some variation in the chronology of their appearance was encountered. It is convenient therefore to number the detected abnormalities that typically occurred so that reference may be made to their chronological appearance in different lambs (table 9).

In all cases, the initial change was an observable increase in the distal metaphyseal trabeculae which became particularly profuse medially (1). Soon afterwards there was considered to be the early appearance of a radiolucent shadow over the metaphyseal aspect of the lateral growth plate (2). At about this time a noticeable increase in thickness of the medial sector of the growth plate occurred (3). The area of radiolucency related to the lateral growth plate became more apparent and was discernible as a lucent band running from the lateral aspect to approximately mid epiphyseal level (4). At this time there was a marked increase in both the number and thickness of trabeculae in the medial distal metaphysis (5). Usually, shortly after these changes were observed slight angular deformity of the limb was noticeable (6) and soft tissue swelling become apparent especially laterally over the site of deformity (7). As the area of lucency increased in size it projected well into the metaphysis and became enclosed in a thin "shell" of increased density on its metaphyseal border (8). At about this time slight peaking of the medial metaphysis and epiphysis was present (9). The lucent defect reached its maximum dimensions (from 3-5mm depth on radiographs) and appeared separated from the growth plate by a thin layer of endochondral bone (10). This thin layer of bone rapidly increased in size until the lucent defect was distant from the growth plate (11). At this time the lateral metaphysis normally contained a mottled density with patchy loss of trabeculae separate from but adjacent to the

defect (12). Marked varus deformity was present by this time (13). With passage of postoperative time the lateral lucent defect came to lie in the region of metaphyseal remodelling. As a consequence its most lateral aspect was removed leaving an irregular cortical defect laterally (14). A mottled density was present around this cortical defect. Gradually the cortical defect was reduced in size but usually left a slight defect in the cortical surface (15).

In a number of lambs a second lucent defect became apparent adjacent to the lateral sector of the growth plate at the same site as the original defect (16). On such radiographs a lucent isthmus appeared to connect the new defect with the original area of lucency. The new defect in turn became separated from the growth plate by a layer of endochondral bone (17) and in two lambs eventually replaced the original defect. The isthmus however remained as a defect in the cortex. (18). At a variable time during the above sequence of changes, the medial metaphyseal and epiphyseal peaking became pronounced (19) and the lateral portion of the distal tibial epiphysis was observably less dense (20). There was also seen to be an increase in density both above (in the metaphysis) and below (in the epiphysis) the medial growth plate sector (21). With passage of postoperative time the medial malleolus became more dense in appearance (22). A very dense appearance with no recognisable trabecular pattern developed in the medial metaphysis (23) and the peaked metaphyseal and epiphyseal edges

became more rounded in contour (24). At this time quite obvious medial growth plate thickening was observed. The medial sector of the growth plate throughout the experimental period had a thickened appearance but ultimately in two long surviving lambs appeared more narrow and dense metaphyseal bone obscured its outline.(25).

Osteophytes were in evidence after a variable period of time particularly on the lateral malleolus and fibular tarsal bone (26). (See table 9).

In one lamb, no 14, a third radiolucent defect appeared to develop at 30 weeks associated with the lateral growth plate sector. This defect however consisted of a number of discrete lucent foci which were equivocally separated from the growth plate by new bone at the time of slaughter (40 weeks)

Weekly monitoring of distal metatarsal growth plates and metaphyses It would be repetitious to describe the sequence of changes noted in the metatarsi of experimental lambs. The radiographic sequences however were not as complete as for the tibia due to the difficulty in small lambs of including the fetlock joint on radiographs. In addition, the x-ray tube had been centred over the distal tibiae and not over the metatarsal site. This made interpretation of radiographs somewhat less accurate.

A considerable number of radiographs were however informative and the sequence of change was as described for distal tibiae.

The chronology of changes was also remarkably similar with for example peaking of the medial epiphysis and metaphysis being noticeable at approximately 8 weeks postoperatively, and medial growth plate thickening being observed at an earlier date.

TABLE 9 CHRONOLOGY OF DISTAL TIBIAL CHANGES

	Lamb Number	52	36	39	55	18	14	12
	Postoperative Survival Time	3mths	5mths	6mths	6mths	8mths	10mths	12mths
DETECTED ABNORMALITY	1	2wks	1wk	2wks	2wks	1wk	2wks	3wks
	2	2	3	2	3	1	5	5
	3	5	3	5	5	4	6	5
	4	7	3	3	5	3	6	6
	5	7	4	5	6	5	6	9
	6	9	5	6	7	4	6	6
	7	10	5	6	7	5	6	6
	8	9	8	6	6	7	10	10
	9	8	5	6	5	5	8	10
	10	12	5	5	7	5	10	12
	11	-	7	6	8	7	15	14
	12	-	7	9	8	9	11	13
	13	-	8	8	9	6	10	14
	14	-	9	9	9	7	12	16
	15	-	11	11	15	18	20	26
	16	-	9	9	14	10	17	22
	17	-	20	16	23	24	24	33
	18	-	-	24	-	-	33	-
	19	-	14	15	9	10	17	15
	20	-	10	6	8	7	11	13
	21	-	13	14	14	14	15	14
	22	-	-	-	22	18	19	19
	23	-	18	17	19	18	18	19
	24	-	19	21	24	19	18	18
	25	-	-	-	-	-	40	44
	26	-	18	18	17	21	29	21

Tibial and Metatarsal Deformity

a) Anteroposterior Radiographs Other information was available from radiographs apart from that reported above. Measurements made on anteroposterior radiographs of the angular deviation of distal metatarsae and tibiae are presented in Append.1, tabs.2,3,4&5.

No accuracy can be ascribed to such measurements which at best can only be considered estimates. Although measurements obtained have been presented in full in Appendix 1 those with pronounced deformities have been represented diagrammatically in figs 16 and 17.

No measurements of tibial deformity could be made in two lambs (nos 45 and 46) in which the tibiae had been sectioned. The first appearance of demonstrable gross deformity (8.5° varus at joint surface) in tibiae was in No 52 at 3 months postoperatively. The most severe deformity occurred in lamb No 14 who was estimated to have 47.5° of varus distal tibial deformity when slaughtered at 10 months postoperatively.

In metatarsae, lamb no 50 was judged to have 7° varus deformity at the distal joint surface when slaughtered at 2 months postoperatively. The most severe deformity occurred in lamb no 17 (estimated 19° varus at joint surface) who was slaughtered at 8 months post-operatively.

Valgus metatarsal deformity was small (estimated 2°, 4°, 5°, 2°, 7° and 6° at joint surface) and occurred in five long surviving lambs all of whom had gross varus tibial deformity. Measurements were made at two sites, the joint surface and the

metaphyseal-growth plate junction and demonstrate that in all cases of deformity the angular deviation at the articular surface was slightly less than that at the metaphysis. This observation was made in cases of both predominantly tibial and predominantly metatarsal deformity.

In control lamb tibiae there was similarly a tendency for the articular surface to be orientated slightly differently from the metaphyseal-growth plate junction. In tibiae the medial sector of the distal tibial epiphysis was normally of slightly greater depth than the lateral portion in both control and experimental lambs.

In metatarsi however epiphyseal abnormalities were apparent. An asymmetry was detectable in 50% of the control lambs but in most cases was of a minor degree. In such cases, there was a discrepancy in the level of the mid articular surface of the medial and lateral epiphyses. Normally such asymmetry was more marked in one limb than the other (Table 10).

TABLE 10 CONTROL LAMB METATARSAL EPIPHYSEAL ASYMMETRY

Lamb Number	Degree of Asymmetry	Limb Involved	Shorter Epiphysis
20	Minimal	RH	Medial
60	Moderate	LH	Medial
48	Moderate	RH	Medial
53	Minimal	RH	Medial
30	Minimal	LH	Medial
35	Minimal	Both	Medial
16	Minimal	RH	Medial

It should be stressed that in cases deemed to show moderate asymmetry the difference of level of epiphyses was only of the order of approximately 1.5mm whereas in those with minimal asymmetry a discrepancy of about 1mm was observed.

Similar discrepancies existed in the epiphyses of some of the experimental lambs in the early postoperative period. In those lambs slaughtered prior to one month postoperatively, nine lambs were detected to have epiphyseal discrepancy. Of this nine, however asymmetry existed in both limbs in six cases with no limb showing more obvious discrepancy. Of the remaining three lambs, all slaughtered prior to 2 weeks, two showed slightly greater discrepancy in the left hind limb, and one in the right hind limb. In all cases the medial epiphysis was slightly shorter.

In longer surviving lambs however a different and more distinct abnormality was detected in the metatarsal epiphyses. In such lambs a medial tilting of the lateral epiphysis occurred. This finding could be considered to represent lateral epiphyseal varus deformity as in the majority of cases the tilting had its origin within the epiphyseal bone substance. Such deformity resulted in a variable amount of apparent shortening of the lateral epiphysis relative to its medial counterpart. This abnormality was detected in seven cases and as soon as 2 months postoperatively (in lamb no 50). This finding was most pronounced in those lambs with severe metatarsal deformity. True bone varus was most apparent in the two longest surviving lambs (nos 17 and 13) in this group.

Epiphyseal asymmetry was minimal in those lambs with metatarsal valgus. Measurements made at the joint surface in these cases revealed no appreciable difference from the angular deviation measured at the metaphyseal-growth plate junction. In all cases, however there was a similar asymmetry to that noted in control lambs except that the lateral epiphysis was slightly shorter.

In all lambs with varus metatarsal deformity the medial epiphysis was slightly broader than the lateral epiphysis. In those lambs with metatarsal valgus, two (nos 18 and 14) had slightly broader lateral epiphyses.

In control lambs also the medial epiphysis was normally slightly broader.

b) Lateral Radiographs Estimation of deformity on lateral radiographs was only performed on tibiae. Radiographic positioning could not be standardised and estimates of deformity on such radiographs are likely to be even more subject to error than on anteroposterior projections.

The estimates of deformity obtained are presented in Appendix 1 tables 6 & 7.

There was no absolute correlation between the posteriorly directed deformity measured on lateral radiographs and the varus measured on anteroposterior plates. With posterior deformity the impression was gained that the angular measurement increased with passage of postoperative time in addition to the degree of varus present. Rotational deformity is undoubtedly of importance

in the production of these findings.

Tibial Torsion

Estimates made of tibial torsion have been tabulated and are presented in Appendix 1, tables 8 & 9.
In all cases torsion was in an internal or medial direction. External torsion was not encountered in any sheep tibia measured. Unfortunately in a number of lambs torsional estimates could not be made due to their tibiae being sectioned before incorporation of this measurement in the experimental method. The operated limb tibia (RH) of lamb no 37 sustained mid-diaphyseal fracture during the experimental period. Fixation was by means of a round section intramedullary pin. A torsional measurement was however made but is unlikely to relate to findings in other lambs.

The torsional measurements have also been represented graphically in fig 18. Measurements for LH and RH limbs are plotted separately.

Control lamb measurements demonstrate that tibial torsion increases gradually with advancement of age. Whereas in younger control lambs between 5 and 11 degrees of medial torsion was detected in longer surviving individuals torsion was of the order of 17-18^{0}. There may be a tendency for the control limbs to possess asymmetry with slightly greater torsion in one limb than the other but discrepancies noted may fall within the error of the technique of measurement.

In experimental lamb tibiae a progressively increasing difference between torsional measurements in each hindlimb occurred.

Such disparity became noticeable very soon after operation. For example one lamb (no 33) slaughtered after 2 weeks had 5^{o} difference in estimated torsion between limbs. Initially, in the early postoperative period, this disparity was due to an increased torsion of the LH (non-operated limb) tibia rather than to a reduction in torsion in the operated limb. With advancement of postoperative survival time however there was a progressive increase in torsion of the non-operated limb and reduction in expected torsion of the operated limb. Torsional deformity was greatest in those lambs with severe varus tibial deformity. In lambs with predominantly metatarsal varus, torsion of the non-operated limb conformed closely with values obtained for comparable aged control lambs in most cases. There was however a reduction in tibial torsion in the operated limb when compared with control measurements.

It was considered useful to identify the limbs of those lambs with predominantly tibial deformity in fig 18. In lamb no 15, who underwent no gross deformity, although the non-operated limb measurement conformed with that of controls, the operated limb had a relative reduction in tibial torsion.

In addition, tibial torsional measurements were made on a total of 58 boiled out bones that had been collected during the course of this investigation. Neither the age nor breed of these lambs was known. Thus, tibial torsion has been plotted against tibial length in fig 19. Such a choice of y-axis is open to criticism. Fig 19 does however demonstrate

torsional variation that existed in mature tibiae, the range being from approximately 9^{o} to 30^{o}.

The torsional measurement tended to be slightly greater in those bones with only a proximal epiphyseal line present than in those with unfused proximal and distal epiphyses. Fusion of epiphyses must represent an increase in age.

Metatarsal Torsion

Metatarsal torsional measurements are similarly recorded in App. 1, tables 10 & 11 and in Fig 20. In all cases metatarsal torsion was in an internal or medial direction.

Figure 20 demonstrates that in control lambs metatarsal torsion was minimal.

In experimental lambs however the LH (non-operated limb) metatarsi underwent an increased degree of torsion with advancement of the postoperative survival time. Metatarsal torsion was marked in all but one of the lambs that underwent predominantly tibial deformity, and in such cases the degree of torsion tended to be greatest in long surviving lambs. In those lambs with pronounced metatarsal deformity, metatarsal torsion was not increased in all cases, and generally was not of such great magnitude. Lamb no 37 with marked varus deformity of the distal left metatarsus has been included in fig 20. It is of interest that this lamb together with no 38, had marked metatarsal angular deformity and yet minimal if any torsional increase (see Appendi 1, tables 5 & 11). It is also noteworthy that four of the five lambs with valgus metatarsal deformity on anteroposterior

radiographs had pronounced torsion of the same bone. There was no marked torsional change in the operated RH metatarsi.

Tibial, Femoral and Metatarsal Length

Measurements made from anteroposterior radiographs of femora, tibiae and metatarsi are presented in tables 12 & 13 App.1 Measurements made from the dissected bones prior to radiography have been regarded as being too subject to error and have not been included.

1) Femoral Length: Femora were measured in two ways (fig 5A) Whereas the measurements made between the summit of the greater trochanter and the lateral femoral condyle could be made on all bones, those from the dorsum of the femoral head to the medial femoral condyle could not be determined in the operated limb of experimental lambs. The operated limb femora of both lambs no 38 and 37 sustained displaced midshaft fractures during the course of this study. Fracture repair was not attempted in either case.

a) Greater Trochanter - Lateral Femoral Condyle Measurement: In control lambs no obvious difference was detected between right and left limbs. In experimental lambs however the operated limb measurement was slightly greater from approximately 1-2 month postoperatively. The disparity tended to increase with advancement of postoperative survival time. It was discovered that in such cases there was both a slight increase in trochanteric dimensions and an increased diaphyseal length between the growth plate of the greater trochanter and the lateral femoral condyle.

There may be a greater disparity between limbs in those cases with the most severe limb deformities as it is interesting that the measurements of both femora were almost equal in lamb no 15 where no gross deformity occurred despite some change in trochanteric dimensions.

b) Femoral Head - Medial Femoral Condyle Measurement:
It is apparent that in control lambs, initially the femoral head measurement exceeded the trochanteric measurement. At a variable age however this situation was reversed and the trochanteric measurement was greater. The difference between each measurement tended to increase with advancement of age.

The same observation was detected in the unoperated limbs of experimental cases. It is possible that the disparity between measurements in some experimental sheep (for example nos 36 and 39) is greater than would be predicted from the control series results.

2) Tibial Length: A remarkable constancy existed in the tibial length measurements of both control limbs. In the experimental lambs however minor discrepancies were present between the operated and non-operated limbs from approximately 3 months postoperatively onwards. In no case however was such disparity pronounced as noted differences never exceeded a few millimetres. In such cases, the nonoperated limb was slightly longer. It seems equivocal whether such measurements can be considered accurate estimates of tibial length.

3) Metatarsal Length: The metatarsi were of equal length in both limbs of the control group. In experimental animals however differences existed between measurements of each limb. In cases with predominantly tibial deformity there was usually a slightly longer metatarsus of the non-operated limb. In those with predominantly metatarsal deformity however the deformed, nonoperated limb, metatarsus was slightly shorter than that of the operated limb. It is likely that measurements on deformed bones are subject to certain errors as is discussed below. In all cases differences in metatarsal length between limbs was at most of the order of a few millimetres.

Joint Mobility:

The goniometric measurements obtained for range of hock and stifle joint movement in both operated and nonoperated limbs together with control measurements are recorded in App.1, tabs 14&15. In addition those with qualitatively assessed reduction in fetlock joint mobility are indicated.

In control lambs there was a slight reduction in the range of stifle joint mobility with advancement of age.

In the experimental lambs however minor discrepancies between stifle mobility in each pelvic limb were detected. Discrepancies of less than 10^{0} may well fall within the expected margin of error of the goniometric method used. Some tentative suggestions can however be made. At about 6 months postoperatively there may be a tendency for reduced stifle movement of the operated limb which is mainly a manifestation of reduced flexion.

Both lambs slaughtered at 12 months postoperatively showed the reverse situation whereby stifle mobility was greater in the operated limb. The increased stifle mobility in the long surviving lambs appeared to be due to both increased flexion and extension ability.

Greater accuracy could be attained in measurements of hock joint mobility. Identification of the greater trochanter, and maintaining the goniometric arm in a straight line between this point and the centre of the stifle joint was probably the main source of error in stifle measurements. In the case of the hock joint however measurements were made on the medial aspect of the limb and it was relatively easy to align the goniometer arms with the relevent bone surfaces which lay subcutaneously.

The measurements obtained of hock joint mobility have been represented in histogram form in fig 21. Only the left hock joint however has been considered and the range of mobility is represented by a column extending from full extension to full flexion.

In the control lambs there was a gradual decline in hock joint mobility involving a progressive limitation of flexion and extension with advancement of age. There was no apparent difference in joint mobility between left and right limbs.

Similarly in experimental lambs slaughtered before 3 months postoperatively a gradual decline in mobility with no appreciable limb difference was detected.

After this time postoperatively however there was a marked

difference between those lambs which underwent predominantly metatarsal deformity and those with tibial deformity. Those lambs with metatarsal deformity demonstrated a progressive decline in mobility similar in character to that seen in control lambs. The experimental lambs with predominantly tibial deformity however demonstrated a severe reduction in hock joint movement of the unoperated limb. This marked reduction was associated with both limitation of joint flexion and extension and tended to increase in severity with passage of postoperative time.

In the case of the right hind, operated limb, there was no marked differences between joint mobility in experimental lambs and their correspondingly aged controls.

The fetlock joint had detectable reduction in range of mobility in all lambs slaughtered at and after 6 months postoperatively. Such reduction in movement was however only assessed qualitatively. The reduction in joint movement was more marked in those lambs with gross metatarsal deformity.

One lamb,no 54,slaughtered after 4 months also had observably reduced fetlock joint mobility. This lamb had a minimal amount of both distal tibial and distal metatarsal deformity, the latter being of slightly greater magnitude.

Tibial Plateau Angle (on anteroposterior radiographs)

The angle made to the tibial long axis by both the joint surface and metaphyseal-growth plate junction were measured. The angular measurements obtained are given in App.1 tabs 16&17.

In addition the results have been represented graphically in figs 22a and b.

a) Angle made to tibial long axis by joint surface: From fig 22a it can be seen that there was a gradual decline in the magnitude of this angular measurement in control lambs. Whereas initially angles of approximately 90^{o} were measured in some cases, towards the end of the experimental period measurements of approximately 85^{o} were obtained in control lambs. Minor differences between limbs were noted in the control group which may be within the margin of experimental error.

In experimental lambs disparity between limbs was detected which tended to be more marked with passage of postoperative time and in those lambs with severe deformity. Little can be inferred from lamb no 37 in which the RH tibia sustained a mid-diaphyseal fracture (the fractured tibia in this case had a relatively low angular measurement). Generally, the longer surviving lambs, with marked deformities demonstrated a reduced angular measurement of the LH nonoperated limb. In the longer surviving lambs disparity of angular measurement between limbs tended to be more marked in those with severe distal tibial deformity. Exceptions to these generalisations occurred however particularly in lambs slaughtered at an earlier age.

b) Angle made to tibial long axis by metaphyseal-growth plate junction: A similar gradual decline in the angle measured occurred with advancement of age in control lambs (fig 22b

The decline however was slightly more rapid than noted for measurements made at the joint surface. Minor differences between limbs were again noted in the control lambs, but such disparities are of doubtful significance.

As with the joint surface measurements, differences were noted between limbs in the experimental group of lambs. In most cases the disparity between limbs paralleled that noted at the joint surface whereas in some it did not. In particular lamb number 18 demonstrated minor disparity between limbs at the metaphysis, but relatively marked limb difference at the joint surface.

The minor degree of valgus orientation of the proximal tibial metaphysis was partially eliminated at the joint surface due to epiphyseal asymmetry. Epiphyseal correction may tend to increase with advancement of age and may be slightly reduced in some long surviving lambs (eg nos. 18 and 12).

Tibial Plateau Angle(on lateral radiographs)

Some difficulties were encountered on estimating the tibial plateau angle on radiographs in this projection.

Despite such difficulties measurements were made and can be regarded as estimates of the tibial plateau angle on lateral radiographs. The results are tabulated in Appendix I, tabs.18&19. In a few cases it was impossible to make adequate measurements. In such cases, no estimated angle has been given. In one lamb the tibia had been sectioned.

There was found to be a gradual increase in the measured

angle with advancement of age in control lambs. Whereas initially, in control lambs, the measured angle was of the order of 73^{o} at the end of the experimental period approximately 80^{o} was recorded. Minor differences occurred between limbs.

The experimental lambs similarly showed a gradual increase in the measured angle with passage of postoperative time. Discrepancy between the angular measurement in each limb however became more marked after approximately 4 months postoperatively. In some cases the measured angle was greater in the operated limb but in a number of the longer surviving lambs the nonoperated limb angle was greater. Because of the difficulties encountered in making these measurements, it is equivocal whether the latter observations can be considered real findings.

Femoral Neck Angle

Measurements obtained of the femoral neck angle are tabulated in Appendix I tables 20 & 21. The method of measurement chosen appeared in all cases to be as close an estimate as was possible.

However, in control lambs no real pattern emerged. The range of recorded measurements was from 57^{o} to 69^{o} but there was no apparent change in femoral neck angle with advancement of age.

In the case of experimental lambs measurements could only be made on the unoperated (LH) limb. Again no pattern emerged. There was a similar wide range of angular measurements (57^{o} to 70^{o}) with no apparent correlation with postoperative survival time.

Greater Trochanteric Base Angle:

Measurements of this angle was also performed. The results are presented in Appendix I tables 22 & 23. The range of observed angle was from 41° to 54.5° in control lambs and from 39° to 55° in experimental animals. Those lambs with severe deformities did have low angular measurements, but it is difficult to ascribe any importance to this observation when there was such wide variation of results in the control group. It was not possible to make comparable angular measurements in femora which underwent excision arthroplasty as in all cases the osteotomy cut included the most medial part of the trochanteric growth plate.

Relative contributions to longitudinal growth of proximal and distal tibial growth plates - Digby's Method.

The location of the tibial diaphyseal nutrient foramen and canal was remarkably constant. In all cases the nutrient canal could be identified entering the lateral aspect of the diaphysis at the proximal end of its middle third.

The results obtained following application of the method described by Digby (1916) have been tabulated and are presented in tables 24 & 25, Appendix I.

With Digby's method, control lambs showed relatively minor differences between limbs, of the distal tibial growth plate's contribution to bone growth. In all cases the contribution

was between 40% and 53.3% of long bone growth. No particular difference was noted with advancement of age.

In the experimental group, measurements could not be made in two cases in which the tibiae had been sectioned prior to incorporation of this technique in the methods. The experimental lambs possessed quite marked variation in the calculated contribution of the distal growth plate to longitudinal tibial growth. Measurements varied between 25.5% and 56.6%.

If this method of measurement is at all plausible a number of findings can be obtained from the results. The tendency in experimental lambs was for a more marked discrepancy between limbs to exist in measurements made from 4 months postoperatively onwards. In the longer surviving lambs,nos 14 and 18, the distal tibial growth plate of the non-operated LH limb produced a markedly reduced growth contribution. Both of these lambs had severe distal tibial deformity. However in most lambs slaughtered at an earlier date the distal growth plate contribution was greater in the nonoperated LH limb.

In all but one of the lambs (no 38) with marked discrepancy between limbs, severe tibial deformity was present. Lamb no 38 had quite marked reduction in growth contribution by the distal tibial growth plate of the nonoperated limb, but no tibial deformity was present.

In cases of discrepancy of distal tibial growth contribution between operated and non operated limbs, usually, associated with greater relative contribution of one limb there was a relative

reduction of contribution by the contralateral limb when compared with control measurements.

Femoral Condylar Measurements

Examination of radiographs revealed no grossly observable change in either the medial or lateral femoral condyles associated with deformity. The femoral condyles were similar in appearance in both control and experimental lambs.

Measurements made of the angle of the fovea capitis relative to the femoral condyles are presented in App.1 tabs 26&27. In control lambs no obvious pattern was observed. In all cases the measured angle was between 8 and 16^{o}.

In experimental lambs this measurement could only be made on the nonoperated limb where the femoral head and neck were intact. As with the control lambs there was no obvious pattern detected, the angle being between 9^{o} and 16.5^{o} in all instances. The lowest observed angle however in long surviving lambs was in lamb number 12.

Anteversion Angle

Estimates were made of the degree of femoral torsion in the manner described in the materials and methods. The results obtained have been tabulated in App.I tabs 28&29. In all cases anteversion and not retroversion was detected. The measured degree of anteversion in control lambs varied between 18^{o} and 26^{o}. Slight differences between each limb occurred but this finding may be due to the innaccuracies inherent in the technique of measurements. There was no apparent increase or decrease in

anteversion with advancement of age.

In experimental lambs measurements could only be made on nonoperated femora. Variation was similarly encountered. The range of variation was however slightly greater being from 14^{o} to 30.5^{o}. The relatively high value of 30.5^{o} anteversion occurred in lamb no 12 slaughtered at 12 months postoperatively and the lowest value of 14^{o} was seen in lamb no 49 at 2 months postoperatively. Long surviving lambs other than no 12 had a comparable degree of anteversion to their corresponding controls. Slightly high values of anteversion were obtained in some lambs (46, and 37 especially) but there was no correlation with degree of limb deformity or postoperative survival period (see figure 23)

Femoral torsional measurements were also made on a total of 30 femoral specimens which had been collected. Results of measurement of these bones has been presented in fig 24 As with tibial bone specimens the age and aetiology of such bones was unknown. Similarly, choice of femoral length as the y-axis in fig 24 is open to criticism as is discussed below. No real pattern emerged. However those bones with a distal femoral epiphyseal line present tended to have a slightly lower anteversion angle than those in which the distal femoral epiphysis was completely unfused. The former bones must be from more skeletally mature lambs. It is impossible however to attach any importance to such a finding as breed differences may exist. The wide range of possible anteversion

in skeletally mature femora is however demonstrated.

Consideration of limb torsion

The torsional measurements for each major long bone of the pelvic limb has been determined. It is useful however to amalgamate the tibial and metatarsal results and obtain a value of total limb torsion between stifle and fetlock joints. In addition it is of interest to compare such torsional change with axial femoral rotation in order to detect any correlation. This has been done and the results are presented in App.1 tabs 30 and 31 for control and experimental lambs respectively.

There is seen to be a gradual increase in the magnitude of medial rotation between stifle and fetlock joints in control lambs. This was largely the result of increased tibial torsion, metatarsal torsion being minimal. No correlation is apparent between the magnitude of anteversion and torsion distal to the stifle joint in control lambs.

In experimental lambs gross increase in medial torsional deformity distal to the stifle joint occurred. This finding was due to an abnormal degree of torsion in both tibiae and metatarsae. As with control lambs there is no immediately apparent correlation between lower limb torsion and femoral anteversion. The high value of anteversion in lamb no 12 and perhaps no 37 has been noted above. The lowest values of anteversion were however encountered in those lambs slaughtered at approximately 1 - 2 months postoperatively.

PART B

Discussion

Browne (1936) stated "There are certain disasters to which the body is liable - for instance cuts, fractures and burns - which could equally well be suffered by a lifeless model with the same physical qualities. There are others such as bacterial infections and disturbances of growth which can only affect living tissues".

Disturbances of growth are by definition affections of the skeleton that manifest during the growth period of an animal, and one of their unfortunate properties is that they tend to progress as long as growth potential remains.

The experimental lambs that have been used in this study demonstrate the reproducibility of the varus limb deformity that was previously noted (Duff 1975; Duff and Campbell 1978). These animals also demonstrate the progression of deformity that occurs with advancement of age following induced limb imbalance. Although one long surviving lamb (no 15) had no gross limb abnormality such a result may well be regarded as being fortuitous and it is interesting to speculate why deformity did not occur.

The experimental plan that has been followed involved unilateral excision of the right femoral head and neck in all cases. Operation on the same limb in each individual has greatly facilitated both the making of observations and the presentation of results but is open to some criticism.

Dominance in both pectoral and pelvic limbs in man has been the subject of a number of reports (Lowrance and Latimer 1957; Latimer and Lowrance 1965; Chhibber and Singh 1970; Pande and Singh 1971). Such studies have involved measurement of both the weight and dimensions of bones and significant differences between right and left limbs is reported. Similarly, Singh (1971) has described one sided dominance in rabbits and frogs and has pointed out that there is no constant relationship in dominance of the pectoral and pelvic limbs. In approximately half of the rabbit and frog skeletons studied a crossed relationship of dominance occurred with for example, a dominant right forelimb being associated with a dominant left hindlimb. Pande and Singh (1971) weighed both bones and muscles in the limbs of human foetuses and found highly significant differences between the right and left sides. Such findings tend to suggest that the use of one limb in preference to another is inherited rather than acquired. It is likely that one sided dominance also exists in other animals. For example, horses are required by their gait pattern to have a leading leg as are dogs. Limb dominance has not been reported to occur in sheep but a certain amount of circumstantial evidence can be gathered from the results of this study. For example in many of the control lambs anteversion angle and tibial torsion estimates were slightly greater in one limb of the animal. Bone length discrepancy between limbs was however practically non existent. Limb dominance could certainly

influence some of the results in experimental lambs undergoing the procedure involved in this study.

In the rat at least, mechanical differences in the strength of the growth cartilage associated with difference of sex appear to be established. The influence of sex on the relative susceptibility of the growth cartilage to injury in other species is not known. One wonders if sex differences in mechanical strength are of importance in producing the marked tendency for some clinical conditions to be commoner in either males or females. In this study the sex ratio of lambs was 2:1 in favour of females. In the group of lambs utilised for part A of this study a similar preponderance of females occurred. Such an unequal distribution was unfortunate but unavoidable as lambs were purchased as they became available and selection would have delayed this study to an impractical extent. There was however no obvious influence of sex on ensuing deformity although all of the long surviving lambs with pronounced deformity were female. A sex difference may be present in the chronology of epiphyseal fusion in sheep which would suggest sex differences have some influence on growth cartilage. There may also be histological differences in the growth cartilage as is reported to occur in other species (Morscher 1968).

From Part A of this study it can be inferred that weekly radiography of the pelvic limbs in both the experimental and control lambs that have been used, is unlikely to have influenced the growth cartilage.

It can also be stated that at the time of operation in experimental lambs rapid growth was occurring. Thus limb imbalance has been imposed on lambs at a period of pronounced growth plate activity. At those bone extremities where deformity occurred, between 10 and 12 months of growth potential existed at the time limb imbalance was produced. The growth plate deformities that have been produced by the procedure described in this study were predominantly varus. Approximately 50% of cases observed after 2 months postoperatively had grossly observable change at the distal tibial site and a similar percentage at the distal metatarsal site. There was no obviously apparent reason why deformity developed at one site and not the other. In many instances however a variable amount of abnormality was noted at the site which was not predominantly involved.

The deformity was progressive and although grossly observable after about 2 months postoperatively was detectable on radiographs prior to this time. The pattern of deformity was essentially the same at both the distal tibial and distal metatarsal sites. There was however no absolute correlation in the degree of deformity and postoperative survival period.

It is interesting that in the longer surviving lambs with gross tibial deformity, a degree of metatarsal valgus developed. This valgus, by producing deformity in the opposite direction to that at the tibial site, effectively brought the foot more into line with the original long axis of the limb. Such metatarsi

had many of the radiographic abnormalities that were noted in those bones which underwent varus deformity. One wonders if the metatarsi of these lambs were in a pre varus state prior to developing demonstrable valgus. In lamb no 12 in which metatarsal valgus was noted an impression was gained from close scrutiny of weekly radiographs that at approximately 30 weeks postoperatively a minor amount of varus deformity was present. No such impression was gained in the other sheep with measurable valgus.

It should be stressed that although estimates have been given of the degree of angular deviation in deformed bones errors are inevitable in attempting to measure three dimensional deformities from two dimensional radiographs. Authors such as Van Nuys (1940) however failed to stress this severe disadvantage of radiographic measurements. Such errors are likely to be much greater when the weekly radiographs, rather than postmortem radiographs, are considered. At least in the latter an attempt could be made to standardise the radiographic projection for the distal extremity of each bone.

The estimations made of varus angular deformity did however reveal that in all cases a minor degree of correction of the deformity occurred between the metaphyseal growth plate junction and the joint surface. The distal tibial epiphysis is normally of slightly greater depth medially. This fact, in conjunction with medial growth plate thickening are possibly the reasons for such correction in cases of tibial deformity. At the

distal metatarsal site a different factor is involved. It should be stressed that in lambs the medial and lateral metatarsal epiphyses are separate entities and thus may respond to deformity differently. It was discovered that in lambs with more severe deformities, the lateral epiphysis underwent varus deformity. The medial tilting so produced resulted in a discrepancy in epiphyseal articular surface level. The lateral epiphysis thus appeared as if it were slightly shorter and the angular deformity recorded at the joint surface was reduced accordingly. In some deformed metatarsi obvious discrepancy existed in dimensions of the medial and lateral epiphysis see plate 23B. The lateral epiphysis was shorter and lesser width than the medial epiphysis in such cases.

In addition to the lateral epiphyseal deformity medial growth plate thickening is likely to account in part for the noted discrepancy between deformity at joint surface and metaphysis. The lateral epiphyseal changes probably account for the greater degree of angular deformity correction at the joint surface in metatarsi. Epiphyseal asymmetry is undoubtedly important in normal bone development. It was noted in both control and experimental lambs that the proximal tibial epiphysis modified the slight valgus orientation of the proximal tibial metaphysis. Thus measurements made at the joint surface demonstrated in most cases a lesser degree of valgus than observed at the metaphyseal growth plate junction. Such a process must involve slightly

greater lateral epiphyseal thickness. That this does indeed occur can be appreciated from scrutiny of radiographs. Thus there is differential growth of the medial and lateral sectors of the proximal tibial epiphysis and there is an indication that asymmetry increases with advancement of age. The lateral epiphysis would appear to grow relatively faster with resulting greater correction of valgus in older lambs.

It has been suggested above however that the distal tibial epiphysis similarly has asymmetry but that the medial sector is thicker at this site. Measurements of angular deviation of the distal tibia obtained in control lambs is of interest. Although the measurements made were small, and although errors were predicted with such measurements, all but a few lambs had a small degree of varus orientation of the distal tibial metaphysis. The fact that only a few cases do not conform to this generalisation tends to suggest the observation is real rather than apparent. If a degree of varus is accepted as being present at the metaphysis the noted epiphyseal asymmetry would tend to reduce or correct angulation at the joint surface. This is seen to occur, as in most cases the varus is reduced to a degree with greater correction being observed in the longer surviving lambs.

It has been mentioned at the commencement of this study that the tibia of the sheep is not perfectly straight but has a sinuous curve when viewed in an anteroposterior direction. The sinuous curve can be seen on radiographs to result in slight metaphyseal valgus proximally with slight metaphyseal varus being observed

distally. The epiphyseal asymmetry that has been discussed above will effectively modify the extremities of the sinuous curve. The result is a closer approximation of the articular surfaces to perpendiculars made to the tibial long axis. It is convenient to refer to such epiphyseal asymmetry as 'physiological', as it would appear to be a normal component of tibial growth at least in the ovine skeleton.

A number of deformities that have been mentioned in the introduction to this section involve gross epiphyseal asymmetry. Such asymmetry is either the cause of deformity or results in accentuation rather than correction of any deformity present in the metaphysis. Notable examples of clinical deformities involving epiphyseal asymmetry include tibia vara and Madelungs deformity in man and genu valgum in giant breeds of dog and in man. At the concavity of the deformity in such cases, there is hypodevelopment of the associated epiphyseal segment. Possible reasons for such conditions arising will be discussed in part D of this study.

In the lambs of this study, 'physiological' epiphyseal asymmetry increased with deformity. Altered epiphyseal growth has been reported to contribute to the spontaneous correction of experimentally produced deformities (Ryoppy & Karaharju 1974; Karaharju, Ryoppy & Makinen 1976).

There was a remarkable constancy in the appearance on radiographs of the growth plate at both distal tibial and

distal metatarsal sites in experimental lambs. Concomitant with the initial detection of angular deformity the lateral growth plate sector had associated with it an area of radiolucency. The lucent area gradually increased in dimensions and eventually became located more proximally in the metaphysis. In many instances this defect gradually came to lie in the region of metaphyseal remodelling and was largely removed.

A condition has been described in growing children which bears an obvious similarity to the observations in sheep. Localised fibrous lesions have been described in the metaphyses of the long bones in children by a number of authors including Hatcher (1945); Ponseti and Friedman (1949), Adams and Gouldner (1953); Cunningham and Ackerman (1956); Campbell and Harkess (1957) and Brower, Culver and Keats (1971). A certain amount of confusion exists due to different authors referring to the same clinical entity by a number of different names. It would seem that the term metaphyseal fibrous defect is most descriptive of the condition. Such lesions were thought to be nonmalignant neoplasms and indeed cortical irregularities which may be a later manifestation of the condition have been considered to simulate malignancy (Kreis and Hensinger 1971; Brower, Culver and Keats 1971). The lesion is seen on radiographs as a localised area of lucency tending to lie eccentrically in the metaphysis. Most commonly the femur is affected and the lesions may be bilateral. The bones of the pelvic limb are normally involved although Jaffe and Lichtenstein (1942) (cited by Hatcher 1945) are reputed to have described a case in an ulna and

Ponseti and Friedman (1949) described the condition in a child's humerus. Lesions become manifest most frequently in children between the ages of 4 and 8 years and are always associated with the period of growth.

It is useful to present the typical radiographic appearance of metaphyseal fibrous defects as given by Hatcher (1945). Initially there is a limited area of reduced density in the metaphysis close to the growth plate. Later the area tends to elongate in the direction of the bone axis and show sharp demarcation from surrounding normal bone by a narrow shell of bone sclerosis. With growth the defect comes to lie further from the growth plate and may eventually occupy a part of the cortex.

Such a description could equally be applied to the findings in the experimental sheep of this study. A line of increased density was similarly seen to demarcate the lesion.

Hatcher (1945) suggests that if situated near to the margin of the metaphysis, longitudinal growth and metaphyseal remodelling will obliterate it.

The proximal humeral case described by Ponseti and Friedman (1949) was observed over a period of thirteen years during which time three separate defects appeared and regressed at the same metaphyseal site. Their regression was associated with their becoming situated progressively further from the growth plate, a fate that has been recorded in the lambs of this study.

Campbell and Harkess (1957) remark that the average duration

of the lesions in children was found by Sontag and Pyle (1941) to be 29 months. Although the lesions noted in experimental lambs normally took between ten and twenty weeks from their recognition to their removal, the rapid skeletal maturation in sheep relative to that in man should be borne in mind. Also , the majority of cases in children appear to result in a defect persisting in the metaphysis whereas in lambs the lesion was much more peripherally situated.

The defect in children is usually medially placed whereas that induced in lambs was lateral. It is likely that in lambs, with progressive varus deformity the metaphyseal defect would come to lie subperiosteally as varus increased (fig 25).

In children the average age of appearance of metaphyseal fibrous defects is reported to be four years. It is perhaps interesting that children may undergo a valgoid phase of limb development which reaches a maximum at approximately this age. With genu valgum, the limb angulation and medially sited lesion would approximate with the varus and laterally sited lesion in sheep. The observation that metaphyseal defects in children can be unilateral or bilateral correlates well, as genu valgum although normally bilateral is stated to be sometimes more marked in one lamb than the other (Dunbar 1958). (That one limb may be more severely affected is also demonstrated in the illustrations of genu valgum presented by Pistevos and Duckworth, 1977;). The question of one sided limb dominance is obviously of relevance here.

Both Hatcher (1945) and Campbell and Harkness (1957) found associated osteochondrosis lesions in approximately one third of cases with metaphyseal fibrous defects. Most frequently Osgood-Schlatters disease, osteochondritis dissecans and patellar osteochondritis were detected. Such an association led Hatcher (1945) to suggest that the metaphyseal defect may result from vascular derangement. A common aetiological factor resulting in osteochondrosis and a metaphyseal fibrous defect would be equally plausible.

A metaphyseal defect is reported to occur in infantile tibia vara. In this condition however the defect occurs in the medial beak-like projection and thus on the concave aspect of the deformity rather than the convex aspect as has been described in experimental lambs. Just as the metaphyseal defect that has been described above in children may be associated with osteochondroses, so tibia vara has been reported concomitant with other deformities such as osteochondritis dissecans, Osgood-Schlatters disease, Perthes disease and Kohlers disease (Lovejoy, Wood and Lovell 1970). It is not know whether such an association is significant or purely coincidental. The possible aetiology of tibia vara metaphyseal defects are considered in more detail elsewhere in this study.

Lateral metaphyseal defects were reported in the distal femora of cases of genu valgum in giant breeds of dog (Riser, Parkes, Rhodes and Shirer 1969). In such dogs, there may also be epiphyseal defects on the lateral aspect of the hypo-

developed lateral femoral condyle. The reported defects however can be considered to be on the concave aspect of the deformity and thus may have some resemblance to the defect reported in cases of infantile tibia vara in children. Some interesting growth plate changes have been described in association with genu valgum in dogs which may offer an explanation for the defects in the metaphysis. This matter is considered in detail elsewhere in this study.

It thus seems that metaphyseal defects can occur on both the concave and convex aspects of a deformity. One wonders whether the metaphyseal fibrous defect that has been described in children is truly comparable to the defect noted in tibia vara. In the majority of radiographs of metaphyseal fibrous defects in children that are present in the literature however, if anything, the knees appear slightly valgoid and not varoid.

Radiolucent metaphyseal defects are also reported to occur in certain developmental abnormalities in man (Kilburn 1973). Such defects consist of cartilaginous tissue. Although Gelbke (1951) noted cyst-like lesions in the proximal ulnar metaphysis of a dog in which traction had been experimentally applied to the associated epiphysis, the exact location of the lesions cannot be discerned from the photographs accompanying his report. It would seem however that the metaphyseal cysts were adjacent to that part of the growth cartilage submitted to the greatest degree of traction, which may make them comparable to those induced in the lambs of this study.

Associated with angular deformity in both metatarsal and tibial bones, an increase in amount of both cancellous and cortical bone became apparent medially. In the introduction to this section a number of bone deformities have been described, the majority of which similarly have associated with them bone hypertrophy on the concave aspect of the angular deviation. Similarly peaking of metaphysis and epiphysis normally occurs in deformities which have their origin in the growth cartilage and result in angulation of the joint surface. Bone hypertrophy in such situations has been the subject of much speculation and experimentation, and will be dealt with more fully in Part D of this study.

Another abnormality commonly encountered in association with angular deformity of a bone is abnormal axial rotation. For example, abnormal tibial torsional deformity occurs in congenital tibial bowing, genu valgum and varum in man and animals. The frequent association of both torsional and angular deformity tends to suggest a common aetiology.

In the lambs of this study both normal and abnormal degrees of tibial torsion were detected. The results obtained have confirmed the suggestion by Lanyon (1977) that tibial torsion in normal lambs is in a medial direction and increases in magnitude with advancement of age. Despite the gradual progression of tibial torsion noted in control lambs a surprising degree of variation was encountered in mature bone specimens which were acquired and 'boiled out'. There is likely to be individual

and breed variation in the degree of torsion and nothing was known of the sheep from which these bones originated.

Use of tibial length as the y-axis in fig 19 is questionable. Tibial length cannot be strictly considered to be an estimate of skeletal age, as individual and breed variations in limb proportions are known to occur (the same criticism can be made of anteversion angle measurements in fig 24). Identification of those tibiae with incomplete epiphyseal fusion tended to correlate with the findings in the control lambs of this study, despite the fact that the tibiae represented in fig 19 are likely to have originated from lambs of different breeds.

The results in experimental lambs demonstrate that torsional abnormality appeared before noticeable angular deformity in two of the lambs slaughtered at 2 weeks postoperatively. Had torsional measurement been possible in more of the short surviving lambs it is quite probable that more cases would demonstrate this finding. It is interesting, that both Rang (1969) and Rudy (1971) comment that in the tibia of children and dogs respectively, torsional deformity often exceeds angular deformity. It is noteworthy however that the metatarsi of two lambs with severe angular deformity had minimal rotational abnormality. The correlation between torsional and angular deformity is therefore far from absolute. It is also apparent that torsional abnormality in tibiae is not necessarily most marked in those with the most severe deformity.

The possible aetiology of both torsional and angular deformity is discussed in Part D of this study.

Metatarsi normally exhibit minimal axial rotation, even in control lambs approaching the time of epiphyseal closure. The remaining major long bone of the pelvic limb, the femur, does however normally have a torsional component.

Measurement of femoral torsion is far from satisfactory, but the best approximations must result from observations on dissected bone specimens as has been performed in this study. Use of the fovea capitis as a reference point however may be open to criticism. Kingsley and Olmsted (1948) observed that in man the femoral head was not centred on the femoral neck in almost 70% of bone specimens examined. Although the same observation was not made in the sheep of this study, if a similar feature was present the estimates of anteversion would be erroneous.

The wide variation of anteversion in control sheep is of interest. Similarly variation is recorded in man by a number of authors including Kingsley and Olmsted (1948). There is known to be a physiological amount of anteversion which is not manifested by axial rotational abnormality at the knee joint. In such cases anteversion is accounted for by the normal position of the femoral head in the acetabulum (fig 26). Thus minor variations in anteversion may be dissipated in two ways. Either there is a slight increase in medial rotation manifested at the stifle joint or, there may be a change in the normal position of the femoral head in the acetabulum (fig 26).

One wonders if the high proportion of femoral heads in man that are not centred over the femoral neck is due to such compensation (fig 26). With extreme values of anteversion in man medial limb rotation is reported (Alvik 1962; Machanik 1968; Harris 1972; Mitchell 1972; Bailey and Wooley 1976) but anterior subluxation of the femoral head may also occur with limb position being normal (Alvik 1962).

It is difficult to apply bipedal findings to the quadrupedal limb. The normally flexed position of all of the major joints of the pelvic limb in the quadruped makes appraisal much more three dimensional. However, assuming that the position of the femoral head in the acetabulum is constant a marked increase in anteversion angle would result in medial distal femoral torsion. Subluxation, if it occurred would be in an anterodorsal rather than an anterior direction. In the dog, abnormally large degrees of femoral anteversion have been associated with clinical hip disease (Nunamaker 1974).

In an earlier publication (Nunamaker, Biery and Newton 1973) the anteversion angles of 34 normal canine femora measured from radiographs was between 12^{o} and 40^{o}. The mean was approximately 27^{o}. In the same year, another worker (Riser 1973) measured femoral anteversion on dissected bones of mature greyhounds and obtained a mean value of 10^{o} with measurements ranging from 3^{o} to 20^{o}. Riser (1973) discovered that in the dog the anteversion angle was 0^{o} at birth but increased gradually with advancement of age. Limited observations were however made.

No such pattern emerged in the sheep of this study. All radiographic techniques of anteversion estimation are subject to error, but Nunamaker, Biery and Newton (1973) claim precision with their method. Although these authors do not state the breeds of dog studied, breed differences may account for the discrepancy between these two studies and for some of the variations encountered in the experimental lambs. In another, earlier study, Riser and Shirer (1966) estimated anteversion in a large number of 1-28 day old Alsatian femora and discovered 0^{o} anteversion in most cases with a range of from 0^{o} to 10^{o}. Thus, initially at least, the dog appears to exhibit minimal anteversion irrespective of breed. No reports of anteversion angle measurement in neonate sheep have been discovered. Possibly the same occurs, but there was found to be little variation in the degree of anteversion achieved at 3 months and 12 months of age.

The range of measurements obtained in control lambs may be regarded as representing normal variation. The slightly greater range of variation encountered in experimental lambs may simply reflect the greater number of individuals in which measurement was made. It is tempting however to consider that the high degree of anteversion encountered in lamb no.12 is significant but it is impossible to do so.

Just as there is physiological anteversion in man, there is probably also a normal position of the ovine femoral head in the acetabulum which would be unlikely to be the same in different individuals. In such a manner the anteversion detected in lambs

may be lost from the limb and thus not result in axial torsion. It is said that in man there is no significant correlation between femoral and tibial torsion (Elftman 1945). The findings of this study suggest the same is true in sheep. In sheep, medial tibial torsion normally occurs with minimal associated metatarsal torsion. It is not known however what contribution the tarsal bones make towards axial rotation in lambs. In man Elftman (1945) reported that with increase in tibial torsion, deviation of the foot increases more slowly. Barnett (1955) stated that the trochlea of the human talus is deviated with respect to the long axis of the foot which would account for Elftmans (1945) observation. In artiodactyls Barnett (1955) described the trochlea of the talus as being parallel to the long axis of the foot. Thus it can be inferred that the talus in sheep is likely to normally have minimal axial rotation. The situation in experimental lambs with gross deformity is unknown.

A final point of interest taken from the human literature on limb torsion is that Alvik (1962) states that in cases of intoeing due to increased femoral anteversion the child tries to overcome the deformity by abducting the forefoot. Clinical flatfoot results. Certain analagies can be drawn between postural flatfoot in children and the metatarsal valgus that occurred in some of the lambs of this study.

A number of authors including Rogers (1934) and Shands and Steele (1958) have described an increase in femoral neck

angle or angle of inclination associated with increases in femoral torsion. The converse coxa vara in association with retroversion has been reported (Shands and Stcole 1958). Rogers (1934) does however remark on the difficulties of estimating the femoral neck angle from radiographs, as increased anteversion itself produces an apparent coxa valga. Klinefelter (1946) determined experimentally the marked influence of positioning on the measurement of the projected bone angle. With such studies in mind, it is difficult to consider in vivo measurements of femoral neck angle as anything but estimates. In addition to anteversion, factors such as anterior femoral curvature undoubtedly influence the measured angle (Dunlap et al 1953). Some of these problems at least have been minimised by ensuring close contact between the proximal femur and radiographic cassette in measurements made on dissected sheep femora. In man, there is reported to be a gradual decline in the femoral neck angle from birth to old age (Von Lonz and Wachsmuth 1938; cited by Amstutz and Freiberger 1962; Bubic and Socerov 1967; Houston and Zaleski 1967) but no such tendency occurred in the lambs of this study. In those lambs with slightly higher values of anteversion there was no relative increase in measured femoral neck angle. If the observedassociation between anteversion and femoral neck angle in man is real, it may well only manifest in connection with truly abnormal angles. Certainly, measurements on lambs femora were not shrouded with the difficulties encountered in making in vivo measurements.

Of most interest however are the findings of Bubic and Secerov (1967) who studied the angle of the femoral neck in ballet dancers. Such hip joints were considered by those authors to be particularly subjected to mechanical stress. Bubic and Secerov (1967) discovered limb differences, the limb most often used for support during dance procedures possessed the least femoral neck-shaft angle. Houston and Zaleski (1967) similarly described the influence of function on femoral neck angle in children.

No comparable findings were discovered in the experimental lambs. The negative findings in lambs may however reflect the different mechanics of the bipedal and quadrupedal pelvic limb. In addition the femoral head and neck in sheep is of a more compact nature and may thus be better able to withstand mechanical stimuli. Applying Bubic and Secerov's (1967) finding to a quadrupedal limb would suggest that overuse of that limb may result in a degree of coxa vara together with an element of retroversion of the proximal femur.

Establishment of femoral neck angle may be influenced by the synchronous or asynchronous growth contributions of adjacent growth plates. Compere, Garrison and Fahey (1940) obtained coxa vara in experimental goats following experimental epiphyseodesis of the capital femoral growth plate. Greater trochanteric epiphyseodesis resulted in coxa valga. Although asynchronous growth may account for the gradual decline of femoral neck angle initially, it cannot explain the continued decline in the mature skeleton. Alterations in femoral neck angle in the

mature skeleton imply bone remodelling is occurring in response perhaps to mechanical factors.

Measurements from radiographs in this study revealed an asynchronous epiphyseal growth of the greater trochanter and femoral head in lambs. Initially the femoral head measurement was greater than that from the trochanter but later this situation was reversed. There was no change in this relationship in the nonoperated limb of experimental lambs.

In operated femora trochanteric changes were observed. There was a slight increase in dimensions of the greater trochanter together with a greater contribution by its growth plate to longitudinal femoral growth. Such findings may result from stimulation of the trochanteric growth plate by adjacent trauma with concomitant changes in the epiphysis. Perhaps this is a case of stimulated bone growth by osteotomy induced hyperaemia. Alternatively, loss of the growth contribution by the capital femoral growth plate may result in compensatory increased activity of the greater trochanteric growth cartilage and perhaps also that of the distal femur. The latter suggestion is in agreement with Hall-Craggs (1968; 1969) postulated intrinsic growth control mechanism of bone. Compare, Garison and Fahey (1940) however obtained only 2mm of increased femoral growth in a goat which sustained partial epiphyseodesis of the greater trochanter. They explain such growth stimulation on the basis of increased coxa valga and suggest hyperaemia may be involved.

If hyperaemia is the cause of the observed increased dimensions and activity of the trochanteric site one wonders

why only minimal length discrepancy (0.5 mm) between limbs was present in lamb no.15. There was however increased trochanteric dimensions of the operated femur. The minimal length discrepancy cannot be explained by either of the two suggestions made above. If any importance is attached to this anomaly, it must be inferred that other factors have some influence on the behaviour of the trochanteric growth plate and epiphysis. Such factors must be associated with contra-lateral limb deformity, as lamb no 15 had minimal skeletal change following excision arthroplasty.

A further test of Hall-Craggs suggestion is provided by measurements obtained of tibiae in the experimental lambs.

It is difficult to know whether Digby's method can be relied upon to provide accurate estimates of growth from each tibial extremity. However, results were obtained which are worthy of comment. The functional tibial length of both the operated and non-operated limbs remained relatively unchanged. The functional length of each bone has been taken as between the proximal and distal articular surfaces. Undoubtedly, had measurements been taken to account for angular deformity, differences between limbs would have been observed. The non-operated limb would be slightly longer but due to innaccuracies inherent in measuring angular deformity such measurements have not been considered. The minor disparity between tibial length of each limb noted in some individuals is probably due in part to magnification, the deformed bones being slightly enlarged

at their distal extremities. Measurements made by Digbys method have similarly been made to the joint surfaces and thus have also not taken angular deformity into account. Two of the three long surviving lambs, with severe tibial angular deformity have a gross reduction in diaphyseal growth contribution by the distal tibial growth plate. It would be reasonable to suppose that growth cartilage changes inherent in angular deformity could result in disruption of normal growth contribution. Such a supposition however did not apply to the longest surviving lamb, no 12. If the reported findings are an accurate representation of the diaphyseal growth from the distal extremity it can be concluded that the contribution of the proximal growth plate has been increased. Increased contribution by the proximal tibial growth plate would comply with Hall-Craggs (1968; 1969) postulated intrinsic bone growth control mechanism. Whatever the true contribution of the distal tibial growth plate in markedly deformed bones is , it is remarkable that the functional bone length is not grossly affected by the bone undergoing axial deviation. To retain functional bone length there must be a controlled increase in total bone length to accommodate angular deformity. Such a finding itself would tend to imply an intrinsic control mechanism in the ovine tibia at least.

It is unfortunate perhaps that bone markers were not incorporated in the tibiae of experimental lambs. On planning the experimental design, it was considered that introduction

of metallic markers would have produced another variable in the tibiae and thus it was decided to retain as near physiological conditions as possible.

Assuming some degree of accuracy of Digby's method, it can be inferred that throughout the period of experimental study there is relatively little change in the contribution made by each extremity to tibial diaphyseal growth in control lambs. It may also be stated that gross derangement of the growth plate, associated with marked deformity is not essential for disturbance of growth contribution. For example, a relatively low contribution to growth by the distal growth plate was estimated in lamb no 38 in whom no gross tibial deformity was observed. This case may however simply represent normal variation. A maximum of 5% discrepancy between estimates of each limb was encountered in control lambs. This finding would tend to imply that the grossly abnormal estimates obtained between limbs in long surviving experimental lambs represent real changes.

Metatarsal length must be entirely derived from the distally situated growth cartilage, assuming that longitudinal interstitial expansion of bone growth, a long discredited theory does not occur. (Longitudinal bone growth has however been described in the absence of growth cartilage; Hellstadius 1950). If the suggestions that have been made on tibial estimations are true, then there could be no correction of bone length discrepancy proximally. Thus, functional bone length would not

be so readily restored or maintained in cases of metatarsal deformity. It is noteworthy therefore that in cases of longstanding metatarsal deformity, a discrepancy was found between metatarsal length of each limb. In all cases the deformed bone was shorter. It may well be that the actual bone shortening is of greater magnitude than that represented in the results, as the enlarged distal extremity of deformed metatarsae would produce magnification on radiographs. Thus the suggestion that proximal tibial growth plate activity may play a major role in maintaining functional bone length of the deformed tibia is augmented.

Of those lambs with metatarsal valgus three failed to show a marked discrepancy in bone length between each limb. Such cases however had minimal angular deformity. The slightly greater observed length of non-operated limb metatarsi in earlier cases of varus deformity may result from magnification on radiographs, be due to error of the measuring technique or represent a minor degree of growth stimulation. It is perhaps relevent that lambs nos 13 and 17 with no tibial deformity had a discrepancy in tibial length of a few mms. between limbs. Such discrepancy was in favour of the non-operated limb. Discrepancy of tibial length in these two cases cannot be accounted for on the basis of magnification on radiographs.

The possibility of diaphyseal fracture resulting in both a local and general bone growth stimulation has been described

briefly in the introduction to this section. In the lamb that sustained tibial fracture shortening relative to the contralateral tibia occurred but it is possible that the intramedullary pin that was inserted has influenced growth. Round section pins are however reported to have no detrimental effect on bone growth even when passed through the growth cartilage. The cases of femoral fracture are more difficult to evaluate because of the considerable overriding that occurred. In lamb No. 38 however the fractured bone ends could be discerned amongst callus on radiographs and although approximately 2cms of overriding had occurred the femoral length discrepancy was only approximately 1cm. Thus the distal femoral growth cartilage and/or trochanteric growth plate must have had increased growth activity as the proximal growth plate had been excised.

It has already been mentioned that the normal ovine tibia has a sinuous curve to its anteroposterior viewed contour. Perhaps this in part explains why a gradual decline was noted in the measured tibial plateau angle with advancement of age Salenius and Vankra (1975) made observations on a large number of anteroposterior radiographs of the proximal tibia in children. These authors found that before the age of one year there was pronounced varus of the proximal tibia which changed into valgus at between $1\frac{1}{2}$ and 3 years of age. Such findings would correlate with the observed varoid and valgoid phases of human pelvic limb development.

Measurements made of the degree of femoral condylar tilt

relative to the fovea capitis were similar in both control and experimental lambs. It can be deduced however that in sheep femora increase in diaphyseal length is matched by increase in length of the femoral neck (fig 27) or that curvature of the femoral diaphysis occurs. Although there was no apparent correlation between the estimates of femoral condylar tilt and those of the proximal tibial plateau, it is perhaps significant that in long surviving lambs the lowest measured proximal tibial angle was in nos. 14 and 12 both of whom had severe distal limb deformity. These cases may perhaps be legitimately referred to as exhibiting a slight increase from normal proximal tibial valgus tilt.

Satisfactory estimates of tibial plateau angle from lateral radiographs were difficult to obtain in lambs. Moore and Harvey (1974) cite Bohler (1958) who apparently found that when viewed laterally the tibial plateau in man slopes posteroinferiorly 5-10^{o} from the horizontal axis of the tibia and thus the plane of the plateau is approximately 80^{o} relative to the tibial long axis. Angles of similar magnitude were measured in experimental lambs which is perhaps surprising considering the habitual semi-flexed posture of the knee joint in the quadruped. It is also apparent from the results obtained that there is a gradual increase in the angular measurement with advancement of age both in control and experimental lambs. Although differences may exist between limbs in long surviving sheep it is not proposed to discuss their possible significance due to the errors that are likely to have been made in their estimation.

Joint mobility measurements also showed changes with advancement of age. The results obtained must be considered estimates of the passive range of movement (as defined by Cynax 1962) as it is not possible to guage active range of movement in animals. Much of the confusion in the literature pertaining to joint measurement has resulted from conflicting methods of assessing joints (Wright and Johns 1960 ; Backlund and Tiselius 1967) and in particular to different definitions of the neutral position of the joint from which measurements were made (Clark 1920). Wiechec and Krusen (1939) cite Rosen (1922) who apparently did not believe that a definite neutral position was possible and that measurement of extremes of joint movement was of more importance. Such has been the approach adopted in this study, with only extremes of movement being estimated.

There was found to be an obvious association between tibial deformity and reduction in joint mobility. Reduction in joint mobility was however influenced by the postoperative survival time. That the association between deformity and joint mobility is not absolute is demonstrated by the fact that reduced fetlock mobility was detected in some lambs in which deformity, if present, was minimal.

It has been noted in experimental lambs that soft tissue enlargement occurred periarticularly very early on in the development of limb deformity. In many cases such enlargement was apparent as soon as 5-6 weeks postoperatively. Swelling was especially marked laterally. At this stage angular limb deformity was becoming apparent but no gross joint derangement was

present. It is generally accepted that muscular, tendinous and fascial structures about joints make up one of their most important stabilisers and add great strength to the human knee joint (Brantigan and Voshell 1941). The same must be true of the stifle, hock and fetlock joints of the sheep which are endowed with strong collateral ligaments. That much of the apparent soft tissue swelling noted in experimental lambs was due to hypertrophy of the periarticular fibrous elements and in particular the lateral collateral ligament, was confirmed on dissection of the limbs.

Peacock (1966) stated that there appeared to be only two basic changes possible in collagenous tissue. Firstly, a change in the amount of collagen present, and secondly changes in the method of assembly of various subunits of collagen. In the experimental sheep of this study the change has apparently been in the amount of collagenous tissue laid down. No comment however can be made on the establishment of cross linkages in collagen around the hock joint.

As hypertrophy of periarticular ligaments has occurred in lambs concomitant with development of deformity the increase in collagen must be a functional adaptation. The increased stresses imposed on the joints as a result of angular deformity has resulted in adaptation of their periarticular elements in such a manner as to withstand stresses imposed. Such stresses would predictably be greatest on the lateral aspect of the joint undergoing progressive varus orientation and this is the site

of maximum hypertrophy. Associated with collagen hypertrophy a progressive reduction in joint movement would predictably occur. That both flexion and extension were found to be reduced suggests the aetiology is not due to a mechanical limitation by the deformed distal bone extremity. No laxity of joint movement was noted and thus it can be assumed that such adaptation has been successful. It can also be inferred that as gross joint laxity was not detected the articulations are not likely to contribute greatly to limb deformity in vivo.

It is impossible to ascribe much importance to the noted differences in stifle joint mobility as accurate standardisation of measurements was impossible.

There may however be a tendency for slight reduction of mobility with advancement of age. The operated limb was seen to have initially a slight reduction in joint mobility but in long surviving lambs to have a relatively increased range of movement. Terminally, the operated limb was progressively reinstituted as a functional limb (see part D of this study) which may have overcome slight previous stiffness due to disuse. The effect of disuse on the development of joint stiffness has been extensively documented (Watson-Jones 1952; Akeson 1961; Peacock 1966; Furlow and Peacock 1967; Clark and Wechesser 1971; Fuller 1975). Such stiffness has been considered to arise from capsular changes and may be due to increased collagen synthesis or establishment of cross linkages between existing collagen fibrils .

It has been noted that control lambs demonstrated a reduction in hock joint mobility, and perhaps to a lesser extent stifle joint mobility, with advancement of age. It is of interest that a similar tendency has been reported to occur in human metacarpophalangeal joints (Wright and Johns 1960a). It is attractive to suggest that increased functional use of a limb with increasing skeletal maturity results in a functional necessity to reinforce periarticular structures. Repeated minor traumata resulting from extremes of joint motion could prompt such reinforcements.

"The method of staining, once having taken root in the animal histologist, grew and grew, till to be a histologist became practically synonymous with being a dyer, with this difference, that the professional dyer knew what he was about, while the histologist with few exceptions did not know, nor does he to the present day".

Marin (1902) cited by Barclay (1951)

Anatomy of the Growth Cartilage

Many standard texts including Lebland & Greulich (1956) Collins (1966); MacLean & Urist (1968); Bloom & Fawcett (1969); Rang (1969); Vaughan (1970); Little (1973); Ham (1974); Warwick & Williams (1975) and Gardner & Dodds (1976) have described the histological appearance of growth cartilage. Fig 28 represents an amalgamation of such descriptions and summarises current conceptions of the morphological changes in cartilage cells that accompany growth.

Description of the growth cartilage is greatly facilitated by division of the cartilage cells into zones according to their morphology. Such a procedure has however resulted in considerable confusion as no classification has been adopted universally by all authors. Thus, one author's conception of the zone of growth for example may differ markedly from that of another. On the assumption that the simplest classifications are usually most useful, the growth plate can be conveniently considered as consisting of three zones, a zone of growth, a zone of cartilage transformation and a zone of ossification. Such an approach is the essence of classifications given by Siffert (1966); Rang (1969) and Warwick

and Williams (1975) although Siffert (1966) differs slightly from the other two authors in his interpretation of what cell morphologies should be included in the growth zone. On the basis of cell morphology each zone has frequently been subdivided into cell layers. Again such a classification aids description but it must be recognised that growth within the plate is a continuous process and that the cartilage cells undergo morphological change as they approach the metaphysis. Similarly the zones of the growth plate will not be static but will be constantly moving in the direction of growth.

The epiphysis gains no growth increment from the growth plate (Payton 1933; Siegling 1941; Siffert 1966) and thus all of the growth cartilage proliferation is ultimately incorporated within the metaphysis. Although proliferation of the growth cartilage has been regarded as the "moving force" of endochondral ossification (Sissons 1971) the growth zone is the only area of the growth plate where longitudinal growth actually occurs (Rang 1969). The growth zone has been interpreted by different authors as containing different cell layers. Closely applied to the epiphysis, resting or germinal cartilage cells are found which act as a stem cell population and do not participate in the growth of the cartilage per se. It would seem appropriate to refer to these cells as a resting or germinal cell layer and not to include them in the broader classification of the growth zone. Many authors including Siffert (1966); and Vaughan (1970) have included such cells in the growth zone. Moving further from

the epiphysis the next cell type is perhaps where most confusion has arisen. The cells of this region undergo cell division and matrix elaboration occurs. Mechanisms controlling bone growth are thought to act by changing the division rate of these cells which constitute the 'proliferating layer' (Kember & Walker 1971) Cell division results in columns of cells being produced and the stacked up form of the cartilage cells that form has been referred to as the 'palisading layer'. Each column is however considered to have a limited life as after a period of time repeated cell division ceases and the column grows out. When this happens intercellular matrix temporarily replaces it prior to the establishment of a new column from germinal cell division (Little 1973). Mitotic figures are commonly encountered in the cells of the palisading layer and the variability of plane of division has been described by Ham (1931). It seems reasonable to include the palisading layer of cells in the zone of growth. Siffert (1966) however considers that such cells are within the zone of cartilage transformation which he regards as preparation of the cartilage for replacement by bone. From the literature it is difficult to agree with Siffert's (1966) classification. The layer of proliferating and palisading cells have been referred to by other names by authors. For example Ham (1974) regards these cells as forming the zone of young proliferating cartilage. The columnar arrangement of cells is thought to be due to their mechanical environment. Little (1973) remarks that surrounding forces cause the cartilage cells to become flattened and that the constricting force of the perichondrium

maintains the vertical disposition of the cartilage cell columns. The longitudinal orientation of collagen fibre bundles in the intercellular matrix is also implicated (Rang 1969; Ham 1974). Ambient forces acting on cartilage cells are undoubtedly important as in their absence unorientated "cell nests" develop.

Moving still further from the epiphysis the cartilage cells are seen to be enlarged and constitute the layers of cell maturation and hypertrophy. Vaughan (1970) adds to the confusion of terminology by referring to these cells as the zone of growth and maturation. The enlarged cells have ceased to produce matrix and their cytoplasm contains a high proportion of fluid to solid and accumulates glycogen (Little 1973; Ham 1974). Although Little (1973) implies that cell hypertrophy results in compression of the surrounding matrix, Ham (1974) considers that expansion of the growth cartilage in a longitudinal direction occurs. This latter suggestion may account for Vaughan's (1970) terminology.

As these cells become further removed from the epiphysis progressive calcification occurs in the surrounding matrix. It is assumed that matrix calcification destroys the cartilage cells ability to obtain diffusable nutrients and thus results in cell degeneration. Empty lacunae result which form a calcified cartilage scaffolding amongst which metaphyseal vascular loops invade. Recognition of vascular invasion marks the zone of ossification. As vascular invasion occurs some of the calcified cartilage scaffolding becomes eroded away which may be the result of osteoclast action or may be due to chondrolytic

activity of the endothelium of the vascular sinusoids. (Warwick and Williams 1975). The transverse bars of calcified cartilage lying between lacunae are removed in preference to the longitudinal bars that separate columns of lacunae. Rang (1969) has recently reviewed the literature which described distinct biochemical differences between the matrix at these two sites. Following invasion of the disrupted lacunae by metaphyseal vascular loops osteoblasts appear and arrange themselves along the surfaces of the cartilage remnants and elaborate osteoid. Such osteoblasts may arise frompluripotential perivascular connective tissue cells or from endosteal cells (Siffert 1966). Calcification of the deposited osteoid occurs which results in a layer of fine fibred new bone over the calcified cartilage remnants. The fine cancellous bone so formed is known as the primary spongiosa which is orientated according to the calcified cartilage "scaffolding" on which bone has been deposited.

Bentley and Greer (1970) observed that not every cartilage column is invaded by a vascular loop as islands of cartilage cells are commonly found within the primary bone trabeculae of the metaphysis. Holtrop (1966) (cited by Holtrop 1967) suggested that such cartilage cells isolated from the growth cartilage may form osteoblasts. However, Bentley & Greer (1970) and Hanaoka (1976) failed to confirm this suggestion. Brighton, Sugioka and Hunt (1973) concluded from their electron microscopic study of rat growth cartilage that the ultimate fate of the growth plate chondrocytes is death. Much progress has been made in the comprehension of the growth cartilage by the application of the

electron microscope to its study. For example the manner of deposition of osteoid and its calcification was described in detail by Robinson & Cameron (1958). The process of calcification in cartilage matrix and osteoid appears to be different as the orientation of crystal to collagen is not the same in the two matrices (Anderson & Parker 1968). Many of the other advances that have been made by use of the electron microscope in studying growth cartilage have been reviewed by Anderson and Parker (1968).

The perichondrial zone

Shapiro, Holtrop and Glimcher (1977) amongst others cite Ranvier, who in 1873 was the first to describe a circumferential groove between the epiphysis and metaphysis of developing long bones. Pratt (1959) however observed that in the rat a well defined groove is not always discernible and in some cases does not completely encircle the growth plate. In close association with the groove Ranvier described the presence of a lamina of bone, the perichondrial bone bark, which encircles the lower portion of the growth plate.

Attention has been centred on these structures in attempts to explain how the growth cartilage grows in width. Some authors (Keith 1919-20; Langenskiold, Rytomaa and Videman 1967; Meikle 1975) believe that the growth plate increases in diameter by means of interstitial growth of cartilage cells whereas others (Gardner 1956; Leblond and Greulich 1956; Solomen 1966; Siffert 1966; Rang 1969; Warwick and Williams 1975; Shapiro, Holtrop and Glimscher 1977) consider appositional growth of the cells in

the ensheathing perichondrium occurs. The comprehensive and rather convincing study of Shapiro, Holtrop and Glimscher (1977) resulted in three groups of cells being discovered within the groove. Densely packed cells were described and found to be associated with bark formation. In addition a group of looser packed cells were found which appeared to add cells to the epiphyseal cartilage above the growth plate by appositional growth. These authors concluded that it was reasonable to assume that some of these cells, which included chondroblast precursors, become incorporated into the growth plate with growth. The third type of cells encountered were fibroblasts and fibrocytes associated with the periosteum. Shapiro and coworkers (1977) also described the manner in which bone synthesis occurs proximally and peripherally on the bone bark with resorption occurring distally. Thus the bone bark grows upwards and outwards in pace with longitudinal bone growth and is influenced by and conforms to the metaphyseal remodelling that occurs. Disruption of the bone bark has been implicated in certain metaphyseal affections (Keith 1919-20: Rang 1969).

Frost (1969) has drawn a distinction between the terms modelling and remodelling as applied to bone. He considers modelling to signify the reconstructive processes concerned with bone growth whereas remodelling refers to processes which turn over lamellar bone. This distinction has not been adopted in this study. Instead the terms surface (or external) and internal remodelling are used to denote Frost's modelling

and remodelling respectively.

Metaphyseal Remodelling

The growth plate produces longitudinal growth and the product of its activity is a layer of newly formed bone which has no medullary cavity and is orientated according to the calcified cartilage scaffolding produced by the matrix that surrounded the hypertrophic cartilage cells. Brookes (1971) considers that the vasculature of the metaphysis is the main influence on trabecular architecture. Certainly it appears that the orientation of the cartilage cell columns does not necessarily bear any relation to that of the endochondral trabeculae. The hypertrophic cells however in general are in alignment with both the metaphyseal trabeculae and vessels. Such bone has been described as the primary or primitive spongiosa consisting of fine cancellous cartilage bone the trabeculae of which are characterised by their possessing a core of calcified cartilage (Pritchard 1971). Remodelling of these trabeculae occurs very rapidly. Sissons (1953) has calculated, from experiments in rats and rabbits, that the primary trabeculae begin to undergo remodelling within 24-36 hours of their formation. Such remodelling results in incorporation of the primary trabeculae either into compact bone or coarse cancellous bone depending on their location in the metaphysis. The coarse cancellous bone represents the secondary spongiosa and is the result of complex remodelling processes. The trabeculae are made up of irregular layers of

bone similar to the interstitial bone found in the diaphyseal cortex. In some trabeculae remnants of calcified cartilage of the growth plate may be detected embedded in the new bone. Cement lines are abundant and signify that both resorption and deposition of bone have occurred (Pritchard 1971). Those endochondral or primary trabeculae which become incorporated in compact bone can be identified by their possessing spicules of calcified cartilage matrix.

The coarse cancellous bone formed in the metaphysis can also become incorporated into compact bone as is described below. In order to understand how this occurs it is necessary to describe the salient features of metaphyseal remodelling of a long bone.

In general terms, Frost (1964b) states "Bone remodelling is the summation of the resorptive and formative processes that occur on bone surfaces and in its interior".

For longitudinal bone growth to occur without loss of normal bone contour it is essential that the metaphysis becomes reduced in width so that it can be incorporated in the much narrower diaphysis. Such a process is the essence of area relocation as described by Enlow (1963) which is represented diagrammatically in fig 28.

The first indication as to how such relocation occurs was gained over a century ago. Enlow (1962a) cites a number of early researchers (Brulle and Huguenу 1845; Flourens 1845 and Loven 1863)

who recognised that periosteal resorption and endosteal deposition occurred in the metaphysis and implicated these processes in metaphyseal remodelling. Lacroix (1951 cited by Enlow 1962) rediscovered the phenomenon of metaphyseal endosteal bone and revived the concept but it was the monumental studies of Leblond, Wilkinson, Belanger and Robichon (1950) which established a detailed understanding of the remodelling process. Leblond and coworkers (1950) visualised metaphyseal remodelling in rats by means of labelled phosphorus precipitation. Subsequent autoradiographic studies (Tomelin, Henry and Ken 1953; Arnold and Jee 1957) confirmed the concept of periosteal resorption and endosteal deposition in metaphyses. More recently accounts of metaphyseal remodelling have been given by a number of authors including Gardner (1956;1971) Leblond and Greulich (1956); Enlow (1962; 1963); Johnson (1966); Ham (1974); Warwick and Williams (1975) and Gardner and Dodds (1976). By far the most comprehensive of these accounts is that given by Enlow (1963). An attempt has been made to summarise the rather complex features of metaphyseal remodelling in fig 29.

As reduction in metaphyseal width occurs, there is endosteal bone deposition in an area already containing coarse cancellous bone. The resulting cortical bone has been termed compacted coarse cancellous bone (Enlow 1962a;1963) and is characterised by possessing irregular convolutions of bone lamellae. The convolutions follow the contour of the underlying coarse cancellous trabeculae. Such bone is regarded as being

formed only by endosteal, and never by periosteal, bone deposition. As a result of remodelling, such bone can become incorporated in a segment of the diaphysis well removed from the growth cartilage and metaphysis.

At the junction of metaphysis and diaphysis, there is usually little or no cancellous bone and thus endosteal bone laid down does not have a convoluted form but is much more evenly deposited. Such bone is referred to as inner circumferential lamellar bone (Enlow 1962a;1963).

When the metaphysis is reduced to the dimensions of the diaphysis the process of periosteal resorption ceases as will endosteal bone deposition. The diaphysis however is also increasing in diameter and thus periosteal growth has to occur. Such a change in direction of bone growth i.e. from reduction in diameter to increase in diameter has been referred to as a "growth reversal" (Enlow 1962a;1963). This concept however is an oversimplification of what actually happens in a bone, as the shape of a bone may well result in metaphyseal prominences developing for muscle attachments. If such is the case, with longitudinal growth metaphyseal remodelling of such regions will involve at least one other growth reversal. A tubercle for example, developing by metaphyseal periosteal deposition on entering the region of metaphyseal remodelling will undergo one growth reversal, followed by another on incorporation of the bone region into the diaphysis.

Wherever periosteal bone lies adjacent to endosteal bone a reversal line is present. Prominent reversal lines usually

separate the layers of bone deposited before and after growth reversals.

The cellular component of metaphyseal remodelling

In a recent review article, Holtrop and King (1977) discuss the role of the osteoclast in bone resorption. These authors state that "resorption occurs continuously in the process of morphogenesis and in the remodelling of adult bone. It is well established that in this slow and well integrated process of resorption and formation osteoclasts play a role in the breakdown of bone". Numerous other accounts including those of Hancox (1971) and Ham (1974) reiterate the accepted concept of osteoclasts being actively involved in bone resorption.

Recently another form of bone resorption has received considerable attention. Such resorption is referred to as osteocytic osteolysis or osteocytic resorption. Extensive reviews have been produced on osteolysis including those by Belanger, Robichon, Migicovsky, Copp and Vincent (1963) and Belanger (1971). Osteocytic osteolysis is considered to represent an active physiological phenomenon acting within the bone under the influence of osteocytes and resulting in modification of bone matrix and loss of bone mineral. Osteolysis may be important in mobilising calcium from the skeleton which is normally a rapid process and there is a certain amount of experimental evidence in support of this suggestion.

Osteolysis is important in hyperparathyroidism but its occurrence under normal conditions remains equivocal.

A related concept has been introduced called bone flow which has been reviewed by Krook, Belanger, Henrikson, Latwak and Shetty (1970). Bone flow suggests that there is a constant cell movement from the surface where bone is being formed towards the area where it is being resorbed. As osteoclasia is a surface phenomenon, the acceptance of osteolysis as fact is required before bone flow can be explained. It has thus been postulated that in cancellous trabeculae bone is formed on the surface, flows toward the deepest portion and is resorbed by osteolysis. Similarly, in osteones it is suggested that bone flows peripherally to be resorped by osteolysis in the peripheral lamellae.

The concept of bone flow also remains equivocal. Studies such as that reported by Olsson (1968) failed to reveal evidence of bone flow. This study however did reveal surface resorption in the absence of demonstrable osteoclasts which led Olsson (1968) to suggest that cells other than osteoclasts seem to have an active role in surface resorption of bone.

In a recent study of metaphyseal remodelling, Whalen, Winchester, Krook, Dische and Nunez (1971) implicated osteolysis. In femora of newborn human infants these authors described convergence of metaphyseal trabeculae as they reached the compact portion of the developing diaphysis. Such convergence was even noted in the peripheral trabeculae and no abrupt cut

off was detected. Histologically osteolysis was judged to be in evidence and the greatest numbers of osteoclasts were found in regions of most marked osteolysis. The distribution of osteoclasts was interpreted as indicating that osteoclasia occurs in bone which is first altered by osteolysis. Whalen et al(1971) also made serial radiographic examinations of femora in children who had ingested lead intermittently. They noted that the shape of the lead line in the metaphysis was similar on serial radiographs but that the size of undulations were smaller at greater distances from the growth cartilage. This finding was concluded to indicate symmetrical shrinkage but such a suggestion is equivocal as is discussed below.

Anatomy of Cortical Bone

Numerous accounts exist of the histological appearance of cortical bone including those by McLean and Urist (1968); Bloom and Fawcett (1969); Vaughan (1970); Clark (1971); Pritchard (1972); Ham (1974); Warwick and Williams (1975) and Gardner and Dodds (1976).

Information from such sources is summarised in fig 30 The anatomy of Haversian systems (or secondary osteones) is well documented in the above texts and a further classical description is considered superfluous and will not be attempted. Haversian systems are considered to be the 'units of structure' of most mammalian tubular bones. Species variations in cortical bone do however occur and are described by a number of authors including Jowsey (1968). Although secondary osteones are often considered to be orientated parallel to the long axis of the bone, Cohen and Harris (1958) described their gentle spiralling course

in dog femora. These authors noted that the direction of spiralling in a proximal distal direction was clockwise in the left femur and anticlockwise in the right bone.

Cement lines delineate the outer margin of the secondary osteone. A cement line forms when a cavity created by resorption, becomes the site of bone deposition as occurs in the formation of secondary osteones. Cement lines have also been referred to as 'reversal lines' (Warwick and Williams 1975) and are analagous to the growth reversal lines described above.

In basic terms, the compact cortical bone is regarded as being composed of bone which has been deposited in layers or lamellae. As can be seen from fig 30 three common patterns of lamellae are found. In the haversian systems the lamellae are arranged concentrically around the haversian canal and between osteones angular fragments of lamellar bone known as interstitial lamellae are found. The periosteal and endosteal surface consist of outer and inner circumferential lamellae respectively. Although osteocytes are trapped within lacunae their fine cytoplasmic processes connect adjacent cells. Studies of the bone cell communication system has led authors such as Weinger, Holtrop and Glimcher (1971) to suggest that bone behaves as a functional syncitium with osteocyte processes permitting intercellular communication being capable of acting as pressure transducers.

The classic description of cortical bone structure that is usually given is grossly oversimplified. As has been suggested above elements of endochondral bone growth can become relocated

in the cortex as a result of the remodelling process. Also, the secondary osteones themselves are the result of remodelling within the substance of the cortex. Remodelling of the diaphysis of a bone is of paramount importance in both bone growth and function and occurs both internally and externally (Lacroix 1971).

Cortical bone remodelling

a) External remodelling As with the metaphysis, cortical bone remodelling involves controlled bone resorption and deposition occurring concurrently. The diaphysis increases in diameter by periosteal bone deposition and in order that the medullary cavity can also increase in width endosteal resorption occurs. Developmental variation may well occur in the process of diaphyseal remodelling as is reported in the human foetus and neonate by Steendijk, Van Eek, Kraas and Bradley (1971). Cortical bone can arise in three ways. Firstly it may be derived from consolidation of the fine cancellous cartilage bone of the metaphysis or from consolidation of fine cancellous membrane bone derived from the periosteal layer. In both instances cylinders of new bone are formed in the vascular spaces of the cancellous bone. Such new bone cylinders are referred to as primary osteones. (Pritchard 1972). Another source of diaphyseal bone is from direct primary formation of solid bone on the periosteal and endosteal surfaces of existing cortex (Pritchard 1972; Ham 1974). A third method of production of cortical bone, namely consolidation of coarse cancellous bone,

has been described briefly above.

Primary osteone bone is described as occurring in association with rapid accumulation of bone (Enlow 1962b) such as in the young animal (Pritchard 1972). Primary osteones are smaller than secondary osteones having only one or two irregularly arranged lamellae of bone around the central canal (Arnold, Jee and Johnson 1956) and no cement lines bounding them.

Enlow (1962b; 1963) described another form of bone, comparable to primary osteones, which is referred to as plexiform bone. Plexiform bone is considered to be common in most artiodactyls and in the dog. It develops in fine cancellous vascular spaces. The central canals are arranged in a closely meshed symmetrical three dimensional plexus rather than in the elongated anastomosing arrangement of primary osteones Plexiform bone is also considered to be associated with regions of rapid accumulation of bone and is commonly associated with periosteal apposition (Enlow 1962a;1963). Currey (1960) used the synonym 'Laminar bone' for plexiform bone and described its more intimate blood supply when compared with Haversian bone.

The pioneer studies of early researchers which has led to the current concept of external or surface remodelling of diaphyses are described by Brash (1934) and Sissons (1971) amongst others. The realisation by John Hunter that bone remodelling and growth necessitates both resorptive and formative processes has been briefly mentioned in the historical introduction

of this study. More recent accounts of diaphyseal remodelling are numerous and include those of Tomlin, Henry and Kon (1953) Enlow (1963); Johnson (1966) and Storey (1972).

b) Internal Remodelling Internal cortical remodelling occurs at a histological level with no disruption of bone shape occurring. Internal remodelling however unlike surface remodelling occurs in compact bone throughout life and has an important mineral homeostasis function. Internal remodelling is also a necessary prerequisite of secondary osteone formation and their characteristic cement lines indicate that resorption has preceded lamellar deposition. The initial process is the formation of a resorption cavity which is the result of osteoclastic activity. The resorption cavity is in fact a three dimensional structure and with progression of osteoclasia a tunnel is formed orientated in the direction that the secondary osteone will occupy. The active front of tunnelling is referred to by some authors as the 'cutting cone' (Johnson 1966). As bone is resorbed, the tunnel is immediately filled with loose connective tissue and is later slowly filled with an osteone by centripetal lamellar bone deposition. Osteoid deposition precedes actual bone formation in the absorption cavity and gives rise to identifiable 'osteoid seams' in bone sections. (Frost and Villaneuva 1960, Frost Villanueva and Roth 1960; Frost 1960c and d; 1962; 1969; Sedlin and Frost 1963; Epker and Frost 1965; Landeros and Frost 1966). Ortner (1975) cites Amprino and Engstrom (1952) who demonstrated that the osteoid matrix of secondary osteones attain approximately 75% of its maximum mineral content very rapidly. Completion of

mineralisation is however very slow (Lacroix 1956). Engfeldt (1958) noted that cortical bone has a very uneven distribution of mineral. The outer and inner circumferential lamellae and interstitial lamellae were found to be usually evenly and highly mineralised but the uneven degree of mineralisation of osteones reflects their protracted acquisition of mineral. Sedlin and Frost (1963) studied human osteone formation and concluded that the volume of bone formed in unit time is initially rapid but thereafter occurs at a much slower rate.

As a result of studies of internal remodelling of cortical bone a narrow zone has been identified in osteones which is similar in appearance but thicker than the cement line. Lacroix (1970; 1971) points out the similarity of the white ring lying between lamellae of the osteone and white lines that are encountered in endosteal circumferential lamellae. The latter are called arrest lines or resting lines because they are thought to result from hypercalcification of the bone surface during a quiescent period. Bone deposition is then considered to be resumed without any preceding bone resorption. In such a manner resting lines become located between endosteal lamellae. Lacroix (1970;1971) reasons that the white ring in osteones may similarly indicate a temporary cessation of bone deposition.

The above description of internal remodelling is of necessity brief. Detailed accounts of various aspects of this process are given by numerous authors including Enlow (1962a; 1963) Jowsey (1963); McLean and Rowland (1963); Sedlin (1964); Frost (1964; 1969) Hattner, Epker and Frost (1965); Johnson (1964,1966)

McLean and Urist (1968); Lacroix (1970;1971) and Ham (1974).

Osseous Drift

Tomlin, Henry and Kon (1953) performed autoradiographic studies on growing rat long bones and made some important observations. They noted that diaphyseal external remodelling was asymmetrical at different levels so that diaphyseal curvature could be established. In the femur for example they noted that the posterior cortical wall was extensively remodelled during early growth whereas the anterior wall grew entirely by bone accretion on both the periosteal and endosteal surface. The cortical diaphysis of a long bone may therefore shift in space due to remodelling which is referred to as lateral, osseous or cortical drift. The direction of drift differs at different levels of the diaphysis and produces both the shape and curvature of the bone. As well as accounting for bone curvature drift can result in a shift of the bones long axis. Such axial drift has been described in the growing human tibia (Garn, Goodspeed and Hertzog 1969). Descriptions of osseous drift are given by Enlow (1962b; 1963) and Johnson (1965) in general terms, and in specific long bones of the rabbit by Stanek (1969) and human ribs by Landeros and Frost (1966). Enlow (1962b; 1963) remarks that by means of osseous drift a bone's cortex becomes relocated without changes in its thickness. Another feature of osseous drift is that it can result in one cortical wall being composed entirely of periosteal bone, the opposite wall being entirely endosteal bone.

The concept of osseous drift is summarised in fig 31. Just as drift is documented in surface remodelling, so transverse drift of the product of internal remodelling, the secondary osteone, has been described. Osteones in which the haversian canal is situated eccentrically have been described by Johnson (1964; 1966) and Epker and Frost (1965). Such "eccentric" or "waltzing" osteones have been defined by Sedlin, Frost and Villanueva (1963 cited by Epker and Frost 1965) as being actively evolving new Haversian systems seen in cross section in which resorption is limited to one side of the absorption cavity while lamellar bone deposition is largely limited to the opposite surface. The eccentric position of the Haversian canal cannot be accounted for by obliquity of the osteone in the plane of section (Epker and Frost 1965).

Methods of study

Histological techniques have been widely applied to the study of bone and cartilage. In addition a number of specialised methods including autoradiography, electron microscopy, microradiography and fluorochrome bone labelling have been utilised and have greatly aided the attainment of present day knowledge. The former two techniques are beyond the scope of this study. It should be acknowledged however that the autoradiographic studies of, for example Leblond, Wilkinson, Belanger and Robichon (1950) andTomalin, Henry and Kon (1953) marked the advent of comprehension of metaphyseal and diaphyseal surface remodelling. The usefulness of both the scanning and

transmission electron microscope in the study of bone is well exemplified in the recent reviews by Boyde (1972) and Cameron (1972). X-ray diffraction and polarized light microscopy have also been successfuly applied to bone studies and will be mentioned where appropriate elsewhere.

Although histological methods have been applied with much success to the study of bone, the necessity to decalcify tissue samples prior to sectioning excludes the study of the mineral conponent of bone. Techniques of microradiography and bone labelling have been developed and permit investigations to be made on bone mineralisation. By necessity however such techniques require the preparation of undecalcified bone sections which because of the hard and yet in many instances brittle nature of bone have been difficult to obtain. Although macroradiographs of slabs of bone have been usefully employed in investigations of some bone disorders (Olsson 1972) for the purposes of bone labelling and microradiographic studies, bone sections of 100u or less are required. Jowsey, Kelly, Riggs, Bianco, Scholz and Gershon-Cohen (1966) consider 100u bone sections to be ideal for microradiography and discuss the disadvantages of thinner and thicker sections. The opinion of these workers is augmented by Wallgren's statement (1957 cited by McQueen, Monk, Horton and Smith 1972) that the thickness of bone section required for maximum radiographic

resolution is 100u.

Methods of preparing undecalcified bone sections

Of the bone sectioning methods developed, perhaps that most similar to routine histological methodology has been the use of microtomes. Bone has been prepared for microtome sectioning by freezing,(Milch, Rall and Tobie 1957; 1958; Kelly, Peterson and Jones 1957; Milch, Tobie and Robinson 1961;)or embedding in plastics and acrylics,(Arnold and Jee, 1954; Bohatirchuk 1963; Delling 1972.) Using such methods, thin undecalcified bone sections of the order of 5-15u are obtained. It has been conceded that the quality of such sections is poor (Johnstone and Tam 1973) and many sections are wasted (Milch, Rall and Tobie 1958).

Frost (1958) realised that the methods of processing bone prior to histological and undecalcified sectioning would have a deleterious effect on the tissue. In an attempt to minimise such artefacts, he developed a method of preparing undecalcified sections which did not necessitate either fixing or embedding the bone specimen. Slabs of bone were produced and ground by hand using wet abrasive paper and liberal water lubrication. The technique was widely used by Frost and coworkers (Frost 1960c,d,and e; 1961; 1962; 1969; Frost and Villanueva,1960; Epker and Frost 1963; 1965; 1966; Sedlin, Villanueva and Frost 1963; Villanueva, Sedlin and Frost 1963; Hattner, Epker and Frost 1965; Landeros and Frost 1966; Rush, Pirok and Frost 1966) and was employed in other studies (Vanderhoeft, Kelly, Janes and Peterson 1963; Landry and Fleisch 1964). Although Frost (1958)

claims that sections of from 3u to 100u can be obtained, Johnstone and Tam (1973) question this and regard a major disadvantage of the method is that bone dust often plugs canaliculi.

Prior to Frosts (1958) report a number of other methods of preparing ground sections had been described which involved either fixation or embedding the bone specimen (Cohen and Lacroix 1953; Kropp 1954). Despite Frosts objections numerous subsequent studies have involved embedding bone specimens which were then ground to the desired thickness (Hallen and Rockert 1958; Hulth and Olerud 1962; 1964; Kornblum and Kelly 1964: Margel 1971).

Johnstone and Tam (1973) amongst others cite Jowsey (1955) who employed a high speed milling machine to section methacrylate embedded bone specimens. This method was widely applied by Jowsey and coworkers (Sissons, Jowsey and Stewart 1960; Harris, Jackson and Jowsey 1962; Jowsey 1963; Kelly, Jowsey and Riggs 1963; Jowsey, Kelly, Riggs, Bianco, Scholz and Gershon-Cohen 1965) and by other researchers (Kelly, Peterson and Janes 1957; Kruse and Kelly 1974). Johnstone & Tam (1973) adapted Jowsey's method by employing what they considered to be a superior embedding medium (Spurrs medium) which they routinely used (Tam, Reed and Cruickshank 1974; Tam, Reed, Campbell and Cruickshank 1974).

Other methods employing high speed sawing have been described (Arnold, Taysum and Jee 1954; Bush 1956; Kropp 1956; Bergendahl and Engfeldt 1960; Young 1963; Rowland 1966; Olsson and Rietz 1965; Rietz 1968; Harris, Haywood, Lavorgna and Hamblen 1968; Seliger 1968; Olerud and Lorenzi 1970; Kwan 1970; Rahn and Perren 1971) which in

many instances have necessitated further grinding of specimens in order to obtain the desired thickness of section.

McQueen, Monk, Horton, and Smith (1972) consider that even when ideally embedded, bone samples tend to shatter when being sectioned with high speed saws. In addition, the much used methyl methacrylate is known to undergo considerable shrinkage on hardening. These authors thus used a low speed 'Macrotome II' machine and embedded their bone specimens in epoxy resin which exhibits minimal shrinkage. The "Macrotome II" machine is equipped with a counterbalanced arm by which the specimen is brought into contact with the blade. Such a design results in much lower stresses being applied to the bone specimen. Some variation in section thickness was however encountered.

The same machine was gainfully employed by Bard, Dickens, Edwards and Smith (1974). The loss of tissue incurred during sectioning due to the thickness of the blade (Kerfloss) is of the order of 200u with the MacrotomeII. This compares favourably with other methods, as the Gillings machine for example, used by Harris, Haywood, Lavorgna and Hamblen (1968) resulted in a Kerf loss of 400u.

A modification of the Macrotome II is equipped with a diamond impregnated blade in an annular, rather than peripheral configuration. The kerf loss in this machine, the Microslice 2, is slightly less than for the Macrotome II. A Microslice 2 machine was successfully used for preparing undecalcified bone sections of 100u and less by Gilbertson (1975a, b, 1977). The cutting blade is stretched

in tension and thus does not 'wander' through the material being sectioned (Gilbertson 1977). As with the Macrotome II the specimen is brought into contact with the blade by an adjustable counterbalance which reduces damage to the bone specimen. A further advantage of the Microslice machine is that there is no necessity to embed the specimen prior to sectioning. Gilbertson (1975b; 1977) obtained 30-50u sections of unembedded cancellous bone but considered that more consistant results were achieved with 80-100u sections.

Microradiography

Microradiography has been defined as being that branch of radiology which deals with the production and study of radiographs of thin sections of tissue (Tirman, Taylor, Banker and Taylor 1951). An enlarged x-ray absorption image is obtained and in principal either primary or secondary magnification of the sample can be performed. With primary magnification methods the sample is placed close to a point source of x-rays and the magnitude of enlargement is given geometrically as the ratio of the source-image to source-object distance. Such a technique has also been referred to as projection microradiography, and as an x-ray microscope is normally employed, x-ray microscopy. Although x-ray microscopy is reported to have some advantages over secondary magnification methods, the technical difficulties inherent in its use have restricted its application to bone (Berhendahl and Engfeldt 1960; Bohatirchuk 1963), Engström (1949); Barclay (1951); Bellman (1953) and Peterson, Kelly and Jones (1953) have

reviewed the historical background to microradiography.
With the attainment of fine grain photographic emulsions, methods employing secondary magnification became a possibility. With secondary magnification methods the specimen is placed in the closest possible contact with the photographic emulsion. The source to specimen distance is adjusted in such a manner as to minimise geometrical blurring of the image and following processing of the microradiogram, an unmagnified image of the specimen is obtained. By examination of the microradiographic image under light microscopy secondary magnification of the specimens structure occurs and can be examined and photographed. Bergendahl and Engfeldt (1960) claim that such methods permit direct magnification of up to 1000 times. Secondary magnification microradiography is also referred to as historadiography or contact microradiography (Bohatirchuk 1963; Gilbertson 1977). Contact microradiography is simpler, gives more constant results and is more widely used than x-ray microscopy. The grain size of the emulsion is the limiting factor in the degree of magnification (resolution) that can be attained with contact microradiography however. Numerous studies have been performed which incorporated the technique of contact microradiography (Engstrom 1949; Graham 1955; Vincent 1954; Kelly, Peterson and Janes 1957; Rowland, Jowsey and Marshall 1959; Sissons, Jowsey and Stewart 1960; Bohatirchuk 1963; Jowsey 1963; Jowsey, Kelly, Riggs, Bianco, Scholz and Gershon-Cohen 1965; Young 1963; Hulth and Olerud 1964; Olsson and Rietz 1966; Ljunggren 1967; Rietz 1968; Gilbertson 1975a and b; 1977). A number of authors

have described the essential factors that are required for the production of high definition images by means of secondary magnification (Peterson, Kelly and Janes 1959; Jowsey, Kelly Riggs, Bianco, Scholz and Gershan-Cohen 1965). These include 1) use of fine grain photographic emulsion 2) a small focal spot 3) thin tissue sections 4) a large target to film distance 5) a small specimen to film distance 6) minimal x-ray scattering and photoelectron emission 7) and absence of motion between focal spot, specimen and film. Techniques have been developed which ensure that these requirements are complied with. Although it might seem desirable to employ bone sections of less than 100u, which would reduce the likelihood of geometric blurring and thus the target film distance and exposure time, this is not so. In thinner sections contrast between areas of different mineral density is decreased and the characteristic features of the bone structure becomes more difficult to recognise. Despite taking all precautions, errors can occur especially in quantitative micro-radiographic studies. Some such errors are discussed by Bohatirchuk (1963). Quantitative microradiography has been extensively applied to the problems of osteoporosis in man and extensive reviews exist, for example McLean and Urist (1968) and Jowsey and Gordon (1971).

In Vivo Bone Labelling

Frost considered that his method of preparing thin undecalcified bone sections and subsequently staining them(Frost 1958; 1969) provided "a means for the microscopic observation of bone that

approaches close to the ideal of observation in vivo".

Another method however has become established which permits visualisation of in vivo bone reconstruction and thus adds a 'dynamic' dimension to histological studies of bone tissue. This method relies on the fact that some substances when administered to the individual, in vivo, become fixed in those parts of the skeleton undergoing active reconstruction at the time of administration.

Although autoradiographic methods have such characteristics little will be added to what has been said above. Hansson (1964) has criticised the application of such methods to studies of bone growth as radioactive substances may, he suggests, influence the rate of growth. Similar criticism can be made of studies in which heavy metals such as lead are administered. Studies of bone growth and reconstruction in man were hampered by the lack of a suitable non-toxic bone label. Some studies have however been possible in man due to for example the fortuotous ingestion of lead by children (Whalen, Winchester, Krook, Dische and Nunez, 1971) or transverse lines being present in metaphyses (Siegling 1941; Garn, Goodspeed and Hertzog 1969). In the former case at least however Hanssons (1964) objection is of relevence.

The discovery of the bone staining properties of madder root has been briefly discussed in the historical introduction of the study. Work with this bone dye continued (Hoyte 1960) and resulted in a number of publications concerned with growth and some aspects of reconstruction in the long bones of pigs (Payton, 1932; 1933; 1934; Brash 1934). As the madder was withdrawn from the

food for varying periods of time a sequential labelling technique was in fact employed by these authors. Payton (1932) refers to this technique as the "indirect madder method" which had been employed by earlier workers. Areas of unstained bone were thus produced interspersed between stained parts of longitudinally cut bones and observations could be made with the naked eye.

The active ingredient of madder root with regard to bone staining is alizarin (Frost, Villanueva and Roth 1960) but the toxicity of this compound precluded its widespread use in bone studies. Despite such a disadvantage sporadic reports appeared by authors such as Schour (1936) who successfully used an Alizarine preparation (Alizarine Red S) in studies of alveolar bone growth in rats. Despite its systemic toxicity Alizarine preparations are known to inhibit bone formation. This has been demonstrated both in vitro (Rahn, Fleisch, Moor and Perren 1970) and in vivo (Coutelier 1964; Harris, Travis, Friberg and Radin 1964; Harris Lavergne, Hamblen and Haywood 1968). For example Harris, Travis, Friberg and Radin (1964) noted marked inhibition of mineralisation at many of the sites of Alizarine Red S deposition in experimental dogs. Similarly in laboratory animals Hoyte (1960) reported a temporary arrest in growth following administeation of Alizarine at doses of 50-100 mg/kg. Some years later Adkins (1965) commented that it was necessary to use low doses of Alizarine Red S (25 mg/kg) in order to avoid producing a fall in weight gain in growing rats. It is relevent that at such low doses, no visible staining of thin bone sections was apparent. The same sections however, when

viewed under ultraviolet light demonstrated red fluorescence corresponding to administration of the label. Detection of other compounds,which become fixed in the skeleton,by subsequent fluorescent microscopy of thin undecalcified sections is the essence of modern day bone label studies. Use of more than one such label (or fluorochrome) greatly aids interpretation of results in sequential labelling methods. Despite the obvious unsuitability of Alizarine Red S in such techniques this compound has been used by a number of authors in sequential bone labelling of experimental animals (Harris 1960; Dhem 1967 cited by Lacroix 1970; Olsson 1968; Rietz 1968). Due to its disadvantages it has been suggested that Alizarine is only suitable as a bone label when administered shortly prior to death (Olerod and Lorenzi 1970) a policy that was employed by Dhem (1967) Olsson (1968) and Rietz (1968) but not by Harris (1960). As a result of investigations attempting to find an alternative to Alizarine Coutelier (1964) described haematoporphyrine which also produced red fluorescence in ultra-violet light. Although Olerod and Lorenzi (1970) incorporated haemotoporphyrine in a triple fluorochrome study they performed Rahn, Fleisch, Moor and Perren (1970) demonstrated in vitro that haematoporphyrine inhibited bone growth significantly. Rahn & Perren (1972) however described Alizarin Complexone which was found, in vitro, to be less toxic than either Alizarin Red S or Haematoporphyrine. This compound was successfully used in a fluorochrome bone label study of experimentally induced osteo-arthritis in dogs by Gilbertson (1975 a and b; 1976) without

signs of toxicity being noted. With Alizarine Complexone a red band of colour is seen in labelled bone under fluorescent light.

Use of calcein and calcein blue as fluorescent bone labels have been described by Suzuki and Matthews (1966) and Rahn and Perren (1970). A purified form of calcein (2-4 bis (N-N (carbomethyl) aminomethyl) fluorescein) DCAF was found by Suzuki and Matthews to fluoresce much brighter than calcein blue following administration of similar doses to rabbits and mice. Rahn and Perren (1970) also remarked on the rapidly feding fluorescence of calcein blue. In vitro although some inhibition of bone growth occurred in the presence of both DCAF, calcein and calcein blue this was not considered to be significant when compared with controls (Rahn, Fleisch, Moor and Perren 1970). DCAF has been utilised in combination with other fluorochromes by Suzuki and Matthews (1966); Olsson (1968); Rietz (1968) Olerud and Lorenzi (1970); Gilbertson (1975a,b, and 1976); and Edwards (1976). With the filter combinations normally used a green fluorescence occurs with DCAF.

Xylenol orange can also be administered in vivo and is visualised as a band of orange coloured fluorescence in bone sections viewed under ultraviolet light (Rahn and Perren 1971). As with calcein and its analogues minimal inhibition of bone growth occurred in vitro with Xylenol Orange (Rahn, Fleisch, Moor and Perren 1970). Although the toxicity of xylenol orange

is low following rapid administration Rahn & Porron (1971) noted hyperventilation and muscular hypertonus in some experimental animals which soon recovered however. The orange fluorescence of this compound is considered to fade less quickly, when exposed to ultraviolet light, than many of the other available fluorochromes. Although xylenol orange was used successfully by Gilbertson (1975 a and b; 1976) with no signs of toxicity in experimental dogs, some variation was encountered between the intensity of fluorescence produced by different batches of dye. This resulted in some difficulties in differentiating the fluorescence of alizarine complexone and xylenol orange.

Perhaps the most important fluorochrome bone labels that have been discovered are the tetracyclines. Their importance rests in the fact that they are commonly administered as therapeutic substances and can therefore be more ethically applied to studies in man.

Andre (1956 cited by Olsson and Rietz 1966) and Rall, Loo, Lane and Kelly (1957 cited by Holmes 1963) originally described the fluorescent properties of tetracylines in bone. Milch, Rall and Tobie (1957) published the results of administration of a number of tetracylines to laboratory animals including dogs. They discovered that soon after parenteral administration a pronounced yellow gold fluorescence was apparent in tissues which gradually disappeared from all sites except the skeleton within 6 hours. The bone fluorescence persisted. In a subsequent

publication (Milch, Rall and Tobie 1958) these authors extended their studies using laboratory animals and also obtained bone samples from human patients who had previously been given tetracyclines orally. The ability to detect tetracycline fluorescence in human bone obtained either surgically or postmortem resulted in numerous other studies of various aspects of human bone physiology. Such studies include those by Frost (1960c and d; 1961; 1962; 1963) Frost, Villanueva and Roth (1960 a and b); Milch, Tobie and Robinson (1961); Bailey and Levin (1962); Kelly, Jowsey and Riggs (1963); Sedlin and Frost (1963); Epker and Frost (1966 a and b); Rush, Pirok and Frost (1965); Lacroix (1970); Joffrey (1973) and D'Ambrosia, Stadalnic, Demardo and Riggins (1976). The strong yellow fluorescence of the tetracyclines contrasts well with that of other fluorochromes and thus they have frequently been utilised in sequential bone label studies in experimental animals (Suzuki and Matthews 1966; Olsson 1968; Rietz 1968; Olerud and Lorenzi 1970; Gilbertson 1975 a and b; 1976; and Edwards 1976).

Tetracyclines by virtue of their availability and ease of administration have also been commonly used alone, either as a single or multiple label, in experimental studies of bone growth and reconstruction (Woodhouse 1962; Hulth and Olerud 1962; 1964; Coutelier, Dhem and Vincent 1963; Vanderhoeft, Kelly, Janes and Peterson 1963; Hansson 1964; Fox 1964; Landry and Fleisch 1964; Lee 1964; Olsson and Rietz 1966; Morgan 1967; Karaharju 1967; Ljunggren 1967; Persson 1967; Sunden 1967; Tam, Reed, Campbell

and Cruickshank 1974; Kruse and Kelly 1974; Enneking, Burchardt Puhl and Piotrowski 1975).

Boyne and Miller (1961 cited by Suzuki and Matthews 1966) reported that there was a difference in the fluorescence of different tetracyclines, chlortetracycline fluorescing a deeper orange colour than other preparations. Similarly Harris, Jackson and Jowsey (1962) reported that the chlortetracycline used by them produced golden fluorescence whereas oxytetracycline fluoresced with a light green colour. Harris, Haywood, Lavorgna and Hamblen (1968) made use of this difference between chlortetracycline and oxytetracycline in their study of cortical bone formation in dogs. In the same year Bohr, Ravn and Werner (1968) studied the fate of bone transplants in rabbits by means of oxytetracycline and chlortetracycline bone labels. Harris (1960) although describing canine osteones labelled with chlortetracycline and oxytetracycline failed to comment on any differences in fluorescence.

Suzuki and Matthews (1966) tried different tetracycline preparations but found that they all fluoresced yellow and could not be distinguished. Similarly Gilbertson (1976) reported that it was not possible, in her study, to distinguish between the yellow fluorescence of tetracycline and oxytetracyline. Filter combinations and perhaps batch differences may in part be the cause of such discrepancies. Ibsen and Urist (1964) review some of the literature pertaining to the observed difference in chlortetracycline fluorescence and suggest that degradation

of the compound occurring during section preparation may be responsible.

A number of descriptions exist of renal toxicity occurring following tetracycline administration in man (Ehlich and Stein 1963; Ibsen and Urist 1964; Benitz and Diermeier 1964) and in the dog Gilbertson (1975 b:1976). Many of these reports contain copious literature reviews and it is not proposed to discuss in detail this phenomenon. Briefly, nephrotoxic changes have been described which in many but not all cases followed administration of improperly stored or outdated tetracyelines. A number of authors have implicated degradation products of tetracyclines (Ibsen and Urist 1964; Benitz and Diermeier 1964; Lowe and Tapp 1966) and in particular anhydro - 4 epitetracycline which has been shown to be nephrotoxic in laboratory animals (Benitz and Diermeier 1964; Lowe and Tapp 1966). Hepatic toxicity has also been reported following tetracycline administration in man (Bottiger 1955, Allen and Brown 1966 and Davis and Kaufman 1966; cited by Harris, Lavorgna, Hamblen and Haywood 1968) and the dog (Gilbertson 1975b 1976).

Tetracyclines are also known to inhibit ossification in vivo (Eger, Gattow and Kammerer 1967; Demers, Fraser, Goldbloom, Haworth, La Rochelle, MacLean and Murray 1968; Harris, Lavorgna, Hamblen and Haywood 1968) and in vitro (Rahn, Fleisch, Moor and Perren 1970). When tetracyclines are administered to laying hens the flucrescent component (fluorophore) enters the egg and the foetal skeleton becomes labelled (Rolle, Bevelander and Fisher 1962).

Similarly in women or laboratory animals given tetracyclines during the last trimester of pregnancy the fluorophore crosses the placenta (Cohlan, Bevelander and Tiamsic 1963). On entering the foetus such tetracycline can result in inhibition of bone growth if present in high enough concentrations. Harris, Lavorgna, Hamblen and Haywood (1968) discovered that low doses of breakdown products of tetracyclines (isochlortetracycline and anhydro-4-epitetracycline) inhibited mineralisation and that this effect was independent of renal toxicity. These authors also noted that doses sufficient to inhibit bone formation in vivo also produced gross signs of systemic toxicity. Theories have been proposed as to how tetracyclines inhibit bone growth and reviews of such literature are given by Eger, Gattow and Kammerer (1967) and Demers, Fraser, Goldbloom, Haworth, La Rochelle, MacLean and Murray (1968). Whatever the fundamental cause the and result is an inhibition of apatite formation.

The Site of Fluorochrome Deposition

Tetracyclines are commonly considered to be deposited at sites of new bone formation where they remain unless that bone is removed by resorption. Thus, labelled bone is more likely to retain its tetracycline fluorescence if it is not being constantly remodelled. Certainly, Frost (1961) reported persistence of fluorescence in mature human bone 109 months following administration of tetracyclines. It may well be however that the much quoted site of deposition of tetracyclines in new bone is an over-simplification of what really occurs.

Harris, Jackson and Jowsey (1962) reported that in addition to intense fluorescence at growing surfaces, a diffuse form of label was also discernible in areas shown by microradiography to contain low mineral density. In addition these authors described a discrete low intensity fluorescence lining the canals of some non-growing osteones, trabeculae and Volkmann's canals. The latter form of fluorescence was associated with a thin rim of increased mineral density which they termed "edge sclerosis".Harris, Jackson and Jowsey (1962) also described fluorescence around resorption sites in animals killed immediately following tetracycline administration. When more than three days separated administration and slaughter however this type of fluorescence was not detected. Olerud and Lorenzi (1970) similarly observed labelling of Howship's Lacunae but suggested that resorption lines were actively labelled by the Fluorochromes they used. In the example given by these authors it would appear that the resorption cavities were labelled from two to three days prior to slaughter which tends to confirm the conclusions reached by Harris, Jackson and Jowsey (1962). Fluorescence was also noted in osteocytic lacunae, but as this site normally exhibited autofluorescence in control animals it was considered that such labelling was possibly an artefact. Hulth and Olerud (1962) however reported similar lacunar fluorescence which they considered to be significant. Urist and Ibsen (1963) similarly observed a diffuse component to bone tetracycline fluorescence occurring in mature osteones

and interstitial lamellae. Hulth and Olerud (1962) concluded that the diffuse labelling by tetracyclines was influenced both by fixation methods and experimental design. With short labelling periods of up to 24 hours a more extensive diffuse component occurred. Urist and Ibsen (1963) distinguish between short term and longterm diffuse uptake by mature bone. With the former, diffuse fluorescence disappeared rapidly within a few days after the serum level of oxytetracycline fell to zero whereas long term diffuse uptake persisted several weeks after exposure to the drug. Urist and Ibsen (1963) cite the work of other authors who observed long term diffuse uptake of oxytetracycline in man and the bird but only faint uptake occurred in the rabbit. Hulth and Olerud (1962) used experimental rabbits and rats in their studies.

Uptake of tetracylines by calcified cartilage has been reported by some authors (Frost, Villanueva and Roth 1960; Hulth and Olerud 1962; Coutelier, Dhem and Vincent 1963; Urist and Ibsen 1963; Hansson 1964) but others have failed to agree with this observation (Milch, Rall and Tobie 1958; Holmes 1963). Tetracycline fluorescence has however also been reported in some areas of cartilaginous neoplasms (Milch, Tobie and Robinson 1961) and in the calcified cartilage of fracture callus (Hulth and Olerud 1964).

Although considerable information has been gathered on bone labelling by the tetracyclines, similar studies on the other available fluorochrome bone labels are sadly lacking. Milch, Tobie and Robinson (1961) and Harris, Travis, Friberg and Radin (1964) report the deposition of Alizarine preparations at

comparable sites to those of tetracycline. Olerud and Lorenzi (1970) noted that both haematoprophyrine and DCAF appeared to have the same affinity for newly formed bone as tetracyclines. Rahn and Perren (1970) however cite an unpublished study in which they observed haematoporphyrine and alizarin red S to be deposited at a somewhat different site from that of tetracycline and DCAF. The same authors subsequently reported that xylenol orange fluorescence occurred at exactly the same site as that of tetracyclines, fluoresceins and calcein blue (Rahn and Perren 1971).

The actual mode of skeletal binding by fluorochromes has been the subject of much speculation and again studies have almost exclusively been concerned with the tetracyclines. Milch, Rall, and Tobie (1957; 1958) noted that fluorescence was associated with new bone formation and suggested a complex interfacial interaction involving tetracycline, calcium and the organic matrix of newly proliferated bone. Titus, Loo and Rall (1957-1958) considered that the fluorophore was probably unaltered tetracycline, a proposal which was subsequently accepted by other researchers. Milch and colleagues later suggested that the tetracycline molecule is bound in unaltered from to superficially situated calcium atoms in crystal nucleation sites which are present in freshly laid down bone (Milch, Tobie and Robinson 1961). In vitro experiments such as those of Finerman and Milch (1962) demonstrated interactions of tetracyclines with mineralised salts and calcified matrices. Further support

for tetracycline binding is afforded by the in vitro experiments reported by Urist and Ibsen (1963). These authors noted that the capacity of the growing crystals for binding oxytetracycline was very much higher than was seen with preformed apatite and bone mineral. This suggested to the authors that there was an initial stage of mineralisation in which the calcium salt was highly reactive to oxytetracycline. Extensive reviews of the process of mineralisation are given by Leblond and Weinstock (1971); Herring (1972); and Cameron (1972). As a result of electron microscopic studies it is considered that inorganic crystals are deposited and held within the collagen fibrils of the matrix. The formation of crystals may occur at regular interband periods along the length of the collagen fibril and later the entire fibril becomes calcified probably by recrystallization (Leblond and Weinstock 1971). The formation of crystal nucleation sites and the importance of collagen fibrils in their development is considerably more complex than has been outlined and is beyond the scope of this study. The literature is however comprehensively reviewed by the above mentioned authors. The manner by which mineralisation occurs (Rang 1969; Bonucci 1971) does however provide possible sources of tetracycline affinity. Ibsen and Urist (1964) presented a number of reasons for why new bone calcium is more available for tetracycline binding. More recently it has been confirmed that the site of deposition of tetracycline in rabbits was the phase of rapid mineralisation of newly formed bone (Tam, Reed and Cruickshank 1974).

An alternative to Chelation was suggested by Frost and Villanueva (1960). As it was possible to stain bone surfaces

in vitro by tetracyclines these authors suggested that the fluorophore is merely cemented in to bone by further mineralisation.

Perhaps of most relevence to this study is the opinion of Rahn and Perren (1971) who stated that "the utility of polychrome sequential labelling for providing time marks is not impaired by the lack of understanding of the exact physiochemical bonding mechanism between labels and tissues".

Authors such as Vanderhoeft, Peterson and Kelly (1962) considered that microradiography in addition to fluorochrome studies are essential for more complete understanding of bone changes. It is undoubtedly true that histological studies should also ideally be included in the experimental design.

As osteoid matrix is a necessary precursor of new bone formation it is predictable that Kelly, Jowsey and Riggs (1963) found a nearly perfect correlation between areas of tetracycline fluorescence and osteoid. It is perhaps more difficult to understand how so perfect a correlation was obtained when Frost's (1960c) observations are considered. Frost (1960c) described a resting state of osteoid seams characterised by arrest in both mineralisation and formation of matrix. Similarly Rush, Pirok and Frost (1966) found that the number of osteoid seams exceeded the number of tetracycline labelled osteones in bone sections. Such reports do however demonstrate the importance of employing other microscopic techniques in conjunction with fluorochrome studies.

It should be mentioned that despite all that has been said above concerning fluorescent bone labels, bone and cartilage normally exhibits a degree of autofluorescence when examined under ultraviolet light. The degree of such autofluorescence can be altered or reduced by use of appropriate filters.

Bone circulation

Kelly, Nelson, Peterson and Bulbulian (1960) remarked that the "Study of the blood supply in any bone ..... is hampered by the structure of bone itself". Such difficulties did not however deter early researchers whose studies and observations are reviewed by authors such as Harris (1929) Tilling (1958); Trueta and Morgan (1960); Trueta (1963); Trueta and Cavadias (1964) and Brookes (1971). More recently considerable advances have been made in studies of bone vasculature mainly as a result of the application of more sophisticated perfusion techniques and electron microscopy. Some of these methods will be discussed below. Initially however some of the more recent concepts will be considered pertaining to the blood supply of the growth cartilage and metaphysis.

As the vascularisation of cortical bone has not been a prime consideration of this study a consideration of the relevant literature has not been presented. Most of the important features of cortical bone vascularity are either reported or reviewed in the following publications (Macnab 1958; Brookes 1958a; 1958b; 1971; Morgan 1959; Cothman 1960a; Nelson,

Kelly, Peterson and Janes 1960; Kelly, Nelson, Peterson and Bulbulian 1961; Brookes, Elkin, Harrison and Heald 1961; Trueta and Cavadias 1964; Rhinelander 1968).

Growth cartilage nutrition

The existence of vascular channels It is normally considered that the growth plate is avascular and acts as a vascular barrier between the epiphysis and metaphysis of a long bone (Harris 1929; Harris and Ham 1955; Trueta and Morgan 1960: Kelly, Nelson, Peterson and Bulbulian 1961; Trueta and Cavadias 1964; Collins 1956; Rang 1969). Such a view has led to the suggestion that the hyaline cartilage of the growth plate obtains its nutrition solely by diffusion. Brodin (1955) reviews the literature that led to this suggestion and to the earlier school of thought which proposed that paths exist for the supply of nutrition to hyaline cartilage.

Vascular channels have however been described traversing the growth plate in foetal and neonatal long bones both in man and animals (Ring 1955; Tilling 1958; Brookes 1958a; 1971; Spira, Farin and Karplus 1963a and b; Spira and Farin 1965; Levene 1965). Brookes (1971) cites the observations of Sharpey and Ellis (1856) which suggested that vascular channels may influence growth plate organisation by producing the columnar arrangement of chondrocytes. Both Ring (1955) and Spira, Farin and Karplus (1963a and b) noted that in the distal ulna and radius of rabbits, the vascular channels that traversed the growth plates contained from four to five blood vessels.

A progressive diminution of vascular channels with advancement of age is suggested by Spira, Farin and Karplus (1963a and b) and Spira and Farin (1965). These authors noted that channels were present in 100% of rabbit growth plate sections at 4 weeks of age, in 42% at 6 weeks, 27% at 8 weeks and at 10 weeks of age only remnants of such vessels were seen. Their observations further showed that the blood vessels were of epiphyseal origin and usually ended in the hypertrophic cell zone of the growth plate. Brookes (1971) considers that such blood vessels originate close to or in the groove of Ranvier from where they radiate over the epiphyseal aspect of the growth cartilage. Some of these blood vessels then penetrate the growth plate and traverse it for a variable distance. Tilling (1958) described anastomoses between epiphyseal and metaphyseal vessels by means of vascular channels in the growth plates of a number of long bones in 2 week old calves. In a detailed study of foetal and neonate bones from a variety of mammalian species Levene (1965) noted that when a vascular channel traverses the growth cartilage the blood vessels it contains form a plexus which is fed by afferent vessels of both epiphyseal and metaphyseal origin. The communication between afferent vessels was observed to occur close to the metaphysis.

The significance of vascular channels in growth cartilage remains equivocal but they may have some nutritive function. Vascular channels of the nature described have frequently been referred to as cartilage canals. Cartilage canals are well

documented and are considered to be important in initiating osteogenesis of secondary ossification centres and indeed appear to delineate the future path of the epiphyseal arteries.

Following epiphyseal union, confluence of epiphyseal and metaphyseal vessels occurs. Prior to closure however the growth cartilage is dependent upon three main vascular supplies, namely the epiphyseal, metaphyseal and perichondrial vessels.

Epiphyseal vessels

Between the growth cartilage and its associated epiphysis is the bone plate (or terminal bone plate) which separates the epiphyseal cancellous bone and the germinal cells. The epiphyseal arteries branch and anastamose freely within the epiphysis and terminal branches pass through foramina in the bone plate. These vessels form terminal expansions which cover the width of up to 12 cell columns (Morgan 1959; Trueta and Morgan 1960). Large veins are then formed which re-cross the bone plate in either the same or a different foramen to the arteriole. Commonly a large epiphyseal vein acts as a collecting vessel for several venules on their passage through foramina and this in turn passes into the epiphyseal sinuses. Trueta and Morgan (1960) point out that there is no apparent difference between the vascularity of the peripheral and central areas of the growth cartilage supplied by epiphyseally derived vessels.

The epiphyseal vasculature that penetrates the bone plate is undoubtedly concerned with nutrition of the growth cartilage.

Metaphyseal vessels

There are two sources of blood supply to the metaphyseal

aspect of the growth cartilage. The nutrient artery by virtue of its ascending and descending branches sends ramifications to supply the growth plate. In the human foetus metaphyseal vessels originating from the nutrient artery are described by Lewis (1956) as being the only available blood supply to the metaphyseal aspect of the growth cartilage. At a later stage of development however metaphyseal arteries which are derived from the periosteum supply the peripheral part of the growth cartilage and the extent of their area of supply progressively increases with advancement of age.

Although there is stated to be no distinction between end vessels derived from each source (Trueta and Morgan 1960) controversy exists over the nature of the vessels that abut on the metaphyseal aspect of the growth cartilage. Some authors describe well defined vascular loops, each related to a column of chondrocytes (Morgan 1959; Trueta and Morgan 1960). The loops are described as advancing towards the last hypertrophic cells of the columns and then turning back to drain into venous sinusoids which form large collecting vessels and veins. Trueta and Morgan (1960) consider that the end vessels of the metaphysis at this site never anastamose. Brookes (1971) summarises his earlier observations in which he noted the end vessels formed clumps with free anastomoses between varicose loops. Blind ending processes arising from such loops were also described. In contrast to the description of discrete well defined loops, Brookes (1971) states that each clump of vessels is related to a number

of cartilage columns. Different species were however studied by these authors.

Further controversy exists as to whether the vascular loops, intimately concerned with the zone of ossification of the growth plate are open or closed. Brookes (1971) adequately reviews the conflicting literature. Whereas some authors have described extravasation adjacent to the vascular loops (Lewis 1956; Trueta and Morgan 1960) others consider that a closed system with no leakage by vessels exists (Brookes 1971). Ham, Hurley, Ryan and Storey (1965) described an attenuated endothelium of metaphyseal capillary loops in rats with wide and numerous gaps present. Anderson and Parker (1968) consider that there is little doubt that the capillaries sometimes rupture and release blood cells, but the difficulty of studying such vessels without producing artefacts remains a problem.

The metaphyseal vessels are considered to have a nutritive function, insofar as they are thought to supply the raw materials for calcification in the hypertrophic zone. In addition it has been suggested that the metaphyseal vessels aid nutrition of the growth cartilage and may influence cartilage column formation (Brookes 1971).

Perichondrial Vessels

The connection between perichondrial vessels and the cartilage canals of the developing long bone has been briefly mentioned above. With development a plexus of small perichondrial vessels persist in the ossification groove of Ranvier and are presumed to have a nutritive function to the adjacent

cartilage cells. In such a manner they may be important in growth and expansion of the growth cartilage in a lateral direction (Brookes 1971). The perichondrial vessels are formed from the superficial anastomoses of the periosteal arteries and are thus connected with both the metaphyseal and epiphyseal arteries (Morgan 1959).

The nutritive role of these vessels was elucidated by Brodin (1955) who injected fluorochrome dyes (3-4' benzpyrene and 3-oxyprene - 5, 8, 10 - tri-sulphonate) into the ear vein of rabbits and subsequently followed their progression around the growth cartilage. Brodin demonstrated rapid diffusion of the fluorophores from the perichondrial plexus and into the edge of the plate. It was suggested however that as the fluorophores were detected in the depth of the growth cartilage very rapidly following administration, a canal system must exist for transport of nutrients within normal cartilage. Such a conclusion has however received an equivocal response.

Despite Brodins (1955) findings Rang (1969) reports that the perichondrial vessels appear to supply only the perichondrial cells and that there is little or no diffusion into the depth of the growth plate. Rang (1969) does however concede that they may be important in latitudinal growth of the cartilage.

Experimental interference with growth cartilage blood supply

The functional importance of the individual vessels which supply the growth cartilage has been elucidated by experimental studies. Many authors including Trueta (1963); Shim (1966) Rang (1969); Brookes (1971); Sissons (1971); and Little (1973)

refer to the interesting experiments by Trueta and Amato (1960) on ablation of the epiphyseal and metaphyseal vessels in growing rabbits. The monumental nature of this study however deserves more than the brief consideration it is usually given.

These authors drilled holes in either the epiphysis or metaphysis of 6 week old rabbits and introduced a spatula in such a manner as to disrupt the vasculature. The cavity so created was then packed with polythene film to deter immediate revascularisation. Following such an insult to the epiphyseal side of the growth plate, the changes noted were related to the degree of disruption produced. With relatively minor damage to the epiphysis, a transient thickening of the growth cartilage was noted which resulted from both an increase in size and number of the hypertrophic cells. Trueta and Amato (1960) explained this finding on the basis of the increase in vasculature noted below the bone plate especially peripherally. Thus in response to injury a transient increase in vasculature reflected in accelerated growth cartilage cell division occurred. If the epiphyseal damage was more extensive, a portion of the growth plate became infarcted with failureof development of normal cartilage columns. With further increase in severity of epiphyseal injury a greater area of the growth plate became infarcted and metaphyseal blood vessels entered the area and bone trabeculae were formed. If massive injury was inflicted rapid death of the whole central part of the plate occurred and metaphyseal vessels entered the devitalised area. Epiphyseodesis resulted although

the peripheral parts of the growth plate were least affected.

With metaphyseal damage the growth plate become grossly thickened due to an increased depth of the hypertrophic cell zone. No calcification of intercolumnar matrix occurred and no degenerative cells or signs of ossification occurred. Revascularisation however was followed by a return to normal calcification and ossification which proceeded at a very rapid rate initially peripherally and finally centrally.

Damage to vessels of the perichondrial ring resulted in the development of a large chondroma which often spread down the metaphysis and adjacent vessels invaded the cartilaginous mass. A disorderly vascular invasion however occurred and bone trabeculae were laid down in a haphazard manner.

Trueta and Amato(1960) interpreted their findings as indicating that nutrition of all the growth cartilage cells is derived from the epiphyseal vessels with the metaphyseal vessels being solely concerned with calcification of matrix, removal of degenerated cells and osteogenesis. Although this suggestion is contradictory to what some authors have thought (Brookes 1971) it is supported by the work of Brighton and Heppenstall (1971). The latter authors measured oxygen tensions in various zones of the growth cartilage in laboratory animals and noted that in vivo a very low oxygen tension occurred in the hypertrophic cell zone. This finding led Brighton and Heppenstall (1971) to suggest that low oxygen tension and calcification may be causally related.

Yabsley and Harris (1965) performed a study of the effect

of periosteal stripping, diaphyseal fracture and nutrient artery destruction in immature rabbit tibiae. Interruption of metaphyseal blood supply resulted in a transient thickening of the central region of the growth cartilage which as noted by Trueta and Amato (1960) was the result of accumulation of hypertrophic cells. Yabsley and Harris (1965) however noted that the epiphyseal vessels associated with the region of thickened growth cartilage increased in diameter and penetrated further than normal into the plate. Yabsley and Harris interpreted this finding as a response by the epiphyseal vessels to the altered metabolic requirements of the thickened plate. Associated with epiphyseal vessel change these authors noted a more rapid maturation of the adjacent cartilage cells so that the proliferative cell layer increased in thickness as well as the hypertrophic cell zone. In some cases the operated tibiae were significantly longer than controls.

A very similar series of experiments were performed by the Jewish workers Spira and Farin (1967). They also experimented with rabbits which were however slightly younger than those of Trueta and Amato, being four weeks of age at operation. Holes drilled in the metaphyses resulted in a transiently thickened zone of hypertrophic cells. Following metaphyseal osteotomy when a large gap was left at the operative site endochondral ossification was impaired. Spira and Farin (1967) noted that the masses of hypertrophic cells were invaded initially by peripherally situated metaphyseal vessels. Their findings thus confirm those of Trueta and Amato (1960) whose work they

appear to have overlooked.

If metaphyseal revascularisation was delayed for more than four or five days Spira and Farin (1967) noted epiphyseally derived vessels traversing the growth cartilage which they considered represented reappearance of vascular channels of the growth cartilage. In contrast to Yabsley and Harris (1965) however Spira and Farin described epiphyseally derived vessels entering the hypertrophic cell zone and initiating osteogenesis. In some cases of epiphyseal drilling Spira and Farin (1967) disrupted the epiphyseal vessels severely and noted an extension of metaphyseal vessels towards the epiphysis.

In the same study Spira and Farin performed diaphyseal osteotomy and resection so as to disrupt the nutrient vessels. Such a procedure resulted in an enlarged hypertrophied cell zone in the distal ulnar growth plate but the distal radial growth plate was similarly affected. It would seem plausible that this observation resulted at least partly from damage to the interosseal artery that supplies both bones. Although these authors described the interosseal artery of the rabbit in detail (Spira, Farin and Karplus 1963b) they attributed this phenomenon to 'reflex reaction' and suggested that further study was necessary.

Anastomoses exist between the metaphyseal and nutrient vessels which can result in no disruption of endochondral ossification following selective destruction of the nutrient artery (Cuthbertson, Siris and Rutherford 1964; Yabsley and Harris 1965). This explains the lack of response by the growth cartilage to nutrient artery destruction noted by Trueta (1953).

Brookes (1957) however unilaterally occluded the principal nutrient canal of rabbit femora and obtained initially slight bone shortening which was followed by equalisation of femoral length. Increased epiphyseal and metaphyseal blood supply was suggested as being the cause of growth stimulation which produced equalisation. Trueta (1953) did however obtain increased bone growth in rabbits following blockage of the medullary canal with bone wax and interference with the periosteal vessels. The inadaquacy of anastomoses (in some individuals) was demonstrated by Fyfe (1961) who obtained no radioisotope uptake by the central metaphyseal aspect of the growth cartilage in some rabbits following selective nutrient artery destruction.

Interest in influencing the vascular supply of the growth cartilage has been because of possible clinical applications to growth stimulation of long bones. The significance of such procedures as periosteal sectioning has been briefly discussed above in connection with the stimulatory effect of diaphyseal fractures.

Certain venous abnormalities are reported to stimulate bone growth. In particular considerable information has been accumulated of experimentally created arteriovenous fistulae (Kelly, Janes and Peterson 1959 ; Vanderhoeft, Kelly, Janes and Peterson 1963; Kelly and Janes 1968; Petty, Winter and Felder 1974) which produce small but unpredictable increases in bone growth. In addition enhanced diaphyseal remodelling

appears to be associated with the vascular derangement that follows their creation. Kelly and Janes (1968) supply possible explanations of these phenomena.

Similarly, but more equivocally, venous stasis has been considered to stimulate bone growth. Increased venous pressure either from venous ligation or application of tourniquets has been reported to stimulate growth of long bones (Hutchison and Burdeaux 1954 cited by Brookes 1971; Colt and Iger 1963; Singh and Brookes 1971) or produce enhanced cortical remodelling (Kelly 1968b; Lilly and Kelly 1970). It should be mentioned however that authors such as Dickinson (1953) failed to obtain increased bone growth in dogs following venous ligation and the influence of increased venous pressure on bones remains inconclusive (Kelly 1968a). Collateral circulation would appear to be of importance.

Factors influencing bone circulation

a) Theories of bone blood flow control Shim (1968); Vaughan (1970) and Little (1973) review the mechanisms that are thought to control bone blood flow.

b) Muscular Activity The influence of muscle activity on the circulation has been briefly reviewed by Trueta (1963;1964). Connections between the vascular systems of bone and soft tissues are well described by Brookes (1971). Shaw (1965) performed blood flow studies on muscle and bone in cats and concluded that changes in bone intramedullary pressure are to a considerable extent dependent on muscle blood flow through the osseomuscular circulation. In the same year Valderrama and Trueta (1965) observed that in dogs, muscle contraction decreased or blocked

the venous outflow of blood resulting in intraosseous vascular engorgement and thus an increase in intramedullary pressure. On cessation of muscle contraction the intra-osseous venous system rapidly emptied and a fall in intra-medullary pressure thus occurred.

It certainly appears that the intra-osseous circulation cannot be efficiently carried out by the heart alone (Trueta 1963) and Little (1973) states that in man the action of the muscles pumping blood out of bone is of almost equal importance to its arterial supply. Little (1973) discusses the importance of muscle activity on bone remodelling. She states that vigorous muscle action results in vigorous osteogenic activity with frequent remodelling occurring in cortical bone adjacent to the greatest muscular activity. Sim and Kelly (1970) have shown the close relationship between cortical bone remodelling and blood flow in canine long bones. Blood flow rates however have been shown to differ in different parts of the long bone which led Brookes(1967) to suggest that probably the pO_2, pCO_2 and pH exert a direct control over local osteogenesis. Trueta (1963) commented on the importance of oxygen availability and bone formation and Sim and Kelly (1970) noted that the percentage of oxygen extraction from arterial blood was similar in high and low bone remodelling states. Thus the blood flow would appear to be the only manner in which a region of bone can acquire an increased oxygen uptake.

c) Mechanical factors and bone circulation Harrison, Shajowicz

and Trueta (1953) studied osteoarthritis of the human hip and noted that in pressure areas bone trabeculae were thicker than normal and healthy lamellae sometimes covered dead bone. In such areas, in addition to increased arterial blood supply, venous engorgement was present. Some years later Trias (1961) noted that persistent pressure applied mechanically to rabbit knee joints resulted in increased subchondral bone density in the compressed area. With prolonged pressure however osteocytic death occurred and new bone lamellae subsequently covered the dead trabeculae. Trueta (1963) considers that repeated persistent compression such as that caused by weight bearing or muscle contraction results in closure of those canaliculi running in a direction perpendicular to the compressing force.Trueta (1963) explains in detail the theory of induction of osteogenesis by liberation of vascular stimulating factor from dying or severely damaged chondrocytes, osteocytes and endothelial cells. He proposes that closure of canaliculi results in cell death and release of vascular stimulating factor which in turn promotes vascular invasion and new bone deposition on the compressed trabeculae.

The increased density or sclerosis of ischaemic bone is well known. Coolbaugh (1952) for example disrupted the blood supply of dog femora and following an initial decreased cortical density of about 5 days duration increased cortical density was observed which was considerably greater than normal bone. An accurate densitometer was used in making such estimates of

cortical density. Trueta (1963) considers that regions of bone sclerosis arise from vascular invasion and new bone deposition on the surface of bone that has been deprived of its blood supply.

Methods of studying bone vascularisation

Studies on bone vascularity can be either qualitative or quantitative the former being concerned with morphological observations and the latter physiological information. Quantitative studies per se are beyond the scope of this study. All involve methods of varying complexity of estimating bone blood flow and haemodynamics. Descriptions of such techniques are given by Brookes (1965; 1967; 1970; 1971) Shaw (1965), Valderrama and Trueta (1965); Shim and Patterson (1967); Shim (1968); Kane (1968); Kato, Bassett and Silver (1970); Sim and Kelly (1970) and Paradis and Kelly (1975). Morphological studies however will provide some quantitative information when pathological specimens are compared with suitable control material.

Morphological studies of bone vascularity

Although dissection of postmortem specimens has frequently been applied to vascular studies the intraosseous circulation cannot be adequately investigated by this method. Researchers soon realised that their studies would be greatly aided by filling of the vascular tree with an easily identified material that contrasted with surrounding tissues. Such materials however were normally injected into major vessels at postmorten. Some techniques of intravital injection were described (Drinker and

and Churchill 1927) but were not applicable to bone studies. With the advent of radiology and the discovery of low toxicity injectable contrast agents in vivo visualisation of bone vascularity became a possibility but has more frequently been employed in arteriographic studies, (Bovill 1963; Kato, Bassett and Silver 1970). Such contrast agents of necessity are removed from the blood vascular system and their dilution by the blood volume can result in technical difficulties.

Most progress has been made by filling of the vascular system under study in experimental animals after death and in human postmortem material. A variety of materials have been used many of which have limitations. For example silicone rubber compounds in particle form have been injected under pressure into vascular trunks (Wilsman and Van Sickle 1970; Urbaniak, Bright, Gill and Goldner 1974) but Copland (1967) found that vessels smaller than 0.5mm diameter were not filled. Chung (1976) noted inconstant filling of vessels with latex methods. Use of such a material would appear to only be applicable,in conjunction with dissection, to studies of gross vasculature and not to the intraosseous circulation. Liquid plastics have also been used, normally in conjunction with digestion of surrounding tissues. Removal of surrounding tissues does however result in severe interpretative difficulties (Chung 1976). Other methods of arteriography employing a mercury emulsion and a coloured injectable paste are described by Rogers and Gladstone (1950) and Lewis (1955) respectively.

Many researchers have combined the injection of an easily

identified material with subsequent clearing of the bone specimen for visualisation of intraosseous vessels. Such a method is dependent on the fact that a block of tissue can be rendered transparent by employing Spalteholz method (Spalteholz 1914) or adaptations of it. (Halliburton, Sullivan, Kelly and Peterson 1958; Morgan 1959; Trueta and Morgan 1960; Nelson Kelly Peterson and Janes 1960; Kelly, Nelson, Peterson and Bulbulian 1961; Trueta and Buhr 1963; Trueta and Cadavias 1964; Copland 1967; Olerud and Danckwardt-Lilliestrom 1968; Wilsman and Van Sickle 1970; Mulfinger and Trueta 1970; Chung 1976; Colles 1976). Blocks of cleared bone can be easily investigated under low power binocular microscopy. Indian ink and Berlin Blue are commonly used in such studies and both have the virtue of being recognisable in histological sections as negative and blue stain respectively (Nelson, Kelly, Peterson & Janes 1960; Trueta and Morgan 1960; Kelly, Nelson, Peterson and Bulbulian 1961; Brookes, Elkin, Harrison and Heald 1961; Brookes and Lloyd 1961; Yabsley and Harris 1965). One further advantage of using either indian ink or berlin blue in the perfusion fluid is that the degree of filling of the limb can be readily assessed by observing the skin of the extremity for signs of colouration.

In general, varying forms of barium sulphate have been most frequently employed in studies of bone vascularity. This material can also be identified in cleared blocks of bone (Spira, Farin and Karplus 1963b) and in histological sections (Larsen, Kelly, Janes and Peterson 1961; Rhinelander and Baragry 1962; Wertheimer and Lopes 1971). Barium sulphate by virtue

of its radiopaque qualities can be used in conjunction with microradiographic techniques to obtain microangiograms. A comparable method to contact microradiography as described above is used. Historical reviews of the development of microangiography as a useful technique in bone vascular studies are presented by Barclay (1951); Bellman (1953); and Peterson, Kelly and Janes (1959). Bellman (Bellman and Engstrom 1952; Bellman 1953) advocated the use of stereoscopic microangiography but a limited number of studies have incorporated this refinement (Peterson, Kelly and Janes 1959 ; Kelly, Peterson and Janes 1959; Gothman 1960a and b). As with conventional microradiography information gained in microangiographic studies is greatly augmented by incorporation of standard histological observations. It is possible to use specimens prepared for microangiography for subsequent histological study as outlined by Kelly, Peterson and Janes (1959). Addition of either indian ink or berlin blue to the perfusion medium permits cleared specimens also to be obtained which provides greater clarity of vessels than if barium sulphate alone is perfused. Normally, the vascular system is prepared for perfusion by heparinisation and subsequent flushing with warm saline. Barium solutions are then injected either by gravity feed (Rietz 1968; Chaplin 1973) or manual pressure by means of a syringe or some other positive pressure system. Peterson, Kelly and Janes (1959) remark that the amount of pressure required varies with the area being studied, but that physiological

pressures are often sufficient. Frequently researchers have incorporated formalin in the final perfusion stages in order to fix the tissues (Gothman 1960a and b; Rhinelander and Baragry 1962; Rhinelander 1968; Rhinelander, Phillips, Steel and Beer 1968; Holden 1972; Chaplin 1973). Incorporation of gelatin in the perfusion medium has been performed which aids retention of the contrast media in specimens (Okawa and Trombka 1956; Halliburton, Sullivan, Kelly and Peterson 1958; Peterson, Kelly and Janes 1959). A number of researchers consider that perfusion is aided by addition of vasodilatory substances, such as sodium nitrate, to the perfusion medium (Halliburton, Sullivan, Kelly and Peterson 1958; Copeland 1967; Kelly and Janes 1968). Although proprietary barium sulphate solutions are most commonly used (Micropaque) Okawa and Trombka (1956) described a method of obtaining smaller particles of barium sulphate which permitted better filling of fine capilliaries. They describe the mixing of solutions of barium chloride and sodium sulphate which resulted in particles of approximately one tenth that of Micropaque particles. Some authors have subsequently mixed solutions of sodium sulphate, barium chloride and micropaque in their perfusion medium (Kelly and Janes 1968). The diameter of barium sulphate particles have been variously described as 0.5u (Gothman 1960a and b), 1u (Okawa and Trombka 1956) and 2u (Brookes 1958b). Thus some controversy exists as to whether barium sulphate traverses the capillary bed. Tilling (1958) for example considers that it does not.

Methods and Materials

Part C

During the post operative period and prior to slaughter four separate fluorochrome bone labels were administered. The fluorochrome dyes used in this study were

1. Alizarin complexone (3-Aminomethyl-alizarin - N_1N - diacetic acid) *
2. Oxytetracycline Hydrochloride **
3. Xylenol Orange ***
4. Fluorescein complexone ****

Each dye was prepared as described by Gilbertson(1975) for use in the dog and as shown in table 11

TABLE 11

Fluorochrome	Dose Rate	Preparation
Alizarin Complexone	30 mg/kg	As a 3% solution in a 2% solution of sodium bicarbonate
Oxytetracycline Hydrochloride	20 mg/kg	supplied in aqueous solution
Xylenol Orange	90 mg/kg	As a 3% solution in a 2% solution of sodium bicarbonate
Fluorescein Complexone	30 mg/kg	As a 3% solution in a 2% solution of sodium bicarbonate

The solutions of Alizarin complexone, xylenol orange and fluorescein complexone were filtered by means of a filter paper and funnel prior to administration.

* BDH Chemicals Ltd. Poole, England
** Terramycin Q. 50mg/ml injectable solution - Pfizer Limited
*** BDH Chemicals Ltd. Poole, England
**** BDH Chemicals Ltd. Poole, England

In all cases administration was by the intravenous route. A 19 guage 1½" needle was placed in the jugular vein and the dye injected by manual pressure at the approximate rate of 20mls/30 seconds. The sequence of dye administration in every case was

1. Alizarin complexone
2. Oxytetracycline Hydrochloride (OTC)
3. Xylenol Orange and
4. Fluorescein Complexone

The time of administration of the dyes in each experimental group is given in table 12. Control lambs were also given fluorochrome bone labels at the same time intervals as the experimental lambs of each group. In one case (Lamb No 59) Alizarin complexone was accidentally given as both the first and second fluorochrome labels. An error was also made with Lamb No 60. In this case xylenol orange was given as the first label and thus alizarin complexone was substituted for xylenol arange as the third label. In all other cases the fluorochromes were administered as shown in table 12.

As mentioned previously, prior to slaughter heparin* was injected (25000 units) into the jugular vein of each lamb. The lambs were then anaesthetised by administration through the same intravenous needle of pentobarbitone sodium** at a dose rate of 20 mg/kg and the ventral abdominal midline was clipped free of wool. An incision was made through the skin and linea alba extending from the xiphisternum to the pelvic brim and the digestive tract was then reflected to the right

* Heparin Injection B.P. 1000 units/ml The Boots Company Ltd.
** Sagatal - Pentobarbitone Sodium 60 mg/ml May & Baker Ltd.

TABLE 12

Day of Alizarin Complexone Admin.,	Day of OTC Admin.,	Day of Xylenol Orange Admin.,	Day of Fluorescein Complexone Admin.,	Experimental Survival Time
-1	1	3	5	7 days = 1 week
6	8	10	12	14 days = 2 weeks
7	11	15	19	21 days = 3 weeks
5	12	19	26	28 days = 1 month
24	34	44	54	56 days = 2 months
24	62	72	82	84 days = 3 months
42	90	100	110	112 days = 4 months
42	118	128	138	140 days = 5 months
42	146	156	166	168 days = 6 months
112	180	201	222	224 days = 8 months
112	236	257	278	280 days =10 months
112	292	313	334	336 days =12 months

Figures correspond to day of experimental period when label given

hand side of the sheeps abdomen. It was found helpful to actually displace the rumen from the abdomen through the midline incision and to subsequently reflect as much as possible of the intestinal tract in a similar manner. This procedure permitted visualisation of both the abdominal aorta and posterior vena cava. The abdominal aorta was then isolated at a point as close as possible to the left renal artery by means of blunt

dissection through the peritoneum and fascia dorsal to the aorta . Two ligatures were then positioned around the isolated segment of aorta and a cannula passed posteriorly down the aorta between them. The cannulae used were 30" lengths of Portex translucent vinyl tubing 2.5mm bore. The distal ligature was tightened in order to secure the cannula in position. Tension on the proximal ligature throughout the procedure of cannulation prevented the flow of aortic blood from obscuring the region. Following insertion and securing of the cannula the proximal ligature was tightened. Euthanasia was then performed by means of an overdose of pentobarbitone sodium* administered intravenously either into the jugular vein or the posterior vena cava. The posterior vena cava was then incised at the same level as aortic cannulation to permit free flow of venous blood from the posterior abdomen and pelvic limbs. It was found useful to position ligatures around both the aorta and posterior vena cava at this stage. By means of the apparatus shown diagrammatically in fig 32 the pelvic limbs were infused with a total of six litres of saline which was fed into the cannula by gravity. By allowing aortic blood to flow in a retrograde fashion out of the cannula and by judicial use of the three way tap (fig 32) and haemostats applied to the cannula the system was kept free of air bubbles prior to saline infusion. Any skin blood vessels incised during the procedure were easily identifiable at this stage by their flow of saline and could be ligated. Following infusion of the saline, flow from the posterior vena cava incision was found to be free of blood and

thus the pelvic limbs were effectively flushed free of heppanised blood. Infusion of saline was performed as fast as possible but this procedure was normally of 40-60 minutes duration. The measurements of joint mobility were made during saline infusion. On completion of saline infusion the pelvic limbs were infused with a contrast medium. Use of the three way tap and haemostats applied to the free end of the cannula enabled the system to be kept free of air bubbles. The contrast medium however was fed into the cannula by means of positive pressure (fig 32). Compressed air at a pressure of 3-5 lbs /sq.in. was used to force the contrast mixture from its container. A total of two litres of contrast mixture was infused which normally took 30-45 minutes. In all cases, after infusion of this quantity there was found to be undiluted flow of contrast medium from the incised vena cava. During contrast infusion it was found useful to reduce the rate of outflow to some extent by applying heamostats to partially occlude the posterior vena cava. Better filling of pelvic limb vasculature resulted. The filling of pelvic limb vessels could be assessed by shaving an area close to the skin horn margin of the digit and examining for blue colouration of the dermis caused by the Berlin Blue coloured contrast mixture. The contrast mixture was prepared by adding

1. 350g Barium Sulphate powder *
2. 70g sodium citrate and
3. 40g Berlin blue

to two litres of saline and mixing the suspension well prior

* Micropaque - Damancy Ltd

to infusion. (This mixture is basically similar to that described by Rietz (1968) except for the addition of Berlin blue. Rietz (1968) infused the contrast by gravity drip however).

Following infusion of the contrast mixture in the experimental lambs of this study, the vena cava and the aorta were occluded by means of the pre-positioned ligatures to prevent emptying of these vessels. At this stage photographs were taken of the limbs of those lambs with observable deformities.

The hindlimbs of all lambs were then radiographed to provide information on the degree of vessel filling obtained. The high definition cassette (24x30cms Siemens Rubin) was used and radiographs were taken of the femoral, tibial and metatarsal regions of each limb in both antero posterior and lateral projections.

Dissection of the individual bones of each pelvic limb was then performed prior to their deepfreeze storage. It was possible to make some observations on the blood supply to the metaphyseal regions from radiographs of the dissected bones.

The bones were sampled for fluorochrome analysis, microradiography, microangiography and histology as shown in fig 33. The bone samples were sectioned by means of a Metals Research Microslice 2 precision saw. This apparatus (Plate 26) is equipped with an annular blade which has an electrometallic diamond cutting surface on its inside edge. Section cutting is effected by rotary movement of the annular blade the spindle speed of which is adjustable between 50 - 1200 r.p.m.

The blade is mounted on a machined shaft and is held in tension from its periphery. The blade can be moved anteriorly or posteriorly in increments of 0.01 mm by means of the control at the front of the machine.

The bone specimen is mounted on the table at the end of a counter balanced arm. A weight is positioned at the opposite end of the arm and can be adjusted to impart a varying degree of load at the specimen/annular blade interface.

This apparatus was successfully used and described by Gilbertson (1975a:1977a) for sectioning of bone specimens.

A plate glass slide $\frac{1}{8}$" thick was attached to the table by means of low melting point dental impression wax* This is in contrast to the technique described by Gilbertson (1977a) where double sided pressure tape was used to attach the glass slide.

Initially double sided tape ** was used in this study but two major drawbacks were discovered. Firstly, use of double sided tape requires re-mounting of the glass slide after sectioning each specimen whereas the wax mounting was re-usable for approximately ten specimens. Secondly, on using the double sided tape ** the lubricant was found to enter and destroy the seal between slide and table. This resulted in the slide together with its attached specimen becoming detached during sectioning and as a consequence an annular blade was damaged. Thus wax mounting was routinely used in this study.

Two sizes of plate glass slides (3" x $1\frac{1}{2}$" x $\frac{1}{8}$" and $2\frac{3}{4}$" x 1" x $\frac{1}{8}$") were used depending on the size of specimen being

* Kerr Impression Compound Type 1 Kerr Sybron Corporation, Michigan, USA

** Scotch double sided tape

sectioned. The dental wax was melted in boiling water prior to use. Pieces of melted wax were removed from the water and flamed in a bunsen flame to remove moisture. It was found necessary to heat the surfaces of the table and glass slide prior to applying the molten wax between them. Once the slide was mounted on the table a thick layer of wax was placed on the upper surface of the glass slide. The bone specimen was positioned on this layer of wax. It was necessary to remove as much fibrous tissue as possible from the cortical bone surface prior to sectioning as if left attached, snagging of the blade occurred which tended to fragment thin bone sections being cut. In addition shallow cuts were made with a hacksaw blade on the bone specimens (fig 34) so that sections could be identified and orientated following sectioning. Unfortunately the depth of the annular blade produces a size limit on specimens that can be sectioned (approximately $1\frac{1}{4}$"). Thus some specimens had to be cut in half with a hacksaw blade and the two specimens so obtained sectioned separately (fig 34).

With the specimen in place on the wax coated glass slide, further pieces of wax were placed so as to completely encase the piece of bone. This procedure resulted in the bone specimen being held rigid while sections were cut. The wax coating around the bone specimens was sealed and smoothed by use of a heated scalpel blade applied over its surface. Prior to cutting it was necessary to allow the wax to cool and thus harden.

The wax encased specimen/glass slide/table assembly was

mounted on the pivot arm (fig 35) and the counterbalance weight and blade speed adjusted to provide a slow controlled speed of sectioning. This normally necessitated a spindle speed of approximately 200 r.p.m. which produced a single cut through the specimen in about 30 minutes. With cortical bone samples however the speed of cutting could be increased and by contrast some specimens of trabecular bone required one hour or more per section due to the fragility of the bone.

The specimens were sectioned as shown in fig 36. Thus a series of thin 100u sections and thick (3-5mm) sections were obtained. In some cases it was impossible to cut 100u sections because of the fragility of the specimen and thus 120u sections were prepared. In the case of epiphyseal/metaphyseal samples 1mm thick sections were made in addition to 100u and 3-5mm sections. In all cases the 100u undecalcified bone sections were placed in labelled containers and transferred to a deep freeze prior to further study. The 1mm and 3-5mm sections however were placed in labelled containers of 4% Buffered Neutral formaldehyde and were subsequently decalcified for approximately six weeks in a mixture of 10% formic acid, 10% formalin and 80% water. Wherever possible a Histette ion exchange apparatus was used for decalcification.

Following decalcification the bone slices prepared for histology were numbered in the manner shown in fig 36. The slices were dehydrated through alcohol and embedded using the following sequence.

1) 70% spirit + 5% phenol	6 hours
2) Methylated spirit	10 hours
3) Absolute Alcohol I (+5% phenol)	6 hours
4) Absolute Alcohol II (+ 5% phenol)	6 hours
5) Equal parts Absolute Alcohol and Amyl Acetate	4 hours
6) Amyl Acetate I	10 hours
7) Amyl Acetate II	10 hours
8) Methyl Benzoate/Celloidin I	20 hours
9) Methyl Benzoate/Celloidin II	20 hours
10) Xylene	4 hours
11) Wax I	14 hours
12) Wax II	20 hours
13) Embed	

Following embedding histological sections were taken from the bone faces corresponding to the stippled areas in fig 35. Sections of 7u ($\pm$ 1u) were obtained by means of a conventional microtome (Spencer-American optical) and were dried overnight in a 56^{o}C oven. The following day sections were stained with toluidine blue, haematoxylin and eosin, Van Giesson and in addition haematoxylin and short duration eosin to aid identification of osteoid. Haematoxylin and short duration eosin entailed exposure of the section to 0.5% aqueous eosin for 10 seconds (Disbrey & Rack 1970). In all cases D.P.X. mountant was used.

Histological sections were examined under transmitted light microscopy in a routine fashion.

Preparation of specimens for vascular studies - Following decalcification and clearing in xylene the 1mm thick sections

were transferred to and stored in a modified Spalteholz solution which comprised of equal parts of Oil of Wintergreen and Benzyl Benzoate. Examination of specimens so prepared was in the first instance performed by low power transmitted light microscopy. It was also possible to obtain photographic and optical enlargement of the vascular supply by mounting specimens between thick glass slides in a suitable mountant. A number of different mountants were tested but only the standard histological preparations (D.P.X. and Harleco-Coverbond T.M) produced consistently good results. Elastic bands were used to ensure that the bone section remained flat during hardening of the mountant. An enlarged image of the bone section was obtained by microscopy and by placing the mounted specimen in a conventional photographic enlarger negatives could be prepared from the projected image. Certain disadvantages were however inherent in this latter method. In particular it was impossible to orientate the specimen in other than the horizontal plane and thus the three dimensional nature of the bone section was both obscured and resulted in difficulties in interpretation. Also, shadows of trabeculae were projected which in some cases could not be easily distinguished from the blue coloured vasculature on monochrome prints.

Finally, microangiographic detail was present in the 100u undecalcified sections prepared for microradiography. In all cases the barium filled vessels were easily discernible but usually cross sections of vessels, in various planes, were visualised by this method.

Preparation of microradiographs - Contact microradiography was performed on the 100u undecalcified bone sections. The apparatus

used was the same as was successfully used and described by Gilbertson (1975b:1977b)and consisted of a Machlett OEG 50A x-ray tube with a tungsten target and a focal spot size of 1.5mm. In all cases a thin layer of polythene film* was interposed between the bone section and the photographic plate.** Close contact between the bone section and photographic plate was ensured by use of clamps and sheets of perspex as described by Gilbertson (1975b: 1977b). A target plate distance of 28cms was used for all microradiographs. Numerous exposure settings were tried but only use of 20KV, 15MA with either a 40 or 50 minute duration proved satisfactory. In all cases the shorter duration setting was chosen. Following exposure, the photographic plate was developed under continuous agitation for 5 minutes or longer in Kodak D19 Developer. In some cases as much as 10 minutes development was required. This was especially so when the developer had been prepared more than a few days previously. The plate was then transferred to a bath of Ilford Hypam fixative for approximately 2 minutes duration under continuous agitation. Finally the plate was washed in a running water bath for approximately 10 minutes prior to being dried in air at room temperature. Microradiographs so prepared were examined under transmitted light microscopy at low power. Photomicrographs were prepared in a routine manner. Adequate definition of bone specimens was obtained in all instances.

* Cling Film
** Kodak - High Resolution Plates

RESULTS PART C

A detailed consideration of the operated right hind limb has been deemed superfluous and will not be described. Only where significant, will changes in that limb be mentioned. Control lamb observations however invariably represent an amalgamation of findings in each limb. Only where significant disparity between limbs was present have they been considered separately.

The appearance of the metaphyseal-growth plate junction in undecalcified and Spalteholz cleared sections

Frequently, in both control and experimental lambs, a minor degree of irregularity was noted in the extreme periphery of the metaphysis at its junction with the growth cartilage. In many control lambs the remainder of the metaphyseal growth plate junction was of relatively regular outline, with epiphyseal bone plate contour paralleling that of the metaphysis. Some anomalies were however encountered. For example, lamb No.44 (3 week control) possessed a step-like defect in the lateral metaphysis of its left distal metatarsus (see plate 27A). Similar defects were noted in the lateral metaphyseal sector of metatarsae in control lambs 53, 16 and 11. Much more frequently areas of undulation were noted in the medial metaphyseal sector of control lambs which was not accompanied by comparable change in the adjacent epiphyseal bone plate. Such undulations were normally present over a limited width of the metaphyseal growth plate junction and were observed in the distal metatarsae of control lambs 59, 60, 53 and 11 and in the distal tibiae of numbers 48, 53, 81, 35, 19 and 11. The growth cartilage

became extremely thin and bone bridges between the epiphysis and metaphysis were seen at the commencement of growth plate closure. In distal metatarsae, growth plate closure was commencing at 11-13 months of age and in distal tibiae at 13-15 months. Evidence of early bone bridges were observed in distal metatarsi of some younger control lambs.

Considerably more irregularity of the metaphyseal growth plate junction was noted in the non-operated limbs of the experimental series. Irregularity of outline of the metaphyseal periphery as seen in control lambs was commonly encountered.

The first observable abnormality seen in undecalcified sections was a line of cleavage in the calcified growth cartilage matrix and primary spongiosa. The line of cleavage traversed a variable number of chondrocyte columns of the lateral growth plate sector and was observed in both the distal tibia and metatarsus of lamb No. 43 (3 weeks postoperative - plate 27B) and distal metatarsus of lamb No. 46 (1 month postoperative - plate 27C). In one lamb (No.33) at only two weeks postoperatively, a similar cleft could be discerned in anterior sections whereas posterior sections possessed a small island of endochondral bone at a comparable site (plate 27D). A similar but smaller defect of lateral metaphyseal outline as noted in control lamb No 44 (plate 27A) was observed in the distal metatarsae of lambs 45 and 46 (1 month postoperative). As in control lambs the medial metaphyseal sector commonly had an undulated contour in experimental lambs slaughtered during this early postoperative period. Such undulation was however more pronounced and was accompanied by increased medial growth plate depth in those with demonstrable clefts in the lateral growth

cartilage sector.

With advancement of the postoperative survival period islands of endochondral bone were more frequently encountered laterally in either or both the non-operated limb metatarsus and tibia. (plates 28 A,B,C). As in lamb No 33 endochondral bone islands were present in sections taken from the posterior aspect of the epiphysis/metaphysis. In those cases in which bone islands were also present in anterior sections they were of smaller dimensions. More frequently anterior sections revealed only a degree of growth plate thickening or minimal observable abnormality. At this time, marked undulation and irregularity of the medial metaphysis was encountered in association with a thickened medial growth cartilage (plates 29 A.B.C). Anteriorly derived sections of distal tibiae normally exhibited greater medial growth plate thickening and more obvious undulation of metaphyseal contour. In distal metatarsae however no such obvious pattern emerged as in some cases the anterior and posterior sections possessed a similar degree of medial growth plate thickening whereas in others asymmetry occurred.

In lambs slaughtered at 4 or more months postoperatively more dramatic abnormalities were present. Those with milder limb deformities were normally of a similar although frequently more marked appearance to that noted above. Thus islands of endochondral bone were observed posteriorly and to a lesser extent in mid epiphyseal/metaphyseal sections.

More extreme deformities possessed a lucent defect located more proximally in the metaphysis especially in posterior sections. The metaphyseal defect was effectively 'isolated' from the growth

cartilage by fusion having occurred between regions of the endochondral bone island and the metaphysis (see plates 29 A and B). By virtue of metaphyseal remodelling such defects normally communicated with the lateral, periosteal, surface of the metaphysis. Mid epiphysis/ metaphysis sections in advanced deformity limbs demonstrated either a discrete endochondral bone island, surrounded by growth cartilage, or a degree of fusion between island and metaphysis. The metaphyseal defect in such cases was not as proximally located in the metaphysis as was noted in posterior sections. Anterior sections similarly possessed either endochondral bone islands, a thickened lateral growth plate or some intermediate appearances. In some severely deformed bone extremities fusion between the endochondral bone island and its adjacent epiphysis was noted (see plates 30 A,B&C). This feature was particularly prevalent in posteriorly sited sections. Although such fusion was commonly encountered in lambs of an age when epiphyseal closure would predictable be commencing, this was not always the case. Thus the distal metatarsus of lamb no 36 (8 months of age see plate 30A) was completely fused over part of its lateral aspect and a similar appearance was encountered in the distal tibiae of lambs Nos 36 & 18 (11 months of age see plates30 B & C). More significantly epiphyseal fusion was relatively less advanced in areas of growth plate thickening in deformed bones than in either the contralateral (operated) limb or in comparably aged controls.

With advancement of deformity the marked undulations noted over the medial metaphyseal growth plate junction became associated with well defined protrusions of epiphyseal bone. Epiphyseal bone protrusions normally correlated with regions of greatest growth plate

thickness. In the longest surviving lambs bone bridges appeared in a comparable manner with epiphyseal bone protrusions traversing the much thinned growth cartilage at some sites. In the longest surviving lambs with gross distal tibial deformity, although epiphyseal fusion had commenced in the medial sector, a lucent defect was present to a variable degree more proximally sited in the metaphysis (plate 30C). Such an appearance bore certain resemblances to that encountered in the lateral metaphysis.

ANGIOGRAPHY

The degree of filling of the pelvic limb vasculature was easily visualised on radiographs taken of the undissected limbs following perfusion (plate 31). Although the method of perfusion employed was successful in all cases, in some the soft tissue vasculature was more obviously demonstrated.

In addition, radiographs taken of the dissected pelvic limb skeleton were assessed. In all cases the nutrient vessels, their ramifications and the intraosseous epiphyseal vessels were visualised, although with greater ease in some specimens than others. Those lambs in which poorer results were obtained were invariably those in which greatest difficulty was experienced in performing perfusion. Following perfusion in the majority of specimens however, the vasculature was uniformly and adequately filled with contrast medium.

The Spalteholz cleared sections of bone prepared specifically for angiographic observations provided the best indication of the success of the method. Even in those cases in which perfusion was judged to be less than adequate meaningful observations were still possible.

It is convenient to describe those observations made on bone vasculature in control lambs prior to considering the grossly abnormal appearance in deformed individuals. Such observations have been devised from scrutiny of

i) Spalteholz cleared sections
ii) microradiographs prepared of 100u undecalcified sections
iii) histological sections
iv) to a lesser extent undecalcified sections examined under ultraviolet light.

A) Control lambs

i) Epiphyseal vessels: The arterial and venous components of epiphyseal vasculature were in many cases not easily distinguished in Spalteholz cleared sections. Normally however the more irregular contour of venous channels and their termination in large, contrast-filled venous collecting channels within the substance of the epiphysis aided identification. The arterial channels however were of more uniform calibre and gradually reduced in diameter as their terminal branches were approached. Identification was further aided by the fact that the contrast medium contained both barium sulphate and berlin blue. Although the barium sulphate did enter the venous system it did not gain such ready access as the berlin blue. Thus the venous system tended to be stained an intense blue colour in both Spalteholz cleared and histological sections. The arterial channels appeared as a paler blue due to the relatively higher proportion of barium sulphate. A similar tendency was noted in microradiographs with venous channels tending to be less radiodense.

As arterial channels approached the growth cartilage, numerous small vessels, derived from a parent arteriole, penetrated the bone

plate through vascular foraminae. The plexus of vessels so formed were often of more irregular outline and appeared in many instances to be blind ending sacculations (plate 32A). Close scrutiny however revealed their ending in discrete loops in at least some cases. Such loops were of variable dimensions, being of such a width as to correspond to one, two or occasionally more, chondrocyte columns. A similar plexus of vessels re-crossed the bone plate to form more irregular outlined vessels which ultimately connected with identifiable venous channels. In most instances however the arterial and venous components of vessels close to the bone plate were not distinguishable. Similarly the plexus of vessels on the growth cartilage aspect of the bone plate could not be readily identified as consisting of arterial and venous limbs.

A remarkable constancy existed between the degree of filling of the vasculature with contrast medium and the depth of the growth plate. Frequently minor differences in thickness existed between the medial and lateral aspects of the growth cartilage. In many Spalteholz cleared sections the medial growth plate sector was thicker and thus the medial epiphyseal vasculature was more prominent (plate 32B). In those few cases in which the lateral growth cartilage was of greater depth the lateral epiphyseal vasculature was generally more pronounced. Where the growth plate was of equal thickness across its width the epiphyseal vasculature was also symmetrical. Such a finding was most easily appreciated by observation of the plexus of vessels penetrating the bone plate and on its growth cartilage aspect.

Plate 27A demonstrates an example of a slightly aberrant growth plate in 3 week control lamb No 44. In the area of growth plate

thickening the plexus of vessels which penetrated the bone plate were of greater dimensions and penetrated the growth cartilage for a greater distance than normal.

Epiphyseally derived vessels also penetrated the growth plate to an abnormal depth prior to epiphyseal closure. At such a time the epiphyseally derived vessels appeared to traverse the much thinned growth cartilage ultimately to anastomose with the metaphyseal vasculature.

ii) Metaphyseal vessels: A degree of difficulty was again encountered in distinguishing the arterial and venous components of metaphyseal vasculature in Spaltcholz cleared sections. Metaphyseal vessels were generally more profusely filled than the corresponding epiphyseal vessels. Those criteria which aided interpretation of epiphyseal vasculature were however also applicable to metaphyses.

In young lambs, the growth cartilage was supplied exclusively from nutrient and thus endosteally derived vessels. In older control lambs however metaphyseal arteries derived from periosteal vessels supplied the peripheral region of the metaphysis and growth plate.

Branches of the metaphyseal vessels ran in an almost vertical manner towards the growth cartilage. Amongst trabeculae however the vascular pattern became very indistinct due to the density of vasculature and the superimposition of venous and arterial systems. As the growth cartilage was reached a plexus of dense irregularly outlined vessels was present. On close examination vessels could be seen to terminate in definite loops in some instances. Such loops were however of only one or two chondrocyte columns width.

In histological sections some intertrabecular spaces contained

contrast which was not delineated by a vessel wall. A similar impression of extravasation was exhibited in some Spalteholz cleared sections.

As with epiphyseal vasculature, a correlation between growth plate depth and density of metaphyseal vasculature which was contrast filled became apparent (plate 32B). Such a correlation was however not absolute with a few cases showing anomalous contrast distribution.

iii) Periosteal - Perichondrial Vessels: Despite removal of the periosteum to aid microslice sectioning of samples, both periosteal and perichondrial vessels could be identified in a number of Spalteholz cleared sections. In such cases, perichondrial vessels were seen to supply branches to the periphery of the growth cartilage (plate 32C). In addition the perichondrial vessels served as a vascular connection between periosteal and epiphyseal vasculature and thus linked the bone components either side of the growth cartilage. In some cases, such anastomoses appeared deeply seated within the growth cartilage periphery (plate 32C). In older lambs the perichondrial/periosteal vessels similarly linked the now more profuse metaphyseal arteries with the adjacent epiphyseal vasculature.

iv) Cortical Vessels: A detailed investigation of cortical vasculature is beyond the scope of this study. Some limited observations on metaphyseal cortical vessel orientation and in some cases extent of periosteal and endosteal vessel supply has been considered relevent.

In a few control lambs sufficient metaphyseal cortex was present and adequately perfused to permit an indication of vessel orientation. In all cases the metaphyseal cortical vessels had an oblique or

vertical course as shown in fig 37. Obliquity when present was considered to be a closer approximation to vertical in the medial cortex (see fig 37).

B) Experimental lambs

i) Epiphyseal vessels: The general morphology and distribution of epiphyseal vessels in the distal tibiae and metatarsae of experimental lambs was as described above. The correlation between degree of contrast filled vasculature and growth plate width was however much more obvious. In almost all cases growth plate thickening was accompanied by more profuse filling of the epiphyseal vasculature especially in the region of the bone plate (plates 33A & C, 34 A). In those limbs which underwent deformity this feature was particularly marked. Where defects developed in the lateral metaphyseal sector, the epiphyseal vasculature was only scantily filled. Despite this appearence, areas of lateral growth plate thickening were accompanied by epiphyseal vessels entering the growth cartilage and traversing its thickness to a variable degree (plate 33 A & D). In limbs which contained gross metaphyseal defects tongues of epiphyseal vessels crossed the entire thickness of the growth cartilage and terminated in the metaphysis in the region of the defect (plate 33 B & H). Such vessels were always orientated perpendicularly to the growth cartilage and thus were parallel to the chondrocyte columns. In a number of the longer surviving lambs not all of the lateral growth plate sector of deformed bones could be distinguished (plates 33 B, E, F, G & H). Areas of epiphyseal fusion, with apparent confluence of epiphyseal and metaphyseal vasculature, were evident at such sites. Generally, in areas of gross metaphyseal

defects in the lateral sector of deformed limbs, the epiphyseal vasculature was relatively more profusely filled.

The medial growth plate sector was however in almost all instances equipped with a gross and much more profusely filled epiphyseal vasculature (plates 33 A & C, 34 B, C, D, & E). With advancement of the postoperative survival period this observation was noted in all cases. In certain areas of the medial bone extremity, especially in long surviving lambs the bone plate was irregular and protrusions of epiphyseal bone occurred within the growth cartilage. Such protrusions were always accompanied by profusely filled epiphyseal vessels and occurred in regions of gross growth cartilage thickening (plates 34, D, E, F, & G) In longer surviving individuals epiphyseal protrusions were similarly observed but confluence of epiphyseal and metaphyseal vessels and the presence of associated bone bridges marked the advent of growth plate closure.

ii) Metaphyseal Vessels: The experimental lambs exhibited a similar distribution of metaphyseal vessels as in control animals. Metaphyseal vessel filling similarly correlated well with growth plate thickness although some aberrations became apparent. In the early postoperative period and in those with minimal deformity there was a relative deficiency of metaphyseal vessel filling over the extreme lateral sector of the growth cartilage (plate 35 A & B). In such areas there was frequently a moderate degree of growth plate thickening but in this instance metaphyseal vessel filling was deficient. With advancement of the postoperative period, the metaphyseal vasculature become relatively more profusely filled associated with the by now gross metaphyseal defect (see plates 33 A to H). In long surviving

lambs metaphyseal vessels also penetrated the defect in a manner and orientation identical to that noted with epiphyseal vessels. Distinct anastomoses between the two circulations, across the metaphyseal defect, were not encountered. In some of the cases in which an 'island' of metaphyseal bone could be identified between the growth cartilage and the metaphyseal defect the vascular supply was clearly established as being of periosteal origin (plate 28B, 33D and G). Thus, in such cases metaphyseal arteries undoubtedly supplied the bone 'island' which had become isolated from endosteally derived vessels. The predominantly endosteal vascular supply to the growth cartilage noted in young control individuals was similarly observed in the experimental series. Establishment of periosteally derived vessels supplying the most lateral aspect of the metaphysis and growth cartilage appeared to be accelerated by the ensuing deformity.

In young lambs, metaphyseal trabeculae and their adjacent vasculature tended to have a common longitudinal orientation approximately parallel to the long axis of the bone. Such an orderly orientation in the direction of growth became less easily discernible in long surviving, grossly deformed, lambs especially in regions of growth plate irregularity and adjacent to the metaphyseal defect (plates 33, B,E,F & G)

iii) Periosteal - Perichondrial vessels: In a number of the deformed bone extremities the periosteal-perichondrial vessels were identifiable. Just as was noted in control limbs, such vessels effectively linked the vasculature of the epiphysis and metaphysis (plate 35A). Perichondrial vessels could be identified supplying the periphery of the growth cartilage both medially and laterally but were

especially distinct and profusely filled over the bulging medial growth plate in grossly deformed lambs (plates 34 B & E). The particular importance of the periosteally derived metaphyseal arteries and veins in the region of the developing lateral defect has been described above. In addition similar vessels could be identified medially in long surviving lambs but generally were less well defined. Endosteally derived vessels could usually be traced supplying all the medial growth plate even in grossly deformed bones. Periosteally derived metaphyseal veins, when present, appeared to anastomose with the endosteal venous system in both the medial and lateral metaphysis.

v) Cortical vessels: In a number of lambs of the experimental series the orientation of distal diaphyseal and metaphyseal cortical vessels could be observed. The site of sectioning precluded meaningful observations in many instances.

The predominant vessel orientation in the medial cortex was vertical. In some lambs with marked deformity the periphery of the medial cortex was supplied by transversely orientated periosteal vessels (see fig 37).

The lateral cortex was endowed with obliquely orientated vessels similar to that noted in control lambs. With advancement of deformity however the lateral cortical vasculature became progressively less oblique and in long surviving individuals tended to be approximately transverse. In some deformed limb extremities two thick bone sections were prepared for angiographic study by the Spalteholz method. Vessel orientation differed in each section. The anterior section of la.b No 18 distal tibia demonstrated a shallow oblique orientation of

lateral cortical vessels. The posterior section however possessed a more haphazard arrangement of lateral cortical vessels with no predominant pattern being identifiable. A similar haphazard orientation was noted in the Spalteholz cleared section of the lateral cortex of the lamb No 55 distal tibia.

Plates 33, B,E & G and 34 B & C demonstrate some of the findings represented in fig 37. Cleared sections also permitted limited observations on the source of cortical vasculature.

In young lambs and those exhibiting minimal deformity the lateral cortex received a relatively large contribution from periosteal vessels. With advancement of deformity a progressive reduction in periosteally derived blood supply occurred and ultimately the entire lateral cortical vasculature was of endosteal origin. The medial cortex however gained an increasing amount of periosteal blood supply with advancement of deformity. Table 13 demonstrates this finding.

Anastomoses between capillaries derived from periosteal and endosteal vessels could be identified. Detailed examination of the arterial and venous components of cortical vascularisation was not performed.

TABLE 13

Lamb number	Postoperative Survival Period (months)	Sample	Angular Deformity	Extent of Endosteal supply to lateral cortex
51	3	LH Distal Tibia	-	At least $\frac{2}{3}$
29	5	LH Distal Tibia	Minimal	At least $\frac{3}{4}$
36	5	LH Distal Tibia	Marked	At least $\frac{7}{8}$
36	5	LH Distal Metatarsus	Moderate	At least $\frac{3}{4}$
37	6	LH Distal Metatarsus	Moderate	Entire width
39	6	LH Distal Tibia	Marked	At least $\frac{2}{3}$
55	6	LH Distal Tibia	Moderate	Entire width
17	8	LH Distal Tibia	Minimal	At least $\frac{3}{4}$
17	8	LH Distal Metatarsus	Moderate	Entire width
18	8	LH Distal Tibia	Extreme	Entire width
14	10	LH Distal Tibia	Extreme	Entire width
14	10	LH Distal Metatarsus	Slight	At least $\frac{3}{4}$
12	12	LH Distal Tibia	Marked	Entire width

* Approximate angular deformity at distal articular surface

\- = 0

Minimal = 0^{o} - 5^{o}

Slight = 5^{o} - 10^{o}

Moderate = 10^{o} - 20^{o}

Marked = 20^{o} - 30^{o}

Extreme = 30^{o}

MICRORADIOGRAPHY

i) Primary Spongiosa In both control and experimental lambs the mineralised cartilage matrix, commonly referred to as the primary spongiosa, could be identified in microradiographs of undecalcified 100u sections. In both groups the primary spongiosa zone was of greatest depth in young animals and extremely thin immediately prior to epiphyseal closure. The primary spongiosa in control lambs contained regular spars of mineralised cartilage matrix, directed approximately to the long axis of the bone. Towards the periphery of some sections however the mineralised spars were directed slightly outwards on their metaphyseal aspect. Those control lambs which possessed minor defects in lateral metaphyseal/growth plate contour had a reduced depth of primary spongiosa at such sites. Undulations of metaphyseal outline over the medial growth plate sector were associated with a primary spongiosa of variable depth. Despite such anomalies many control lambs possessed a more or less equal depth of primary spongiosa medially and laterally.

The primary spongiosa was also of almost equal depth over the majority of the medial and lateral growth plate aspects in the non-operated limbs of experimental animals. Anomalies were however encountered associated with the development of deformities. In short surviving lambs the lateral primary spongiosa zone, close to the periphery, was sometimes of slightly increased but more frequently decreased depth (plate 27D). Variation was detected in the same bone extremity depending on whether the section was anteriorly or posteriorly derived.

A distinct cleft traversing the lateral spongiosa from the periphery

was detectable in two lambs (plates 27 B & C) slaughtered in the early postoperative period.

Those cases with a distinct bone 'island' located in the periphery of the lateral growth cartilage showed more obvious derangement of the associated primary spongiosa. Frequently the 'island' possessed a thin zone of primary spongiosa but in some cases this was barely perceptable (plates 28 A, B & C). The outwardly directed columns of the most peripheral primary spongiosa were more frequently observed in experimental than control animals and were also noted in the lateral growth plate 'islands' of mildly deformed bones (see plates 27D & 28B). The primary spongiosa was also grossly reduced in depth or absent in the metaphysis adjacent to the bone island (plates 27D, 28, A,B & C). In addition such spongiosa appeared less well mineralised and was often not orientated according to the bones long axis (plates 28 A & C). The normal, orderly, columnar arrangement of primary spongiosa was absent at this site with varying degrees of disorganisation being noted. In longer surviving cases, with lateral metaphyseal defects, the primary spongiosa was observed over the extremity of the defect and thus at its most proximal site in the metaphysis. At this site the spongiosa zone was of variable depth and organisation and when columns were present they were directed approximately parallel to the bones long axis. Primary spongiosa lined other aspects of the metaphyseal defect to a variable extent but rarely showed any measure of organisation.

The medial primary spongiosa zone was frequently of slightly greater depth in the non operated limbs of experimental lambs. Varying degrees of disorganisation of the spongiosa were however noted in

association with undulation of the metaphyseal/growth plate contour. In areas of growth cartilage protrusion into the metaphysis, the columns of the primary spongiosa were less inclined to approximate to the bones long axis. In addition, the spongiosa zone was of reduced depth over such protrusions. A clear columnar arrangement to the mineralised cartilage spars was often difficult to observe at such sites and over much of the medial growth plate in longer surviving individuals. Associated with the discrete metaphyseal protrusions, commonly present in grossly deformed bones, the primary spongiosa columns were frequently absent but when discernible clearly bore no relation whatsoever to the bone's long axis. The three longest surviving lambs with gross tibial deformity possessed a variable amount of primary spongiosa more proximally located in the metaphysis in addition to that noted adjacent to the growth cartilage. The mineralised cartilage spars at both sites, but in particular those more proximally located, were relatively disorganised in appearance.

ii) Metaphyseal bone trabeculae In the metaphysis, immediately adjacent to the primary spongiosa zone bone had accumulated around the mineralised cartilage spicules. The remnants of the primary spongiosa contained in such trabeculae were normally of greater density in microradiographs and thus relatively easily observed. In control lambs and in those bones of experimental lambs with no observable deformity, the bone trabeculae adjacent to spongiosa were orientated parallel to the bones long axis and thus in an identical manner to the mineralised cartilage spicules. Such trabeculae were however less numerous and of greater dimensions than the cartilage spicules which formed their core. In those cases in which the spongiosa columns

were slightly outwardly directed at the periphery of the bone, the adjacent metaphyseal trabeculae had a similar orientation (plates 27C & 28B). Generally the metaphyseal trabeculae accentuated this feature and thus were of a slightly curved profile. On occasions, in young experimental lambs with minimal deformity, the metaphyseal trabeculae were only deviated in the extreme lateral region of the metaphysis. In addition, in other individuals a deviated profile to trabeculae was noted adjacent to the spongiosa over parts of the medial metaphysis but not at its periphery. More proximally in the metaphysis the trabeculae were much fewer in number but were of greater dimensions. In control and undeformed experimental lambs such trabeculae were orientated approximately parallel to the bones long axis. Remnants of mineralised cartilage spicules could be identified in trabeculae which had become consolidated and incorporated in the metaphyseal cortex. The most proximally sited trabeculae were separated by intertrabecular spaces which were orientated according to the blood vessels that supplied its adjacent cortex (see fig 37)

The most proximally sited trabeculae of grossly deformed bones were similarly orientated according to the cortical blood supply. Similarly, in those lambs in which a marked periosteal circulation was noted medially, a more horizontal orientation of adjacent trabeculae was observed. In such instances a similar trabecular orientation was frequently noted peripherally quite close to the spongiosa zone. Generally however the medial metaphyseal trabeculae demonstrated a predominantly vertical orientation (plate 29A) approximating to the long axis of the bone. In regions of the medial spongiosa where mineralised spicules were disorganised and variously orientated

adjacent metaphyseal trabeculae were of irregular contour (plate 29B). Such trabeculae initially approximated to the orientation of the adjacent spongiosa but were of such a contour that their most proximal region conformed to the disposition of more mature trabeculae. Metaphyseal trabeculae were usually much wider and more numerous medially in deformed bones. In grossly deformed bones the most proximally sited trabeculae frequently formed coarse struts of bone separated by wide intertrabecular spaces orientated as for the principal blood supply of the region (plates 29A & B, & 30A.). Those cases in which primary spongiosa was detectable at two levels in the medial metaphysis typically possessed closely packed metaphyseal trabeculae (plate 30C).

The somewhat shallow spongiosa zone noted laterally in deformed bones was associated with adjacent metaphyseal trabeculae of more modest dimensions. Adjacent to the metaphyseal defect in more advanced deformities however, trabeculae had a less organised appearance with a predominant orientation being difficult to discern. As elsewhere the initial orientation approximated to that of the mineralised spicule disposition when a distinct spongiosa zone was present. Trabeculae associated with the most proximal aspect of the defect typically possessed an almost parallel orientation but approximating to the long axis of the bone prior to its angular deformity. More mature trabeculae were directed in a manner appropriate to the predominant vasculature at that site. In long surviving grossly deformed bones the lateral metaphysis frequently contained trabeculae of equal dimensions to those present medially (plates 30 B&C).

iii) <u>The bone plate</u> In control lambs the bone plate was of more

or less regular thickness across its width. Those cases with localised increases in growth cartilage thickness possessed patchy radiolucency of the adjacent, more roughened, equivocally thickened-bone plate.

In experimental lambs, a roughened contour and patchy radiolucency of the bone plate invariably accompanied areas of growth plate widening both medially and laterally. In one instance a defect in the extreme lateral primary spongiosa was accompanied by a much thinned bone plate (see plate 27C). With advancement of deformity, the lateral bone plate was seen to be abnormally thin, invariably of irregular contour and in many instances trabeculated (plates 29 B,C, & 30A). Some areas of the lateral bone plate in deformed limbs appeared abnormally radiodense. Areas of increased radiodensity were especially evident in association with a roughened bone plate contour.

With the advancement of deformity the medial bone plate by comparison increased in thickness and protrusions of epiphyseal bone plate into the growth cartilage possessed areas of increased radiodensity. Medial bone plate thickening resulted in the foramina, through which epiphyseal blood vessels passed, being of greater depth medially. In some cases a distinct impression of both an increase in number and width of medial bone plate foramina was observed. Foramina of the lateral bone plate however appeared in some instances to be sealed by new bone thus prohibiting the passage of epiphyseal vasculature to the growth cartilage.

iv) Bone density As mentioned above irregularities of the bone plate frequently possessed areas of increased radiodensity. In particular where protrusions of the medial bone plate into the growth

cartilage occurred, areas of increased radiodensity containing large lacunae were often present. Dense bone also formed in the medial peak or spur of the epiphysis that accompanied severe bone deformity.

Areas of radiodensity of a more amorphous nature, with no distinct lacunae were noted in the lateral bone island that formed. Bone bridges which marked the advent of epiphyseal union were also relative radiodense and similarly possessed few or no lacunae.

v) Distal metaphyseal cortical bone All of the cortical bone types reported to be present in compact bone could be identified in undecalcified sections. Thus both primary and secondary osteones, plexiform and coarse compacted cancellous bone could be recognised and the proportions of each type was largely dependant on the site of section. Compacted coarse cancellous bone was detected on the endosteal aspect of distally sited sections and thus those closest to the growth cartilage. Endosteally and periosteally derived bone was typically of a plexiform nature although when scant, as in control lambs, consisted mainly of primary osteones. In severely deformed cases in which marked periosteal bone deposition had occurred a pronounced plexiform arrangement was evident. Usually however such cases demonstrated primary osteones in the most deeply sited periosteal bone. The relative proportion of secondary osteones to primary osteones increased with advancement of age in control lambs and with advancement of deformity in experimental animals. Primary longitudinal vascular canals could be observed in all of the cortical bone types but were most readily identified in plexiform and primary osteonal bone.

Areas of osteonal remodelling were easily visualised by the

presence of resorption cavities in cortical bone microradiographs. Such resorption cavities also aided in determining the principal regions of secondary osteone formation. Figs 38 A & B represent the principal sites in cortical bone sections in which osteonal remodelling was profuse. In control animals, osteonal remodelling was more widely present throughout the cortex with the advancement of age. In younger lambs resorption cavities were normally present at a particular site with larger areas of the cortex being composed of primary osteones. Primary osteones were similarly less frequently recognised in longer surviving individuals. Resorption cavities tended to be less numerous in distal sections in which an ever increasing proportion of cortical bone consisted of compacted coarse cancellae.

Periosteal and endosteal bone tended to be deposited asymmetrically in lambs of the experimental series. In control lambs although a much greater proportion of the cortical circumference possessed endosteal and periosteal bone deposition normally more profuse at a particular site. Thus a direction of bone drift could be established which corresponded to the site of greatest periosteal bone deposition. In the wake of periosteal drift endosteal bone was more profusely deposited on the opposite cortex. Figures 39A & B represent such patterns of drift and thus refer to the sites of greatest depth of periosteal and endosteal bone. Figures 39A & B do not however indicate the relative thickness of, for example, periosteal bone present in individual sections. In all cases drift was more marked and endosteal/periosteal bone more profuse in experimental lambs, being

greatest in those with gross deformity. The direction of drift was not identical in all sections from the same bone extremity. By way of example more proximally sited sections of the left distal tibia of control lamb 60 possessed profuse periosteal bone anteriorly and to a lesser extent medially and posteromedially. More distally sited sections possessed periosteal bone solely on the anterior cortical aspect. Similarly although the deformed distal tibial metaphysis of lamb No 18 possessed profuse periosteal bone medially in proximal sections, more distally sited sections were endowed with predominantly posteromedial periosteal bone deposition. Figures 39 A & B thus represent only the predominant direction of drift.

A close scrutiny was made of cortical bone microradiographs for the presence of screw osteones. Although osteones of such a morphology were infrequently encountered, no particular site predisposition in either control or experimental lambs was detected. Invariably however where a Volkmann's canal entered the resorption cavity that aspect furthest from its point of entry was more profusely endowed with lamellar bone deposition. Asymmetrical filling of resorption cavities by secondary osteonal lamellae was occasionally seen being thickest in the direction of drift. Even in severely deformed bones with pronounced drift patterns symmetrical secondary osteones were usually present adjacent to the deep layers of periosteal bone.

In cortical bone microradiographs interstitial lamellae were normally the most radiodense. Remnants of mineralised growth cartilage matrix contained within compacted metaphyseal trabeculae also appeared extremely dense in microradiographs. Adjacent secondary

osteones were invariably of unequal density and maturity whereas adjacent primary osteones normally possessed an equal degree of mineralisation. At their formative bone surface primary osteones were relatively less radiodense however.

The most peripherally sited osteocytic lacunae in secondary osteones were often larger. Even in actively forming secondary osteones with asymmetrical filling the most deeply sited osteocytic lacunae were on occasions enlarged. Interstitial lamellae possessed lacunae of greater dimensions than those observed in the concentric lamellae of osteones.

Scrutiny of microradiographs failed to reveal a 'white line' of hypermineralisation within osteones. An infrequent finding however was a band of reduced density between adjacent concentric lamellae (see plate 36 F). Such bands of reduced density or 'dark lines' were encountered in microradiographs of cortical bone samples from both experimental and control lambs but were more frequently seen in younger individuals.

Cement lines were usually difficult to identify clearly, but when observed possessed greater radiodensity than the adjacent concentric lamellae.

Plates 36 A - F demonstrate many of the features of cortical bone that have been described above.

HISTOPATHOLOGY

A) CONTROL LAMBS No marked disparity was noted in growth cartilage morphology of males and females or between distal tibiae and distal metatarsi. Each zone of the growth plate chondrocytes could be discerned and the relative proportion of each was typically as shown in plate 37A. Growth plate cartilage was not however uniform either in thickness or morphology and aberrations from its typical appearance (plate 37A) were noted across its width.

The resting cell zone could be readily discerned by its denser staining matrix both in H&E and Van Giesson Sections. This zone was of inconstant depth but always of greater thickness towards the medial and lateral peripheries of sections. In long surviving lambs the resting cell zone was much thinned and conversely of greatest dimensions in young growing animals. Whenever a sector of the growth cartilage was of greater depth or thickness than elsewhere an increased depth of the associated resting cell zone occurred. Mitotic figures could be identified in the resting cell zone but with moderate difficulty. At a number of sites across the growth plate width, protrusions, or bullae, of resting cell zone cartilage into the epiphysis were noted. Such bullae were of variable dimensions in some instances being associated with three or more chondrocyte columns and in others having no clear association with any cell column. Bullae of resting cell cartilage were noted with equal frequency medially and laterally and resulted in the bone plate/growth cartilage interface being not a smooth surface but one in which the two surfaces were keyed together.

In addition septae originating from the resting cell zone/bone plate interface traversed the growth cartilage to a variable depth. Such septae were eosinophilic, were always orientated approximately parallel to the direction of growth and separated adjacent cell columns. Although encountered in lambs of all ages such septae appeared with greatest frequency in young individuals. In many cases a distinct cleft, widest at its epiphyseal aspect, was present in septae. The clefts appeared to contain amorphous material including formalin stain, but close scrutiny invariably revealed the presence of red blood cells. In some instances the intravascular Berlin Blue particles could be identified in epiphyseally derived vessels which traversed the bone plate and fed smaller vessels within the cleft. Such clefts were concluded to represent continuations of the epiphyseal vasculature into the growth cartilage (see plate 37B). There was no marked difference in mitoses of cells adjacent to the cleft and those more distantly located. In most cases no marked asymmetry was noted between medial and lateral sectors of the growth plate with regard to such clefts and septae. In long surviving control lambs however the medial growth plate sector of distal tibiae possessed more septae than the lateral sector. In addition wherever an area of growth cartilage was of greater depth due to its protruding into the metaphysis clefts were invariably present. The bone plate was incomplete in areas where clefts existed. Larger foraminae through the bone plate existed at such sites.

The growth zone typically possessed orderly columns of palisading or proliferating cells where mitoses were frequent. The classical description of flattened 'palisading' chondrocyte nuclei was not

however always observed. In some control lambs a moderately pronounced asymmetry was noted between the medial and lateral sectors. That sector which was of greatest depth (thickness) invariably possessed not flattened but more rounded chondrocytic nuclei (plate 37C). In the most extreme cases a definite columnar arrangement of such rounded nuclei could not be appreciated and toluidine blue sections revealed greater matrix elaboration at such sites. In sections where both the medial and lateral growth plate sectors were of almost equal thickness, more rounded chondrocyte nuclei could still be identified interspersed with flattened palisading nuclei, the latter composing most of the growth cartilage. Towards the periphery the normal palisading appearance was similarly observed. Both medially and laterally chondrocytic nuclei had assumed an oblique orientation which was usually more obvious and extensive laterally in distal tibial sections. The filiform nuclei were orientated with their long axis approximating to that of the bone (see plate 37D). In cell columns which exhibited palisading, although the plane of division was typically transverse, (perpendicular to the direction of growth) variations were encountered. The plane of division was far from constant in areas of growth plate thickening with rounded chondrocytic nuclei where all planes could be identified.

In distal tibiae the growth plate tended to be of greater depth medially in anteriorly derived sections. Thus, typically more rounded chondrocyte nuclei were detected medially in such sections with palisading and pronounced columns being more appreciable laterally. Sections derived from the middle of the bone extremity were normally of fairly uniform growth plate depth and consequently palisading and

discrete columns were more regularly present across its width. In at least some of the control lambs posteriorly derived sections possessed a slightly greater growth cartilage thickness laterally with less obvious palisading at this site.

In some control lambs this generalisation did not apply and the medial growth plate sector was thicker both in anterior and posterior sections. The morphological changes noted above were more pronounced in such cases and thus poorly defined columns and rounded cell nuclei were readily distinguished medially. In these instances protrusions of growth cartilage into the metaphysis were common resulting in localised areas of growth plate thickening. Rounded cell nuclei were readily discernible and epiphyseally derived clefts, septae and a thickened resting cell zone were invariably present. The bone plate was also observably thickened medially. Although all zones of the growth cartilage were of greater depth associated with such metaphyseal protrusions, the most pronounced change was in the maturing and hypertrophic cell layers. At this site, a defective or delayed degeneration of chondrocytes was sometimes appreciable which resulted in primary spongiosa formation being relatively retarded. The intense staining of osteoid in Van Giesson sections proved useful in its identification and far excelled results gained by short duration eosin staining. Cell columns within the growth cartilage were not always parallel to the direction of growth. Curved columns were frequently encountered especially in regions of the interdigitations of metaphysis and epiphysis (Plate 37E). At the periphery of the growth cartilage cell columns were often curved outwards and thus the primary spongiosa also was orientated

somewhat obliquely. Across most of the width of the growth cartilage any curving of columns resulted in the maturing/ hypertrophic cell zones being orientated approximately in the direction of growth. The curved profile of individual cell columns was continued in the spongiosa where a primary spongiosa orientated in the direction of growth was deposited. Transverse bars of cartilage matrix were always removed prior to osteoid deposition. Metaphyseal bone trabeculae resulted from remodelling of such spongiosa.

Although chondrocytes could be identified within cancellous trabeculae their death soon ensued as they became located more proximally in the metaphysis.

Trabeculae

Some metaphyseal and epiphyseal trabeculae appeared to possess lacunae devoid of osteocytes. Such a finding was uncommon and may have represented artefact. More frequently vascular channels, comparable to primary longitudinal canals of compact bone, were observed especially in regions of dense trabecular pattern. In both epiphyseal and metaphyseal dense trabecular bone, longitudinal canals possessed Berlin blue stained blood vessels.

B) EXPERIMENTAL LAMBS In the early postoperative period those features noted in the growth cartilage of control lambs could be readily discerned and often with greater ease. Thus the noted regional difference in proliferating cell nuclear shape could be readily appreciated as could the propensity for resting cell zone bullae to be present medially. Bending of peripherally located

chondrocyte columns was more pronounced. With the onset of angular bone deformity comparable histological changes were noted in both distal tibiae and distal metatarsae. It is convenient to describe separately the sequence of changes that occurred in the medial and lateral growth plate sectors with advancement of the postoperative period.

i) The Lateral Growth Plate Sector At the lateral periphery of the growth cartilage attenuated chondrocytic nuclei with a longitudinal plane of division could normally be discerned in short surviving lambs. In a few instances the cytoplasm around such nuclei appeared denser staining in both H&E and Van Giesson section.

As soon as one week postoperatively marked aberrations from control lamb findings were present. In lamb no 58, slaughtered after this period of time, a 'core' of growth cartilage penetrated the metaphysis in posteriorly derived sections of the distal left tibia (plate 38A). Anteriorly derived sections however demonstrated only enlarged rounded proliferative cell nuclei medially and more regular, flattened nuclei laterally with no metaphyseal protrusion. Although the growth cartilage protrusion, noted posteriorly, possessed a thickened proliferative cell layer the increased cartilage depth was also accommodated within the cartilage transformation zone . Hypertrophic cell degeneration appeared to be delayed and the primary spongiosa that ultimately formed was defective (see plate 38A). On the epiphyseal aspect of the thickened growth plate epiphyseally derived blood vessels traversed the bone plate and protruded further than normal into the resting cell zone (plate 38A). Other lambs slaughtered at this time showed lesser change.

Lamb no 33 slaughtered after 2 weeks possessed a discrete bone island in the lateral tibial growth plate periphery in posteriorly derived sections. More anterior sections however possessed no endochondral bone island but longitudinal clefts, between adjacent cell columns, filled with fibrous tissue, and amorphous material amongst which red blood cells could be identified. Such clefts disrupted the primary spongiosa and terminated amongst metaphyseal trabeculae. In lamb no 33 and many other distal tibiae and metatarsi of lambs slaughtered prior to one month postoperatively, the primary spongiosa appeared more attenuated and less well developed laterally. In some regions less profuse osteoid deposition occurred laterally.

Mineralisation of osteoid matrix laterally in these short surviving lambs was not as extensive as noted medially. With passage of postoperative time the converse applied with regions of more orderly spongiosa, osteoid deposition and mineralisation.

Distal tibiae and metatarsae of lambs slaughtered three or more weeks postoperatively elucidated the pathology of the lateral growth plate sector. A transverse cleft or 'fracture line' was noted extending from the lateral periphery to a variable extent (plate 38 B & C). Amongst the amorphous material present in this cleft, fibrin and in particular red blood cells were identifiable. The cleft traversed the cartilage columns and normally occurred through the hypertrophic cell layer or its junction with the maturing cell layer. Occasionally the primary spongiosa and more rarely recently formed metaphyseal trabeculae were disrupted. Although the cleft was always more pronounced and extensive in posteriorly derived

sections, in longer surviving individuals a discrete bone island formed peripherally (plate 38 D). Anteriorly derived sections typically possessed either a fracture line traversing the growth cartilage (plate 38C) or curved cell columns interspersed to a variable degree, with fibrous filled clefts orientated in the direction of growth (plate 38E). In many instances, and particularly in longer surviving individuals, a relative displacement between epiphysis and metaphysis could be appreciated. Although occasional pyknotic nuclei could be identified adjacent to the fracture line (transverse cleft) mitotic figures and increased nuclear and cytoplasmic staining density were more frequently observed. This was particularly so on the epiphyseal aspect of the fracture line. Chondrocytic nuclei adjacent to the cleft were of variable shape some being elongated, some enlarged and rounded. Cells adjacent to the cleft formed 'nests' being clumped together and surrounded by apparently acellular matrix (plate 38B). A variable degree of disorganisation of cell columns accompanied such changes.

The bone island that formed could be seen to be derived from continued maturation and subsequent hypertrophy and degeneration of cells on the epiphyseal side of the original line of cleavage (plate 38D). Growth cartilage cells on the metaphyseal side of the fracture line had a different fate. Generally with the advancement of angular deformity chondrocytes adopted a filiform shape their longitudinal axis being parallel to the direction of growth. In some regions maturation and hypertrophy proceeded with ultimate formation of primary spongiosa. In other instances, cell nests which formed from clumping of rounded chondrocytes, became located proximally in

the metaphysis and hypertrophy and degeneration of such cells was defective (plate 38F). Normally the entire defect between bone island and metaphysis consisted of attenuated chondrocytes interspersed with regions of collagen in which true fibrocytes could be identified (plate 38G). The attenuated chondrocytes resembled fibrocytes but close scrutiny revealed the presence of attenuated lacunae surrounding their nuclei and metachromasia was still present in toluidine blue sections (plate 38G). With the elaboration of collaginous fibrous tissue such metachromasia was lost (plate 38 H). Primary spongiosa was patchy in distribution being frequently absent but more commonly encountered in anteriorly derived sections. Generally the metaphyseal defect consisted of a progressively greater amount of fibrous tissue with the advancement of postoperative survival time. In those long surviving lambs with advanced deformity the fibrous component of the metaphyseal defect blended with that of the periosteum. Some individuals possessed an area of chondrocytes which had become inverted and thus contributed to growth of the metaphyseal island (see plate 38 I). A profuse blood supply accompanied the establishment of a gross metaphyseal defect. In many sections in which much of the metaphyseal defect consisted of true fibrous tissue, no chondrocytes could be identified proximally and no primary spongiosa was formed. Ultimately, the growth cartilage between epiphysis and bone island became much thinned and demonstrated defective metachromasia. Fusion of the bone island to the adjacent epiphysis ensued with pyknotic chondrocyte nuclei prevailing.

ii) The Medial Growth Plate Sector As noted in control lambs wherever thickening of the medial growth plate sector occurred palisading of proliferative cells was absent or limited. Larger more rounded nuclei were noted with little columnar arrangement being apparent in both short and long surviving individuals. The proliferative cells underwent frequent mitoses with division occurring in all planes. In short surviving cases no marked disparity between the number of mitoses medially and laterally could be discerned although an indication was gained of greater frequency medially in cases of severe deformity with gross medial growth plate thickening. Toluidine blue sections revealed a greater deposition of matrix by medially located cells. Some mitoses could be identified in maturing zone cells especially those nearest the epiphysis and in particular, cases demonstrating gross deformity.

Anteriorly derived sections of distal tibiae typically displayed more severe changes than posterior sections. Such asymmetry was not however so obvious in distal metatarsae where the whole medial growth plate was often almost equally affected.

In almost all cases a profuse blood supply could be identified, associated with the medial primary spongiosa, irrespective of the postoperative survival period.

Generally features such as bullae of resting zone cells and epiphyseally derived clefts and septae were more numerous in medial growth plate sections. In association with medial growth plate thickening an increased resting cell zone depth was usually found especially in longer surviving individuals. In cases with marked deformity, the lateral resting cell zone was invariably much thinned.

Protrusions of growth cartilage into the metaphysis were a frequent occurrence medially and were identifiable in the early postoperative period. Such protrusions resulted in localised areas possessing a relatively thick portion of growth cartilage. Because of the change of nuclear size and shape it was difficult to discern which zone of the growth cartilage contributed most to growth plate thickening. It seems probable that all zones were affected. Limited primary spongiosa formation occurred at such sites and the degeneration of hypertrophic cells appeared somewhat delayed. Trabeculae that formed were frequently misshapen.

In the early postoperative period epiphyseally derived clefts containing fibrin and blood vessels penetrated the corresponding bone plate in association with eosinophilic septae.

In distal metatarsae as early as 2 to 3 weeks postoperatively transverse clefts or fracture lines could be identified traversing the medial growth cartilage for a variable distance (plate 39A). In some cases the fracture line communicated with the medial periphery of the growth cartilage but in others did not. The fracture line was comparable to that seen laterally but could not be identified in distal tibiae until at least 5 months postoperatively. In bone extremities in which only one growth plate sector possessed a fracture cleft, marked bending of cell columns in the opposite sector was present. In longer surviving individuals in which fracture lines were present both medially and laterally the two lines of cleavage did not communicate and thus an isthmus of intact cartilage always persisted in the midline. Medial fracture lines differed slightly from those noted laterally. Whereas lateral clefts normally traversed the maturing/hypertrophic

cell zone, those medially possessed a more variable line of cleavage. This finding is well demonstrated in the distal metatarsus of, for example, lamb no 38 in which anteriorly derived sections possessed a fracture line traversing the resting and palisading cell zones whereas in posterior sections it traversed between maturing and hypertrophic cells. The medial cleft contained amorphous material amongst which fibrin, red blood cells and occasionally discrete blood vessels could be identified entering the cleft from epiphyseally derived eosinophilic septae. On either side of the cleft cells were of similar appearance to those noted adjacent to lateral clefts. Thus rounded nests of cells were invariably present and large rounded chondrocytic nuclei could be identified. Some nuclei adjacent to the cleft adopted a filiform shape orientated parallel to the line of cleavage (plate 39B). Wherever an underlying columnar arrangement of chondrocytes could be identified gross bending and distortion of columns was apparent. Some nuclei stained poorly or not at all in both H & E and Van Giesson sections whereas others possessed mitotic figures. Areas of amorphous matrix with no nuclear staining were present medially even in cartilage not traversed by a fracture line. Occasional pyknotic nuclei were present immediately adjacent to the fracture line. Displacement of epiphysis relative to metaphysis could be identified in some cases with modarate or gross deformity.

A degree of bone plate irregularity normally accompanied gross disruption of the medial growth cartilage. Epiphyseally derived septae and clefts containing blood vessels were frequently encountered often in association with epiphyseal bone protrusion (plate 39C).

In addition metaphyseally derived clefts, identical in appearance, were frequently encountered penetrating the growth cartilage to a variable extent. Such clefts also contained blood vessels and as they communicated with epiphyseal vessels in some sections, probably represent anastomoses of the two circulations (plate 39D).

With advancement of the postoperative period disruption of normal growth cartilage architecture was more severe. Some cell nests which formed adjacent to the medial fracture line failed to degenerate and become located proximally in the metaphysis (plate 39E). Similarly, in other regions more orderly hypertrophic cells failed to degenerate normally and a somewhat haphazard primary spongiosa was formed. Matrix metachromasia was uneven especially in association with cell nest formation. Frequently in markedly deformed individuals disruption resulted in hypertrophic cells being orientated at varying angles. The primary spongiosa that resulted was similarly orientated and bore little relation to the direction of growth (plate 39D). In such instances rapid remodelling occurred. Wherever osteoid was deposited it underwent mineralisation fairly rapidly. Some primary spongiosa however appeared to collapse with fragments of it and adjacent metaphyseal trabeculae being present.

The distal tibia of lamb no 18 (8 months postoperative) demonstrated a double growth cartilage line in some sections with an endochondral bone island separating each component.

Lamb no 17 (8 months postoperative) displayed a unique metatarsal growth cartilage appearance in which the lateral sector was separate from its medial counterpart, the former being located more proximally.

Those individuals with gross deformities frequently possessed

elongated, filiform, chondrocyte nuclei in some regions of the medial growth cartilage. Although such nuclei bore a strong resemblance to fibrocytes in no instance was there a loss of metachromasia.

Grossly deformed bones also demonstrated mitoses in the peripheral zone of the growth cartilage adjacent to the perichondrial region. Although a similar zone was appreciable in control lamb sections, the enhanced appearance in experimental animals was easily identified as was the gradual transition of nuclear shape at this site (see plate 39F).

Those changes noted in control lamb trabeculae were similarly observed but more marked. Thus vascular canals penetrated areas of dense trabecular pattern and were especially obvious medially in the region of the bone plate. An increased proportion of such canals accompanied increase in trabecular density. In cases with marked medial trabecular increase, areas of apparently dead trabeculae could be discerned. In such areas empty lacunae were present and frequently appeared enlarged. In some instances pyknotic nuclei were present within lacunae. In Van Giesson and short duration Eosin stained sections such trabeculae were seen to be coated with viable cancellous bone which possessed in at least some instances a relatively high content of osteoid. These findings were present in lambs from 3 months postoperatively onwards and were always most marked in the medial epiphyseal sector. Occasionally empty lacunae were identified laterally.

Another feature of Van Giesson stained sections was that within areas of increased trabecular density a somewhat deranged matrix

pattern was encountered in some cases. Deranged matrix pattern was in the form of unorientated collagen distribution and was always associated with trabeculae identified as being non-viable. A distinct lamellar orientation of collagen within trabeculae was present except at these sites.

Cortical bone sections It is not proposed to describe in detail observations made on distal metaphyseal cortical sections. In all instances such sections supplemented information gained from scrutiny of undecalcified sections. Some limited observations which are however considered relevant will be reported.

Control lamb distal metaphyseal cortex . In metaphyseal cortical sections primary longitudinal canals were more commonly encountered. Lacunae immediately adjacent to such canals tended to be of lesser dimensions than those further sited.

Similarly lacunae contained within bone lamellae in Haversian systems were smaller adjacent to the central vascular canal. More peripherally sited lacunae in osteones were frequently larger and interstitial lamellae contained osteocytic lacunae of relatively gross dimensions. Evidence was not gained supporting the classical concept of osteocytic osteolysis. Some lacunae in interstitial lamellae and peripherally in osteones contained pyknotic nuclei or were empty but such findings were uncommon. In more distally sited metaphyseal sections remnants of the growth cartilage persisted in the form of cores of matrix stained material contained within compacted cancellae. Although such cores were numerous, in none were viable chondrocytes detected. The presence of such matrix cores diminished when more proximal metaphyseal sections were examined. This was

especially marked in areas where internal remodelling and bone deposition was occurring.

In regions where surface remodelling was known to be occurring osteoclasts were identifiable. Osteoclasts were not however readily observed in the resorption cavities formed in conjunction with secondary osteone formation.

Experimental lamb distal metaphyseal cortex Those features noted in control lamb metaphyseal cortices were similarly present. Primary longitudinal vascular canals were encountered in both plexiform, primary osteonal and secondary osteonal bone. The characteristic pattern of plexiform bone was easily discerned both endosteally and periosteally in cases undergoing marked osseous drift and frequently osteoclasts on resorptive surfaces were more readily identified than in control lamb sections.

SEQUENTIAL BONE LABELLING

Identification of fluorochrome labels in undecalcified sections greatly aided both microradiographical and histological observations. In particular however the dynamics of the developing changes were more easily visualised. The following abbreviations will be used in this section

DTE = Distal tibial epiphysis/growth plate/metaphysis sections
DME = Distal metatarsal " " " "
DTM = Distal tibial cortical bone section
DMM = Distal metatarsal " " "
AC = Alizarine Complexone = First label administered
OTC = Oxytetracycline = Second label administered
XO = Xylenol Orange = Third label administered
FC = Fluorescein Complexone = Fourth label administered

Control Lambs - OTE, DME. Normally a degree of pale green autofluorescence was exhibited by the growth cartilage which aided in its identification. In addition contrast filled blood vessels were easily visualised by their intense blue black colouration.

In young lambs all labels could usually be identified but AC and XO labels fluoresced less intensely being a deep red and dark orange colour respectively with the filter combination used. Both FC and especially OTC fluoresced an intense turquoise blue and bright yellow respectively.

In older lambs AC label was located high in the metaphysis and in many instances was difficult to identify in metaphyseal trabeculae. FC label was always administered two days prior to slaughter and in all cases was identified in the primary spongiosa and adjoining metaphysis. In young lambs a greater depth of unlabelled calcified cartilage primary spongiosa separated the growth cartilage matrix from the FC label. A less intense zone of FC label extended into the metaphysis and blended with a zone of predominantly XO fluorescence. Within the XO zone frequently thin bands of FC label could be identified coating the metaphyseal trabeculae. Moving proximally the XO zone similarly blended with one of predominantly OTC fluorescence which was always relatively more intense. Within the OTC zone similar strands of XO and occasionally FC label were present on trabeculae. More proximally still, in young lambs a distinct AC zone could be identified, but the coating of such trabeculae by the more intense OTC label often masked the AC label.

Thus broad zones of each label were encountered, but the most distal part of each zone (i.e. that nearest the growth plate) always

displayed more intense fluorescence. This band of intense fluorescence was of greater thickness in younger individuals. Proximally high in the metaphysis more patchy labelling was normally present and distinct endosteal bone deposition in the region of metaphyseal remodelling could be discerned. Endosteal bone deposition also displayed the sequential labels with FC coating the endosteal aspect and AC being more deeply located in the metaphyseal cortex. Endosteal bone deposition diminished with establishment of a diaphysis and in some instances a distinct asymmetry of metaphyseal cortical bone was apparent. In such cases the medial cortex was always thicker.

Many control lambs possessed a degree of undulation of the metaphyseal/growth plate junction over its medial sector. Such undulation was normally associated with an irregular depth of labelled bone. In many instances the contralateral limb possessed similar undulations over the medial growth plate sector. Usually undulations were more marked in one limb than the other.

A lateral primary spongiosa defect was noted in control lamb no 44 microradiographs and was found to be associated with a defect in FC labelling. XO label was present and abnormally thick. A comparable but less marked defect at the same site was noted in the contralateral limb. As the OTC seam was intact in this lamb it is possible to state that the defective mineralisation at this site must have developed within the 10 days prior to slaughter in both limbs.

Other lambs also demonstrated minor metaphyseal protrusions of growth cartilage both medially and laterally. Usually medial protrusions possessed a fairly even FC label in the adjacent spongiosa.

In some cases with gross medial sector undulations patchy and diffuse FC label was encountered at the lateral metaphyseal periphery.

Generally a close correlation existed between DTE and DME findings. Thus distal tibiae with for example, gross medial sector undulations typically had comparable changes in the distal metatarsus of that limb.

Epiphyseal trabeculae possessed varying amounts of label. More profuse labelling was encountered peripherally and especially at the bone plate and over the subchondral bone underlying articular cartilage. Where asymmetry in bone plate thickness was present a greater amount of label was present on the thicker side. In addition such bone was laid down as lamellae.

Experimental Lambs - DTE, DME. The minor abnormalities noted in control lambs were similarly observed in bones of experimental lambs which underwent no demonstrable deformity.

The increased growth plate depth laterally in lamb no 58 is of interest. In addition to blood vessels traversing the bone plate the metaphysis contained all four fluorochromes although they were patchy and diffuse and located at an abnormally great distance from the bone plate. More anteriorly located sections possessed more normal labelling which was however thicker and there was a greater depth of unlabelled spongiosa present (see plate) Plate 4

In the first two weeks postoperatively lateral metaphyseal labels in posteriorly sited DTE sections typically were of slightly increased depth. Conversely labels in the medial metaphysis were of lesser depth when compared with control animals. There was a greater

rate of longitudinal bone growth in the early postoperative period. A similar increased label depth laterally was noted in posteriorly sited DME sections at two, three and four weeks postoperatively. However in many anterior sections of lambs slaughtered at one and two weeks postoperatively, medial metaphyseal labels were more intense. In such instances lateral FC labels were observed to be thin and defective in the metaphyseal periphery.

Lamb no 33 DTE was the first to possess a discrete endochondral bone island located in the lateral growth plate periphery. The island of cancellous bone was of irregular mineralisation as demonstrated by the blurred fluorochrome labels it contained. As OTC, XO and FC labels could be identified in the bone island mineralisation must have been occurring as soon as 6 days postoperatively. In this individual medial growth plate/metaphysis undulation and irregular medial metaphyseal label thickness were present in DME sections. Labels confirmed that such undulation, which was much more gross than observed in control lambs, had commenced by day 6 postoperatively. Distal tibiae similarly possessed medial sector undulation which was present in label administered 11 days postoperatively but obviously may have commenced prior to that time.

At 3 - 4 weeks postoperatively the lateral periphery of the metaphysis typically possessed defective FC labelling but greater total width. The densely labelled region of each fluorochrome zone (that nearest to the growth plate) was typically thicker medially but the distance between labelled zones was less than noted laterally.

At this time medial sector undulation was gross and the adjacent FC label zone was of variable thickness being thinner where growth

cartilage protruded into the metaphysis.

From 2 months postoperatively defects in medial sector labels were common. Typically the densely labelled region of, in particular, the FC zone was incomplete when in association with gross undulation of the growth plate. In one case (no 50) which possessed metatarsal deformity at this time a distinct fracture line and island of cancellous bone was present medially in DME sections. The cancellous island contained blurred labels (XO & FC) and mineralisation must have been present at 44 days postoperatively.

This individual also demonstrated periosteal bone deposition on the medial aspects of both metaphysis and epiphysis in DME sections. As three distinct seams of periosteal new bone (OTC, XO and FC) were presented such deposition must have occurred at or prior to 34 days postoperatively.

At 3 months postoperatively defects were present in all labels over the undulated medial sector. Disruption of mineralisation at this site must have been present at least on day 24 postoperatively. By this time it was obvious that not all defects appeared with the same rapidity. Thus, for example, an individual possessing minimal deformity at a bone extremity possessed changes typical of other individuals in which defects appeared at a younger age.

By 3 months postoperatively the depth of unlabelled spongiosa between growth cartilage and FC label, was greater laterally than medially in many regions.

With advancement of deformity the lateral metaphyseal defect became more pronounced and adjacent trabeculae typically possessed a haphazard, disorderly uptake of bone labels. Blurred FC, XO and OTC

were generally found to encircle the metaphyseal defect and bone islands when present showed a similar pattern of mineralisation. Elsewhere, remodelling of existing trabeculae became more apparent with advancement of deformity. With establishment of severe deformities the thickened medial growth plate contained protrusions of epiphyseal bone and gross label disruption in the adjacent metaphysis. Total label depth medially was variable sometimes being grossly reduced and sometimes of relatively normal dimensions. Profuse blood vessels were present both medially and laterally with advancement of deformity.

Epiphyseal union was commencing in distal metatarsi from 3 - 4 months postoperatively when bone bridges were identifiable.

In some distal tibiae early closure of the lateral growth plate sector occurred which effectively resulted in the bone island being fused to the adjacent epiphysis. In such cases as well as diffuse labelling adjacent to the metaphyseal defect, FC label was commonly found patchily deposited around all junctions of the defect with trabecular bone. In some instances an FC coated isthmus connected the proximally located metaphyseal defect with the growth cartilage.

In association with angular deformity the lateral metaphyseal cortex was found to be composed almost entirely of endosteal bone. The medial cortex however was hypertrophied and consisted of a variable proportion of endochondral and periosteally derived bone.

In long surviving individuals with a double growth cartilage appearance medially, a band of irregularly deposited FC label was present at each site.

From 8 months postoperatively severely deformed distal tibiae

and metatarsi possessed a grossly thickened medial bone plate. In addition to the generalised label associated with bone plate trabeculae such cases often possessed a discrete layer of FC labelled bone immediately adjacent to the resting cell zone of the growth plate. Such F.C label traversed the bone plate for a variable distance from its medial periphery but was typically located in the region of epiphyseal 'peaking' or 'spurring'.

The distal metatarsus of lamb no 18 is of interest as slight valgus deformity was present. Some sections of this individual revealed moderate undulations of the lateral metatarsal growth plate/metaphysis.

The distal metatarsus of lamb no 15 possessed a slightly defective FC label at its lateral metaphyseal periphery with slightly more intense label fluorescence medially.

Table 14 summarises some of the noted features and provides an indication of their chronology. Of necessity however the figures given do not represent the actual commencement of each change but simply the time of label administration when the feature was unequivocally present.

As in control lambs epiphyseal trabecular label was especially pronounced on the articular subchondral bone aspect and over the bone plate. Bone plate label was profuse medially in those lambs which underwent deformity and by 3 months postoperatively was typically of an orderly 'lamellar' nature. Laterally a less organised and scant pattern of bone plate trabecular label was observed.

TABLE 14 CHRONOLOGY OF FLUOROCHROME OBSERVATIONS

FEATURE	PRESENT AT DAYS POSTOPERATIVE	
	DTE	DME
1) Defective label periphery lateral metaphysis	1 day	1 day
2) Defective label medial metaphysis	11 days	6 days
3) Increased depth labelled zones laterally	6 days	6 days
4) Increased intensity of fluorescence medially	6 days	6 days
5) Bone island mineralisation laterally	6 days	42 days
6) Bone island mineralisation medially	180 days	44 days
7) Undulation of medial growth plate/metaphysis	11 days	6 days
8) Epiphyseal periosteal bone deposition	42 days	34 days
9) Metaphyseal periosteal bone deposition	42 days	34 days

Assessment of Bone Growth

Use of a calibrated eyepiece greatly aided observations of metaphyseal bone growth. Variation occurred in endochondral bone growth over the medial and lateral growth plate sectors and although minimal in control lambs marked variation was observed in those limbs which underwent deformity. In a few control lamb distal tibiae endochondral bone growth was judged to be greater laterally in anteriorly derived sections but slightly greater medially in posteriorly derived sections. It is noteworthy that in such cases the sector of growth cartilage which was of relatively greater thickness was associated with a reduction in endochondral bone growth rate. Initially left hind distal tibiae of experimental lambs showed a similar although more marked asymmetry, with however greater bone growth laterally in all sections irrespective of their site. In such instances the medial growth plate was associated with a more marked reduction in bone growth rate when compared with comparably aged control lambs. Experimental lambs in the early postoperative period typically possessed a widened medial growth plate, but similar and often more pronounced 'widening' laterally due to the developing metaphyseal defect. In association with more marked distal tibial deformity, although medial growth rate was judged to be less than normal a slowing down of increased lateral bone growth occurred. Especially when a large metaphyseal defect was present, lateral bone growth was either approximately comparable to the contralateral limb and control animals, or appeared to be slightly less than normal.

In lambs undergoing distal metatarsal deformity similar changes in bone growth rate were observed. Thus, initially bone growth rate

was relatively greater laterally despite medial growth plate thickening. Lateral bone growth continued to be greater with advancement of deformity but, as with distal tibiae, the appearance of a pronounced metaphyseal defect heralded a reduction in this differential growth rate. The end result of this marked reduction in lateral metaphyseal growth is well demonstrated in plate 23E where a staggered growth plate line has developed. The epiphysis underwent comparable but less obvious changes in growth as noted in the adjacent metaphysis. Thus, lateral metaphyseal growth became relatively reduced, as did the adjacent epiphyseal sector. With advancement of deformity the medial distal tibial and metatarsal epiphyses gained increased dimensions by bone deposition on their medial aspect. With advancement of deformity, the medial epiphyseal sector both in distal tibiae and distal metatarsae obtained further expansion by bone deposition on the surface of the bone plate.

Control Lambs - DTM, DMM. The compilation of figs 38 & 39 were greatly aided by examination of sequential bone labelled sections. Both sites of active internal bone remodelling and periosteal/endosteal bone deposition were easily visualised. Even in control lambs variation between sections occurred depending on their location in the metaphysis and it must be stressed that figs 38 & 39 represent only the predominant pattern.

The amount of labelled periosteal and endosteal new bone was slight in control lambs and as mentioned above was principally primary osteonal bone. Such bone was deposited in resorption cavities in a more or less regular fashion with labels administered after the same time interval being equidistant with each other in the resulting osteone

This finding was in contrast to the secondary osteone where asymmetry was encountered. In fully formed secondary osteones, typically a large amount of unlabelled bone separated the lamellae containing OTC and XO/FC.

In more distally sited sections the osteonal pattern was confused by the presence of densely labelled compacted cancellous trabeculae. Around such trabeculae the labels administered could be identified. Especially when viewed under incident UV light osteocytic lacunae exhibited a degree of peripheral fluorescence. Such fluorescence was blue-green in colour and was more easily visualised in some sections than others. In addition some sections exhibited similar fluorescence lining the innermost edge of secondary osteones irrespective of their stage of completion. Such fluorescence was less intense but separate from that of fluorescein complexone, the last label administered.

Experimental Lambs - DTM, DMM. Those features noted in control lambs equally apply to the experimental series but were more pronounced in the latter.

Profuse endosteal and periosteal new bone was present especially in those with most marked angular deformities. In many instances the total thickness of periosteal new bone became greater in more distally sited metaphyseal sections. The asymmetry noted between sections taken from different metaphyseal sites was more marked. This feature was especially apparent when the thickness of periosteal and endosteal bone deposition was noted in each section. Such asymmetry occurred in the early postoperative period. For example, although endosteal bone deposition was principally anteromedial in proximal DMM sections of lamb no 45, it was principally medial in the adjacent more

distally located section. The profuse periosteal and endosteal bone in experimental lambs was almost exclusively plexiform in character. Plexiform bone was normally devoid of internal remodelling. In some lambs with severe angular bone deformities limited resorption cavities and secondary osteone formation was noted in plexiform bone. Sequential labelling permitted an estimation to be made of the advent of such remodelling. As XO/FC labelled secondary osteones were present in the most distal DMM sections of lamb no 29, internal remodelling of plexiform bone was not encountered before 128 days postoperatively. By contrast an increase in periosteal/endosteal bone deposition could be observed in DTM sections from 11 days and in DMM sections from 24 days postoperatively. The thickness of labelled periosteal bone was greatest in the direction of drift and with advancement of deformity each successive label was incorporated in a greater thickness of periosteal bone. Thus, the thickness of FC labelled bone was greater than that labelled by, for example, OTC. Such asymmetry was noted from at least 42 days postoperatively in DTM sections and at a slightly later date in distal metatarsae. Generally, the periosteal AC label was more diffuse, sometimes containing primary osteones and sometimes undergoing remodelling in the formation of secondary osteones.

Secondary osteone labels typically showed the asymmetry noted in control lambs with labelled lamellae not being equidistant. More profuse osteonal remodelling was noted medially in some distal metatarsae of lambs slaughtered at 7 days and in distal tibiae of lambs slaughtered at 14 days postoperatively. This finding was present in the more distally sited metaphyseal sections.

The dark lines noted infrequently in some osteones could be equated with lines of fluorochrome label. In such cases the only label which corresponded to their location in each osteone was OTC. Osteocytic lacunae and Haversian canals exhibited similar fluorescence to that noted in control lamb sections.

On occasions asymmetrical filling of resorption cavities could be identified. This feature was most apparent in long surviving experimental lambs and the site of maximal deposition corresponded to the direction of drift.

Some of the findings described above are illustrated in plates 40 A to F & 41 A & B.

DISCUSSION

Some preliminary statements can be made with regard to the morphology of growing ovine tibiae and metatarsi.

Clearly the classical description of growth cartilage consisting of regular chondrocyte columns and possessing flattened palisading nuclei does not always apply. In control lambs variation occurred across the width of the growth cartilage and morphological differences were encountered depending on the sections location in the bone extremity. Growth cartilage thickness differed between medial and lateral sectors especially in distal tibiae. Salentijn (1974) has considered that growth cartilage thickness was an accurate reflection of bone growth rate and the fluorochromes administered to lambs allowed this hypothesis to be tested.

It is interesting that differential bone growth occurred in some control lamb tibiae between medial and lateral metaphysis. Such differential growth would predictably affect morphology of the bone extremity as is discussed below. In control lambs growth cartilage thickening medially was associated with a reduction of bone growth a feature which was more easily observed in deformed limbs. Thus widening may be thought to correlate with reduced bone growth. Relatively increased growth however occurred in the lateral metaphysis again in association with growth cartilage thickening. From histological observations growth cartilage thickening at these two sites is known to differ and thus in order to make Salentijn's (1974) statement tenable, the cell morphology must be considered. Whenever enlarged rounded chondrocyte nuclei with no obvious columnar orientation were present endochondral bone growth was relatively

retarded. The elongated filiform type of nuclei noted laterally could be associated with relatively greater bone growth rate but this correlation was not absolute. With advancement of deformity and establishment of an enlarged lateral metaphyseal defect bone growth was either approximately equal medially and laterally or reduced laterally.

Epiphyseal growth in length followed a similar pattern to that noted in the adjacent metaphysis.

It is concluded therefore that the supposed correlation between growth plate thickness and bone growth in length is far from absolute. The cell morphologies noted in experimentally deformed bones were encountered in control lambs and thus may account for the differential growth rate noted in control tibiae.

The junctions between growth cartilage, bone plate and metaphysis were not even lines but frequently consisted of cartilage protrusions into each bone surface. Those protrusions into bone plate resulted in an interdigitation of cartilage and bone. Comparable protrusions or herniations through the bone plate followed application of a threaded screw across the growth plate between epiphysis and metaphysis (Campbell, Grisolia and Zanconato 1959).

The metaphyseal/growth plate junction is of particular interest as when gross irregularities in its outline were noted in control lambs, normally both pelvic limbs were affected.

The primary spongiosa was thickest in young individuals. The rate of incorporation of such spongiosa within metaphyseal trabeculae must therefore be relatively less rapid in young lambs. This observation could be explained by osteoid deposition and its subsequent mineralisation

being less rapid but it is known from part A of this study that growth rate is relatively greater in young lambs. Thus, this finding may imply that osteoid deposition and mineralisation occurs at a more or less constant rate throughout growth and that when rapid bone growth is occurring a relatively greater depth of spongiosa is produced. Spongiosa thickness was also influenced by the contour of the growth plate - metaphyseal junction as undulations were normally associated with an uneven spongiosa thickness.

Towards the periphery of control lamb sections a mild outward curving profile of cell columns and spongiosa was a not infrequent finding. Morscher (1958) similarly described the common orientation of cartilage cell columns and spongiosa. In experimental lambs however this relationship was disturbed and frequently disorientated trabeculae accompanied growth plate column derangement.

The ultimate fate of the spongiosa was its incorporation in metaphyseal trabeculae which were subsequently remodelled by bone resorption and deposition. Although occasionally chondrocytes trapped within cancellous trabeculae could be identified their viability appeared doubtful. Certainly on their becoming located higher in the metaphysis such chondrocytes were undoubtedly dead. Death of entrapped chondrocytes supports the experimental evidence of Bentley & Greer (1970) which failed to demonstrate their acting as osteoblast precursors in the rabbit. Holtrop (1966) had previously proposed that hypertrophic cells should be considered as one of the precursors of osteoblasts in endochondral ossification.

The greater radiodensity of the cores of metaphyseal trabeculae

is of interest. Different authors have failed to agree whether fluorochrome bone labels become incorporated within mineralising growth cartilage (Milch, Rall and Tobie 1958; Frost and Villanueva 1960; Hulth and Olerud 1962). The intense band of fluorescence noted in the lambs of this study could represent either calcified matrix spicules or mineralised osteoid acquiring mineral at the time of administration. In no instance was calcified matrix within the growth plate labelled, but 2 days had elapsed between fluorescein complexone administration and slaughter. The endochondral growth that had occurred during this interval was visualised as unlabelled spongiosa. As the calcified matrix cores of trabeculae were extremely radiodense it seems that the intense fluorescence noted may well represent mineralised cartilage matrix and not mineralised osteoid. The profuse remnants of cartilage matrix in this region was noted in histological sections. It is known that endochondral osteoid and growth cartilage matrix contain different collagen types and undergo mineralisation independantly (Von der Mark and Von der Mark 1977). The collagen of osteoid probably has a number of possible orientations depending on its site and is considered to have a woven arrangement in primary spongiosa (Rang 1969). Matrix calcification occurs around the hypertrophic cells closest to the invading metaphyseal capillaries but has been reported as occurring principally in the longitudinal septae. Van der Hoof (1964; cited by Rang 1969) has proposed that a different collagen type occurs in longitudinal as apposed to transverse septae. Recently accumulation and release of calcium by chondrocyte mitochondria has been implicated in matrix mineralisation (Brighton and Hunt 1978). If an independent process of mineralisation occurs in

cartilage matrix on a substrate differing from that of osteoid, a different mineral density would not be unusual. If mineralsied cartilage matrix does label with administered fluorochromes, then a band of increased fluorescence corresponding to the zone of increased mineral density (the cartilage matrix) would be expected. Although Hulth and Olerud (1962) reported a comparable zone of strong tetracycline fluorescence in growing rabbits they assigned it to intense osteoblastic activity in the region of the primary spongiosa. Certainly, the intense fluorescence noted medially in experimental lambs may indicate the intense osteoblastic activity involved in spongiosa remodelling.

The epiphysis is reported to gain no growth from the growth plate (Payton 1933; Siegling 1941; Siffert 1966). In control lambs the bone plate possessed evidence of remodelling. It is interesting however that when disparity existed between bone plate thickness medially and laterally, labelled bone was lamellar in nature and more profuse on the thicker side. As all labels were more profuse in all ages of control lambs with this feature, such asymmetrical bone plate hypertrophy must have occurred throughout the labelling period. This feature was much more apparent in experimental lambs which underwent limb deformity. The medial bone plate was always the site of increased bone deposition. In association with marked deformities the bone plate gained most bone from its growth cartilage surface. The origin of such bone remains uncertain but may represent growth cartilage contribution to epiphyseal growth. It is tempting to postulate that bone plate hypertrophy is the origin of epiphyseal scars. This suggestion correlates well with the findings in part A of this study.

The growth in width of the growth plate has been described as either latitudinal or interstitial (Langenskiold, Rytomaa and Videman 1967; Rang 1969; Brookes 1971). Certainly, these are the only two ways in which cartilage growth can occur (Pribylova and Hert 1971). Scrutiny of histological sections of the growth cartilage periphery in lambs revealed a gradual transition from fibrocyte to chondrocyte or vice versa. In addition mitoses in peripherally located chondrocytes could be identified. Although such findings could equally be explained by interstitial or latitudinal growth, the gradual change in cell shape was reminiscent of that noted laterally in deformed bone extremities. Perhaps the aetiology of such cell shapes is a consequence of their mechanical environment (see part D of this study).

A feature of both control and experimental lamb growth cartilage was the presence of eosinophilic septae running parallel to the direction of growth and traversing the growth plate for a variable distance from its epiphyseal aspect. Comparable structures have been described as eosinophilic streaks in clinically and radiographically normal pigs at sites of growth cartilage thickening (Fell, Jones and Boyne 1967; Thurley 1969). Similar 'eosinophilic streaks' have been observed in both articular and growth cartilage of pigs suffering from osteochondrosis (Grondalen 1974a) and in the growth plate of children with adolescent coxa vara (Mickelson, Ponseti, Cooper and Maynard 1977). The identification of berlin blue in at least some of the clefts in lambs has led to the conclusion that they may represent the course of vascular channels in the ovine growth plate. Comparable vascular channels traversed rabbit growth cartilage which underwent experimental epiphyseolysis (Spira and Farin 1967). Such channels may be of

particular significance in younger individuals and eosinophilic septae perhaps are their vestiges. Septae may however have the power of regeneration into more clearly defined vascular channels whenever growth cartilage thickening requires their presence. Undoubtedly, the pig growth cartilage possesses similar structures.

The mildest changes noted in experimental lambs were essentially an exaggerated form of control lamb growth cartilage morphology. More pronounced medial growth plate thickening was a constant early feature in experimental lambs and there was an associated thickened resting cell zone. The oblique orientation of lateral periphery chondrocytes whose nuclei adopted a filiform appearance was more marked.

A necessary prerequisite of further pathological change was the appearance of a fracture line traversing a variable number of cell columns from their lateral periphery. Although a medially located fracture line was identifiable at a later stage in distal tibial sections it was an early feature of distal metatarsal samples. In no instance did the medial and lateral fracture lines communicate, there always being an isthmus of undisrupted growth cartilage mid section. It would appear that an S-shaped bending of chondrocyte columns may be a prefracture state in growth cartilage. Certainly, in those bone sections where a fracture line was only present posterolaterally, bending of cell columns normally occurred elsewhere.

The fracture line was not constant in its direction but laterally normally traversed the hypertrophic cell/maturing cell zone occasionally involving the primary spongiosa. A similarly sited fracture line has been reported in upper femoral epiphyseolysis in children (Mickelson, Ponseti, Cooper and Maynard 1977). The medial fracture line was

even less constant and typically traversed a number of cartilage cell zones depending upon where the section was sited in the extremity. Morscher (1968) claims that resting proliferative cell zones are never involved in the fracture line but clearly this statement cannot be applied to the lambs of this study.

Brashear (1959 a and b) produced closed adduction fractures of distal femora in rats and found that on the distraction (convex) side the fracture line traversed the hypertrophic cell zone. The compressed (concave) side possessed a fracture line traversing metaphyseal trabeculae. These results cannot be equated with those of the lambs as Brashear produced a fracture line traversing the whole width of the growth plate and the insult to the bone was manually induced. The fracture line in sheep was incomplete and produced by in vivo conditions. Morscher (1968) considers that the weakest layers of the growth plate against traction forces are the hypertrophic and provisional calcification zones. Certainly the junction between mineralised and unmineralised matrix would appear to be susceptible to injury in many cases. Palfrey (1973) performed a detailed study of the bone cartilage junction of articular cartilage in rats. His findings suggested that the junction between cartilage and bone was stronger than the adjacent articular cartilage matrix. Although the morphology of the junction he describes undoubtedly contributed to this suggestion, a comparison may be possible with the lambs of this study. Although the rather tenuous primary spongiosa was sometimes fractured, more commonly the hypertrophic cell zone or the maturing hypertrophic cell junction was traversed. It thus appears that the mineralised matrix scaffolding is less prone to failure than the non mineralised matrix.

It is known that the soft tissues that surround the growth cartilage are important in maintaining its integrity (Harsha 1957; Morscher 1968; Chung, Batterman and Brighton 1976). Their stabilising effect however must be considered inadequate despite their remaining intact, as in some lambs relative displacement of epiphysis and metaphysis occurred.

The histological features that accompany upper femoral epiphyseolysis in children bear a remarkable resemblance to those induced in the experimental lambs of this study. Histologically the proximal femoral growth cartilage has been described as thickened, with column disorganisation and formation of cell nests or clusters of chondrocytes. Necrotic debris is contained within clefts in the growth cartilage and islands of cartilage cells have been described in the adjacent metaphysis (Ponseti and McClintock 1956; Mickelson, Ponseti, Cooper and Maynard 1977). In some histological sections of upper femoral epiphyseolysis, although no fracture line was present in the growth plate, chondrocyte columns were described as appearing to "flow in the direction of slippage" (Mickelson, Ponseti, Cooper and Maynard 1977). These changes are typical of those noted in the medial growth plate sector of experimental lambs. Further described changes in upper femoral epiphyseolysis, include disorganised primary spongiosa and prominent longitudinal eosinophilic septae. These changes have similarly been noted over the medial growth plate sector in lambs of this study. Similar histological features have been described in clinically normal pigs and those suffering from osteochondrosis (Fell, Jones and Boyne 1967; Grondalen 1974a). In such animals in addition to metaphyseal chondrocyte nests and growth cartilage

thickening, areas of growth cartilage necrosis, defective column formation and chondrocytic clumping have been described. Cell necrosis was evident in experimental lambs and in particular in advanced deformities large areas of amorphous matrix were noted medially. Rang (1969) reviews the literature pertaining to infantile coxa vara in children. In some ways this condition is comparable to slipped upper femoral epiphysis (adolescent coxa vara). The pathogenesis of infantile coxa vara is discussed by Rang (1969) and of most interest is the triangular fragment of metaphyseal bone that develops ventrally in relation to the displaced epiphysis. It is postulated that this fragment is derived from continued endochondral ossification following cleavage of the growth cartilage during the process of epiphyseal displacement. A comparable island of bone has been described in the experimental lambs following growth cartilage fracture and it undoubtedly represented continued endochondral bone deposition. Following the appearance of the lateral fracture line in sheep mitoses were commonly noted in the adjacent cartilage which still maintained contact with the epiphysis. Some chondrocytes undoubtedly died as a result of the fracture cleft as occasional pyknotic nuclei were identified. The mitoses on the epiphyseal side of the fracture line suggested continued proliferation and maturation of existing cell columns with eventual production of a trabecular bone island. The experimentally produced growth cartilage fractures of Brashear (1959 a and b) have been briefly described above. In rats this author noted continued maturation and endochondral bone formation by cells on the epiphyseal aspect of the fracture line. An island of trabecular bone thus formed and separated the two components of the

growth cartilage at the site of the original fracture. Brashear (1959 a and b) however considered endochondral bone island formation was contributed to by both the metaphyseal and epiphyseal growth cartilage aspects and thus postulated a reversal of direction of normal cartilage degeneration. Ultimately the more proximally located growth cartilage line was removed.

The experimental findings of Brashear bear a close similarity to those noted following medial growth cartilage fracture in experimental lambs but no observable contribution to endochondral island growth occurred from the proximally sited growth cartilage line. In those lambs possessing a double medial growth cartilage line primary spongiosa was always present on the metaphyseal aspect. By contrast, the bone island that developed laterally had a far from regular bone label pattern. Normally all labels were present in a diffuse and irregular manner, but in some long surviving individuals the proximal growth cartilage line or metaphyseal defect possessed labelled bone on both its metaphyseal and epiphyseal aspects. In some cases at least remnants of growth cartilage become reorientated. Nests of chondrocytes were occasionally encountered laterally but were most frequently present medially in longer surviving individuals.

The fusion of lateral bone island to adjacent metaphysis, and ultimately to epiphysis together with metaphyseal defects and cell nests gave rise to the somewhat confusing appearance of the lateral growth plate sector in radiographs. In vivo observations suggested that the lateral metaphyseal defect appeared, became located in the metaphysis and then a new defect formed. This appearance was however due to the relatively more advanced stage of defect formation

posteriorly within the extremity, resulting in a superimposition of images. The outstanding feature of the metaphyseal defect in lateral sections was the attenuation of chondrocytes until they resembled fibrocytes. With passage of postoperative time such chondrocytes were invariably replaced by a greater proportion of true fibrocytes. The metaphyseal fibrous defect reported in children has been described in part B of this study. Histologically this condition essentially consists of whorls and sheets of fibrous tissue (Hatcher 1945; Caffey 1966; Cunningham and Ackerman 1956; Campbell and Harkness 1957). Brower, Culver and Keats (1971) reported a cortical irregularity containing fibrous tissue in children which was continuous with the periosteum. This condition may represent a metaphyseal fibrous defect that has been largely removed as a result of metaphyseal remodelling and growth. The metaphyseal defect in children therefore bears most resemblance to that noted laterally in experimental lambs. Thus, the two forms of metaphyseal defect postulated in part B of this study have been recognised. On that side with a convex metaphyseal cortical aspect fibrous tissue and /or elongated chondrocytes predominate whereas in association with a concave cortical outline nests of chondrocytes or protrusions of growth cartilage into the metaphysis may occur. The metaphyseal protrusions noted medially were common and especially with passage of postoperative time were frequently associated with concomitant epiphyseal bone plate protrusion into the growth cartilage. Protrusion of the epiphysis into growth cartilage has been described as a common feature in older children, particularly in the weight bearing bones (Little 1973). In addition, protrusions have been

described in osteochondrosis in pigs (Grondalen 1974a) which as mentioned above share many of the features noted medially in experimental lambs. Langenskiold (1955) made some interesting observations on human epiphyses compensating for metaphyseal defects which resulted from osteomyelitis or tuberculosis. Protrusions of epiphysis into growth cartilage appeared at sites of metaphyseal defects. Langenskiold suggested that such as adaptive change in epiphyseal bone plate contour would maintain functional stability of the bone extremity. Grondalen (1974a) cites Zimmerman (1959-1960) who also suggested epiphyseal protrusions would reinforce the growth plate. In all the cases described by Langenskiold a thickened potential growth cartilage space existed between epiphysis and metaphysis. Thus in many ways the situation is analogous to that noted in the lambs of this study in which epiphyseal bone protrusions occurred.

Although on occasions certain fluorochrome labels, in particular alizarine complexone and xylenol orange, were difficult to discern in metaphyseal sections, this cannot be interpreted as being evidence of decreased or absent mineralisation when they were administered. Other labels regularly produced distinct bands of fluorescence. Thus no evidence suggesting a biorhythm of bone deposition as described by some authors (Swinson 1973; 1974; Simmons, Sherman and Lesker 1974) was found. Label defects were only noted in one control lamb (no 44).

Cortical bone sections revealed that bone drift could occur in a different direction in different ages of control lambs. Experimental lambs typically demonstrated similar but more pronounced patterns of drift. The fact that direction of drift could be different in proximal and distal sections of the same bone extremity implies that

the direction of drift changes with increase in bone length. Distal tibial drift was typically anterior initially and progressed to a medial or posteromedial direction even in control lambs. Metatarsal drift by contrast was typically posterior which was encountered in the majority of control lambs. A degree of change of drift to a posteromedial direction was seen in two longer surviving control lambs. Medial metatarsal drift however was reserved for experimentally deformed individuals with gross angulation of the bone extremity. Thus, whereas a change in drift was normally encountered in control distal tibiae it was uncommon in control distal metatarsi. Relevent to this observation is the fact that asymmetry in growth plate width depending on the site of the section in the bone extremity was commonly observed in distal tibiae. On occasions a discrepancy between growth plate activity across its width was present and it seems probable that in many control lambs the discrepancy was so slight that it was not recognised. Internal torsion was a normal component of tibial growth but not of metatarsal growth. One is led to the conclusion that asymmetrical growth plate activity, internal torsion and bone drift are intimately linked.

This suggestion is augmented by the observation that internal remodelling was also located in different sectors of the cortex depending on the site of section in the metaphysis.

As osteonal remodelling and thus the replacement of primary by secondary osteones was approximately the same as the direction of drift a spiralling of remodelling occurred in the distal metaphysis.

Whatever the stimulus for conversion to secondary osteonal bone was it undoubtedly had a similar influence on the site of periosteal bone deposition. The accelerated drift and changed remodelling pattern encountered in experimental animals was concentrated within that part of the metaphysis produced during the experimental period. Thus the changes noted were a direct result of the experimental procedure and its effect on bone growth and remodelling. If the sites of osteonal remodelling in different metaphyseal sections represent linked Haversian systems, a situation similar to that described by Cohen and Harris (1958) is present. Carrying this reasoning further, in distal tibiae osteonal spiralling in control and experimental animals may be an indication of how internal limb torsion increases with advancement of age or deformity. A similar explanation can be proposed for experimental metatarsi where internal torsion was frequently produced. Thus the spiralling of osteones noted by Cohen and Harris (1958) in canine femora may develop during growth with internal metaphyseal cortical remodelling resulting in femoral torsion. Femoral torsion (anteversion) may therefore be derived from the distal femoral metaphysis.

The basic concepts involved in this explanation are not new. For example Kummer (1959), Arkin (1949;1964), and Johnson (1964; 1966) briefly mention differential growth plate activity and uneven distribution of osteonal remodelling has been observed by Enlow (1963); Marotti (1963) and Harris, Haywood, Lavorgna and Hamblen (1968). In support of the suggestion that osteonal remodelling in adjacent sections is linked, Marotti (1963) noted a gradual change in location of osteones in serial sections of canine long bones.

Internal remodelling of cortical bone is known to be profuse in young animals and children (Sissons, Jowsey and Stewart 1960; Epker and Frost 1965; Landeros and Frost 1966; Lacroix 1970). A drift pattern in Haversian systems is reported with asymmetrical bone deposition within resorption cavities (Johnson 1964; 1966; Epker and Frost 1965; Landeros and Frost 1966; Lacroix 1970). Such scew osteones were encountered in the lambs of this study and when present the greatest bone deposition was directed in the direction of drift. Thus in these instances the factors predisposing to surface bone drift and osteonal remodelling may have a further influence on the process of internal reconstruction.

The periosteal and endosteal bone deposition that occurred in association with bone drift in experimental and many control lambs was plexiform in nature. In some control lambs however bone at these sites more closely resembled less regular primary osteones. Plexiform bone is well documented (Enlow 1962 a and b; 1963) and has been described as a variation of primary bone. In experimental lambs the thickness of plexiform bone labelled by successive fluorochromes increased with passage of postoperative time but was profusely deposited from at least 11 days postoperatively. Enlow (1962a) describes plexiform bone as developing in response to the need for large amounts of bone rapidly as occurs in rapid skeletal growth. By contrast slower skeletal growth is characterised by lamellar bone deposition with or without primary osteones. This concept is augmented by the findings in experimental lambs. Plexiform bone is reported to typically result from periosteal bone deposition although it has also been noted endosteally. Normally the plexiform

bone deposited in lambs did not possess osteonal remodelling. Although osteonal remodelling increased with age in experimental lambs, only in cases with marked deformities were secondary osteones present or developing within periosteal plexiform bone.

Although secondary osteone formation would appear to be a consequence of those factors controlling drift, Amprino (1948) favours the hypothesis that they develop and remodel in response to the need for mineral homeostasis. Whereas this is undoubtedly important, the rapid remodelling rate of young animals may produce the adult distribution of secondary osteones which are successively removed and replaced in the process of internal remodelling. It must be conceded that the mechanical and physiological significance of secondary osteones is uncertain (Ortner 1975) but some further comments will be made in part D of this study. One suggestion that has been proposed (Enlow 1962b) is that secondary osteones may develop as a regenerative process in areas of bone necrosis. Osteocytic death and empty lacunae were equivocally present in regions of cortical bone remodelling in experimental lambs but were judged to represent artefact. Enlow (1962b) does concede however that secondary osteones can develop independantly of necrosis during the process of bone remodelling during growth.

Cement lines delineating the periphery of each secondary osteone were difficult to discern. Sokoloff (1973) reviews the literature which proposes that cement lines do not exist in all mammalian species and that their existence has been questioned. They do however stain positively with a number of histological stains including haematoxylin, but not with Van Giesson. A number of authors have described cement

lines as being more radiopaque than adjacent lamellar bone (Cohen and Lacroix 1953; Lacroix 1971; Pritchard 1972) but even this observation has been questioned. Scrutiny of microradiographs in this study occasionally revealed what appeared to be hypermineralised cement lines. Peripherally located fluorescent bands in osteones were never of the dimensions of the cement lines and were thus assumed to represent concentric lamellar label.

A few lambs demonstrated lines of reduced density within osteones visualised in microradiographs. Although differing density between adjacent concentric lamellae has been noted (Cohen and Lacroix 1953; Lacroix 1956) the site of relative radiolucency corresponded with the oxytetracycline labelled lamellae.

Similar lines of radiolucency in tetracycline labelled osteones have been reported by Harris, Lavorgna, Hamblen and Haywood (1968) and deranged osteogenesis and mineralisation in tetracycline labelled bone by Eger, Gattow and Kammerer (1967) and Demers, Fraser, Goldbloom, Haworth, La Rochelle, MacLean and Murray (1968). Degradation products of tetracyclines are known to be systemically toxic and it is possible that the few lambs with radiolucent lines within osteones were given a different batch of oxytetracycline. Certainly the dose of oxytetracline administered was within the therapeutic range and only a few lambs were affected. No signs of systemic toxicity were noted following administration. Another possible explanation is that individual variation exists in the oxytetracycline dose required to inhibit mineralisation in vivo.

Osteocytic lacunae differed in their relative dimensions depending on their proximity to their blood supply. Thus lacunae of peripherally

located osteonal osteocytes and those of interstitial lamellae were larger. Similarly those osteocytic lacunae immediately adjacent to primary longitudinal vascular canals were smaller than those more distantly located. Despite such observations no obvious evidence of osteocytic osteolysis was detected. Although some primary longitudinal vascular canals resembled grossly enlarged osteocytic lacunae close scrutiny revealed that they contained blood vessels. The absence of observable osteocytic osteolysis does not deny its existence. Both species and age factors may be of relevance and lacunar enlargement as described could be interpreted as representing osteocytic resorption.

The method of microangiography employed in this study worked remarkably well. The experimental technique would however have been greatly improved by incorporation of a manometer in the infusion circuit such that known physiological pressures could be utilised. The method employed however did not result in either petechial or ecchymotic areas of extravasation and thus it appears unlikely that the positive pressure system used was excessive.

The strong correlation noted between growth plate thickness and epiphyseal and metaphyseal vascular filling is worthy of comment. It is arguable that this pattern may represent artefact due to medial vascularisation differing anatomically from that laterally with perhaps an easier passage of contrast media into that sector. It is noteworthy however that a few sections revealed more profuse vascularity in the lateral sector. In no instance of relative growth plate thickening was a reduced vascularity noted. On occasions no asymmetry between medial and lateral sectors were encountered but

generally growth plate thickening was accompanied by increased vascular filling.

All microangiography, Spalteholz, bone sections were obtained from the middle/anterior region of the bone extremity. At this site in distal tibiae the medial growth plate was predictably of greater depth which would account for the relatively greater medial vascularity. The more marked asymmetrical vascularity noted in experimental lamb tibiae and metatarsae also tends to support the suggestion that this is a real finding.

One interesting observation was that frequently when there was an increase in growth plate depth, epiphyseal derived vessels traversed the resting cell zone and entered the growth cartilage to a variable extent. The growth plate is normally considered avascular and has been reported as producing a barrier between epiphyseal and metaphyseal vasculature. Trueta and Morgan (1960) stated that in no species that they had studied did blood vessels traverse the fully developed growth plate. The introduction of this section reviews the literature which reports vascular channels traversing growth cartilage in young animals. The observations of in particular Spira, Farin and Karplus (1963 a and b) and Spira and Farin (1965) suggest a diminution in number of such channels with advancement of age. The conclusion has been drawn in the experimental lambs that eosinophilic septae represent the course of such vascular channels. Although eosinophilic septae diminished in number with increase in age, the vascular infiltration noted in areas of growth plate thickening occurred

in association with eosinophilic septae. Whether such septae remain in a grossly reduced form and are able to become re-established if necessity requires, can only be speculated. Irrespective of whether growth cartilage thickening occurred in the medial or lateral sector, epiphyseally derived vessels were encountered within growth cartilage. Normally such vessels were of wide calibre medially although gross lateral metaphyseal defects possessed profuse epiphyseal vessel infiltration. Yabsley and Harris (1965) produced metaphyseal vasculature disruption experimentally in dogs and noted epiphyseal blood vessels penetrating growth cartilage. They interpreted their findings as resulting from increased metabolic demands of the thickened growth plate. In a later publication Harris (1968) proposed a similar aetiology of epiphyseal vessel hyperaemia in rickets. A similar conclusion would be applicable to the findings in lambs reported in this study. If this suggestion is accepted then further evidence is obtained for the epiphyseal vessels being the only source of growth cartilage nutrition. This statement however does not take into account the possible nutritive role of perichondrial vessels. Although some authors have considered that perichondrial vessels supply only the perichondrial cells (Rang 1969) others have suggested a nutritive supply to chondrocytes located in the growth cartilage periphery (Trueta and Amato 1960; Brookes 1971). If the suggestion that increased metabolic demands of growth cartilage results in increased vascular supply is accepted then it would be predictable that increased growth in width of growth cartilage would result in more prominent perichondrial vessels. This would only be true if perichondrial vessels contributed to nutrition of the growth cartilage

periphery. The medial growth plate periphery in experimental lambs with angular deformities demonstrated pronounced perichondrial vasculature which supports both hypotheses. Such anutritive role of perichondrial vessels would be greatly aided by vascular channels rather than a reliance on diffusion. Brodin (1955) has suggested the existence of such channels and a further possibility is plausible in light of the findings in experimental lambs. Brookes (1971) suggests that the source of vascular channels traversing growth cartilage is from ramifications derived from perichondrial vessels. If this suggestion is true then maintenance of such vascular channels whatever their dimensions would afford a rapid pathway to cells concerned with growth in width of the growth cartilage. Such a hypothesis would also support the contention that the growth plate expands in width by interstitial cell growth. Examination of histological sections in experimental lambs revealed that perichondrial vessels were present at the site of transition of cell nuclear shape but in addition eosinophitic septae were often present in the medial growth cartilage periphery. The manner of growth cartilage growth in width remains equivocal but in lambs interstitial growth would appear to be more plausible. Certainly areas of mitoses within the growth plate periphery were identifiable in the region of unequivocal chondrocytes.

The concept of increased metabolic demands equally explains the findings of both the medial and lateral growth plate sectors of experimental lambs.

The medial sector has been observed to consist of a thickened resting cell zone and a growth and maturing zone of increased chondrocyte number and matrix elaboration. Laterally, whenever

a metaphyseal defect appeared, it consisted of attenuated chondrocytes which were still elaborating matrix except when they were replaced by fibrous tissue. The resulting grossly increased depth of growth cartilage would of necessity have increased nutritive requirements. With the development of gross metaphyseal defects and the elaboration of true fibrous tissue an additional source of vasculature derived from metaphyseal vessels appeared. Metaphyseal vasculature was never directly involved in medial growth plate nutrition except where epiphyseally derived vessels traversed the growth cartilage and appeared to anastomose with their metaphyseal counterpart.

Although metaphyseal vessels similarly tended to correlate with growth plate thickness, this was not always so. In the early postoperative period and in some cases with minimal angular deformity, the lateral metaphyseal vasculature was relatively less profuse or scant despite a degree of growth cartilage thickening.

As could be predicted, the metaphyseal blood vessels and their adjacent trabeculae possessed the same orientation. Some authors have suggested that metaphyseal blood vessels provide the main influence on trabecular architecture (Brookes and Lloyd 1971; Brookes 1963; 1971). Such a concept does not seem unreasonable. In the human foetus femoral metaphyseal vasculature has a radiate appearance with all vessels converging at approximately mid diaphyseal level (Brookes 1971). An identical distribution of metaphyseal trabeculae has been described in human neonate femora by Whalen, Winchester, Krook, Dische and Nunez (1971). Considerable evidence exists against the once popular belief that metaphyseal trabeculae derive their orientation from that of the growth cartilage columns. Such evidence is documented by Brookes (1971)

amongst others and has been augmented by the findings in lambs of this study. It is attractive to postulate that the vasculature is indeed of primary importance and certainly in the developing long bone, trabecular pattern would appear to be secondary to that of the local vasculature.

In young lambs, endosteally derived vessels derived from nutrient artery and vein, were the only source of nutrition of the growth cartilage/metaphyseal junction. This finding is in agreement with the reports of other authros (Lewis 1956; Brookes 1971). In older lambs metaphyseal arteries and veins were identifiable. The controversy that exists over whether metaphyseal vasculer loops abutting on the growth cartilage are open or closed could not be elucidated. Apparent extravasation was identified but it was impossible to detect whether this was artefact.

Observations in lambs of metaphyseal cortical vessel orientation were of interest. The findings are broadly in agreement with Brookes (1965; 1971) who reports on the orientation of vessels in endochondral, endosteal and periosteal bone. Vessels within endochondral bone, he states, are longitudinally orientated and thus run parallel with the bones long axis. Periosteal bone is reported (Brookes 1971) to possess a radiate pattern of vessels in which the centre of radiation corresponds to the site of the original diaphyseal ossification centre. (Lewis (1956) described a different orientation). By contrast endochondral bone is similarly equipped with a radiate pattern of vessels but in this instance the centre of radiation is at a point in space exterior to the diaphysis. Brookes (1971) provides evidence for the concept that cortical and trabecular vascular patterns are

morphogenetically determined. Brookes (1971) emphasises the importance of the 'bone-forming system' on vessel orientation. Support for this concept is found in the lambs of this study. Only if vessels form in a constant orientation relative to the distal periosteum or endosteum can the pattern in fig 37 be accounted for even in the presence or gross deformity. The orientation of endochondral bone vasculature would appear similarly to be grossly controlled by the orientation of the growth plate.

The laterally located island of cancellous bone that developed following fracture of the growth cartilage obtained its blood supply initially from adjacent periosteal vessels. Such vessels can be considered to be the nearest available supply and were abnormally prominent. Their prominence can be explained by virtue of increased metabolic demands resulting from growth plate pathology.

Johnson (1966) stated "As evil is inappropriate good, so disease is normality out of temporal, spatial or quantitative context". Such a concept undoubtedly applies to histopathological findings in the lambs of this study where various degrees of exaggerated normality were encountered.

Aetiology of Deformity

"Unlike muscle, bone is hard; yet it is the most plastic tissue in the body".

(Merchon 1935 cited by Storey 1972)

INTRODUCTION

1) Intrinsic Bone Shape

In vitro studies have demonstrated that skeletal rudiments deprived of their normal vascular and muscular attachments retain the capacity for self-differentiation (Fell and Robison 1929; Murray & Selby 1930; Murray 1936; Fell 1956; 1969; Amtmann 1971; Reynolds 1972; Ascenzi and Bell 1972). The pioneer study of Fell and Robison (1929; cited by Fell 1969) involved tissue culture of isolated femora taken from 5½ day chick embryos. Such explants grew rapidly in culture and acquired a surprisingly normal shape despite the absence of their normal environment. It is interesting that embryonic femoral explants taken from mice homozygous for an inherited femoral deformity developed similar malformation in tissue culture (Burda and Center 1969 cited by Amtmann 1971).

As a result of experiments in which chick femoral explants were grown on chorio-allantoic membranes, Murray and Selby (1930) concluded that whereas intrinsic factors produced the gross form of the appendicular skeleton, extrinsic factors were necessary to produce the final perfection of form required of the functioning skeleton. These authors also suggest that in later stages of development and particularly after birth and

in adult life, the extrinsic influences become of relatively greater and intrinsic forces of relatively less, importance. Fell (1969) tended to agree with Murray and Selby's (1930) conclusions but emphasised that development in isolation applies only to the early cartilaginous model and that mechanical and other extrinsic factors are extremely important in determining the form of true bone.

The early explant studies however did result in many interesting observations being made. Deformity of bone shape occurred in some of the chick femoral explants grown on chorio-allantoic membranes (Murray and Selby 1930; Murray 1936) and it was suggested that such abnormalities resulted from mechanical pressures acting upon the graft in its abnormal environment. Of special interest is the fact that abnormal curvature resulted in the development of a hypertrophied layer of bone on the concave side of the diaphyseal curvature. In the deformed femora a peculiar architecture was noted in the bone that formed at the point of greatest concavity. At this site a radiating arrangement of trabeculae was detected and attempts were made by a number of authors to explain this finding on the basis of the abnormal mechanical environment. Murray (1936) however offered an alternative explanation which suggested that the radiate appearance was an exaggeration of normal ossification in this region. Fell (1925) cited by Evans (1957) found that the trabeculae in the tibial shaft of late embryos or newly hatched chicks normally have an oblique orientation.

It is interesting that a similar radiate appearance has been described in a case of congenital tibial bowing and angulation in a human infant (Angle 1954).

Murray (1936) suggested that the bone hypertrophy on the concave aspect of the malformed femoral graft may be due to the production of an enlarged subperiosteal space. The observation was also made that chick femora are normally endowed with a femoral curvature and that the diaphyseal cortex of the concave aspect is thicker.

2) Experimental mechanical stimuli applied to explants

The fact that maldevelopment of chick femora occurred on chorio-allantoic membranes in some cases has been interpreted as indicating the importance of extrinsic forces even during the stage of "self-differentiation" (Murray and Selby 1930). Numerous reports exist of the effects on limb rudiments of experimentally produced mechanical stimuli. It is impossible to review such literature without paying homage to the early studies of workers and in particular Glucksmann (1938, 1939, 1942) and Weiss and Amprino (1940). The relevence of such studies to the understanding of bone deformities is well demonstrated by Glucksmann (1942) who stated "the effect of mechanical stresses on skeletal tissue developing under the extremely simplified conditions of culture in vitro are essentially the same as those produced by similar mechanical factors on skeletal tissue developing in vivo where the situation is complicated by the presence of muscles, blood vessels and nerves. This affords evidence that many of the

structural effects resulting from mechanical stresses in vivo are due to the direct action of the stresses on the skeletal tissue itself".

Glucksmann (1938;1939; 1942) performed a large number of experiments on the effects of tension and compression on osteogenesis in chick skeletal rudiments. In the experiments reported in 1938 Glucksmann implanted tibial endosteal explants between adjacent ribs in vitro. As the intercostal muscles degenerated the rib segments became drawn together and thus applied compression to the endosteal culture. In early preosseous explants Glucksmann noted a "compression structure" with trabeculae arranged parallel with the ribs. In more advanced and thus better ossified cultures a "resistence structure" with trabeculae orientated perpendicular to the ribs was detected. The compression structure was orientated along lines of tension and the resistance structure along lines of pressure. In the same paper Glucksman (1938) reported the results of tension applied to endosteal culture. The osteogenic fibres that developed were found to be arranged parallel to each other in the presence of tension. Glucksmann (1939 and 1942) reported further experiments in which mechanical forces were applied to embryonic chick metatarsae, phalanges, femora and tibiae. Such experiments revealed that limb bone rudiments placed in such a way that their growth resulted in pressure being applied to one another resulted in cartilage formation at the site of pressure in both the epiphyseal

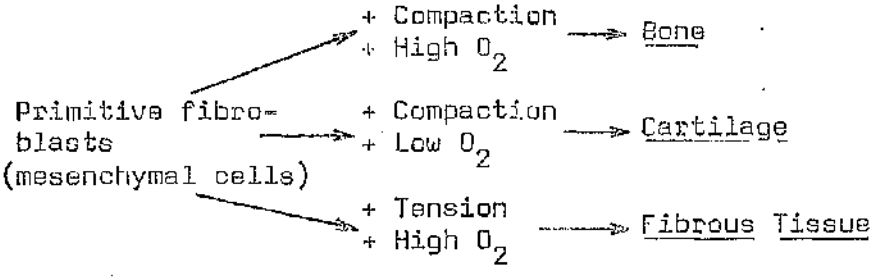
perichondrium and diaphyseal periosteum. Obstruction of elongation of the explants resulted in a bent skeletal rudiment which possessed little or no bone on the convex aspect but an abnormally large amount of bone formed on the concave surface. Glucksmann (1942) reasoned that the elasticity of the perichondrium drew it away from the shaft cartilage on the concave aspect thus producing a space which became filled with bone. Such an explanation is comparable to that offered by Murray (1936) for the development of excess bone on the concavity of chick femora malformed during growth on CAM. In older rudiments Glucksmann (1942) found that obstruction of growth only produced a lesser degree of bending. In such explants however the periosteum remained firmly attached and ossification was always greater on the convex aspect. The increased bone formation on the convex surface followed tension stresses. The stimulatory effect of tension on osteogenesis was verified by Glucksmann (1942) when he either totally or partially removed one epiphysis from a skeletal rudiment. Total removal resulted in a reduction in the amount of diaphyseal subperiosteal bone whereas partial epiphyseal removal produced impaired ossification only on the affected aspect.

In 1940 Weiss and Amprino noted that strong tensions tend to suppress the differentiation of chick prescleral mesenchyme into cartilage. Collagenous fibres formed orientated along the lines of tension. Lesser tensions, although not suppressing cartilage formation did result in a thinner cartilaginous plate differentiating.

Bassett (1962) cites Selye, Lemine and Bajusz (1960) who obtained bone formation in rat subcutaneous tissue in some shapes of culture tube but not in others. These authors apparently postulated that the dimensions of the tube determined the mechanical forces acting within it and thereby influenced tissue form.

Bassett (1962) by selective excision of parts of grafts allowed cultures to develop their own compressive force. Tension was produced by stretching the cultures during the growth phase. The central region of the cell mass was excised and the remaining ring of tissue was stretched over a cylindrical or square silicone rod. Periodically, during the growth phase the cultures were stretched over successively larger silicone rods. In compacted areas of the cell mass cells were found to be spheroid whereas in areas of tension fusiform cells developed. Under tension the cultures, in addition to possessing fusiform cells orientated with their long axis parallel to the main lines of tension, developed bundles of collagen and resembled tendon or fascia. Chondroid features were never detected in such cultures. Bassett (1962) suggests that it seems possible that such alterations in a cell's shape may alter its function due to altered organelle disposition. Scapinelli and Little (1970 cited by Little 1973) reached the same conclusion. Bassett(1962) further suggested that in view of previous literature the number of cells present may be important in influencing matrix production and properties of

the extracellular matrix by affecting the polymerisation pattern of collagen and/or mucopolysaccarides. Such suggestions are however dependent on the assumption that a pluripotential cell mechanism exists. Bassett (1962) remarked that although the wealth of evidence would suggest such a mechanism, it is possible that genetically distinct fibroblasts, chondroblasts and osteoblasts are present and that the multiplication of each is favoured preferentially by the various environmental conditions. The results of tissue culture experiments employing primitive fibroblasts (or mesenchymal cells) from bone have been summarised in the following diagram given by Bassett (1962).



Bassett (1964) cites the work of Weiss (1962) which suggests that such changes in cell function are probably reversible (modulation) and not permanent (differentiation). In vivo experiments are reviewed by Bassett (1964) which produced comparable results to those obtained in vitro. In the presence of membranes which inhibited the passage of nutrients to the osteogenetic zone chondrogenesis was favoured whereas bone formed in the absence of such interference.

Skeletal organogenesis under the influence of mechanical stimuli

Whereas many of the experiments summarised above have been concerned with the response of excised portions of a developing bone to mechanical stimuli, the response of intact and thus complete, immature, long bones to such factors has also been investigated. Murray and Selby (1930) inadvertently produced such experimental conditions by culturing chick femora on chorio-allantoic membrance but the mechanical environment could only be postulated. Fell (1956) reported similar deformities of skeletal rudiments due to the resistance of plasma clots on which they were cultured. Glucksmann (1939; 1942) was unable to quantify the forces involved in producing deformed skeletal rudiments. Recently, Sabelman, Hayes, Jones and Burton (1976) applied known forces to foetal mouse tibiae in vitro and reached the conclusion that the stress threshold for initiation of ossified tissue remodelling was less than that required to grossly deform the cartilaginous tissue.

Monson and Felts (1961) implanted humeri of neonatal mice subcutaneously in adults of the same inbred strain. To some of these humeri they attached semicircular wire appliances in such a manner as to apply compression to the ends of the bones. It was possible to determine the amount of force associated with a given deflection of the appliance and thus the force produced by elongation of the bone could be estimated. Although some differences were noted in development of normal mouse humeri and those implanted (without compression appliance) subcutaneously, there was no difference in for example the time

of epiphyseal closure. Differences of both size and shape were however encountered between those implants fitted with the compression appliance and those without. Compressed implants showed progressive flattening against the appliance and widened extremities. Ossification and fusion of the distal epiphysis occurred before gross deformation was possible but marked changes occurred proximally. Proximal epiphyseal fusion occurred earlier in the compressed implants. Centrally, there was a failure of development of the proximal secondary centre of ossification which these authors attributed to impaired nutrition caused by the appliance. Evidence of nutritional insufficiency of this region was seen histologically. Some implants possessed marked angular deformities of the proximal extremity which was explained on the basis of poor seating in the appliance.

Monson and Felts (1961) estimated the normal load borne by humeri at different ages by measuring body weight in a sample of growing mice and quartering the results. From calculated forces imposed on humeri by the compression appliance they concluded that the force applied to the bone exceeded the quarter weight early in the period of rapid deflection of the appliance. At such a time although progressive deformation of cartilage occurred there was no marked effect on longitudinal growth. Forces greatly in excess of normal were required to suppress growth and result in early epiphyseal fusion although no major deformation of the diaphysis occurred. Implants were

found to have a lesser mid diaphyseal width than comparable normal mouse humeri. The reduction in width was seen in both implants with and without a compression appliance.

Effect of mechanical stimuli on cartilage development of explanted bones

Monson and Felts (1961) noted that initially there was no obvious difference in either depth or contour of the growth cartilage in those implanted mouse humeri with and without a compression appliance fitted. However with progression of growth, the growth plate of the compressed humeri became more irregular in contour, slightly reduced in depth but of greater width.

The development of organ culture techniques permitted the study of the response of bones to externally applied forces without necessitating the use of attached appliances. McMaster and Weinert (1970) employed such a technique in investigating growth of the distal tibia of 11 day old chick embryos. These authors cite Fell (1925) who described all longitudinal growth of the avian tibia as occurring as a result of cartilage proliferation at the articular surface with no secondary ossification centre or true growth plate being present. McMaster and Weinert (1970) utilised centrifugation of distal tibiae placed in tubes of culture medium to exert force on the cartilage surface. Their results demonstrated the ability of compression and traction to modify the growth of embryonal cartilage. Initially specimens subjected to either compression or traction

showed a significantly greater growth than unstressed specimens. Two components of this response were identified which occurred concurrently. Firstly there was a stimulation of cartilage proliferation in the mitotically active surface layer and secondly there was a flow of the newly formed cartilage cells in the direction of the force applied. The cells in their matrix behaved like a fluid of high viscosity flowing centrally with traction and peripherally when compressed. It was suggested that progressively increasing the force eventually resulted in cessation of cartilage growth, as the increase in mitotic rate was exhausted in some longer duration experiments. A greater longitudinal growth was seen in response to traction which was explained by the physical response of the newly formed cartilage.

Comparable results were obtained by Wunder, Briney, Kral and Skaugstad (1960) who exposed young mice to continual centrifugation and then studied their femora. Initially, there was a decrease in body weight which could be assigned to reduced food consumption. At the end of one week body mass was approximately the same as that noted initially, prior to centrifugation. By this time however femora had continued to grow as fast, if not faster than control bones. The femora also developed a more circular cross section.

Effect of mechanical stimuli applied to growth plate cartilage

a) Compression In considering the influence of mechanical factors on cartilage structure the observation of Benninghoff (1925b cited by Murray 1936) is of interest. In young rabbits

and dogs Benninghoff (1925) found that the first rib cartilage cannot be bent as much as the seventh although the two were of about the same thickness. The difference of elasticity was explained on mechanical grounds as the respiratory movements of the 7th rib are greater than those of the first. Benninghoff thus suggested that the mechanical properties of the rib cartilages have been developmentally adapted to the forces acting.

Murray (1936) also cites early workers who established that fragments of cartilage were much more able to resist compression than tension.

That pressure applied to a bone's long axis can affect its rate of growth has long been recognised (Delpech 1829 cited by Lewin 1929; Hueter 1862; Volkmann 1862; Appleton 1929). Gelbke (1951) demonstrated the controversy of the morphological effects of increased pressure on the growth cartilage by citing Muller (1928) and Maas (1922; 1924; 1927 a and b). Muller (1928) is reported to have stated that growth cartilage is narrowed under pressure, whereas Maas (1922; 1924; 1927 a and b) considered that widening occurred. Some of this controversy has been elucidated by more recent studies.

Compression of growth plates in vivo was performed in dogs by Haas (1945) and was suggested as a useful corrective technique for human limb length discrepancy. Insertion of wire loops in such a manner as to compress the growth cartilage resulted in a reduction of longitudinal bone growth. Following

following application of a wire loop to only the medial distal radial growth plate Haas (1945) obtained obliquity of the distal articular surface. In a subsequent paper Haas (1948) produced growth cartilage compression by applying staples rather than wire loops. Staples also produced retardation of bone growth but following their removal Haas noted an increased rate of growth which was however not as rapid as normal. By contrast Duben (1956) observed that following the removal of a wire loop in dog distal femora, growth was of the same rate as in the contralateral limb and that there may in fact be an ability to partially compensate for the induced reduction in bone length. Blount and Zeier (1952) observed a growth spurt of a few months duration, following removal of staples in children. Haas (1948) reported that although growth was prevented in the aspect of the growth cartilage which was stapled, a lesser amount of growth retardation on the opposite aspect of the growth plate also occurred. In some of Haas's (1948) experiments there was evidence of premature epiphyseal fusion although observations were made near to the time of normal growth plate closure. Growth inhibition and eventual epiphyseodesis has been described in detail by Siffert (1956) in experimental rabbits. Siffert (1955) considered that the effect of stapling was a direct pressure inhibition of the proliferating stage of endochondral growth. Gradual thinning of the growth cartilage in the region of stapling is well documented. Use of staples to control endochondral growth

has been extensively reported both in the medical and veterinary literature (Blount and Clarke 1949; Brockway, Craig and Cockrell 1954; Heinze 1966; Poirier 1968; Herzog 1971; Carlson, Lohse and Hughbanks 1972; Pistevos and Duckworth 1977).

Shortly after the publication of Haas's results Gelbke (1951) described a series of experiments in which stainless steel wire loops were passed through the distal femora of dogs in such a manner as to apply compression to the distal growth cartilage. Gelbke noticed that the compressed growth plate was narrower approximately 3 months after insertion of the wire loop. Haas's observations of femoral shortening following distal growth plate compression were confirmed by Gelbke. Gelbke's (1951) experiments resulted in marked narrowing of the growth plate following compression which was confirmed histologically. In one case illustrated by him there was gross disruption of the growth cartilage with no apparent columnar chondrocyte arrangement and an irregular plate of growth cartilage was observed. Bone bridges were present. By estimating the force required to deform the wire loop Gelbke (1951) calculated that a force equivalent to 50% of body weight could be borne by the growth cartilage without radiographic changes being observed.

Other experiments in which compression has been applied across growth plates in vivo have involved implanting spring loaded pins into both the epiphysis and metaphysis of experimental animals. Strobino, French and Colonna (1952), by using two different force measuring devices discovered that the proximal

tibial growth plate of calves could withstand forces greater than body weight without growth impairment. In subsequent experiments growth arrest was however produced in calves using a modified compression devise with a force slightly in excess of body weight. Similar results were obtained by Strobino, Colonna, Brodey and Leinbach (1956). It is interesting however that after growth arrest for a period of 6 months the growth plate remained viable and was able to resume growth following removal of the appliance.

Following application of a spring loaded clamp between pins placed in the metaphyses of rabbit stifle joints, Trueta and Trias (1961) noted an increased thickness or height of the growth cartilage. The earliest changes occurred in both distal femoral and proximal tibial growth plates after only three days of compression. After one week of pressure most animals demonstrated widening of the whole growth plate with signs of disorganisation of the columnar arrangement of cartilage cells. In those rabbits in which clamps were applied for approximately three weeks the growth plate became thinner and bone bridges developed. Similar disorganisation of growth cartilage was described by Sijbrandij (1963) who applied a comparable devise to the proximal tibiae of rabbits. This author discovered that a compressing force of approximately 3 kg. ($1\frac{1}{2}$ - 2 x body weight) was required to arrest growth. Sijbrandij (1963) however reasons that compression forces are unlikely to influence bone growth under normal circumstances.

A compression force was applied across the proximal tibial growth plate of guinea pigs by Hinrichson and Storey (1968) with a slight initial reduction in growth rate being noted.

Hert (1964) produced growth inhibition in rabbit radii and ulnae following the application of staples. The radius and ulna was connected by means of the staple which resulted in overloading of the proximal radial and distal ulnar growth plates and reduced loading of the distal radial growth cartilage. The compressed proximal radial and distal ulnar cartilages displayed decreased growth activity whereas growth was enhanced at the unloaded distal radial growth plate. In another series of experiments Hert (1964) placed staples connecting the radial diaphysis and the distal ulnar epiphysis. Such a situation resulted in reduced pressure being applied to the proximal radial growth plate. When the proximal radius retained its contact with the humerus the activity of its growth cartilage was increased but loss of contact resulted in decreased activity. Thus axial pressure was necessary for increased growth plate activity whereas loss of function of the articular surface resulted in diminution of growth cartilage activity.

Although the studies of researchers such a Strobino et al (1952; 1956) suggest an all or none inhibition of bone growth by compression, Arkin and Katz (1956) were of the opinion that minor degrees of pressure could slow growth without stopping

it entirely.

A more physiological approach to overloading growth cartilage was taken by Fyfe (1961) who anaesthetised growing rabbits and subjected the proximal tibiae to 2 - 3 lbs pressure intermittently for periods of 1 second to 3 minutes through femoral and tibial diaphyseal pins. Increased growth occurred whereas control limbs with pins inserted but no pressure applied showed an increase but over a longer period. Microradiography of pressurised tibiae showed increased vascularity and irregularity of trabeculae. The hypertrophied cell region of growth cartilage was widened with the cells more vesicular and often forming nests rather than columns.

That overloading of growth cartilage can occur if animals are overfed is demonstrated in the findings of Andreae and Dammrich (1972) and Seibel, Dammrich and Andreae (1973) in bulls and by Paatsama, Jussila and Alitalo (1972) and Dammrich and Unshelm (1975) in pigs. Local growth plate disturbance was noted as were articular cartilage changes.

Although various devices have been applied in such a manner as to compress the growth cartilage, overloading of one of the bones of the antebrachium or tibio-fibula has been produced by selectively removing part of its paired bone. This was the approach adopted by Wermel in a whole series of experiments (Wermel 1934; 1935 a-f) which have been extensively reviewed by Murray (1936). In summary Wermel produced growth retardation in the remaining bone and abnormal bone curvature was produced which will be described in detail below.

More recently Tschantz, Taillard and Ditesheim (1977). produced mechanical overloading of the distal ulnar growth plate in dogs by resecting part of the radial diaphysis. A fracture line in the growth cartilage was produced with disorganisation of the adjacent metaphyseal aspect of the growth plate. Their findings were interpreted as representing an epiphyseal tilt.

b) Tension In addition to studying the influence of pressure on bone growth Gelbke (1951) also applied traction to epiphyses. In one experiment Gelbke attached a wire loop to the patella and femur in such a manner as to apply tension on the tibial tuberosity. No change was however noted either histologically or radiographically in the growth cartilage of the tibial tuberosity. When Gelbke (1951) attached a wire loop through an epiphysis (the olecranon) and attached it to the humerus in full elbow extension, changes were noted. The growth cartilage was narrowed and irregular and small 'cysts' were noted in the metaphysis. Gelbke (1951) remarked on the similarity of the effects of tension and compression on the growth cartilage.

Smith and Cunningham (1957) applied distraction forces to the proximal tibial growth plate of calves by inserting epiphyseal and metaphyseal pins joined by a turnbuckle. The distracting force was increased at intervals of 10 - 14 days. Considerable difficulties were encountered and results were only obtained in two animals. A thickened growth cartilage was observed and one animal possessed a slight increase in bone length.

Histologically traction was associated with large elongated chondrocytes and clefts were found between the columns of cartilage in the distracted growth plate.

In 1968 Hinrichsen and Storey spplied tension across the proximal tibial growth plate of guinea pigs. Deviation of growth was induced. Hert (1969) installed spring distractors across the proximal tibial growth plate of rabbits. Acceleration of tibial growth occurred when forces of between $\frac{1}{4}$ and $\frac{1}{2}$ of body weight were applied. In confirmation of Smith and Cunningham's (1957) findings, the growth cartilage was thickened and a larger number of cell columns was observed. Hert (1969) remarked that Smith and Cunningham's description of prolongation of cells suggests passive deformation caused by traction. Hert's (1969) experiments resulted in acceleration of tibial growth until 3 months postoperatively when growth became slower and finally stopped despite continued growth of control growth plates. The distal growth plate had reduced growth activity in a number of cases which may suggest disuse.

Recently Porter (1978) applied distraction springs to the proximal tibial growth plates of lambs and calves. The confusion that arises from synonymous usage of the word epiphysis for growth cartilage is well demonstrated by this report. Of ten lambs utilised seven demonstrated increased bone growth of 1 - 4mms with angular deformity being produced in nine. Longitudinal overgrowth was recorded in the two calves subjected to parallel distraction.

Induced postural change and growth cartilage

Dubrul (1950) defines posture as "the normal habitual bodily position to which the animal consistently returns in the flowing continuum of its response to an impinging environment". Dubrul further states that "the so-called characteristic postures may be more apt to reflect the balanced mechanical stresses operating upon the bodily architecture".

Experimentally, posture has been altered in a number of different animals and the changes noted in bones have been recorded. Colton (1929) appears to have been one of the first investigators to amputate the forelimbs in young rats and thus force them to adopt a bipedal posture. In such rats Colton (1929) found increased fibular bowing and a slight valgus deformity of the distal tibial extremity. More recently Goff (1957) described the preparation of bipedal rats and his technique which included tail amputation has been adopted by a number of workers with variable bone changes resulting. Thus Sakamato (1959) observed irregularities of both vertebral and hindlimb growth plates in bipedal rats. Inhibition of bone growth was observed and the severity of changes gradually increased with the duration of bipedalism. Ushikubo (1959) in identical experiments discovered that the femora and tibiae were shorter and thicker and both enhancement and reduction in the degree of normal vertebral curvatures were noted. Although Ushikubo (1959) failed to record vertebral body changes, abnormalities were observed in intervertebral discs. The mouse similarly has been subjected

to bipedalism by means of Goff's method. In such animals Sato (1959) reported scoliotic spinal curvatures developing and irregular growth plate development.

The work of Appleton (1934) in which postural deformity was produced in rabbits has been described above in the consideration of limb torsion. Such postural deformity was produced by tenotomy of the obturator tendon and by the application of splints which induced medial thigh rotation. The torsional deformities that were produced have been summarised above. As Appleton (1934) failed to produce appreciable skeletal changes in adult animals, he deduced that the primary effect of postural abnormality was on the growth plates. He also concluded that asymmetrical loading of the growth plate occurred which was associated with an asymmetrical reduction in metaphyseal growth.

Arkin and Katz (1956) performed similar experiments by applying plaster casts to one pelvic limb of young rabbits. The cast was moulded in such a manner as to apply a valgus force to the proximal tibia which was of gradually increasing severity when plaster changes were performed at weekly intervals. Valgus deformity of the proximal tibia resulted and these authors emphasised that all of the deformity occurred in the part of the bone laid down since the commencement of the experiments, the diaphysis being unaffected. Arkin and Katz (1956) concluded that the portion of the growth plate that was compressed by the induced valgus grew relatively more slowly whereas the opposite

side grew more rapidly. The bend in the fibula showed similar characteristics. No significant differences in growth cartilage thickness was noted.

It is interesting that Arkin and Katz (1956) attempted to discover whether or not postural abnormality in the absence of direct proximal tibial stress, could result in deformity. Plaster casts were again applied which although not including the proximal tibia, enforced a habitual postural valgus. A milder form of proximal tibial valgus resulted which these authors interpreted as being the result of the animals weight and the weight of the cast.

Murray (1936) cites Wermel (1935c) who noted a postural change in the distal extremity of rabbits following partial or complete removal of the ulna. The paw was rotated outwardly and functioning of the limb resulted in distal radial deformity. In the absence of functional use of the limb, or adoption of abnormal foot posture, radial deformity failed to develop.

The implication of postural abnormalities in clinical growth disorders

Knight (1954) stated that "the importance of certain sitting and sleeping habits of the infant and child deserve emphasis; these habits may interrupt the normal developmental pattern of the lower extremities and even cause deformities". The adoption of abnormal posture may cause abnormalities of limb growth, or exacerbate a pre-existing congenital deformity. A number of orthopaedic conditions have been considered to result from

abnormal pressures on the foetal skeleton in utero (Gaenslen 1915; Chapple and Davidson 1941; Thelander and Fitzugh 1942; McComas 1949; Broune 1965; Harris 1972; Ralis 1974). Post-natally such deformities can result in a characteristic posture being adopted which accentuates the defect (Sell 1941; McComas 1949; McNamara 1949; Harris 1972). Deformities may however be acquired rather than congenital and be predisposed to by abnormal postural habits. For example certain sleeping and sitting postures can predispose to tibial torsions (Knight 1954; Kite 1954; 1967) and abnormal bipedal posture may be important in some cases of idiopathic scoliosis (Steindler 1935; Anon 1955). One study reported that 79% of examined cases of idiopathic scoliosis had abnormalities of equilibrium which increased during the growth period with progression of the scoliotic curves (Yamada, Yamamoto, Ikata, Nakagawa; Kinoshita, Tezuka and Tamura 1971). The same group of workers produced lesions in the brain stem (in the postural reflex system centre) of bipedal rats and almost half of the animals developed marked scoliosis (Yamada, Yamamoto, Tamura and Tezuka 1974). In cases of juvenile brain injury which produced a motor deficit Clark (1974) observed scoliosis which however became less severe as the motor deficit improved.

Millikin (1914) remarked that any abnormality of normal limb posture will result in a state of muscle imbalance. Certainly, developmental or acquired defects in myogenesis have been suggested as a possible aetiological factor in the

development of limb deformities (Middleton 1934; Browne 1936; Silver and Simon 1959; Drennan and Sherrard 1971; Fuller 1975; Dykes 1978) and muscle imbalance has been implicated in scoliosis (Virchow 1914; Carey 1932; Schwartzmann and Miles 1945; Miles 1947; Riddle and Roaf 1955; Lindahl and Raeder 1962; James 1970; Roaf 1971; Hoogmartens and Basmajian 1973; Fidler and Jowett 1976).

The importance of muscle action on the skeleton

Although the intrinsic factors influencing skeletal differentiation are of undoubted importance, mechanical factors such as body weight, muscle action and tension exerted by adjacent tissues are also involved in determining final bone morphology. Although experimentally it has been shown that mechanical loading can influence endochondral bone growth, muscle action is thought to contribute to such forces in vivo. Contrary to the belief of early workers such as Koch (1917) muscles can exert great force and indeed Frost (1964) stated that most of the physical loads on bones are due to muscle action and that body weight usually produces loads of less than 0.3 the magnitude of muscle loads. That muscles can exert great force was known by early researchers (Evans 1957) and recently muscle action has been considered of more importance in the production of stress fractures than load bearing (Bertram 1971). A subsequent report by Baker, Frankel and Burstein (1972) concluded that muscle fatigue produced altered stress distributions on the skeleton which resulted in

microfractures appearing. These authors further suggested that fatigue microdamage may be a normal stimulus for the production of properly orientated new haversian bone exactly where it is most needed to increase the structural strength of a bone in response to exercise. In a study of muscle action and bone formation, Geiser and Trueta (1958) found evidence that during muscle action pressure forces are transmitted through the bone and that the presence or absence of such forces conditioned the balance between bone formation and removal. Support for this suggestion has been found by many authors and will be discussed in more detail below.

Of most interest to this study is the possible action of muscles on endochondral bone growth and the activity of the growth cartilage. Johnson (1964) stated that the conflicting action of muscle attachments to the epiphysis and metaphysis of the humeral head produce a torque on the growth plate and cartilage cell columns which results in a slight spiral growth. Salentijn (1974) tentatively suggested that muscle action may be one of a number of factors producing differing amounts of compression loading on growth plates and resulting in their differential growth. Kummer (1959) appears to propose a similar aetiology of abnormal tibial and femoral curvature in humans who adopt a habit of resting, sitting on the heels. Arkin (1949; 1964) on the basis of experimental knowledge of the effects of pressure on growth plate activity suggested that scoliosis may result from asymmetrical epiphyseal pressure.

Nachlas and Borden (1950) succeeded in producing scoliosis in experimental dogs following unilateral tethering of vertebral bodies with resultant asymmetrical epiphyseal compression. Muscle imbalance may produce such asymmetry in some naturally occurring scoliotic patients.

Support is found for this suggestion by the studies of both Olsen and Rosen (1974) and Bobechko (1974) who successfully produced and corrected scoliotic curves in experimental animals by unilateral electrical stimulation of the paraspinal musculature.

If muscles are to be implicated in the biomechanical control of bone form their action must influence bone remodelling. Some early researchers cited by Evans (1957) described the maintenance and growth of bone in totally paralyzed infantile limbs and concluded that intrinsic factors were of paramount importance in influencing bone structure. Such bones were however slower growing and contained less osseous tissue than normal bones and thus cannot be considered to contradict the suggested importance of biomechanical factors. Although it is commonly considered that muscle action stimulates bone deposition with the possible formation of tubercles at points of muscle attachment a serious objection to this view has been proposed. Both Enlow (1965) and Hoyte and Enlow (1966) pointed out that quite commonly muscles are attached to resorptive periosteal surfaces in bones undergoing osseous drift. Thus it seems unlikely that muscle tension always

results in subperiosteal bone deposition. Johnson (1966) remarked that areas of bone that have no muscle tendon or ligament attachments remodel later than those with them.

The reaction of cortical bone to mechanical stimulation

Ample evidence exists of bone form being influenced by function which Rashevsky (1955) considered to result in optimal design, a concept which he applied to many different examples of organic form. Examples of the response of cortical bone to function are given by Pottorf (1916); Howell (1917); Friel (1926); Bell (1956); Scott (1957); Evans (1957) Bunak and Klebanova (1960); Enlow (1963; 1968); Frost (1964); Avdiunitcheva (1964); Epker and Frost (1966); Nikitiuk (1968); Harris, Morrissy, Weinberg and Mack (1971); Chamay and Tschantz (1971); Ascenzi and Bell (1972); and Lanyon, Goodship and Baggott (1977) amongst others.

It has been stated (Ascenzi and Bell 1972) that muscle action is the most important biomechanical factor acting on bone and the very close parallel between muscle mass and bone mass was recorded by Johnson (1966). Frankel (1974) likewise stated that muscles produce the largest component of load on the human knee joint. In addition to muscle, tendon and ligament action, in the limbs of quadrupeds and lower limbs of man body weight is an additional source of mechanical force acting upon the skeleton.

The kinds of pure mechanical force that can act on the skeletal system, namely tension, compression and shear and the concepts of bone stress and strain are comprehensively described by Evans (1957; 1973) and Frost (1964) amongst others. If

bone deformation results from application of a force, bone strain of the bone specimen can be measured but stress can only be computed. Most studies of bone stress and strain have been performed on specimens of bone under laboratory conditions which despite limitations have provided information on the biomechanical properties of bone tissue (Walmsley and Smith 1957; Smith and Walmsley 1957; 1959; Evans and Lebow 1957; Evans 1957; 1973; Dempster and Coleman 1961; McElhaney 1966; Mather 1967; Ascenzi and Bonucci 1968; Swanson, Freeman and Day 1971; Bynum, Ledbetter, Boyd and Ray 1971; Griffiths, Swanson and Freeman 1971; Ascenzi and Bell 1972; Bargren and Bassett 1973; Panjabi, White and Southwick 1973; White Panjabi and Hardy 1974; Reilly and Burstein 1974; Burstein, Zika, Heiple and Klein 1975; Yu, Siu, Shim, Hawthorne and Dunbar 1975; Burstein, Reilly and Martens 1976; Carter and Hayes 1977; Shah, Hampson and Jayson 1978; amongst others). Such studies have revealed considerable variations in the biomechanical strength of cortical bone samples taken from different sites of the same diaphysis (Evans 1957; 1961; 1973) and from individuals of differing age (Curry and Butler 1975). Bone specimens taken in the direction of the diaphyseal long axis are of greater strength, as judged by a number of biomechanical parameters, than samples taken in a radial direction. The histological structure of bone can also be equated with its mechanical properties (Evans 1958; 1973; Currey 1959; Evans and Bang 1967) and its vascular pattern may influence its elasticity (Smith and Walmsley 1959).

In addition studies have been made on models of bones

(Pauwels 1948; 1960; Fessler 1957; Smith 1962a and b; Solares, Gaxiola and Olivares 1969) but have little direct application to the functioning skeleton and have been severely criticised (Evans 1957). Other methods of studying the biomechanical properties of cortical bone have been devised and are extensively reviewed by Evans (1957) and Asling (1961) amongst others.

Attempts to correlate in vivo forces with bone form necessitated measurements to be made in the intact, living animal.

Abnormal forces have been applied to bones in vivo by resecting the diaphysis of one member of paired bones (Rutishauser and Majno 1949; Tschantz and Rutishauser 1967; Chamay and Tschantz 1972), by prohibiting normal functioning of one limb (Lesshaft 1882; Pottorf 1916; Howell 1917; Vigliani 1955; Geiser and Trueta 1958; Bunak and Klebanova 1960) or by use of transverse pins, applied to the diaphysis which could be subsequently loaded (Liskova 1965; Hert, Liskova and Landrgot 1969; Hert, Liskova and Landa 1971; Liskova and Hert 1971; Hert, Pribylova and Liskova 1972; Chamay and Tschantz 1972). Some authors have implanted magnets as a means of applying continuous compression to bone surfaces (Cushing 1961; Ackerman, Cohen and Cohen 1966) but not in tubular bones.

Such studies have demonstrated cortical hypertrophy following loading, with overloading resulting in internal derangement of bone structure. Of most interest perhaps is the significant

increase in both the number of osteones and remodelling in loaded cortical bone (Hert, Pribylova and Liskova 1972). The stimulus for activation of remodelling was intermittent rather than continuous loading (Liskova and Hert 1971; Hert, Pribylova and Liskova 1972; Chamay and Tschantz 1972) and bone may react similarly to both intermittent tension and compression (Liskova and Hert 1971; Hert, Pribylova and Liskova 1972). Bone resorption occurred under continuously applied traction to cortical bone (Hert, Liskova and Landrgot 1969; Chamay and Tschantz 1972) Initial signs of cortical remodelling were noted in canine ulnae after only 48 hours of increased loading by Tschantz and Rutishauser (1967) who also reported plastic bone deformity with lamellar bending in the compressed cortex. Chamay (1970) noted similar plastic deformity of canine ulnae in vitro. The stress-strain characteristics of cortical bone specimens have been shown in vitro to also be influenced by the duration frequency and rate of loading (see Evans 1973). The rather unphysiological experiments of Seireg and Kempke (1969) which produced intermittent bone loading in rats followed by their laboratory analysis, failed to demonstrate cortical hypertrophy.

The in vitro experimental findings described above conform well with the postulated functional adaptation of bone that has been put forward by Frost and coworkers (Frost 1964; Epker and Frost 1965; Epker Henny and Frost 1968). These authors have suggested that bone deformation controls the manner of cortical

bone drift. The cortical aspects that become concave are considered to undergo bone formation whereas convex surfaces undergo resorption. In such a manner Frost (1964) proposes that abnormal bone aggulation can be corrected by biomechanical control of cortical or osseous drift. Frost (1964) also introduced other concepts such as'waisting' which he considered to be of importance in maintaining vertebral shape. Support for such concepts are found in subsequent publications by Frost and coworkers and in the experiments of Hammond and Storey (1970 cited by Storey 1972). The latter authors demonstrated that asymmetrical development of rat coccygeal vertebrae followed alteration of action of associated tendons. Stanek (1969) analysed the drift pattern in rabbit long bones and found that the mid diaphyseal cortex shifted during growth towards the concavity of the longitudinal curvature but that towards the extremities of the bone drift was towards its convexity.

Considerable advances were made following the development of wire resistance strain guages which could be applied to bone surfaces in vivo (Solares, Gaxiola and Olivares 1969; Lanyon and Smith 1969; 1970; Barnes and Pinder 1974; Lanyon, Hampson, Goodship and Shah 1974; Turner, Mills and Gabel 1975; Lanyon and Hartman 1976; Lanyon and Baggott 1976). In vivo strain guage studies have enabled the measurement of bone surface strain under dynamic as well as static conditions an advantage which will be elucidated below.

Lanyon and Baggott (1976) demonstrated by strain-guage recordings that the slight cranially convex curvature of the ovine radius resulted in compressive strain on the caudal cortex and tensile strain on the cranial cortex. The compressive strain was always larger than the tensile strain and was seen to be associated with a greater amount of osteonal remodelling in young animals. Cortical thickness did not however reflect the difference in cortical deformation. In a subsequent publication Lanyon, Goodship, and Baggott (1977) amplified this finding by stating that although cortical thickness did not reflect the imposed stress, even a slight consistent increase in strain imposed during locomotion resulted in cortical hypertrophy. Such cortical hypertrophy was not however proportional to the overload. These authors proposed therefore that the deformation customarily imposed on an individual bone's surface was critical and that any changes from its expected mechanical environment resulted in adaptive change. Lanyon, Goodship and Baggott (1977) further considered that it was not possible to infer the loading regime of a bone solely by examining its shape and the position of its adjacent musculature.

The reaction of cancellous bone to mechanical stimuli

For over a century the formation of trabecular patterns at the extremities of long bones have been studied. The controversial trajectorial theory of trabecular structure suggested that trabeculae were laid down along the lines of maximum internal stress and is well reviewed by Murray (1936)

and Evans (1957) amongst others. Many studies have been concerned with proximal femoral trabecular pattern (Dixon 1910; Koch 1917; Ochsner 1933; Wright 1934; Townsley1948; 1949; Fitzgerald 1962) and have suggested the importance of mechanical stimuli on internal bone architecture. For example Wright (1934) noted that the proximal femur of the sloth did not demonstrate the usual weight bearing trabecular pattern seen in other animals. Similarly Townsley (1948; 1949)and more recently Nikolic, Ruszkowski and Vucetic (1970) described the acquisition of normal trabecular architecture with weight bearing and functioning of human femora which was not present in pre-ambulatory specimens. The bone atrophy associated with disuse or immobilisation has been described by numerous authors including Cooke (1955); Siffert (1957); Singh, Nagrath and Maini (1970) and Kranendonk, Jurist and Lee (1972). Although it is accepted that mechanical stimuli influence trabecular orientation the exact nature of such stimuli has been interpreted differently by different authors. For example Koch (1917) considered muscle action to be of relatively little importance in influencing bone form, but Carey (1929) was of the opposite opinion and introduced the theory that muscle action produced back pressure vectors which controlled trabecular orientation. Other theories of trabecular bone formation were proposed and have been reviewed by Murray (1936); Evans (1957) and Scott (1957). Recently

Lanyon (1974) produced rather convincing support for the trajectional theory following the application of strain guages to the fibular tarsal bone of sheep. Two main trabecular tracts were described, one corresponding to the principal compressive strain, and the other to the principal tensile strain that was recorded. Just as with cortical bone, it appears that both compressive and tensile forces may regulate trabecular architecture, and indeed some authors have distinguished between compressive and tensile trabeculae in the proximal femur (for example Fitzgerald 1962).

The biomechanical properties of cancellous bone samples have been less extensively studied due to difficulties in their preparation. Some parameters have however been measured by for example Shoji, Walker and Behrens (1974); Townsend, Raux, Paul, Miegel, Rose and Radin (1974); Pugh, Radin and Rose (1974) and Klawitter and Weinstein (1975). Such studies have shown trabecular organisation to be the most significant factor in cancellous bone strength. Studies on patellar spongiosa by photoelastic methods have demonstrated that trabecular architecture is dependant upon the stresses and strains acting (Heasters 1974).

Statics and Dynamics

When a body is at rest it may be considered to be in static equilibrium as opposed to dynamic equilibrium which is a requirement of coordinated locomotion. It has long been known however that even the standing posture in man is an

extremely dynamic event with a continuous postural sway being adjusted in order to maintain stability (Cotton 1931; Thomas and Whitney 1959a & b; Murray, Wood, Seireg and Sepic 1975). In addition electromyographic studies have greatly elucidated the importance of muscles in maintaining postural stability (Joseph and Nightingale 1954; Portnoy and Morin 1956; Houtz and Walsh 1959; Houtz and Fischer 1961; Pauly 1965; Gray and Basmajian 1968). Investigations of the postural mechanism of joints have demonstrated the contribution of ligaments and periarticular soft tissues to maintenance of joint stability. (Smith 1956; 1967; Warren, Marshall and Girgis 1974) and their ability to prevent distraction (Basmajian 1961). Joint mechanoreceptors have been described and are considered to be important in maintaining postural stability (Boyd 1954; Andersson and Stener 1959; Freeman and Wyke 1964; 1965; 1966; 1967). There is evidence that when passive mechanisms, such as ligaments, counteract gravitational pressure, muscle contraction may be unnecessary (Denslow and Gutensohn 1967). Postural sway does however result in fluctuations above and below body weight being recorded at the stance (Murray, Wood, Seireg and Sepic 1975) which are of greater magnitude when voluntary movement occurs (Murray, Seireg and Scholz 1967).

Goldthwait (1914) remarked that "the human body is like a machine whose parts are all correlated and finely adjusted to operate without strain or waste of energy". Gardner and O'Connel (1961) concluded from electromyographic studies that as the skill to perform an activity increased, muscular activity

was reduced and thus no energy was wasted on antagonistic muscular action. Similarly economy of energy expenditure is important in normal human posture and gait (Steindler 1935; Kummer 1962) and has resulted in functional adaptation during quadrupedal evolution (Gregory 1912; Manter 1938; Gray 1943; 1961). In conditions producing pathological gait, although energy consumption is increased it is considered to be kept as low as possible by exaggerated motions at unaffected levels (Steindler 1935; Saunders, Inman and Eberhardt 1953; Ewald 1962; Corcoran 1971; Burke, Miller, Kearney, Milsum and Burstein 1972). The abnormal gaits adopted in certain human clinical conditions have been documented (Steindler 1935; Klain 1970; Bleck 1971; Burke, Miller, Kearney, Milsum and Burstein 1972; Gore, Murrey, Sepic and Gardner 1975).

In view of the known adaptive response of bone to mechanical stimuli, the dynamics of locomotion become extremely important. Problems are however encountered in the biomechanical analysis of dynamic forces acting upon the skeleton. Despite such difficulties the application of strain guage, force plate and other biomechanical techniques have been fruitful.

Methods and Materials Part D

Load Bearing All of the lambs utilised in parts B & C of this study were placed on a simple platform scale apparatus (Plate 42A&B)at weekly intervals throughout the experimental period. In such a manner information was obtained on the relative weight bearing by each limb in both normal lambs and those undergoing excision arthroplasty. Varnished wooden frames were constructed into which the four platform scales could be placed. In addition 'spacers' were assembled which could be inserted between the two sets of scales and which thus adapted the apparàtus for use with larger animals. It became necessary to use one of the 'spacers' with the lambs of the experimental series as they became older. The platform scales purchased for this apparatus were "Weylux" self-indicating Counter Platform Machines, model number 1500 with a 14" x 12" plate and 45 kilograms x 200g capacity. The scales proved to be perfectly adequate for the experimental procedure employed. Non-slip self-adhesive material (Safety Walk-Minnesota Mining Company) was applied to both the scale platforms and surrounding framework and spacers. Standard weights were used to ensure the platform scales were reading true prior to their use. Paul (1976) advised the use of piano wires under tension to minimise lateral deflections of the platforms when in use. It was recommended that the pianowires were placed close to but not touching the platform on all four sides. The depth of the platform on the scales, and the frames that had been constructed

however made it technically difficult to achieve this ideal and it was found that the wires tended to impede the platforms and thus produce false readings. As a compromise, two short strips of hardwood were secured at either end of each side of the frame surrounding the platform. Lateral deflections were substantially reduced by their application with no impedance of free movement of the platform vertically. Using the standard weights, a check was made of the balance readings at monthly intervals to prevent false recordings being made. The main difficulty encountered was that straw, faecal pellets and other foreign material gradually accumulated in the space between frame and platform and had to be cleared at weekly intervals to prevent impeding free movement of the platform scales.

The lambs were placed with one limb on each scale. The head was steadied but not held. Similarly the forelimbs were steadied by fingertip pressure over theshoulder region but not supported. All the lambs soon became accustomed to the procedure and stood with the minimum of restraint. The lambs were placed on the platform scale apparatus on several occasions prior to commencing the experimental period to familiarise them with the procedure. On beginning the experiment, platform scale recordings were made for each experimental lamb pre-operatively, 1 day postoperatively, 3 days postoperatively, 1 week postoperatively and thereafter at weekly intervals until slaughter. Recordings of control lambs were similarly made at weekly intervals.

It was found that constant variations occurred in the recorded weight bearing by each limb as the lamb maintained postural stability at the stance. In an attempt to standardise readings, it was found convenient to equalise the forelimbs so that each bore an equal proportion of weight. The hindlimb readings were then recorded. Further comments on this method of standardisation of readings are made in both the results and the discussion of this section, where its possible implications are remarked upon. As has been explained above, platform scale measurements were made on the same occasions as weekly pelvic limb radiographs were obtained.

Posture and Gait

Posture Immediately prior to slaughter each lamb was photographed in a caudal view so that any postural abnormality which followed unilateral excision arthroplasty could be recorded. In addition the normal postural stance of control lambs was obtained. Limited information was also available from observations made during the experimental period on the adoption of abnormal postural attitudes. Some clinical cases with postural abnormalities were examined during the course of this investigation which augmented information obtained from the experimental lambs.

Gait The time limit of this study permitted some limited observations on gait in one control lamb and in two lambs which underwent unilateral hip excision arthroplasty. The lambs used for this purpose were purchased at a much later date than those described in parts A,B & C of this study. All however

were from the same farm of origin, were of comparable parentage and were kept under identical housing and husbandry regimes.

In addition to obtaining general information on gait, it was proposed to make some appraisal of limb torsion during locomotion. Permanent reference points were inserted in the form of self-tapping orthopaedic screws in the left pelvic limb of five lambs. The chosen sites were firstly the wing of the ilium and secondly the fibular tarsal bone. In each case general anaesthesia was performed in an identical manner to that described above. Following routine preparation of the surgical sites, stab incisions permitted a 7/64" bone drill to be inserted and directed transversely into both the wing of the ilium and the fibular tarsal bone. Self-tapping screws (9/64") were then inserted. Although screws placed in the fibular tarsal bone tightened well, those in the ilium were invariably less soundly fixed. The screws were placed in such a manner as to protrude 0.5-1.0cms from the skin surface. Following insertion of the screws into the skeleton an unforeseen delay in obtaining equipment necessary for the gait analysis occurred. During the course of this delay many of the screws loosened and thus new screws were re-applied prior to commencement of the experiment. Routine antibiotic therapy had only been instituted during the initial postoperative period following the installment of the first set of screws. Constant antibiotic therapy was therefore provided after reoperation and a number of different antibiotics

were used, each for one weeks duration. Such a precaution appeared to substantially reduce the number of screws that became loose. Following insertion of screws into the fibular tarsal bones, a padded dressing was applied to the hock region. The screw inserted into the ilium was however left undressed and aided in detection of any developing infection. Despite such precautions, one of the five lambs so prepared shed its iliac screw and was discarded from this experimental series. A further lamb was also excluded from the experiment due to a mild but persistent hock lameness in the limb in which screws had been inserted.

The apparatus used in this study is shown in plate 43A An electrically driven conveyor belt, designed for use with greyhounds* proved ideal as a 'moving road' for experimental lambs. The experimental lambs were lifted onto the'moving road' twice daily prior to commencement of the experiment in order to familiarise them with the apparatus. In all cases and on all occasions the speed of the machine was kept constant and at the lowest setting available. Scaffolding was constructed around the apparatus in such a manner as to permit cinematograph filming to be performed from above. A second camera enabled concurrent filming in a lateral view. A bulb equipped flashgun was fixed in such a manner that its firing would register on both cameras and thus permit synchronisation of the walking cycle in both views. The scaffolding also permitted fixing of studio lights in the same location on each filming occasion and thus

* Ayrshire Elevator Company

standardisation of lighting conditions. Both the cameras employed in this study were Bolex H16 Reflex cameras. Ektachrome* film was routinely used at a speed of 64 frames per second.

Following familiarisation with the apparatus all three lambs were clipped free of wool and indian ink was applied in the form of large dots along the easily palpable spinous processes of vertebrae. In such a manner the spinal movements during locomotion were represented and available for further study. Dots were similarly applied to the skin overlying the greater trochanteric, mid-stifle and mid-fetlock regions of the left hindlimb to permit joint flexion/extension angles to be calculated. The time duration of this study has precluded much of this information being presented. The main purpose of this experiment has been to provide some subjective indication of both normal and pathological gait and a similar appraisal of limb torsion during locomotion.

Perspex extension bars were constructed (Plate 43B) and were applied by means of plastic 'sleeves' to the screw heads that protruded from the skeleton. The extension bars were easily identifiable on films taken of both the dorsal and left lateral aspects of the lambs. Films were made of all three lambs as described above. On the following day two lambs (nos 3&4) underwent unilateral right hip excision arthroplasty and the third lamb (no 2) was retained as a control. Subsequently one, two and three weeks later, further films were taken of all three lambs.

* Eastman Ektachrome Video News film 7240.Tungsten

Films obtained were projected by means of a variable speed projector onto a standard screen. In addition analysis was aided by observing each film in a manually wound 'editor'[1] and by means of an analyser cine projector[2].

At the conclusion of this experiment still photographs were taken of the three lambs when walking voluntarily on a concrete surface. In each lamb an attempt was made to photograph each phase of the stride taken by the nonoperated left hindlimb.

1 Murray - Teleray

2 Specto Mark III Motion Analysis Projector

RESULTS Part D

Platform Scale Measurements

All lambs adjusted well to the procedure of being placed on the platform scale apparatus. Minimal restraint was necessary even on the first occasion they were placed on the scales. Postural sway however was a severe problem in recording estimated load bearing by each limb. In all cases obvious discrepancies between limbs were easily discerned. Accurate quantitative measurements however were severely hampered by constant shifting of body weight between limbs which occurred during maintenance of postural stability both in control and experimental lambs.

The modified method of recording weight bearing described above, namely equalisation of forelimb load distribution, was routinely employed in obtaining the results presented below. By employing such a modification, postural fluctuations in recordings were considerably reduced and more standardised measurements were obtained. Predictably however, equalisation of forelimb loading resulted in erroneous estimates of weight bearing by each limb. In order to assess such errors, twenty consecutive recordings were made in six lambs over a period of approximately two minutes. Such an exercise also demonstrated the degree of fluctuation in platform scale measurements that could be expected to result from postural stabilisation. The consecutive measurements obtained in the six lambs, five of which had undergone unilateral excision arthroplasty, are presented

in figs 40 a to f. Included in the histograms are both the arithmetic mean of the twenty measurements and recordings made on the same occasion in which equalisation of forelimb loading had been employed. Table 15 has been compiled to emphasize differences between the calculated arithmetic mean of free standing measurements and those made with forelimb equalisation. It can be seen that in almost all cases forelimb equalisation has resulted in overestimation of left forelimb and left hindlimb measurements but underestimation of right forelimb and right hindlimb measurements.

TABLE 15

	LAMB NO	RESULT OF FORELIMB LOAD EQUALISATION WHEN COMPARED WITH FREE STANDING MEASUREMENTS			
		LF	RF	LH	RH
	16 (Control)	↑ O	↓ O	↓ O	↓ O
	55 (Exptal	↑ O	↓ O	↑ O	↑ O
Unilateral (RH) Excision Arthroplasty	14 (Exptal)	↑ O	↓ X	↑ O	↓ O
	15 (Exptal)	↑ O	↓ O	↑ O	↓ X
	12 (Exptal)	↑ O	↓ X	↓ O	↓ O
	13 (Exptal)	↑ O	↓ X	↑ X	↓ O
	Total	6↑	6↓	4↑	5↓

↑ = Overestimation
↓ = Underestimation
LF = Left forelimb
RH = Right hindlimb etc
O = Forelimb equalised measurement within range of freestanding variation
X = Forelimb equalised measurement not within range of freestanding variation

Figures 40 a to f demonstrate that the degree of overestimation or underestimation following forelimb balancing tended to be greater in those lambs with limb deformity. The largest errors occurred in right forelimb load bearing estimates. The range of free standing measurements are also given in figures 40 a to f . In addition whether or not the measurement made following forelimb equalisation is within this range is represented in Table 15 . Whereas in both hindlimbs and the left forelimb, estimates of load bearing following forelimb equalisation were in almost all cases within the range of free-standing variation, greatest error was encountered in right forelimb estimates.

The results of weekly platform scale measurements employing forelimb load bearing equalisation have been presented in three forms.

Firstly histograms have been prepared in order to represent each weekly measurement for both control lambs and those which underwent unilateral (right hind limb) excision arthroplasty. The majority of such histograms are included in Appendix II. By way of example, figures 41 a to f demonstrate the results of platform scale measurements in six lambs. As growth would confuse the presentation of measurements in absolute units of weight, the recorded weight bearing by each limb has been represented as a percentage of the total body weight on that occasion. In figs 41a to f such percentages are given above each histogram column. Although comparable histograms are presented

for all other lambs in Appendix II individual percentage figures above columns have not been provided.

Secondly, as a large number of measurements were obtained in different lambs of the same age, both from birth and commencement of the experiment, arithmetic means could be calculated. Thus, fig 42represents the arithmetic means of load bearing by each limb in the lambs at various times postoperatively. A total of 256 measurements were made in control lambs throughout the experimental period. The arithmetic mean of control lamb measurements for each limb could thus be calculated (fig 42). In Fig 42 the circled number indicates the total number of measurements from which the arithmetic mean was calculated, and the figures above each column refer to the percentage weight bearing by each limb.

Finally, Table 16 has been prepared. The arithmetic mean of ten consecutive hindlimb measurements for each lamb at various stages of the postoperative period was calculated. This estimate was performed in all experimental lambs slaughtered after three or more months postoperatively (see table 16)

The histograms demonstrate a number of findings. There can be seen to be weekly variation in the platform scale measurements even in control lambs. For example fig 41 a demonstrates that in lamb no 16 the percentage weight bearing by the left hindlimb was 19% on some occasions and as high as 27% on another. Following forelimb equalisation, with postural sway minimised the hindlimb measurements were found

TABLE 16

AVERAGES OF WEIGHT BEARING MEASUREMENTS AT VARIOUS STAGES OF POSTOPERATIVE PERIOD. EXPRESSED AS % OF TOTAL WEIGHT BEARING BY ALL LIMBS

Lamb Number	Limb	Average Weight Bearing 1-10 wks	10-20 wks	20-30 wks	30-40 wks	Postoperative Survival Time
51	LH	31.65%	-	-	-	3 months
	RH	6.20%	-	-	-	
52	LH	37.55%	-	-	-	3 months
	RH	4.60%	-	-	-	
38	LH	35.65%	-	-	-	4 months
	RH	2.30%	-	-	-	
54	LH	31.75%	-	-	-	4 months
	RH	8.05%	-	-	-	
29	LH	35.85%	37.45%	-	-	5 months
	RH	2.85%	7.65%	-	-	
36	LH	34.15%	28.15%	-	-	5 months
	RH	3.70%	8.75%			
37	LH	38.45%	32.65%	-	-	6 months
	RH	1.25%	6.95%	-	-	
39	LH	29.95%	27.55%	-	-	6 months
	RH	8.70%	12.75%	-	-	
55	LH	31.30%	27.15%	-	-	6 months
	RH	8.80%	7.90%	-	-	
17	LH	35.15%	33.45%	29.55%	-	8 months
	RH	2.20%	5.65%	8.60%	-	
18	LH	31.80%	29.40%	25.10%	-	8 months
	RH	2.20%	4.30%	5.05%	-	
14	LH	34.65%	36.15%	31.05%	23.80%	10 months
	RH	2.70%	4.25%	6.60%	10.25%	
15	LH	36.80%	34.15%	33.10%	25.80%	10 months
	RH	2.50%	6.05%	8.55%	8.80%	
12	LH	35.05%	29.50%	33.10%	26.05%	12 months
	RH	4.65%	4.30%	13.25%	15.20%	
13	LH	31.30%	26.20%	26.90%	22.95%	12 months
	RH	4.90%	13.20%	13.30%	11.55%	

in some cases to be equal. In those cases where this was not true, the application of fingertip stabilisation, without undue pressure, to the hip regions equalised hindlimb weight bearing in all cases. Thus it can be seen that in all control lambs the limbs of each side of the animal contributed equally to supporting body weight.

In experimental lambs the immediate effect of excision arthroplasty can be seen to be a dramatic reduction in load bearing by the operated limb, and a comparable increase in loading of the contralateral limb. In most cases almost all of the operated limbs contribution to load bearing was accommodated for by the contralateral limb and thus frequently the left hindlimb had a 100% increase in loading. Curiously, in many instances the maximum loading of the left hindlimb did not occur immediately but was noted after 3 days or more postoperatively. Variation in usage of the operated limb postoperatively occurred. Some lambs failed to attempt load bearing at one day postoperatively although in many cases limited usage occurred at least initially. This fact is borne out by fig 42. With passage of postoperative time a gradual return of function of the operated limb was noted and this was normally accompanied by a comparable reduction in excess loading of the contralateral hindlimb. In many cases a gradual increase in forelimb loading occurred with passage of postoperative time which was often of greater magnitude than had been encountered in any of the control lambs. Increased forelimb loading was however negligible in those long surviving

lambs in which the operated limb was contributing almost normally to load bearing.

No correlation exists between the severity of deformity and the degree of increased loading sustained by the left hindlimb. This fact is well demonstrated in lamb no 15 who although exhibiting no gross deformity, underwent similar load bearing changes to those noted in severely deformed cases (see fig 41d and table 16). Similarly no absolute correlation can be detected between increased loading and the site of principal deformity. Those lambs which underwent predominantly metatarsal deformity had similar load bearing changes to those in which tibial deformity was mainly manifested. In some lambs with metatarsal deformity however there may have been a more persistent period of excess loading by the left hindlimb.

Postural changes associated with developing deformities

The changes in posture noted in photographs taken of the lambs prior to slaughter are summarised in fig 43. Normal control lambs typically demonstrated the hindlimb posture shown in fig 43 Thus control lambs had a 'cow-hocked' stance with slight outward rotation of the pelvic limb and a medially placed hock joint relative to the stifle joint. With onset of skeletal maturity however the 'cow-hocked' stance was much less apparent and medial placement of the hock joint was minimal.

Immediately postoperatively there was a change in hindlimb posture (fig 43 plate 44A). The unoperated left hindlimb was adducted to bring the foot to a point midway beneath the pelvis.

This action necessitated a degree of internal rotation of the limb so that the os calcis pointed posteriorly rather than posteromedially. Tracings made from photographs of lambs at this time postoperatively demonstrated that a perpendicular drawn from midway between the acetabuli to the ground passed through, or slightly medial to, the new foot position of the left hindlimb. At this time the operated, right hindlimb was similarly adducted but held in an advanced position relative to the non-operated limb (fig 43 plate 44A). The limited weight bearing of the right hind limb was demonstrated by only the toes of each digit touching the ground. This hindlimb posture was observed in all lambs slaughtered prior to 2 months postoperatively. In one lamb, no 45, slaughtered after one month, postural limb torsion appeared particularly marked although minimal skeletal change was noted post mortem (plate 44B).

In the lambs slaughtered at two months postoperatively the right hind limb was held less far advanced (fig 43). Lamb no 50 demonstrated metatarsal varus deformity of the left hind limb at this time (plate 44C).

The lambs slaughtered at 3 and 4 months postoperatively all showed limb deformities. A more obvious internal rotation of the left hind limb was noted. The right hind limb was progressively less far advanced, relative to the non-operated limb, and less adducted than noted previously. In those lambs slaughtered at 4 months postoperatively a perpendicular drawn to the ground through the left hip joint passed approximately through the left

foot, both in cases with tibial and in cases with metatarsal deformity (fig 43 and plate 44D). The right hind foot was either in a position vertically below the right hip joint or slightly laterally placed. Slight inward rotation of the right hindlimb was noted in the experimental lambs at about this time.

At 5 months postoperatively, increased angular limb deformities were noted (plate 44E). Inward rotation of both hindlimbs, but especially the nonoperated limb was noted. From this time until 10 months postoperatively there was progressive abduction of both hindlimbs (plates 44E & F). With advanced deformity the left hindfoot was placed in a position well lateral to a perpendicular drawn to the ground through the hip joint. The right hind limb was similarly more laterally placed (fig 43 and plate 44F). One lamb, no 39, (6 months postoperative survival) showed increased fetlock sinking of the right hind limb demonstrating the increased contribution to load bearing made by the operated limb at this time.

The experimental lambs slaughtered at 12 months postoperatively showed adduction of the right hindlimb to a position more closely approximating to normality (fig 43 and plate 44G) This finding was despite extreme lateral displacement of the left hind foot in lamb no 12. In lamb no 13 however the right hind limb was similarly adducted, but in this case the left hind foot was less severely laterally displaced (plate 44G).

No definitive record was made of postural changes during

the postoperative period prior to the development of gross deformity. In all cases however the immediate postoperative stance recorded in fig 43 and plate 44A was noted. After a variable period of time internal limb rotation became manifested and subsequently characteristic postures became adopted depending on the site of ensuing deformity. In those lambs which developed metatarsal deformity postures initially as in lamb no 45 (plate 44B) and later as in lamb no 50 (plate 44C) were adopted. In those with tibial deformities postures similar to that of lamb no 54 (plate 44D) were seen. Such postures were observable at a variable time in the experimental period up to approximately 2-3 months postoperatively. Lamb no 15 was unique in that although initially adopting the characteristic posture shown by all lambs postoperatively, subsequently failed to adopt any abnormal posture of the left hindlimb. This was despite limited usage of the operated limb. The right hindlimb subsequently underwent the postural changes noted in all lambs namely from adduction to abduction to adoption of a more normal postural attitude.

Analysis of gait

Perhaps surprisingly, lambs soon adjusted to walking on the moving road apparatus. Locomotion was relatively normal on approximately the third day of familiarisation with the apparatus and from this time two lambs voluntarily jumped on to the moving road themselves. Following excision arthroplasty lambs showed a varying degree of difficulty in achieving the speed of locomotion

required by the apparatus. By one week postoperatively however lambs 3 and 4 were able to maintain their balance and provide results. The speed of the machine was however probably faster than the sheep would voluntarily have progressed.

A degree of difficulty was encountered with adequate fixation of the perspex extension bars to the protruding screw heads. Inevitably perhaps progressive loosening of the junction occurred with protracted locomotion on the moving road. Despite this difficulty filming could normally be carried out for approximately 2 - 3 minutes prior to loosening occurring. Loosening of the extension bars was more of a handicap in analysis of pathological gait but again filming could be performed for a sufficient duration for results to be obtained.

The 'editor' proved much more useful in visualising gait than the adjustable speed projector.

The control lamb (no 2) demonstrated a number of features. Initially, on the first occasion of filming a mild although detectable lameness was present in the nonoperated limb in which screws had been inserted. On subsequent films the gait was considered normal.

Normal gait involved a regular sequence of protraction and retraction of each hindlimb. A torsional component to hindlimb gait was observed. At the extreme of protraction and immediately prior to the foot strike, a degree of internal limb torsion occurred. By contrast at the extreme of retraction of the left hindlimb outward or external torsion occurred. The degree of external torsion appeared greater than internal torsion and occurred over a greater period of time. External torsion progressed slowly as the limb

was retracted and increased rapidly at the point of full limb retraction.

In both lambs 3 and 4 a similar sequence of events occurred prior to excision arthroplasty. Following unilateral right hip excision arthroplasty a similar gait pattern was noted in both lambs. Unilateral surgery resulted in a more rapid protraction of the nonoperated limb to enable it to contribute as much as possible to hindlimb weight bearing. In addition increased hock flexion and fetlock extension was noted in the mid-phase and retraction phase of the left hindlimb stride especially in lamb no.4.

The torsional components noted in the control lamb no 2 were also observed in lambs 3 and 4. On full limb protraction a rapid, although accentuated, phase of internal torsion occurred. With progressive retraction of the limb external torsion was detected which was of greater magnitude than that noted pre-operatively. The noted observations were more pronounced in lamb no 4 than lamb no 3. Hock hyperflexion and especially fetlock hyperextension were particularly marked in this lamb and appeared to become more pronounced with passage of postoperative time.

In both lambs the operated limb contributed to weight bearing from approximately four days postoperatively.

Some of the information gained from still photographs is reproduced in plates 45A-D. Although each phase of the stride is not represented some of the observations noted above are demonstrated. The numerals on each plate are not intended to denote comparable phases for each lamb but simply to place each frame in some form of order.

In addition the typical stance adopted by each lamb at the conclusion of the experiment is presented.

DISCUSSION PART D

Johnson (1964) remarked "Disease processes never involve any strange or new reactions but only altered relationships in normal reactions"

Intrinsic factors are of undoubted importance in establishing basic bone form, but the influence of mechanical factors, or ambient forces, on bone growth and development are of most relevance to this study.

Although forces acting upon the distal metaphyses and growth plate are likely to be complex during the dynamics of locomotion it is possible to make some tentative suggestions. Hert (1964) stated that growth plate activity is principally regulated by mechanical factors. He observed that increased pressure inhibited growth, decreased pressure accelerated growth but that complete unloading or tension resulted in growth cessation. Experimental compression of growth cartilage produced comparable histopathology to that noted medially in lambs that underwent deformity (Fyfe 1961; Trueta and Trias 1961; Bright, Burstein and Elmore 1974; Tschantz, Taillard and Ditesheim 1977). Experimentally applied tension produced a thickened growth cartilage containing elongated chondrocytes (Smith and Cunningham 1957) which have been interpreted as representing passive deformation (Hert 1969). Trueta and Amato (1960) produced areas of infarcted growth plate which resulted in the cartilage being placed under tension. Such chondrocytes can be seen to be elongated. From what has been said above it can be assumed that the medial growth plate sector in lambs underwent

relative compression whereas the lateral sector sustained tension. This conclusion is augmented by asymmetry of cortical thickness and scrutiny of bone shape.

A clue towards the local mechanical environment of some control lambs is gained from their possessing areas of enlarged rounded chondrocytes with indistinct column formation. As this feature was typical of the medial growth plate sector of deformed individuals relative compression at such sites can be postulated. Hert (1964) however described tension producing larger chondrocytes whereas small cells occurred with compression. The converse was identified in lambs.

The mechanical environment is considered to produce the columnar arrangement of chondrocytes (Rang 1969; Lercenthal 1971; Little 1973) and it has been noted that prenatal growth cartilage lacks the organisation seen postnatally (Collins 1966). Similarly, the orientated collagen present between columns may result from ambient forces and the intercellular matrix could conceivably be altered as a result of compression. The physical and mechanical characteristics of connective tissues are considered to be largely dependent upon differences in the amount or type of glycosaminoglycans and upon the type and aggregation of collagen fibres (Flint 1977). Tendons are well suited to resist tension and contain orientated collagen with relatively little glycosaminoglycans content. Pressure applied to tendons results in a markedly increased glycosaminoglycans production (Flint 1977). Changes in physical loading of tissues can therefore result in altered synthetic activity and cell shape (Bassett 1962; 1971a; Flint 1977).

In tissue culture experiments, the development of mesenchymal cells was largely influenced by environmental factors (Bassett 1962; 1964). The prime source of undifferentiated mesenchymal cells is uncertain but Bassett (1971a) favours the perivascular connective tissue cell as opposed to endothelial cell origin (Trueta 1963). In the presence of tension undifferentiated cells developed into fibroblasts and a similar aetiology can be proposed for the fibrous tissue encountered laterally in experimental lambs. Bassett (1971a) considers that cells can re-enter the mesenchymal cell pool from a previous state of specialised function. Tension influencing cell shape and function can also explain the gradual transition in cell morphology noted at the perichondrial region. Peripherally the periosteum is considered to be attached to the perichondrial region and is under tension. Thus a gradation of ambient forces from tension to relative compression can be envisaged which would explain the cellular morphology. The transition from chondrocyte to fibrocyte can be explained in two ways. Firstly, chondrocytes may adopt an elongated appearance in response to tension and ultimately change their synthetic activity. Secondly, tension may again produce elongated chondrocytes but adjacent undifferentiated progenitor cells may add fibrocytes to the cartilage surface. The progressively greater fibrocyte content of metaphyseal defects with passage of postoperative time favours the former suggestion. In addition, the smooth transition from one cell type to the other with a gradual change in matrix staining with toluidine blue would imply a change in cell function secondary to cell shape.

Establishment of a metaphyseal defect effectively increased

the thickness of the lateral sector growth cartilage. Gelbke (1951) was unable to produce growth cartilage thickening following experimentally applied traction. This author attached a wire to the proximal ulnar epiphysis. It is interesting that in lambs, in vivo, growth cartilage thickening occurred in association with another traction epiphysis, the tibial tuberosity. Smith (1962a and b) and Ogden (1974) described the histological appearance of such fibrous epiphyseal plates which contain fibrous tissue on their tensile aspect. Histological information of tibial tuberosity growth cartilage in experimental lambs is not as yet available. It would not be unreasonable to predict that a transition of cell morphology is present at this site which can be explained in a similar manner to that proposed for pressure growth plates.

In vitro behaviour of mesenchymal cells can also be used to explain bone plate protrusions noted in the medial growth plate sector.

In the presence of high oxygen tension, compaction of progenitor cells resulted, in vitro, in bone formation whereas low oxygen tension and compaction produced cartilage (Bassett 1962). Bone plate protrusions accompanied pronounced ingrowths of epiphyseal vasculature which would conceivable produce raised oxygen tension in association with compaction.

The vascular effect of experimentally produced compression and tension on growth cartilage is less well documented. Trueta (1958) reported metaphyseal ischaemia following excessive pressure although Hert (1964) stated that no vascular changes accompanied increased or decreased pressure applied to growth cartilage. Trueta and Trias

(1961) however recognised profuse epiphyseal vessels traversing the bone plate following application of continuous compression. Following removal of the compression device these authors observed increased vascularity around the growth plate. The effects of continuous and intermittent compression cannot be assumed to be identical and the latter would be closer to the in vivo situation in sheep. Fyfe (1961) experimentally applied intermittent pressure to growth cartilage and recorded a noticeably increased vascularity in the region.

Clearly increased vascularity does not necessarily produce increased growth which suggests that growth stimulation by induced vascular changes is of limited clinical application. The increased vascularity noted medially in control and experimental lambs can be explained in a number of ways. It would appear unlikely that this finding was purely artefact as has been discussed above. The epiphyseal hyperaemia can be explained as a functional requirement of increased growth cartilage thickness. The supposed stimulatory effect of dead or dying bone on vascularity must also be considered (Trias 1971; Trueta 1963). Certainly areas of empty lacunae were encountered within epiphyseal trabeculae in experimental lambs, but much less obviously in control animals. Dee (1964) concluded that although bone necrosis can stimulate remodelling it was not the only factor. The pronounced remodelling per se could result in the functional need for increased metaphyseal vasculature or conversely increased vasculature could result in intense remodelling. The effect of mechanical factors on blood vessels has been the subject of much discussion (Lura 1952) and theories of bone remodelling secondary to a vascular response

to mechanical environment have been proposed (see Brookes 1936; Evans 1957). Perhaps of more importance is the effect on blood flow. At least three mechanisms are thought to control blood flow but one, the metabolic control mechanism, has gained much experimental support (Shim 1968). The metabolic control mechanism suggests that pO_2, pCO_2, pH and acid metabolite levels are involved in controlling blood flow rates (Shim and Patterson 1967; Shim 1968) and bone remodelling (Brookes 1967; 1973). Sim (1970) and Sim and Kelly (1970) have shown the importance of changes in blood flow which occurs in response to the metabolic demands of bone. The experimental evidence would appear to be weighted in favour of vascular changes being secondary to the requirements of remodelling rather than the converse. Certainly Jee (1964) concluded that local alteration in vascularity is not the sole causal factor of bone remodelling.

Trabecular bone remodelling has been constantly discussed and some of the literature has been reviewed in the introduction to this section. Much of the literature is concerned with the orientation of trabeculae in which the mechanical environment is undoubtedly important. The manner in which such forces may modulate cellular activity has been the subject of many theoretical considerations.

Glegg and Leblond (1953) proposed that pressure results in dissolution and redistribution of bone crystals, a theory based on the physicochemical fact that pressure increases the solubility of crystals. A similar approach has been adopted by Justis and Loft (1970) who postulated that altered loading of bone produced altered hydroxyapatite crystal solubility which provided a negative feedback

message to the bone cells thus controlling their activity.

Other authors have proposed a direct effect of mechanical factors on the bone cells thus modulating their activity (Scott 1957; Johnson 1964; Hattner, Epker and Frost 1965). One way in which such control may be exerted is by piezoelectricity.

Piezoelectricity may be defined as electricity resulting from stress on crystals (Bassett 1971a). Piezoelectricity is not only a feature of bone but has been described in cartilage (Bassett and Pawluk 1972; Lotke, Black and Richardson 1973; 1974) muscle (Lokietek, Pawluk and Bassett 1974) various collagenous structures (Athenstaedt 1970), plants (Cerquiglini, Cignitti, Marchetti and Salleo 1967) and many other materials (Bassett 1971b).

It is well documented that when bone is mechanically deformed, or loaded, electric potentials are produced with a charge difference being produced on each bone surface. (Bassett 1965a and b, 1971a, b and c; Cerquiglini, Cignitti, Marchetti and Salleo 1967; McElhaney 1967; Gillooly, Hosley, Matthews and Jewett 1968; Cochran, Pawluk and Bassett 1968; Dwyer and Matthew 1970; Steinberg, Busenkell, Black and Korostoff 1972; 1974; Zengo; Pawluk and Bassett 1973). The concave bone surface possesses a relatively negative charge. As a result of recognition of piezoelectricity it has been suggested that bone converts mechanical energy to an electric signal and changes in the environment of mesenchyme cells may control their mitotic and functional activity to a large extent (Becker, Bassett and Bachman 1964; Shamos and Lavine 1964; Bassett 1965a and b; 1968; 1971a and b: Becker and Bachman 1966). This concept has been accepted by some as at least a possible agent in the transducer mechanism controlling

cell activity (Frost 1964b; Johnson 1964) but has been greeted with scepticism by others (Brookes 1971). Brookes (1971) points out that it is difficult to explain, by piezoelectricity, simultaneous bone resorption and deposition on the same bone surface. Although this objection would appear reasonable bone would predictably consist of a "mosaic" of electrical charge which would be constantly changing in a functional limb. In addition a number of different sources of electric potentials contribute to a bones bioelectric response to deformation (Bassett 1971 a and b). These include streaming potentials (Cerquiglini, Cignitti; Marchetti and Salleo 1967; Anderson and Eriksson 1970; Bassett 1971 a and b) which are due to fluid movement either within vessels or within bone tissue. Thus, changes in vasculature would predictably affect measured electrical potential in bone. Typically, the site of greatest electronegativity is in the metaphyses (Friedenberg and Brighton 1966; Dyer and Friedenberg 1970; Harlow, Heppenstall, Friedenberg and Brighton 1971) but Dyer and Friedenberg (1970) were unable to correlate piezoelectric changes with anatomical shape of a long bone. The metaphyses greater electronegativity may be a reflection of their being actively remodelled. It is relevant that Purdy and Sheard (1931) noted a definite correlation between metabolic rate and differences of electric potential in skin.

The collagen of bone is important in its piezoelectric properties perhaps due to its electret behaviour with bound water (Moscarenhas 1974). It is interesting that Stainberg, Kom, Korostaff and Pollack (1975) obtained reduced bone potentials in experimental lathyrism.

Induced potentials were investigated in conjunction with strain guage evaluation in vivo by Lanyon and Hartman (1976). These authors noted that the degree of polarisation was linearly proportional to deformation but that slow walking was insufficient in producing appreciable change in charge. As these authors conclude, it is uncertain whether piezoelectricity is a functionally irrelevant byproduct of bone deformation or whether it is instrumental in controlling cellular activity.

Perhaps the most convincing fact in support of electric potentials influencing bone shape is the realisation that electric currents can promote osteosynthesis. The literature which supports this finding is voluminous (Cieszynski 1964; Bassett, Pawluk and Becker 1964; Minkin, Poulton and Hoover 1968; Becker and Murray 1967; O'Connor, Charlton, Kirby and Woods 1969; Friedenberg, Andrews, Smolenski, Pearl and Brighton 1970; Andrews and Friedenberg 1970; Lavine, Lustrin, Shamos and Mose 1971; Friedenberg, Roberts, Didizian, and Brighton 1971; Levy and Rubin 1972; Jurgensen 1972; Connolly, McPhail, Eaton, Ortiz and Bayuzick 1974; Lemons, Reymonn and Niemann 1974; Brighton and Friedenberg 1974; Friedenberg, Zemsky, Pollis and Brighton 1974; Bassett, Pawluk and Beaumont 1974; Rinaldi, Lavine, Lustrin and Shamos 1975; Brighton, Friedenberg, Zemsky and Pollis 1975; Becker and Shapiro 1978).

In vitro experiments with electrical fields applied to costochondral growth cartilage resulted in growth stimulation but no gross histological change was noted (Brighton, Cronkey and Osterman 1976). In vivo experiments utilising a different order of current flow across growth cartilage failed to stimulate growth

(Friedenberg and Kohanim 1968).

In light of the demonstrable effect of electric currents on cellular activity it is difficult to totally dismiss the concept of electric potentials and bone form.

The concept that osteosynthesis occurs in connection with negative surface charge can be proposed as an explanation of cortical bone drift (Frost 1964; Epker and Frost 1965; 1966; Enlow 1968; Chamay and Tschantz 1972). Adaptive hypertrophy of the concave surface of a deformed bone is a common feature of many of the conditions described in part 8 of this study. Such adaptive hypertrophy has however been reported to occur only with intermittent and not continuous bone deformation (Roux 1895; cited by Liskova and Hert 1971; Chamay and Tschantz 1972). Chamay and Tschantz (1972) noted that use of lesser forces resulted in hypertrophy of both cortices as both were subjected to a degree of compression. With greater forces the concave cortex was under compression and the convex surface under tension. This may in part explain the apparent trophic influence of both compression and tension noted by Liskova and Hert (1971) or as with trabecular bone both stimuli may be able to promote adaptive hypertrophy. In the lambs of this study drift was predominantly in the direction of the concave bone surface. In addition massive periosteal bone deposition occurred at this site. The tension convex surface also contributed to drift in metaphyseal sections by acquiring endosteal bone deposition. No comments can be made on mid-diaphyseal sections which although demonstrating little change in normal surface contour were not examined in detail. The mechanical forces acting on the distal

metaphysis would appear to control drift and adaptive cortical hypertrophy. Compression is the principal trophic stimulus for periosteal bone deposition. It was noted however that the site of cortical hypertrophy was not constant but had a gradual spiral form in the direction of bone torsion. If compression is the stimulus for periosteal bone drift, then either the trophic influence has a gentle spiral orientation or the continued growth in length of the bone re-aligns the site of maximal compression. Pure unidirectional forces acting on metaphyses in vivo are unlikely.

The spiral nature of the trophic stimulus is suggested by the same orientation of profuse internal remodelling and cortical drift. A number of authors have observed internal remodelling being predominantly in that cortex possessing a concave surface, (Hert, Pribylova and Liskova 1972; Lanyon & Baggott 1976) or in the thicker cortex (Marotti 1963). In this connection it is of interest that application of compression plates to cortical bone are reported to initially stimulate skeletal renewal and secondary osteone formation (Perren, Huggler, Russenberger, Allgower, Mathys, Schenk, Willenegger and Muller 1969; Harris, Coutts, Davis and MacKenzie 1972). Similarly increased loading of compact bone results in profuse internal remodelling (Hert, Pribylova and Liskova 1963) whereas decreased function is accompanied by a reduction in secondary osteone formation (Avdiunitcheva 1964). Such findings suggest that internal remodelling is largely dependant on mechanical factors in addition to its mineral homeostasis function. Whether internal remodelling and drift are stress or strain dependant is debatable. Lanyon and Baggott (1976) noted osteones in sheep

radii were parallel to the direction of principal strain but remarked that this correlation may be coincidental. The findings in experimental lambs of this study suggest that a real correlation exists with the trophic stimulus, stress or strain, imposing the orientation of both drift and secondary osteones. Such a concept is in essence that described by Frost (1964a) who considered that resorption cavities develop principally in response to compression. Frost (1964a) remarked that when the bone is loaded in torque in addition to compression a spiral orientation is produced. The significance of this statement is discussed below. Lanyon, Goodship and Baggott (1977) suggested that cortical hypertrophy appeared to be directed towards restoring the strain regime of the whole bone following its deformation. Secondary osteone formation may similarly be influenced during attempts to maintain a structure best equipped to endure imposed forces. The formation of secondary osteones is reported to result in a reduction of biomechanical strength of bone (Amprino 1948; Currey 1959; 1960; Enlow 1962b; Hert, Kucera, Vavna and Volenik 1965). Certainly a large number of cement lines reduces the tensile strength of bone samples (Evans 1958; 1973; Evans and Bang 1967). Some authors consider that the formation of secondary osteones is a necessity following localised bone necrosis despite the loss in biomechanical strength that results (Enlow 1962b; Hert, Kucera, Vavra and Volenik 1965).

The result of bone drift in lambs was a profuse deposition of plexiform bone which is relatively well vascularised when compared with secondary osteonal bone (Currey 1960).

Presence of a vascular gradient in cortical bone results in its being

more flexible (Smith and Walmsley 1959) and this fact may alter the biomechanical strength of distal metaphyses. In addition, young bone has been considered to be more able to absorb energy due to its ability to undergo plastic deformation (Currey and Butler 1975). In light of such observations it is difficult to ascribe much biomechanical significance to limited areas of secondary osteone formation in distal metaphyses.

Undoubtedly another factor of importance is collagen orientation within osteones. Observations of osteonal collagen orientation have not been made in experimental lambs, but are a proposed future study using the material available. Briefly, however, it is thought that collagen orientation within lamellar bone substantially affects its biomechanical properties (Maj and Toajari 1937; Evans 1957; 1958; 1973; Ascenzi and Bonucci 1965; 1968; Evans and Vincintelli 1969).

Prior to gross deformity one of the fundamental lesions developing in the growth plate of lambs was a fracture line. Fig 44 summarises the morphological changes that followed. The matrix of growth cartilage has been described as providing the strength of the growth plate (Rang 1969) and in particular its resistance to shear. Rang (1969) considers that probably only shearing and avulsion forces are capable of separating an epiphysis and Morscher (1968) stated that the hypertrophic cell zone and the zone of provisional calcification were least able to resist traction. In slipped upper femoral epiphysis it is suggested that separation occurs when the force applied exceeds the shear strength of the growth plate (Rang 1969; Chung, Batterman

and Brighton 1976) although others have suggested a primary matrix lesion (Ponseti and McClintock 1956; Mickelson, Ponseti, Cooper and Maynard 1977). Certainly the matrix and cells of cartilage are much more vulnerable to applied forces than is bone with its rigid intercellular matrix (Little 1973) and the importance of surrounding soft tissues in maintaining cartilage integrety has been demonstrated (Chung, Batterman and Brighton 1976). The tensile strength of growth cartilage may differ at different sites (Wagner 1974). Either tension or shear could account for the noted growth cartilage separation in experimental lambs. The concomitant medial and lateral clefts, with an intact central isthmus may imply a torsional or shearing aetiology with the central region undergoing elastic or plastic deformation. The aetiological factor would be unlikely to be a pure force but the bending of cell columns and epiphyseo-metaphyseal displacement suggests a principal torsion or shear component. Moreland (1975) applied torsion experimentally to growth cartilage and produced a 'flowing' or bending of chondrocyte columns with similarly deformed primary spongiosa.

Some indication of the possible aetiology of such forces is provided by the experimental results of this section. Load bearing by the unoperated limb was predictably increased post.peratively. Gradually, with advancement of postoperative time the operated limb returned to almost normal weight bearing. The ability of a limb sustaining unilateral excision arthroplasty to regain full function has been recently demonstrated by force plate analysis of canine gait (Dueland, Barter and Antonson 1977).

The method of estimating load bearing by forelimb equalisation

is open to criticism. Obviously the results would have been improved by freestanding measurements had this been practical. The most serious error was in estimates of the right forelimb (RF) which could be predicted when the'three limb support' mechanism is considered (Gray 1943; 1961; Hildebrand 1966). In order to maintain equilibrium, if there is a greater vertical thrust, due to load bearing, by the nonoparated LH limb there must of necessity be a comparably increased thrust by the contralateral RF limb (Gray 1943; 1961). The necessity for such equilibrium explains the errors produced by forelimb equalisation. If increased load bearing or thrust by a limb was sufficient to produce deformity, then it might be expected that abnormal bone growth would be encountered in the RF limb. This suggestion is augmented by the known forward shift of centre of gravity in some experimental lambs. Forelimb deformity was however not grossly apparent. No hindlimb deformity followed excision arthroplasty in lamb no 15 despite a similar load bearing pattern to that noted in comparable grossly deformed individuals. It can be deduced that increased load bearing per se cannot be the sole aetiological factor.

The other obvious factor is muscle action which is intimately involved with posture and gait.

Excision arthroplasty resulted in adoption of a characteristic posture of the contralateral hindlimb. Increased LH load bearing necessitated adoption of a new limb position whereby the foot was adducted to a position more suited to maintaining equilibrium. In addition limb adduction resulted in internal rotation of abnormal extent. Adoption of a one foot stance in man similarly

results in postural changes including tibial rotation (Mitchell 1971). Such a posture in association with increased load bearing was present when the first growth cartilage abnormalities were identified. An abnormal degree of joint angulation, hock flexion and fetlock hyperextension, was also noted at this stage. Increased angulation in association with greater vertical forces was similarly noted in the quadruped by Jayes and Alexander (1978) and flexion deformities are considered common in pathological gait (Saunders, Inman and Eberhardt 1953). Some of the features noted in lambs are presented diagramatically in figs 45 a, b and c. The vertical line in these figures is purely a reference point and does not represent any particular axis. If the mechanical axis is considered however, joining the centre of pressure and the hip joint, then its relationship to the long bone diaphyses will alter with adoption of abnormal posture. The torsional component of abnormal posture will result in an altered or at least accentuated loading regime in the tibial and metatarsal extremities. Gait analysis revealed both internal and external torsional components during locomotion which were accentuated in pathological gait. Steindler (1935) and Brooks, Burstein and Frankel (1970) have described limb torsion in normal human gait. It would seem probable that the limb rotation occuring during the stages of the weight bearing cycle could account for a torsional force in the bone extremities. Such torque could explain the establishment of normal tibial torsion in lambs as is discussed below. With increased joint angulation any torsional force will be accentuated as a transverse moment will be produced when the mechanical axis is further removed from

the bone extremity. The same transverse moment will result in amplification of the effect of increased load bearing. The most lateral aspect of the metaphyses will be subjected to relatively greater tension or traction with adoption of abnormal posture. This effect as well as that of limb torsion will be sustained by both distal tibiae and distal metatarsae. The production of a transverse moment may explain how deformity can progress despite an even increasing contribution to load bearing by the operated limb in longer surviving lambs. Despite a relative reduction in LH load bearing the presence of progressive angular deformity will perpetually increase the transverse moment. Although the mechanical axis has been chosen in discussing changes of posture, the projection of vertical thrust through the centre of pressure would undergo comparable excursions relative to the bone diaphyses. The effect of altered ambient forces would be equally felt by the growth cartilage and the metaphyses. Establishment of a transverse moment would appear to be crucial in propogation of deformity. Sokoloff (1969) cites Simonet, Maquet and DeMarchim (1963) who analysed the moments in human clinical valgus and varus deformities and concluded that the loads may be increased severalfold. The loads exerted at bone extremity level during locomotion are likely to grossly exceed static load bearing. Paul (1967) measured human hip joint forces and noted that hip load can rise to 3 or 4 times body weight at heel strike and toe off. Thus the estimates of load bearing made in lambs are unlikely to bear much relation to the forces encountered during locomotion especially when pathological gait occurred. The magnitude of forces that can be sustained by joints is further exemplified by

Smith (1972 cited by Wright and Dowson 1976) who demonstrated human knee load to be up to 25 times body weight following a vertical drop of one metre.

All of these factors make it impossible to accurately predict the mechanical environment imposed on the bones of experimental lambs from the data available. Some limited conclusions can however be drawn.

It has been stated that cartilage is much less resistant to deforming forces than bone (Murray 1936; Little 1973) and is more resistant to compression than tension (Murray 1936). Of all the forces that could conceivably act on the growth cartilage and metaphysis, namely compression, tension, bending/shear and torsion, each could be produced by the altered posture and gait adopted by lambs in this study. As has previously been envisaged, it is improbable that a pure force is instrumental in producing growth cartilage separation. It would be more conceivable to envisage a "mosaic" of forces acting on the skeletal system a concept which is supported by strain guage studies (Lanyon and Smith 1970; Lanyon Hampson, Goodship and Shah 1974; Barnes and Pinder 1974; Lanyon, Goodship and Baggott 1977). The fracture line in growth cartilage could result from a combination of torsion, shear and tension. Evans (1957; 1973) has reviewed the effects of torsion applied to a column. Evans cites Timoshenko and Young (1962) who proposed that torsion of a homogeneous column results in a spiral of force at 45° around the column. Torsion results in tensile and compressive forces developing in addition to shearing. Bone however cannot be considered homogeneous (Currey 1964) which explains why torsional

fractures produced in bone specimens were less than 45^{o} to the bones long axis (Sammarco, Burstein, Davis and Frankel 1971). By contrast, cartilage is more homogeneous in structure than bone which may explain the relatively constant plane of cleavage encountered. If this argument is valid, the aetiological factors of each fracture line can be concluded to be comparable. Evans (1973) describe Timoshenko and Young's (1962) observation that in a column of material the magnitude of torsional shearing stress progressively increases from the centre to the periphery of its cross section. This observation may explain the relative immunity of the central growth cartilage region to separation. For the existence of a compression and tension aspect of the growth cartilage asymmetrical loading of the bone extremity must occur. The adoption of altered posture would of necessity produce asymmetrical loading. Fig 46 represents a hypothetical case of such asymmetry. If cartilage and bone are considered to be more resistant to compression than tension or shearing, the lateral growth plate must be predisposed to failure following adoption of postural change. The medial growth cartilage may however undergo a degree of necrosis prior to its failure. It is uncertain from the data available whether such necrosis is a necessary prerequisite to failure. Certainly some cases demonstrated a greater degree of amorphous matrix adjacent to the medial fracture line but this may represent pathological change secondary to cleavage.

In support of the proposed requirement of postural change prior to deformity the experiments of, in particular, Arkin and Katz (1956) are noteworthy. The experiments of these authors and others

who have induced torsional deformities in growing limbs following postural change are reviewed in the introduction of this section.

Whether or not the torsional forces described can result in bone torsion and dictate patterns of predominant internal remodelling and drift can only be surmised. Certainly Lanyon and Smith (1970) stressed the fact that theoretical computations of forces due to body weight, muscle action etc; are of doubtful significance unless supported experimentally.

It is interesting however that tibial torsion normally developed in lambs whereas metatarsal torsion did not. Metatarsal torsion however did develop in association with deformity of that bone. It is attractive to postulate that normal bone torsion has a similar aetiology to that produced pathologically in metatarsi. Adoption of abnormal posture has been concluded to produce asymmetrical growth cartilage loading and limb torsion of abnormal magnitude during locomotion. Both of these factors may be instrumental in producing normal bone torsion. If the mechanical axis of the limb is considered, or a vertical drawn through its centre of pressure, then the distal tibial site is more distant from each axis than the distal metatarsal growth plate. This factor in itself would amplify the forces produced by load bearing at the distal tibial site. A lesser degree of asymmetrical loading probably occurs in at least some normal distal metatarsae. This suggestion is augmented by the findings on metatarsal radiographs of normal lambs in part A of this study (see plate 12 A, B & C). It is unknown whether such bones possessed an appreciable degree of torsion. Compression is likely to be the major force acting upon pressure growth plates in long bones. Thus in association with

pressure epiphyses the bone extremity is probably exposed to lesser tension, shear and torsional stresses. Simon (1970) has postulated a similar normal mechanical environment in articular cartilage. Undoubtedly however anatomical differences exist, dependant upon the shape and dimensions of individual growth plates and upon their associated musculature and ligaments. The action of musculature cannot be ignored, but must be considered in conjunction with load bearing in providing the mechanical environment of each bone. It would be presumptive to assess the importance of musculature on purely anatomical terms. A strong hereditary pattern has been suggested in normal bone curvature (Murray and Selby 1930; Murray 1936; Walensky 1965). Others such as Pauwels (1948) and Amtmann (1971) have proposed that human femoral shape is mechanically controlled as a result of physiological bending.

The proposed asymmetrical growth cartilage loading in lambs of this study suggests another possible aetiology of normal curvature. Asymmetrical loading by either slowing or increasing endochondral bone growth could effectively model the bone diaphyseal curvature. Bhaskar, Weinmann, Schour and Green (1950) similarly proposed differential growth plate activity as being instrumental in producing rat tibial curvature.

General Discussion and Concluding Remarks

It is considered that the experimental model of angular bone deformity in immature lambs far surpasses many previous attempts recorded in the literature. In particular, the fact that no surgical interference occurred in the limb undergoing deformity makes the procedure much more 'physiological'. The importance of the experimental model produced is further suggested by the occurrence of meniscal and articular cartilage changes which although not described in this study will be the subject of future publications.

An explanation has been offered for at least two types of metaphyseal defect. One contained fibrous tissue, but the other consisted of hypertrophied chondrocyte protrusions into the metaphysis. The latter occurred as a result of compression and the retained cartilage core described in ulnae of large and giant dogs by Riser & Shirer (1965) may have a similar aetiology. It is probable that the distal ulna in such dogs is a site of potential overloading as one author (Zontine 1972) described metaphyseal trabecular stress fractures in conjunction with a retained cartilage core in the distal ulna of a Great Dane. A similar lesion was described in the distal ulna of a young puma (McMullin 1970) and although this author was unaware of the condition in the dog, an aetiology as postulated above could be logically proposed. It is tempting to suggest that in cases of osteodystrophy the potential for overloading of growth cartilage would be much greater due to the mechanical inadequacy of the skeleton. Thus, the pathology of

rickets, for example, (Trueta & Buhr 1963; Riser 1964; Storey 1965; Ehrlich, Weiss, Mankin, Treadwell & Sanzone 1973) may in part be explained purely on mechanical grounds.

Avian long bones grow in length from their articular surfaces, and it is interesting therefore that a comparable lesion to that noted in experimental lambs has been described in the 'Shaky Leg Syndrome' in turkeys. In this condition, a metaphyseal protrusion of uncalcified growth cartilage is described in association with trabecular collapse in the femoral neck (Wise and Ranaweera 1978). These authors suggest that periosteal traction is the aetiology of such lesions but overloading of growth cartilage would appear more feasible in light of the discussion above.

Overloading of articular cartilage may in certain circumstances similarly result in protrusion of chondrocytes into subchondral bone as a result of their failing to degenerate. Certainly the pathology of osteochondritis dissecans is in many ways reminiscent of that noted in experimental lambs (Craig & Riser 1965; Grondalen 1974).

Metaphyseal defects are reported to occur in the beak-like metaphyseal region in infantile tibia vara. This condition has been described in some detail in part B of this study. Assuming that the medial proximal tibia of such cases are subjected to overloading, the pathology can be explained on purely mechanical grounds.

It would appear that the marked epiphyseal asymmetry that occurs in infantile tibia vara is a result of the relative skeletal immaturity at the time the condition manifests. One

wonders if similar epiphyseal asymmetry would result from over-loading of bone extremities in experimental animals of comparable skeletal immaturity.

If the pathology described in experimental lambs is purely a result of limb imbalance,with excessive and asymmetrical loading of growth cartilage producing deformity, naturally occurring cases might be envisaged. During the course of this study a Border Leicester ewe lamb was presented for treatment with an angulated malunion tibial fracture of the left hindlimb. A comparable postural abnormality to that induced experimentally was present in the contralateral right hind limb (see plate 46). Radiography of the right hind limb revealed comparable growth plate/metaphyseal changes to those produced experimentally (plate 47).

A similar postural abnormality was seen in experimental lambs in which the contralateral hindlimb received a carbon fibre implant following achilles tendon section.

The early postural abnormality noted in lambs of this study, consisted of internal torsion and adduction. If this stance is an effort to maintain postural stability it might be expected to occur in other quadrupeds with unilateral hindlimb disability. This was found to be true in the bovine, equine and canine species especially in cases of contralateral hindlimb fracture. By way of example, plate 48 demonstrates the stance adopted by an 8 month old Irish Setter puppy 3 days following fracture of the right hind tibia. Following internal fixation of the tibial fracture this case made a rapid recovery and by four weeks postoperatively possessed an almost

normal hindlimb posture. During the early postoperative period there was judged, on radiographs, to be a mild degree of growth plate thickening medially in the left hind tibia. The increased growth cartilage thickness was noted in both the proximal and distal tibial growth plates.

One critical factor in determining whether deformity occurs must be the age of the animal and the duration of limb imbalance. In addition species variation undoubtedly exists and the ability of the animal to shift its centre of gravity forwards may well be important. When the diversity of limb lengths and relative body weights of quadrupeds are considered, it is easy to imagine that some would be able to accommodate centre of gravity shifts easier than others. It is known that experimental lambs in this study possessed a degree of increased forelimb loading following hip excision arthroplasty but this attempt must be deemed inadequate.

The ovine species may be particularly susceptible to the type of deformity produced. Recently, lambs have been presented for investigation which possess varying degrees of carpal valgus or varus. The site of angulation in such individuals is at two sites, the distal radius and ulna and the distal metacarpus. Bone specimens from affected lambs have been acquired and are currently undergoing investigation but some of the changes described in this study have already been detected. Recently, Hidiroglou, Dukes, Ho and Heaney (1978) have described a 'bent limb syndrome' in housed lambs which similarly involves forelimb valgus and varus and appears to be the same clinical entity.

These authors have produced histological sections of the distal radius or metacarpus of affected inidiviuals. Areas of 'fibrous dysplasia' are described extending into the diaphysis and protrusion of growth cartilage into the metaphysis was noted. The pathology described by these authors closely resembles that induced in this study but unfortunately they fail to identify the location in the deformed bone extremities of each finding. Hidiroglou et al (1978) describe the syndrome as complex and postulate that inhibited Vit. D. synthesis may contribute to its development. The similarly affected lambs presented at Glasgow were from hill flocks kept under a totally different husbandry regime to that described by Hidiroglou et al (1978) in Canada. The pathology can certainly be explained purely on mechanical grounds. The affected lambs seen at Glasgow were placed on the platform scale apparatus and estimates of load bearing by each limb made. Total forelimb load bearing was slightly greater than would have been expected from the results of this study.

One regime where persistent postural asymmetry would predictably produce asymmetrical growth cartilage loading is in the human spine. Scoliosis is mainly a problem in the biped and is rare in quadrupeds where it is usually associated with congenital vertebral malformations. Scoliosis has however been produced in experimental animals with variable success (Bisgard 1935; Haas 1939; Bisgard & Musselman 1940; Schwartzman & Miles 1945; Nachlas & Borden 1950; Langenskiold & Michelsson 1962; Ottander 1963; Michelsson 1965; Robin 1965; Karaharju 1967; Alexander, Bunch & Ebbesson 1972;

MacEwen 1973; Ritsila & Alhopuro 1975; Robin & Stein 1975). Authorities such as James (1970) however consider that experimental scoliosis has added little to our knowledge of the natural disease. Histopathological studies of experimental scoliosis however are of interest and are worthy of brief mention.

Michelsson (1965) produced scoliosis in rabbits a species which has growth cartilage on both the cranial and caudal vertebral surfaces. Histological sections of the growth cartilage reveal loss of distinct chondrocyte columns towards the concave surface of the deformity whereas the chondrocytes are more attenuated on its convex aspect. In some areas the chondrocytes of the concave aspect appear to have formed nests or clumps rather than columns.

Karaharju (1967) produced scoliosis in pigs and rabbits and described similar differences in chondrocyte column morphology on the concave and convex aspects of the curve. In addition this author noted growth cartilage thickening on the concave aspect in both pigs and rabbits.

The histological findings in experimental scoliosis thus bear a close similarity to those noted in experimental lambs undergoing distal tibial or distal metatarsal deformity. The conclusion of Arkin (1949) that the "vertebral deformation of structural scoliosis can be explained by an asymmetrical disturbance of epiphyseal growth by pressure" would thus appear tenable.

INDUCED ABNORMAL BONE GROWTH WITH PARTICULAR REFERENCE TO THE GROWTH PLATE.

(in 2 volumes)

VOLUME II

A THESIS submitted for THE DEGREE

OF DOCTOR OF PHILOSOPHY

in

The FACULTY OF VETERINARY MEDICINE

of

THE UNIVERSITY OF GLASGOW

by

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August 1979

FORMAT

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APPENDIX 1 TABLE 1

Experimental Number	Ear Tag Number	Sex	Date of Birth	Birth Weight	From Ewe Number	No. of lambs born to ewe in that pregnancy	Twins
20	TG072	F	23.3.76	9lbs	15	2	Twins
59	226	F	1.12.76	6lbs	1	2	
21	TG073	M	23.3.76	10lbs	15	2	
58	229	F	3.12.76	7lbs	2	2	Twins
56	TG184	M	24.7.76	6lbs	59	2	Twin to no.48
57	228	M	3.12.76	8lbs	2	2	
25	TG081	F	24.3.76	6lbs	35	2	
24	TG050	F	24.3.76	7lbs	48	2	
33	TG155	F	29.5.76	5lbs	62	2	Reared single
40	TG195	F	28.7.76	11lbs	21	2	Twin to no.54
41	TG190	M	25.7.76	9lbs	19	3	Sibling to nos 49 and 52
44	TG173	F	23.7.76	9lbs	41	2	
42	TG174	F	23.7.76	7lbs	37	3	Twins
43	TG176	F	23.7.76	6.5lbs	37	3	
60	222	F	29.11.76	9lbs	60	2	
45	TG177	M	23.7.76	11lbs	40	1	
46	TG181	F	23.7.76	9lbs	61	1	
48	TG183	F	24.7.76	8lbs	59	2	Twin to no.56
49	TG191	M	25.7.76	9lbs	19	3	Sibling to nos41&52
50	TG187	F	25.7.76	6lbs	32	3	Twins
53	TG193	M	25.7.76	14lbs	28	1	
51	TG188	F	25.7.76	9lbs	32	3	
52	TG192	M	25.7.76	10lbs	19	3	Sibling to nos.41 & 49
30	TG168	F	2.6.76	10.5lbs	5	2	
38	NT28	M	2.6.76	12.5lbs	64	2	
54	TG194	M	28.7.76	10lbs	21	2	Twin to no.40
31	TG161	M	30.5.76	7lbs	2	2	
29	TG160	F	30.5.76	8lbs	66	2	Reared Single
36	TG166	F	1.6.76	10lbs	63	1	
35	TG154	M	29.5.76	9lbs	9	2	Twins
37	TG155	F	29.5.76	8lbs	9	2	
39	NT44	F	29.5.76	10lbs	38	1	
55	TG189	F	25.7.76	10lbs	22	1	
19	TG070	F	23.3.76	6lbs	14	3	Triplets
17	TG068	F	23.3.76	8lbs	14	3	
18	TG069	F	23.3.76	8lbs	14	3	

Experimental Number	Ear Tag Number	Sex	Date of Birth	Birth Weight	From Ewe Number	No.of lambs born to ewe in that pregnancy	Twins
16	TG067	F	22.3.76	6lbs	46	2	
14	TG064	F	22.3.76	7lbs	55	2	Twins
15	TG066	M	22.3.76	7lbs	46	2	
11	TG055	M	21.3.76	7lbs	54	2	Twins
12	TG056	F	21.3.76	7lbs	54	2	
13	TG060	F	21.3.76	6lbs	49	2	

APPENDIX 1 TABLE 2

DISTAL TIBIAL DEFORMITY (ON ANTEROPOSTERIOR RADIOGRAPHS) — CONTROL LAMBS

Lamb Number	Limb	Angle I	Angle II	Experimental Survival Time
20	LH	2.0°M	1.0°M	1 week
	RH	2.0°M	1.0°M	
59	LH	2.5°M	2.0°M	1 week
	RH	2.5°M	2.0°M	
57	LH	0°	0.5°M	2 weeks
	RH	0°	0.5°M	
25	LH	1.0°M	0.5°M	2 weeks
	RH	1.0°M	0.5°L	
44	LH	1.0°M	1.0°L	3 weeks
	RH	1.0°M	1.0°L	
60	LH	2.0°M	1.5°M	1 month
	RH	2.0°M	2.0°M	
48	LH	0.5°M	1.5°L	2 months
	RH	0.5°M	1.5°L	
53	LH	1.0°M	0°	3 months
	RH	1.0°M	0°	
30	LH	1.5°M	1.0°M	4 months
	RH	1.0°M	0°	
31	LH	0.5°L	1.0°M	5 months
	RH	0°	0.5°M	
35	LH	1.0°M	1.0°L	6 months
	RH	1.0°M	1.0°L	
19	LH	2.0°M	0.5°L	8 months
	RH	2.0°M	0.5°L	
16	LH	1.5°M	1.0°M	10 months
	RH	1.0°M	0°	
11	LH	1.5°M	1.5°L	12 months
	RH	1.5°M	1.5°L	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular facets

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 3

DISTAL TIBIAL DEFORMITY
(ON ANTEROPOSTERIOR RADIOGRAPHS) EXPERIMENTAL LAMBS

Lamb Number	Limb	Angle I	Angle II	Postoperative Survival Time
21	LH	2.5°M	1.0°M	1 week
	RH	2.0°M	1.0°M	
58	LH	1.5°M	1.0°L	1 week
	RH	1.5°M	1.0°L	
56	LH	1.5°M	1.0°M	1 week
	RH	2.0°M	1.5°M	
24	LH	2.5°M	2.0°M	2 weeks
	RH	2.5°M	2.0°M	
33	LH	2.5°M	2.0°M	2 weeks
	RH	2.5°M	2.0°M	
40	LH	2.0°M	0°	2 weeks
	RH	2.0°M	0°	
41	LH	1.0°M	1.5°L	2 weeks
	RH	1.0°M	1.5°L	
42	LH	3.5°M	1.5°M	3 weeks
	RH	2.0°M	1.0°M	
43	LH	2.5°M	0°	3 weeks
	RH	2.0°M	0°	
45	LH	Sectioned		1 month
	RH	Sectioned		
46	LH	Sectioned		1 month
	RH	Sectioned		
49	LH	3.0°M	1.5°L	2 months
	RH	1.0°M	0.5°L	
50	LH	2.5°M	1.5°L	2 months
	RH	1.5°M	0.5°L	
51	LH	1.5°M	1.0°L	3 months
	RH	1.5°M	0°	
52	LH	11.0°M	8.5°M	3 months
	RH	1.0°M	0°	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular facets

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 3 (contd)

DISTAL TIBIAL DEFORMITY
(ON ANTEROPOSTERIOR RADIOGRAPHS) EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Angle I	Angle II	Postoperative Survival Time
38	LH	1.5°M	0°	4 months
	RH	1.0°M	0°	
54	LH	5.0°M	1.0°M	4 months
	RH	0°	1.5°L	
29	LH	4.5°M	3.0°M	5 months
	RH	1.0°M	0°	
36	LH	33.0°M	29.5°M	5 months
	RH	3.0°M	1.0°M	
37	LH	0°	2.0°M	6 months
	RH	3.0°M	0°	
39	LH	28.5°M	25.0°M	6 months
	RH	1.0°M	4.0°L	
55	LH	17.0°M	15.5°M	6 months
	RH	2.5°M	1.5°M	
17	LH	7.0°M	3.0°M	8 months
	RH	1.0°M	2.0°L	
18	LH	44.0°M	42.5°M	8 months
	RH	2.0°M	1.0°M	
14	LH	49.5°M	47.5°M	10 months
	RH	2.0°M	1.0°L	
15	LH	1.5°M	1.5°L	10 months
	RH	0°	0°	
12	LH	29.0°M	25.0°M	12 months
	RH	4.0°M	1.5°M	
13	LH	4.0°M	1.0°L	12 months
	RH	1.0°M	1.0°L	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular facets

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 4

DISTAL METATARSAL DEFORMITY
(ON ANTEROPOSTERIOR RADIOGRAPHS) CONTROL LAMBS

Lamb Number	Limb	Angle I	Angle II	Experimental Survival Time
20	LH	0.5°M	0.5°M	1 week
	RH	0.5°M	0.5°M	
59	LH	1.0°M	1.0°M	1 week
	RH	1.0°M	1.0°M	
57	LH	0°	0°	2 weeks
	RH	0°	0°	
25	LH	0.5°M	0.5°L	2 weeks
	RH	0°	0°	
44	LH	0.5°M	0.5°L	3 weeks
	RH	0°	0.5°L	
60	LH	1.0°M	0.5°L	1 month
	RH	1.5°M	0.5°M	
48	LH	0°	2.0°M	2 months
	RH	0.5°M	2.5°M	
53	LH	0.5°L	0°	3 months
	RH	0.5°L	0°	
30	LH	1.5°L	0.5°M	4 months
	RH	1.0°M	1.5°M	
31	LH	0°	0.5°L	5 months
	RH	0°	0.5°M	
35	LH	1.0°L	0.5°M	6 months
	RH	0.5°L	1.5°M	
19	LH	0°	0°	8 months
	RH	0°	0°	
16	LH	0.5°M	0.5°M	10 months
	RH	0.5°M	0.5°M	
11	LH	0°	0.5°L	12 months
	RH	0°	0°	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular surface

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 5

DISTAL METATARSAL DEFORMITY (ON ANTEROPOSTERIOR RADIOGRAPHS) EXPERIMENTAL LAMBS

Lamb Number	Limb	Angle I	Angle II	Postoperative Survival Time
21	LH	0.5°L	1.5°M	1 week
	RH	0.5°M	1.0°M	
58	LH	0.5°M	0.5°M	1 week
	RH	0.5°M	0.5°M	
56	LH	1.0°L	1.0°M	1 week
	RH	0°	1.0°M	
24	LH	1.0°M	1.5°M	2 weeks
	RH	0.5°L	1.5°M	
33	LH	1.0°M	0.5°M	2 weeks
	RH	0.5°L	0.5°M	
40	LH	0°	1.0°M	2 weeks
	RH	1.0°M	1.0°M	
41	LH	0.5°L	0°	2 weeks
	RH	0.5°L	1.0°M	
42	LH	1.0°M	1.5°M	3 weeks
	RH	1.0°M	2.0°M	
43	LH	2.0°M	3.5°M	3 weeks
	RH	0°	3.5°M	
45	LH	2.0°M	0.5°M	1 month
	RH	0.5°M	0.5°M	
46	LH	1.0°M	1.0°M	1 month
	RH	0°	2.0°M	
49	LH	1.0°M	1.0°M	2 months
	RH	0°	1.0°M	
50	LH	10.5°M	7.0°M	2 months
	RH	0°	1.5°M	
51	LH	5.0°M	1.0°M	3 months
	RH	0.5°M	1.5°M	
52	LH	5.0°M	4.0°M	3 months
	RH	0.5°M	3.0°M	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular surface

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 5 (contd)

DISTAL METATARSAL DEFORMITY (ON ANTEROPOSTERIOR RADIOGRAPHS) EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Angle I	Angle II	Postoperative Survival Time
38	LH	13.0°M	10.5°M	4 months
	RH	1.0°M	1.5°M	
54	LH	8.5°M	3.0°M	4 months
	RH	0°	0°	
29	LH	13.5°M	8.5°M	5 months
	RH	0.5°M	1.0°M	
36	LH	13.0°M	11.0°M	5 months
	RH	0.5°M	1.0°M	
37	LH	21.0°M	18.5°M	6 months
	RH	0.5°M	2.0°M	
39	LH	2.0°M	2.0°L	6 months
	RH	0.5°M	0.5°M	
55	LH	5.0°L	4.5°L	6 months
	RH	0.5°M	2.5°M	
17	LH	26.0°M	19.0°M	8 months
	RH	0.5°M	1.0°M	
18	LH	3.0°L	2.0°L	8 months
	RH	0°	2.0°M	
14	LH	7.0°L	7.0°L	10 months
	RH	1.0°M	4.0°M	
15	LH	2.5°L	1.0°L	10 months
	RH	1.0°M	2.0°M	
12	LH	6.0°L	6.0°L	12 months
	RH	2.5°M	5.0°M	
13	LH	12.5°M	2.5°M	12 months
	RH	1.0°L	3.0°L	

Angle I = Angle to long axis made by perpendicular drawn from growth plate/metaphysis junction

Angle II = Angle to long axis made by perpendicular drawn from distal articular surface

M = medial angular deformity L = lateral angular deformity

APPENDIX 1 TABLE 6

DISTAL TIBIAL DEFORMITY

(ON LATERAL RADIOGRAPHS)

CONTROL LAMBS

Lamb Number	Limb	Posterior Angle	Experimental Survival Time
53	LH	0.5°	3 months
	RH	1.5°	
30	LH	1.0°	4 months
	RH	1.5°	
31	LH	1.0°	5 months
	RH	1.5°	
35	LH	1.0°	6 months
	RH	< 1.0°	
19	LH	1.0°	8 months
	RH	1.5°	
16	LH	2.0°	10 months
	RH	2.0°	
11	LH	1.0°	12 months
	RH	< 1.0°	

Posterior angular deformity relative to tibial long axis

APPENDIX 1 TABLE 7

DISTAL TIBIAL DEFORMITY (ON LATERAL RADIOGRAPHS) EXPERIMENTAL LAMBS

Lamb Number	Limb	Posterior Angular Deformity	Postoperative Survival Time
51	LH	3.0°	3 months
	RH	1.0°	
52	LH	11.0°	3 months
	RH	1.0°	
54	LH	1.0°	4 months
	RH	< 1.0°	
29	LH	5.0°	5 months
	RH	1.5°	
36	LH	17.5°	5 months
	RH	1.0°	
39	LH	14.5°	6 months
	RH	1.5°	
55	LH	21.0°	6 months
	RH	1.5°	
18	LH	29.0°	8 months
	RH	< 1.0°	
14	LH	49.0°	10 months
	RH	1.5°	
12	LH	41.0°	12 months
	RH	1.5°	

Posterior angular deformity relative to tibial long axis

APPENDIX 1 TABLE 8

TIBIAL TORSION - CONTROL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
20	LH	5.0°	5.0°	5.0°	5.0°	1 week
	RH	sectioned	-	-	-	
59	LH	11.0°	11.0°	11.0°	11.0°	1 week
	RH	8.0°	9.0°	9.0°	9.0°	
57	LH	7.0°	7.0°	7.0°	7.0°	2 weeks
	RH	8.0°	8.0°	8.0°	8.0°	
25	LH	8.0°	7.0°	7.0°	7.0°	2 weeks
	RH	5.0°	7.0°	5.0°	5.0°	
44	LH	sectioned	-	-	-	3 weeks
	RH	sectioned	-	-	-	
60	LH	11.0°	10.0°	10.0°	10.0°	1 month
	RH	8.0°	8.0°	7.0°	8.0°	
48	LH	7.0°	7.0°	7.0°	7.0°	2 months
	RH	8.0°	8.0°	7.0°	8.0°	
53	LH	10.0°	10.0°	12.0°	10.0°	3 months
	RH	13.0°	12.0°	12.0°	12.0°	
30	LH	12.0°	11.0°	12.0°	12.0°	4 months
	RH	10.0°	9.0°	9.0°	9.0°	
31	LH	11.0°	11.0°	12.0°	11.0°	5 months
	RH	9.0°	9.0°	10.0°	9.0°	
35	LH	18.0°	18.0°	18.0°	18.0°	6 months
	RH	16.0°	17.0°	16.0°	16.0°	
19	LH	17.0°	18.0°	18.0°	18.0°	8 months
	RH	16.0°	17.0°	17.0°	17.0°	
16	LH	17.0°	17.0°	17.0°	17.0°	10 months
	RH	18.0°	17.0°	16.0°	17.0°	
11	LH	17.0°	18.0°	18.0°	18.0°	12 months
	RH	17.0°	17.0°	18.0°	17.0°	

APPENDIX 1 TABLE 9

TIBIAL TORSION - EXPERIMENTAL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
21	LH	9.0°	8.0°	9.0°	9.0°	1 week
	RH	8.0°	8.0°	8.0°	8.0°	
58	LH	10.0°	10.0°	10.0°	10.0°	1 week
	RH	8.0°	8.0°	6.0°	8.0°	
56	LH	sectioned	-	-	-	1 week
	RH	sectioned	-	-	-	
24	LH	16.0°	16.0°	16.0°	16.0°	2 weeks
	RH	12.0°	13.0°	12.0°	12.0°	
33	LH	12.0°	12.0°	12.0°	12.0°	2 weeks
	RH	6.0°	7.0°	8.0°	7.0°	
40	LH	sectioned	-	-	-	2 weeks
	RH	sectioned	-	-	-	
41	LH	sectioned	-	-	-	2 weeks
	RH	sectioned	-	-	-	
42	LH	sectioned	-	-	-	3 weeks
	RH	sectioned	-	-	-	
43	LH	sectioned	-	-	-	3 weeks
	RH	sectioned	-	-	-	
45	LH	sectioned	-	-	-	1 month
	RH	sectioned	-	-	-	
46	LH	sectioned	-	-	-	1 month
	RH	sectioned	-	-	-	
49	LH	15.0°	15.0°	15.0°	15.0°	2 months
	RH	8.0°	7.0°	8.0°	8.0°	
50	LH	10.0°	10.0°	10.0°	10.0°	2 months
	RH	6.0°	6.0°	5.0°	6.0°	
51	LH	14.0°	14.0°	14.0°	14.0°	3 months
	RH	7.0°	8.0°	7.0°	7.0°	
52	LH	20.0°	19.0°	18.0°	18.0°	3 months
	RH	7.0°	6.0°	6.0°	6.0°	
38	LH	16.0°	17.0°	17.0°	17.0°	4 months
	RH	8.0°	7.0°	8.0°	8.0°	
54	LH	15.0°	15.0°	17.0°	15.0°	4 months
	RH	5.0°	4.0°	4.0°	4.0°	
29	LH	15.0°	15.0°	14.0°	15.0°	5 months
	RH	7.0°	6.0°	6.0°	6.0°	
36	LH	38.0°	37.0°	37.0°	37.0°	5 months
	RH	8.0°	8.0°	7.0°	8.0°	

APPENDIX 1 TABLE 9 (contd)

TIBIAL TORSION - EXPERIMENTAL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
37	LH	23.0°	23.0°	25.0°	23.0°	6 months
	RH	23.0°	23.0°	22.0°	23.0°	
39	LH	29.0°	29.0°	29.0°	29.0°	6 months
	RH	9.0°	10.0°	9.0°	9.0°	
55	LH	26.0°	25.0°	25.0°	25.0°	6 months
	RH	8.0°	9.0°	10.0°	9.0°	
17	LH	17.0°	15.0°	16.0°	16.0°	8 months
	RH	7.0°	6.0°	7.0°	7.0°	
18	LH	46.0°	47.0°	47.0°	47.0°	8 months
	RH	8.0°	8.0°	8.0°	8.0°	
14	LH	30.0°	30.0°	29.0°	30.0°	10 months
	RH	11.0°	12.0°	12.0°	12.0°	
15	LH	19.0°	18.0°	17.0°	18.0°	10 months
	RH	4.0°	4.0°	6.0°	4.0°	
12	LH	48.0°	49.0°	50.0°	49.0°	12 months
	RH	12.0°	12.0°	12.0°	12.0°	
13	LH	11.0°	11.0°	11.0°	11.0°	12 months
	RH	9.0°	10.0°	9.0°	9.0°	

APPENDIX 1 TABLE 10

METATARSAL TORSION - CONTROL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Experimental Survival Time
20	LH	2.0°	2.0°	2.0°	2.0°	1 week
	RH	2.0°	2.0°	2.0°	2.0°	
59	LH	2.0°	4.0°	3.0°	3.0°	1 week
	RH	3.0°	2.0°	3.0°	3.0°	
57	LH	1.0°	2.0°	3.0°	2.0°	2 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
25	LH	1.0°	2.0°	2.0°	2.0°	2 weeks
	RH	2.0°	1.0°	1.0°	1.0°	
44	LH	2.0°	2.0°	2.0°	2.0°	3 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
60	LH	2.0°	2.0°	2.0°	2.0°	1 month
	RH	3.0°	2.0°	2.0°	2.0°	
48	LH	2.0°	1.0°	2.0°	2.0°	2 months
	RH	2.0°	2.0°	2.0°	2.0°	
53	LH	2.0°	3.0°	2.0°	2.0°	3 months
	RH	3.0°	2.0°	3.0°	3.0°	
30	LH	2.0°	2.0°	2.0°	2.0°	4 months
	RH	2.0°	2.0°	3.0°	2.0°	
31	LH	3.0°	4.0°	3.0°	3.0°	5 months
	RH	4.0°	3.0°	4.0°	4.0°	
35	LH	2.0°	2.0°	2.0°	2.0°	6 months
	RH	2.0°	2.0°	2.0°	2.0°	
19	LH	2.0°	2.0°	2.0°	2.0°	8 months
	RH	2.0°	3.0°	2.0°	2.0°	
16	LH	2.0°	2.0°	2.0°	2.0°	10 months
	RH	2.0°	2.0°	2.0°	2.0°	
11	LH	5.0°	5.0°	4.0°	5.0°	12 months
	RH	5.0°	5.0°	5.0°	5.0°	

APPENDIX 1 TABLE 11

METATARSAL TORSION - EXPERIMENTAL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
21	LH	2.0°	2.0°	2.0°	2.0°	1 week
	RH	2.0°	2.0°	2.0°	2.0°	
58	LH	3.0°	3.0°	2.0°	3.0°	1 week
	RH	2.0°	2.0°	2.0°	2.0°	
56	LH	2.0°	2.0°	2.0°	2.0°	1 week
	RH	2.0°	2.0°	2.0°	2.0°	
24	LH	1.0°	1.0°	1.0°	1.0°	2 weeks
	RH	2.0°	1.0°	1.0°	1.0°	
33	LH	2.0°	2.0°	2.0°	2.0°	2 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
40	LH	3.0°	3.0°	3.0°	3.0°	2 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
41	LH	4.0°	4.0°	4.0°	4.0°	2 weeks
	RH	3.0°	3.0°	3.0°	3.0°	
42	LH	2.0°	2.0°	2.0°	2.0°	3 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
43	LH	2.0°	2.0°	2.0°	2.0°	3 weeks
	RH	2.0°	2.0°	2.0°	2.0°	
45	LH	6.0°	6.0°	6.0°	6.0°	1 month
	RH	5.0°	4.0°	5.0°	5.0°	
46	LH	3.0°	3.0°	3.0°	3.0°	1 month
	RH	3.0°	2.0°	3.0°	3.0°	
49	LH	8.0°	8.0°	8.0°	8.0°	2 months
	RH	2.0°	3.0°	3.0°	3.0°	
50	LH	2.0°	3.0°	3.0°	3.0°	2 months
	RH	2.0°	2.0°	2.0°	2.0°	
51	LH	14.0°	14.0°	14.0°	14.0°	3 months
	RH	1.0°	2.0°	2.0°	2.0°	
52	LH	13.0°	13.0°	13.0°	13.0°	3 months
	RH	3.0°	3.0°	3.0°	3.0°	
38	LH	1.0°	2.0°	2.0°	2.0°	4 months
	RH	1.0°	2.0°	2.0°	2.0°	
54	LH	13.0°	12.0°	13.0°	13.0°	4 months
	RH	5.0°	6.0°	5.0°	5.0°	

APPENDIX 1 TABLE 11 (contd)

METATARSAL TORSION - EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
29	LH	18.0°	17.0°	18.0°	18.0°	5 months
	RH	6.0°	6.0°	6.0°	6.0°	
36	LH	14.0°	13.0°	14.0°	14.0°	5 months
	RH	1.0°	2.0°	2.0°	2.0°	
37	LH	5.0°	5.0°	5.0°	5.0°	6 months
	RH	4.0°	4.0°	5.0°	4.0°	
39	LH	18.0°	18.0°	18.0°	18.0°	6 months
	RH	2.0°	2.0°	2.0°	2.0°	
55	LH	18.0°	18.0°	18.0°	18.0°	6 months
	RH	5.0°	4.0°	5.0°	5.0°	
17	LH	14.0°	14.0°	13.0°	14.0°	8 months
	RH	4.0°	5.0°	4.0°	4.0°	
18	LH	6.0°	7.0°	7.0°	7.0°	8 months
	RH	3.0°	3.0°	2.0°	3.0°	
14	LH	21.0°	23.0°	21.0°	21.0°	10 months
	RH	2.0°	2.0°	2.0°	2.0°	
15	LH	9.0°	9.0°	9.0°	9.0°	10 months
	RH	2.0°	2.0°	2.0°	2.0°	
12	LH	28.0°	27.0°	29.0°	28.0°	12 months
	RH	3.0°	2.0°	2.0°	2.0°	
13	LH	5.0°	5.0°	5.0°	5.0°	12 months
	RH	2.0°	2.0°	2.0°	2.0°	

FEMORAL, TIBIAL & METATARSAL LENGTH
(ON ANTEROPOSTERIOR RADIOGRAPHS) CONTROL LAMBS

Lamb Number	Limb	Femoral Length G.T. (cms)	Femoral Length F.H. (cms)	Tibial Length (cms)	Metatarsal Length (cms)	Experimental Survival Time
20	LH	12.30	12.50	14.55	9.90	1 week
	RH	12.30	12.50	14.55	9.90	
59	LH	12.70	13.00	14.40	9.45	1 week
	RH	12.70	13.0	14.40	9.45	
57	LH	11.20	11.10	12.75	8.30	2 weeks
	RH	11.20	11.10	12.75	8.30	
25	LH	12.80	12.65	14.50	9.35	2 weeks
	RH	12.80	12.65	14.50	9.35	
44	LH	11.55	11.70	13.90	9.15	3 weeks
	RH	11.55	11.70	13.90	9.15	
60	LH	13.40	13.70	15.40	9.70	1 month
	RH	13.40	13.70	15.40	9.70	
48	LH	13.50	13.30	15.30	10.60	2 months
	RH	13.50	13.30	15.30	10.60	
53	LH	16.20	16.30	18.70	12.20	3 months
	RH	16.20	16.30	18.70	12.20	
30	LH	15.10	15.15	17.80	12.40	4 months
	RH	15.10	15.15	17.80	12.40	
31	LH	15.55	15.40	17.80	12.15	5 months
	RH	15.55	15.40	17.80	12.15	
35	LH	16.60	16.30	18.60	11.75	6 months
	RH	16.60	16.30	18.60	11.75	
19	LH	16.85	16.55	19.10	12.0	8 months
	RH	16.85	16.55	19.10	12.0	
16	LH	16.70	16.50	18.90	11.40	10 months
	RH	16.70	16.40	18.90	11.40	
11	LH	18.85	18.15	22.0	14.30	12 months
	RH	18.80	18.05	22.0	14.30	

APPENDIX 1 TABLE 13

FEMORAL TIBIAL & METATARSAL LENGTH
(ON ANTEROPOSTERIOR RADIOGRAPHS)

EXPERIMENTAL LAMBS

Lamb Number	Limb	Femoral Length G.T. (cms)	Femoral Length F.H. (cms)	Tibial Length (cms)	Metatarsal Length (cms)	Postoperative Survival Time
21	LH	13.30	13.50	15.70	10.10	1 week
	RH	13.25	Excised	15.70	10.10	
58	LH	12.20	12.20	14.0	8.80	1 week
	RH	12.15	Excised	14.0	8.80	
56	LH	12.35	12.60	14.90	11.20	1 week
	RH	12.40	Excised	14.90	11.25	
24	LH	13.80	13.80	15.80	10.20	2 weeks
	RH	13.80	Excised	15.80	10.20	
33	LH	14.10	14.25	16.30	11.0	2 weeks
	RH	14.10	Excised	16.25	10.95	
40	LH	12.15	12.10	13.50	9.15	2 weeks
	RH	12.30	Excised	13.50	9.15	
41	LH	13.15	13.20	15.50	10.90	2 weeks
	RH	13.15	Excised	15.50	10.90	
42	LH	13.20	13.25	15.40	10.75	3 weeks
	RH	13.25	Excised	15.40	10.65	
43	LH	14.30	14.30	16.40	11.10	3 weeks
	RH	14.35	Excised	16.35	11.0	
45	LH	14.25	14.30	Sectioned	10.70	1 month
	RH	14.25	Excised		10.65	
46	LH	13.10	13.20	Sectioned	10.50	1 month
	RH	13.25	Excised		10.40	
49	LH	14.80	14.70	16.80	11.20	2 months
	RH	14.80	Excised	16.80	11.20	
50	LH	14.40	14.40	16.70	10.90	2 months
	RH	14.70	Excised	16.60	10.80	
51	LH	15.40	15.20	17.50	11.30	3 months
	RH	15.60	Excised	17.45	11.30	

Femoral Length G.T. = Greater Trochanteric Measurement

Femoral Length F.H. = Femoral Head Measurement

APPENDIX 1 TABLE 13 (contd)

FEMORAL TIBIAL & METATARSAL LENGTH
(ON ANTEROPOSTERIOR RADIOGRAPHS) EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Femoral Length G.T. (cms)	Femoral Length F.H. (cms)	Tibial Length (cms)	Metatarsal Length (cms)	Postoperative Survival Time
52	LH	15.70	15.90	18.50	12.60	3 months
	RH	16.0	Excised	18.20	12.60	
38	LH	14.75	14.95	17.20	11.55	4 months
	RH	13.80	Excised	17.10	11.50	
54	LH	15.70	15.50	17.90	11.60	4 months
	RH	15.90	Excised	17.70	11.45	
29	LH	14.70	14.55	16.80	11.55	5 months
	RH	14.70	Excised	16.70	11.70	
36	LH	16.60	16.00	19.30	12.55	5 months
	RH	17.00	Excised	19.20	12.50	
37	LH	15.90	15.80	18.45	12.0	6 months
	RH	12.70	Excised	17.50	12.40	
39	LH	16.60	16.15	19.10	12.0	6 months
	RH	16.90	Excised	18.80	12.10	
55	LH	18.20	18.0	21.10	13.80	6 months
	RH	18.60	Excised	20.80	13.55	
17	LH	17.0	17.0	19.80	12.15	8 months
	RH	17.30	Excised	19.55	12.35	
18	LH	16.70	16.85	19.60	12.40	8 months
	RH	16.85	Excised	19.50	12.35	
14	LH	15.50	15.40	18.30	12.75	10 months
	RH	16.0	Excised	18.25	12.65	
15	LH	16.35	15.70	17.90	12.50	10 months
	RH	16.40	Excised	17.70	12.40	
12	LH	17.50	17.20	20.45	12.70	12 months
	RH	17.85	Excised	20.40	12.40	
13	LH	17.10	16.80	20.10	11.70	12 months
	RH	17.30	Excised	19.70	11.90	

APPENDIX 1 TABLE 14

HOCK AND STIFLE JOINT MOBILITY

CONTROL LAMBS

Lamb Number	Limb	Hock Full Fl*	Hock Full Ex*	Range of Hock Movement	Stifle Full Fl*	Stifle Full Ex*	Range of Stifle Movement	Experimental Survival Time
20	LH	25°	180°	155°	30°	145°	115°	1 week
	RH	25°	180°	155°	30°	145°	115°	
59	LH	25°	175°	150°	30°	145°	115°	1 week
	RH	25°	175°	150°	30°	145°	115°	
57	LH	30°	175°	145°	30°	150°	120°	2 weeks
	RH	30°	175°	145°	30°	150°	120°	
25	LH	20°	180°	160°	25°	140°	115°	2 weeks
	RH	20°	180°	160°	25°	140°	115°	
44	LH	35°	180°	145°	27°	135°	108°	3 weeks
	RH	35°	180°	145°	30°	135°	105°	
60	LH	25°	170°	145°	30°	150°	120°	1 month
	RH	25°	170°	145°	30°	150°	120°	
48	LH	30°	170°	140°	25°	130°	105°	2 months
	RH	30°	170°	140°	25°	130°	105°	
53	LH	40°	170°	130°	30°	135°	105°	3 months
	RH	40°	170°	130°	30°	130°	100°	
30	LH	45°	170°	125°	30°	130°	100°	4 months
	RH	45°	170°	125°	30°	130°	100°	
31	LH	30°	175°	145°	40°	140°	100°	5 months
	RH	30°	175°	145°	40°	140°	100°	
35	LH	35°	160°	125°	45°	140°	95°	6 months
	RH	35°	160°	125°	45°	140°	95°	
19	LH	35°	170°	135°	35°	145°	110°	8 months
	RH	35°	170°	135°	35°	145°	110°	
16	LH	25°	170°	145°	35°	140°	105°	10 months
	RH	25°	170°	145°	35°	140°	105°	
11	LH	40°	170°	130°	35°	140°	105°	12 months
	RH	40°	170°	130°	35°	140°	105°	

* Fl = Flexion
* Ex = Extension

APPENDIX 1 TABLE 15

HOCK AND STIFLE JOINT MOBILITY

EXPERIMENTAL LAMBS

Lamb Number	Limb	Hock Full Fl*	Hock Full Ex*	Range Of Hock Movement	Stifle Full Fl*	Stifle Full Ex*	Range of Stifle Movement	Experimental Survival Time
21	LH	25°	180°	155°	35°	145°	110°	1 week
	RH	25°	180°	155°	35°	145°	110°	
58	LH	27°	170°	143°	35°	145°	110°	1 week
	RH	25°	170°	145°	35°	145°	110°	
56	LH	30°	180°	150°	25°	140°	115°	1 week
	RH	30°	180°	150°	25°	140°	115°	
24	LH	20°	180°	160°	25°	140°	115°	2 weeks
	RH	20°	180°	160°	25°	140°	115°	
33	LH	25°	180°	155°	35°	140°	105°	2 weeks
	RH	25°	180°	155°	35°	140°	105°	
40	LH	35°	170°	135°	25°	140°	115°	2 weeks
	RH	35°	165°	130°	25°	140°	115°	
41	LH	30°	170°	140°	25°	140°	115°	2 weeks
	RH	30°	160°	130°	25°	140°	115°	
42	LH	30°	170°	140°	25°	140°	115°	3 weeks
	RH	30°	170°	140°	25°	140°	115°	
43	LH	35°	170°	135°	30°	130°	100°	3 weeks
	RH	35°	170°	135°	35°	135°	100°	
45	LH	33°	170°	137°	30°	140°	110°	1 month
	RH	32°	170°	138°	37°	140°	103°	
46	LH	25°	170°	145°	30°	140°	110°	1 month
	RH	25°	170°	145°	30°	140°	110°	
49	LH	30°	170°	140°	25°	130°	105°	2 months
	RH	30°	170°	140°	25°	135°	110°	
50	LH	25°	170°	145°	20°	140°	120°	2 months
	RH	20°	170°	150°	20°	145°	125°	
51	LH	25°	175°	150°	40°	140°	100°	3 months
	RH	25°	175°	150°	35°	140°	105°	

APPENDIX 1 TABLE 15 (contd)

HOCK AND STIFLE JOINT MOBILITY

EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Hock Full Fl*	Hock Full Ex*	Range of Hock Movement	Stifle Full Fl*	Stifle Full Ex*	Range of Stifle Movement	Postoperative Survival Time	
52	LH	45°	160°	115°	30°	130°	100°	3 months	
	RH	35°	160°	125°	30°	140°	110°		
38	LH	40°	170°	130°	30°	135°	105°	4 months	
	RH	30°	170°	140°	30°	140°	110°		
54	LH	40°	165°	125°	35°	145°	110°	4 months	R.F.M.
	RH	30°	165°	135°	35°	145°	110°		
29	LH	45°	170°	125°	35°	145°	110°	5 months	
	RH	30°	170°	140°	35°	145°	110°		
36	LH	50°	155°	105°	35°	150°	115°	5 months	
	RH	30°	170°	140°	35°	150°	115°		
37	LH	45°	160°	115°	35°	145°	110°	6 months	R.F.M.
	RH	55°	160°	105°	55°	160°	105°		
39	LH	85°	150°	65°	35°	145°	110°	6 months	R.F.M.
	RH	40°	165°	125°	50°	140°	90°		
55	LH	77°	157°	80°	33°	155°	122°	6 months	R.F.M.
	RH	25°	165°	140°	35°	150°	115°		
17	LH	50°	165°	115°	35°	145°	110°	8 months	R.F.M.
	RH	25°	170°	145°	35°	145°	110°		
18	LH	50°	130°	80°	25°	145°	120°	8 months	R.F.M.
	RH	30°	170°	140°	35°	145°	110°		
14	LH	70°	125°	55°	30°	145°	115°	10 months	R.F.M.
	RH	35°	170°	135°	35°	145°	115°		
15	LH	35°	170°	135°	35°	145°	110°	10 months	R.F.M.
	RH	25°	170°	145°	35°	145°	110°		
12	LH	85°	140°	55°	30°	140°	110°	12 months	R.F.M.
	RH	25°	170°	145°	30°	155°	125°		
13	LH	40°	170°	130°	30°	130°	100°	12 months	R.F.M.
	RH	25°	170°	145°	30°	145°	115°		

Fl* = Flexion

Ex* = Extension

R.F.M. = Reduced Fetlock Joint Mobility

APPENDIX 1 TABLE 16

TIBIAL PLATEAU ANGLE
(ON ANTEROPOSTERIOR RADIOGRAPHS)

CONTROL LAMBS

Lamb Number	Limb	Angle I	Angle II	Experimental Survival Time
20	LH	84.5°	86.5°	1 week
	RH	84.5°	87.0°	
59	LH	87.0°	90.0°	1 week
	RH	86.0°	90.5°	
57	LH	86.0°	88.5°	2 weeks
	RH	85.0°	89.0°	
25	LH	83.0°	86.5°	2 weeks
	RH	83.0°	88.0°	
44	LH	85.5°	87.5°	3 weeks
	RH	85.0°	87.0°	
60	LH	82.0°	87.5°	1 month
	RH	81.5°	89.0°	
48	LH	83.5°	87.5°	2 months
	RH	83.5°	87.0°	
53	LH	83.0°	85.0°	3 months
	RH	83.0°	85.0°	
30	LH	83.5°	87.0°	4 months
	RH	83.0°	87.0°	
31	LH	85.0°	86.5°	5 months
	RH	85.0°	87.0°	
35	LH	83.5°	86.0°	6 months
	RH	82.0°	85.5°	
19	LH	81.0°	87.5°	8 months
	RH	81.0°	86.0°	
16	LH	79.0°	86.0°	10 months
	RH	79.0°	85.0°	
11	LH	78.5°	84.5°	12 months
	RH	78.5°	84.5°	

Angle I = Angle to long axis made by growth plate/metaphysis junction
Angle II = Angle to long axis made by articular surfaces of condyles

APPENDIX 1 TABLE 17

TIBIAL PLATEAU ANGLE
(ON ANTEROPOSTERIOR RADIOGRAPHS)

EXPERIMENTAL LAMBS

Lamb Number	Limb	Angle I	Angle II.	Postoperative Survival Time
21	LH	82.5°	85.5°	1 week
	RH	83.5°	86.5°	
58	LH	86.5°	87.0°	1 week
	RH	85.5°	88.5°	
56	LH	83.0°	85.0°	1 week
	RH	82.0°	85.0°	
24	LH	83.5°	86.5°	2 weeks
	RH	84.0°	88.0°	
33	LH	82.5°	86.5°	2 weeks
	RH	83.5°	87.5°	
40	LH	82.0°	87.0°	2 weeks
	RH	82.0°	87.0°	
41	LH	83.0°	87.0°	2 weeks
	RH	84.0°	87.5°	
42	LH	83.0°	86.0°	3 weeks
	RH	84.5°	87.0°	
43	LH	81.0°	83.5°	3 weeks
	RH	81.5°	83.0°	
45	LH	Sectioned	-	1 month
	RH	Sectioned	-	
46	LH	Sectioned	-	1 month
	RH	Sectioned	-	
49	LH	85.0°	86.5°	2 months
	RH	86.0°	86.0°	
50	LH	77.5°	80.5°	2 months
	RH	82.0°	84.5°	
51	LH	80.0°	83.0°	3 months
	RH	83.0°	86.0°	
52	LH	79.0°	82.0°	3 months
	RH	83.0°	86.0°	

Angle I = Angle to long axis made by growth plate/metaphysis junction

Angle II = Angle to long axis made by articular surfaces of condyles

APPENDIX 1 TABLE 17 (contd)

TIBIAL PLATEAU ANGLE

(ON ANTEROPOSTERIOR RADIOGRAPHS)

EXPERIMENTAL LAMBS

Lamb Number	Limb	Angle I	Angle II	Postoperative Survival Time
38	LH	84.5°	85.5°	4 months
	RH	88.0°	88.0°	
54	LH	82.5°	87.5°	4 months
	RH	82.5°	86.0°	
29	LH	78.5°	82.0°	5 months
	RH	86.0°	89.0°	
36	LH	79.5°	82.5°	5 months
	RH	80.5°	83.5°	
37	LH	87.0°	88.5°	6 months
	RH	78.0°	81.0°	
39	LH	80.5°	83.5°	6 months
	RH	84.0°	87.5°	
55	LH	77.5°	82.0°	6 months
	RH	81.5°	86.0°	
17	LH	78.0°	82.5°	8 months
	RH	80.0°	84.5°	
18	LH	80.0°	82.0°	8 months
	RH	82.0°	87.0°	
14	LH	72.0°	77.0°	10 months
	RH	78.5°	83.0°	
15	LH	76.0°	85.0°	10 months
	RH	79.0°	88.5°	
12	LH	77.5°	80.5°	12 months
	RH	84.0°	88.5°	
13	LH	75.5°	82.0°	12 months
	RH	75.0°	83.0°	

Angle I = Angle to long axis made by growth plate/metaphysis junction

Angle II = Angle to long axis made by articular surfaces of condyles

APPENDIX 1 TABLE 18

TIBIAL PLATEAU ANGLE
(ON LATERAL RADIOGRAPHS) CONTROL LAMBS

Lamb Number	Limb	Tibial Plateau Angle	Experimental Survival Time
20	LH	73°	1 week
	RH	73°	
59	LH	73°	1 week
	RH	74°	
57	LH	-	2 weeks
	RH	76°	
25	LH	72°	2 weeks
	RH	72°	
44	LH	74°	3 weeks
	RH	76°	
60	LH	72°	1 month
	RH	73°	
48	LH	77°	2 months
	RH	77°	
53	LH	80°	3 months
	RH	80°	
30	LH	77°	4 months
	RH	77°	
31	LH	79°	5 months
	RH	78°	
35	LH	77°	6 months
	RH	77°	
19	LH	76°	8 months
	RH	76°	
16	LH	82°	10 months
	RH	82°	
11	LH	79°	12 months
	RH	77°	

APPENDIX 1 TABLE 19

TIBIAL PLATEAU ANGLE
(ON LATERAL RADIOGRAPHS) EXPERIMENTAL LAMBS

Lamb Number	Limb	Tibial Plateau Angle	Postoperative Survival Time
21	LH	73°	1 week
	RH	71°	
58	LH	73°	1 week
	RH	73°	
56	LH	72°	1 week
	RH	-	
24	LH	74°	2 weeks
	RH	73°	
33	LH	75°	2 weeks
	RH	75°	
40	LH	75°	2 weeks
	RH	75°	
41	LH	74°	2 weeks
	RH	74°	
42	LH	74°	3 weeks
	RH	74°	
43	LH	75°	3 weeks
	RH	75°	
45	LH	Sectioned	1 month
	RH	Sectioned	
46	LH	76°	1 month
	RH	76°	
49	LH	73°	2 months
	RH	75°	
50	LH	74°	2 months
	RH	74°	
51	LH	78°	3 months
	RH	78°	
52	LH	78°	3 months
	RH	77°	
38	LH	74°	4 months
	RH	79°	
54	LH	78°	4 months
	RH	79°	
29	LH	76°	5 months
	RH	77°	
36	LH	76°	5 months
	RH	78°	
37	LH	82°	6 months
	RH	-	
39	LH	-	6 months
	RH	77°	
55	LH	79°	6 months
	RH	78°	
17	LH	76°	8 months
	RH	76°	
18	LH	83°	8 months
	RH	78°	
14	LH	81°	10 months
	RH	78°	
15	LH	79°	10 months
	RH	75°	
12	LH	81°	12 months
	RH	-	
13	LH	79°	12 months
	RH	79°	

APPENDIX 1 TABLE 20

FEMORAL NECK ANGLE - CONTROL LAMBS

Lamb Number	Limb	Femoral Neck Angle	Experimental Survival Time
20	LH	57.0°	1 week
	RH	sectioned	
59	LH	62.0°	1 week
	RH	62.0°	
57	LH	68.0°	2 weeks
	RH	67.5°	
25	LH	64.5°	2 weeks
	RH	64.5°	
44	LH	66.5°	3 weeks
	RH	65.0°	
60	LH	59.0°	1 month
	RH	61.0°	
48	LH	66.5°	2 months
	RH	67.5°	
53	LH	64.0°	3 months
	RH	63.5°	
30	LH	61.0°	4 months
	RH	61.5°	
31	LH	66.5°	5 months
	RH	67.5°	
35	LH	63.0°	6 months
	RH	62.5°	
19	LH	61.0°	6 months
	RH	59.0°	
16	LH	60.0°	10 months
	RH	59.5°	
11	LH	68.0°	12 months
	RH	69.0°	

APPENDIX 1 TABLE 21

FEMORAL NECK ANGLE - EXPERIMENTAL LAMBS

Lamb Number	Limb	Femoral Neck Angle	Postoperative Survival Time
21	LH	59.0°	1 week
	RH	Excised	
58	LH	65.0°	1 week
	RH	Excised	
56	LH	70.0°	1 week
	RH	Excised	
24	LH	58.5°	2 weeks
	RH	Excised	
33	LH	60.5°	2 weeks
	RH	Excised	
40	LH	64.0°	2 weeks
	RH	Excised	
41	LH	63.0°	2 weeks
	RH	Excised	
42	LH	65.0°	3 weeks
	RH	Excised	
43	LH	63.0°	3 weeks
	RH	Excised	
45	LH	60.5°	1 month
	RH	Excised	
46	LH	64.5°	1 month
	RH	Excised	
49	LH	66.0°	2 months
	RH	Excised	
50	LH	62.5°	2 months
	RH	Excised	
51	LH	66.0°	3 months
	RH	Excised	
52	LH	61.5°	3 months
	RH	Excised	
38	LH	60.5°	4 months
	RH	Excised	
54	LH	62.0°	4 months
	RH	Excised	
29	LH	62.0°	5 months
	RH	Excised	
36	LH	67.0°	5 months
	RH	Excised	
37	LH	61.5°	6 months
	RH	Excised	
39	LH	64.5°	6 months
	RH	Excised	
55	LH	65.0°	6 months
	RH	Excised	
17	LH	57.0°	8 months
	RH	Excised	
18	LH	60.5°	8 months
	RH	Excised	
14	LH	65.0°	10 months
	RH	Excised	
15	LH	66.0°	10 months
	RH	Excised	
12	LH	65.5°	12 months
	RH	Excised	
13	LH	67.0°	12 months
	RH	Excised	

APPENDIX 1 TABLE 22

GREATER TROCHANTER BASE ANGLE
(ON ANTEROPOSTERIOR RADIOGRAPHS) CONTROL LAMBS

Lamb Number	Limb	Greater Trochanter Base Angle	Experimental Survival Time
20	LH	41.5°	1 week
	RH	Sectioned	
59	LH	43.0°	1 week
	RH	43.0°	
57	LH	50.0°	2 weeks
	RH	50.0°	
25	LH	47.0°	2 weeks
	RH	47.0°	
44	LH	51.0°	3 weeks
	RH	50.0°	
60	LH	42.5°	1 month
	RH	42.0°	
48	LH	49.5°	2 months
	RH	52.5°	
53	LH	48.0°	3 months
	RH	50.0°	
30	LH	43.0°	4 months
	RH	48.0°	
31	LH	50.0°	5 months
	RH	50.5°	
35	LH	42.0°	6 months
	RH	41.0°	
19	LH	43.5°	8 months
	RH	42.0°	
16	LH	43.0°	10 months
	RH	45.0°	
11	LH	53.0°	12 months
	RH	54.5°	

APPENDIX 1 TABLE 23

GREATER TROCHANTER BASE ANGLE
(ON ANTEROPOSTERIOR RADIOGRAPHS)

EXPERIMENTAL LAMBS

Lamb Number	Limb	Greater Trochanter Base Angle	Postoperative Survival Time
21	LH	43.5°	1 week
	RH	Excised	
58	LH	46.5°	1 week
	RH	Excised	
56	LH	55.0°	1 week
	RH	Excised	
24	LH	41.5°	2 weeks
	RH	Excised	
33	LH	44.5°	2 weeks
	RH	Excised	
40	LH	47.5°	2 weeks
	RH	Excised	
41	LH	51.0°	2 weeks
	RH	Excised	
42	LH	47.5°	3 weeks
	RH	Excised	
43	LH	51.5°	3 weeks
	RH	Excised	
45	LH	43.5°	1 month
	RH	Excised	
46	LH	48.5°	1 month
	RH	Excised	
49	LH	49.0°	2 months
	RH	Excised	
50	LH	48.0°	2 months
	RH	Excised	
51	LH	46.5°	3 months
	RH	Excised	
38	LH	44.0°	4 months
	RH	Excised	
54	LH	42.0°	4 months
	RH	Excised	
29	LH	43.0°	5 months
	RH	Excised	
36	LH	46.0°	5 months
	RH	Excised	
37	LH	42.0°	6 months
	RH	Excised	
39	LH	46.0°	6 months
	RH	Excised	
55	LH	41.5°	6 months
	RH	Excised	
17	LH	39.0°	8 months
	RH	Excised	
18	LH	43.0°	8 months
	RH	Excised	
14	LH	42.5°	10 months
	RH	Excised	
15	LH	51.0°	10 months
	RH	Excised	
12	LH	42.0°	12 months
	RH	Excised	
13	LH	50.0°	12 months
	RH	Excised	

APPENDIX 1 TABLE 24

RELATIVE CONTRIBUTION OF GROWTH PLATES - DIGBY'S METHOD

CONTROL LAMBS

Lamb Number	Limb	X - PAS = L1 cms	X - DAS = L2 cms	L1 + L2 (cms)	$\frac{L2}{L1 + L2} \times 100$	Experimental Survival time
20	LH	7.2	6.8	14.0	48.5%	1 week
	RH	7.4	6.5	13.9	46.7%	
59	LH	7.4	6.2	13.6	45.6%	1 week
	RH	7.5	6.1	13.6	44.8%	
57	LH	6.5	5.5	12.0	45.8%	2 weeks
	RH	6.8	5.3	12.1	43.8%	
25	LH	7.7	6.2	13.9	44.6%	2 weeks
	RH	7.2	6.6	13.8	47.8%	
44	LH	6.6	6.6	13.2	50.0%	3 weeks
	RH	6.7	6.5	13.2	49.2%	
60	LH	8.7	5.8	14.5	40.0%	1 month
	RH	8.0	6.5	14.5	44.9%	
48	LH	7.0	7.6	14.6	52.0%	2 months
	RH	7.3	7.3	14.6	50.0%	
53	LH	8.6	8.8	17.4	50.6%	3 months
	RH	8.9	8.7	17.6	49.4%	
30	LH	8.7	8.1	16.8	48.2%	4 months
	RH	8.1	8.7	16.8	51.8%	
31	LH	8.8	8.2	17.0	48.2%	5 months
	RH	9.0	8.0	17.0	47.1%	
35	LH	8.7	9.0	17.7	50.8%	6 months
	RH	9.1	8.5	17.6	48.3%	
19	LH	9.2	9.2	18.4	50.0%	8 months
	RH	8.5	9.7	18.2	53.3%	
16	LH	9.7	8.2	17.9	45.8%	10 months
	RH	10.2	7.8	18.0	43.3%	
11	LH	11.3	9.8	21.1	46.4%	12 months
	RH	11.4	9.4	20.8	45.2%	

x = Reference Point (see fig 9)

PAS = Proximal Articular Surface

DAS = Distal Articular Surface

APPENDIX 1 TABLE 25

RELATIVE CONTRIBUTION OF GROWTH PLATES - DIGBY'S METHOD

EXPERIMENTAL LAMBS

Lamb Number	Limb	X - PAS = L1 (cms)	X - DAS = L2 (cms)	L1 + L2 (cms)	$\frac{L2}{L1 + L2} \times 100$	Post Operative Survival Time
21	LH	7.7	7.2	14.9	48.3%	1 week
	RH	7.3	7.6	14.9	51.0%	
58	LH	6.8	6.4	13.2	48.5%	1 week
	RH	6.2	6.9	13.1	52.7%	
56	LH	6.9	7.2	14.1	51.0%	1 week
	RH	7.4	6.7	14.1	47.5%	
24	LH	8.1	7.0	15.1	46.3%	2 weeks
	RH	8.2	6.8	15.0	45.3%	
33	LH	8.2	7.4	15.6	47.4%	2 weeks
	RH	8.0	7.6	15.6	48.7%	
40	LH	6.8	6.2	13.0	47.7%	2 weeks
	RH	7.0	5.9	12.9	45.7%	
41	LH	7.5	7.3	14.8	49.3%	2 weeks
	RH	7.8	6.9	14.7	46.9%	
42	LH	7.8	7.0	14.8	47.3%	3 weeks
	RH	7.9	6.7	14.6	45.9%	
43	LH	7.8	7.9	15.7	50.3%	3 weeks
	RH	7.7	8.0	15.7	50.9%	
45	LH	-	-	-	-	1 month
	RH	-	-	-	-	
46	LH	-	-	-	-	1 month
	RH	-	-	-	-	
49	LH	8.1	7.8	15.9	49.0%	2 months
	RH	7.8	8.1	15.9	50.9%	
50	LH	7.6	8.0	15.6	51.3%	2 months
	RH	7.6	7.9	15.5	50.9%	
51	LH	8.5	8.1	16.6	48.8%	3 months
	RH	8.5	8.0	16.5	48.5%	
52	LH	8.7	8.7	17.4	50.0%	3 months
	RH	8.6	8.7	17.3	50.3%	
38	LH	10.0	6.4	16.4	39.0%	4 months
	RH	8.5	7.6	16.1	47.2%	

APPENDIX 1 TABLE 25 (contd)

RELATIVE CONTRIBUTION OF GROWTH PLATES - DIGBY'S METHOD (Cont)

EXPERIMENTAL LAMBS

Lamb Number	Limb	X - PAS = L1 cms	X - DAS = L2 cms	L1 + L2 (cms)	$\frac{L2}{L1 + L2} \times 100$	Post Operative Survival Time
54	LH	8.8	7.9	16.7	47.3%	4 months
	RH	9.3	7.2	16.5	43.6%	
29	LH	8.8	6.9	15.7	43.9%	5 months
	RH	9.1	6.7	15.8	42.4%	
36	LH	9.75	8.5	18.2	46.7%	5 months
	RH	11.2	7.0	18.2	38.5%	
37	LH	9.5	8.0	17.5	45.7%	6 months
	RH	8.7	8.4	17.1	49.1%	
39	LH	8.6	9.1	17.7	51.4%	6 months
	RH	9.9	8.0	17.9	44.7%	
55	LH	10.7.	9.2	19.9	46.2%	6 months
	RH	11.5	8.0	19.5	41.0%	
17	LH	10.4	8.1	18.5	43.8%	8 months
	RH	9.8	8.8	18.6	47.3%	
18	LH	13.7	4.7	18.4	25.5%	8 months
	RH	9.6	8.7	18.3	47.5%	
14	LH	10.2	6.8	17.0	40.0%	10 months
	RH	7.5	9.8	17.3	56.6%	
15	LH	9.6	7.4	17.0	43.5%	10 months
	RH	9.3	7.5	16.8	44.6%	
12	LH	11.7	7.8	19.5	40.0%	12 months
	RH	11.0	8.4	19.4	43.3%	
13	LH	10.7	8.1	18.8	43.1%	12 months
	RH	9.9	8.6	18.5	46.5%	

x = Reference Point (see fig 9)

PAS = Proximal Articular Surface

DAS = Distal Articular Surface

ANGLE OF FOVEA CAPITIS RELATIVE TO FEMORAL CONDYLAR PLATEAU

CONTROL LAMBS

Lamb Number	Limb	Angle of Fovea Capitis to Femoral Condylar Plateau	Experimental Survival Time
20	LH	13.0°	1 week
	RH	13.0°	
59	LH	8.0°	1 week
	RH	8.5°	
57	LH	11.5°	2 weeks
	RH	11.0°	
25	LH	15.0°	2 weeks
	RH	16.0°	
44	LH	14.0°	3 weeks
	RH	13.0°	
60	LH	10.0°	1 month
	RH	9.5°	
48	LH	12.0°	2 months
	RH	11.5°	
53	LH	10.5°	3 months
	RH	11.5°	
30	LH	11.5°	4 months
	RH	11.0°	
31	LH	11.0°	5 months
	RH	11.0°	
35	LH	15.0°	6 months
	RH	15.5°	
19	LH	13.0°	8 months
	RH	12.0°	
16	LH	14.0°	10 months
	RH	14.0°	
11	LH	14.0°	12 months
	RH	15.0°	

APPENDIX 1 TABLE 27

ANGLE OF FOVEA CAPITIS RELATIVE TO FEMORAL CONDYLAR PLATEAU

EXPERIMENTAL LAMBS

Lamb Number	Limb	Angle of Fovea Capitis to Femoral Condylar Plateau	Postoperative Survival Time
21	LH	13.5°	1 week
	RH	Excised	
58	LH	12.0°	1 week
	RH	Excised	
56	LH	10.0°	1 week
	RH	Excised	
24	LH	12.0°	2 weeks
	RH	Excised	
33	LH	12.0°	2 weeks
	RH	Excised	
40	LH	15.0°	2 weeks
	RH	Excised	
41	LH	9.0°	2 weeks
	RH	Excised	
42	LH	10.0°	3 weeks
	RH	Excised	
43	LH	10.0°	3 weeks
	RH	Excised	
45	LH	13.0°	1 month
	RH	Excised	
46	LH	10.0°	1 month
	RH	Excised	
49	LH	10.5°	2 month
	RH	Excised	
50	LH	11.0°	2 months
	RH	Excised	
51	LH	13.0°	3 months
	RH	Excised	
52	LH	11.0°	3 months
	RH	Excised	

ANGLE OF FOVEA CAPITIS RELATIVE TO FEMORAL CONDYLAR PLATEAU

EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Angle of Fovea Capitis to Femoral Condylar Plateau	Postoperative Survival Time
38	LH	15.0°	4 months
	RH	Excised	
54	LH	13.0°	4 months
	RH	Excised	
29	LH	14.0°	5 months
	RH	Excised	
36	LH	12.0°	5 months
	RH	Excised	
37	LH	15.0°	6 months
	RH	Excised	
39	LH	16.5°	6 months
	RH	Excised	
55	LH	15.0°	6 months
	RH	Excised	
17	LH	13.0°	8 months
	RH	Excised	
18	LH	13.0°	8 months
	RH	Excised	
14	LH	15.0°	10 months
	RH	Excised	
15	LH	16.0°	10 months
	RH	Excised	
12	LH	10.5°	12 months
	RH	Excised	
13	LH	14.0°	12 months
	RH	Excised	

APPENDIX 1 TABLE 28

FEMORAL ANTEVERSION ANGLE - CONTROL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Experimental Survival Time
20	LH	26.50°	26.0°	26.0°	26.0°	1 week
	RH	Sectioned	-	-	-	
59	LH	23.5°	23.0°	23.0°	23.0°	1 week
	RH	22.5°	22.0°	22.0°	22.0°	
57	LH	23.5°	23.5°	23.5°	23.5°	2 weeks
	RH	22.0°	22.0°	21.5°	22.0°	
25	LH	22.5°	22.0°	22.0°	22.0°	2 weeks
	RH	21.5°	21.5°	21.5°	21.5°	
44	LH	26.0°	26.5°	26.0°	26.0°	3 weeks
	RH	25.5°	25.5°	25.5°	25.5°	
60	LH	23.0°	21.5°	21.0°	22.0°	1 month
	RH	22.5°	21.0°	21.5°	21.5°	
48	LH	21.50°	21.50°	21.50°	21.50°	2 months
	RH	21.50°	21.50°	21.75°	21.50°	
53	LH	22.0°	21.5°	22.5°	22.0°	3 months
	RH	22.0°	21.5°	22.5°	22.0°	
30	LH	25.5°	24.5°	26.5°	25.5°	4 months
	RH	22.5°	22.5°	22.5°	22.5°	
31	LH	18.5°	18.0°	18.0°	18.0°	5 months
	RH	18.0°	17.75°	18.0°	18.0°	
35	LH	19.5°	19.5°	20.5°	19.5°	6 months
	RH	18.5°	19.5°	19.5°	19.5°	
19	LH	22.25°	22.50°	23.0°	22.50°	8 months
	RH	20.50°	20.50°	21.50°	20.50°	
16	LH	22.0°	22.0°	22.0°	22.0°	10 months
	RH	21.0°	21.0°	21.0°	21.0°	
11	LH	24.5°	23.5°	24.0°	24.0°	12 months
	RH	23.5°	23.5°	24.0°	23.5°	

APPENDIX 1 TABLE 29

FEMORAL ANTEVERSION ANGLE - EXPERIMENTAL LAMBS

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
21	LH	20.5°	19.5°	20.0°	20.0°	1 week
	RH	Excised	-	-	-	
58	LH	30.0°	28.0°	28.0°	28.5°	1 week
	RH	Excised	-	-	-	
56	LH	27.0°	26.25°	27.50°	27.0°	1 week
	RH	Excised	-	-	-	
24	LH	25.0°	25.0°	25.0°	25.0°	2 weeks
	RH	Excised	-	-	-	
33	LH	23.0°	23.0°	23.0°	23.0°	2 weeks
	RH	Excised	-	-	-	
40	LH	25.0°	24.5°	25.0°	25.0°	2 weeks
	RH	Excised	-	-	-	
41	LH	25.25°	26.0°	25.25°	25.5°	2 weeks
	RH	Excised	-	-	-	
42	LH	24.5°	23.5°	24.5°	24.5°	3 weeks
	RH	Excised	-	-	-	
43	LH	18.0°	17.5°	17.25°	17.5°	3 weeks
	RH	Excised	-	-	-	
45	LH	17.25°	16.75°	17.5°	17.0°	1 month
	RH	Excised	-	-	-	
46	LH	28.0°	27.75°	27.0°	27.5°	1 month
	RH	Excised	-	-	-	
49	LH	14.0°	14.0°	14.0°	14.0°	2 months
	RH	Excised	-	-	-	
50	LH	19.0°	19.0°	19.0°	19.0°	2 months
	RH	Excised	-	-	-	
51	LH	24.0°	24.0°	23.5°	24.0°	3 months
	RH	Excised	-	-	-	
52	LH	16.0°	16.0°	15.0°	16.0°	3 months
	RH	Excised	-	-	-	
38	LH	22.5°	22.5°	22.5°	22.5°	4 months
	RH	Excised	-	-	-	
54	LH	25.0°	25.0°	25.0°	25.0°	4 months
	RH	Excised	-	-	-	

APPENDIX 1 TABLE 29 (contd)

FEMORAL ANTEVERSION ANGLE - EXPERIMENTAL LAMBS (Cont)

Lamb Number	Limb	Measurement I	Measurement II	Measurement III	Average of I-III	Postoperative Survival Time
29	LH	16.0°	15.5°	16.0°	16.0°	5 months
	RH	Excised	-	-	-	
36	LH	18.5°	18.0°	17.75°	18.0°	5 months
	RH	Excised	-	-	-	
37	LH	27.0°	27.5°	27.5°	27.5°	6 months
	RH	Excised	-	-	-	
39	LH	25.0°	25.0°	25.0°	25.0°	6 months
	RH	Excised	-	-	-	
55	LH	20.0°	21.5°	21.0°	21.0°	6 months
	RH	Excised	-	-	-	
17	LH	18.5°	19.5°	18.5°	18.5°	8 months
	RH	Excised	-	-	-	
18	LH	20.5°	20.5°	19.5°	20.5°	8 months
	RH	Excised	-	-	-	
14	LH	19.0°	19.0°	20.0°	19.0°	10 months
	RH	Excised	-	-	-	
15	LH	23.0°	22.0°	21.5°	22.0°	10 months
	RH	Excised	-	-	-	
12	LH	29.5°	31.0°	30.5°	30.5°	12 months
	RH	Excised	-	-	-	
13	LH	20.0°	20.0°	20.0°	20.0°	12 months
	RH	Excised	-	-	-	

APPENDIX 1 TABLE 30

EVALUATION OF LIMB TORSION - CONTROL LAMBS

Lamb Number	Limb	Metatarsal Torsion	Tibial Torsion	Total Internal Torsion between stifle & fetlock joints	Anteversion Angle	Experimental Survival Time
20	LH	2.0 (I)	5.0 (I)	7.0	26.0	1 week
	RH	2.0 (I)	-	-	-	
59	LH	3.0 (I)	11.0 (I)	14.0	23.0	1 week
	RH	3.0 (I)	9.0 (I)	12.0	22.0	
57	LH	2.0 (I)	7.0 (I)	9.0	23.5	2 weeks
	RH	2.0 (I)	8.0 (I)	10.0	22.0	
25	LH	2.0 (I)	7.0 (I)	9.0	22.0	2 weeks
	RH	1.0 (I)	5.0 (I)	6.0	21.5	
44	LH	2.0 (I)	-	-	26.0	3 weeks
	RH	2.0 (I)	-	-	25.5	
60	LH	2.0 (I)	10.0 (I)	12.0	22.0	1 month
	RH	2.0 (I)	8.0 (I)	10.0	21.5	
48	LH	2.0 (I)	7.0 (I)	9.0	21.5	2 months
	RH	2.0 (I)	8.0 (I)	10.0	21.5	
53	LH	2.0 (I)	10.0	12.0	22.0	3 months
	RH	3.0 (I)	12.0	15.0	22.0	
30	LH	2.0 (I)	12.0 (I)	14.0	25.5	4 months
	RH	2.0 (I)	9.0 (I)	11.0	22.5	
31	LH	3.0 (I)	11.0 (I)	14.0	18.0	5 months
	RH	4.0 (I)	9.0 (I)	13.0	18.0	
35	LH	2.0 (I)	18.0 (I)	20.0	19.5	6 months
	RH	2.0 (I)	16.0 (I)	18.0	19.5	
19	LH	2.0 (I)	18.0 (I)	20.0	22.50	8 months
	RH	2.0 (I)	17.0 (I)	19.0	20.50	
16	LH	2.0 (I)	17.0 (I)	19.0	22.0	10 months
	RH	2.0 (I)	17.0 (I)	19.0	21.0	
11	LH	5.0 (I)	18.0 (I)	23.0	24.0	12 months
	RH	5.0 (I)	17.0 (I)	22.0	23.5	

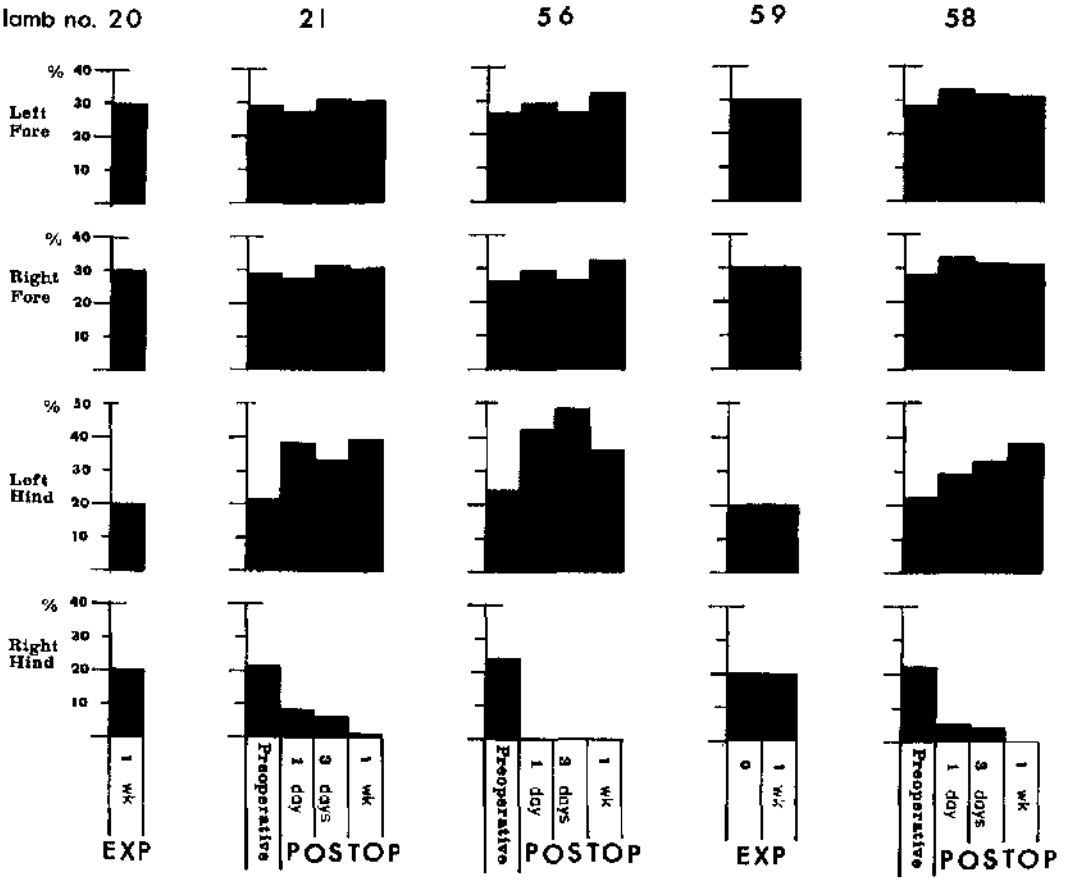
(I) = Internal Torsion

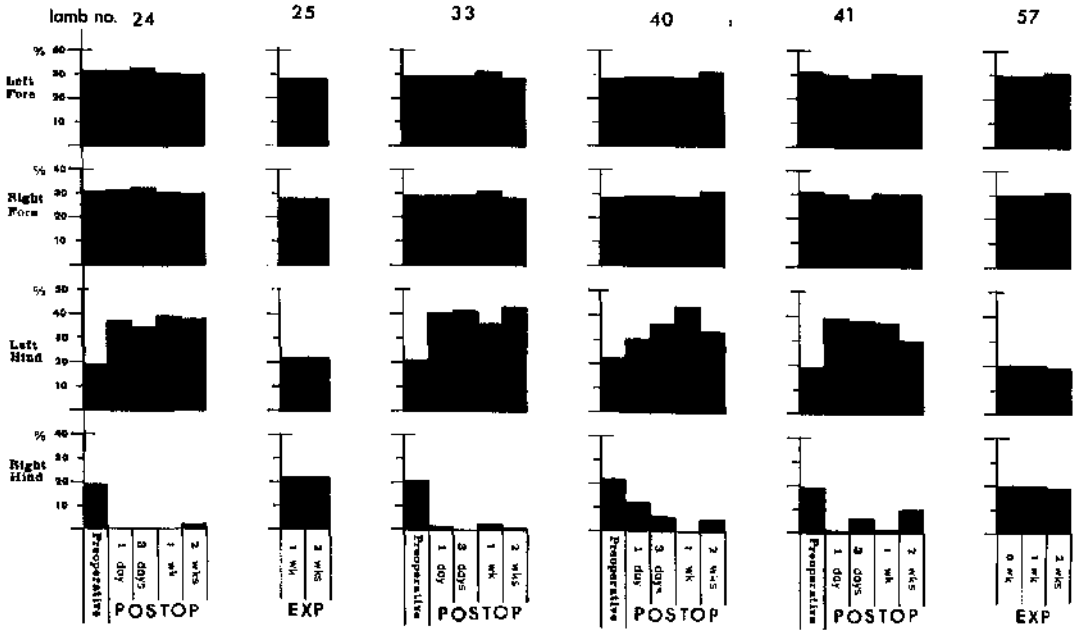
APPENDIX 1 TABLE 31

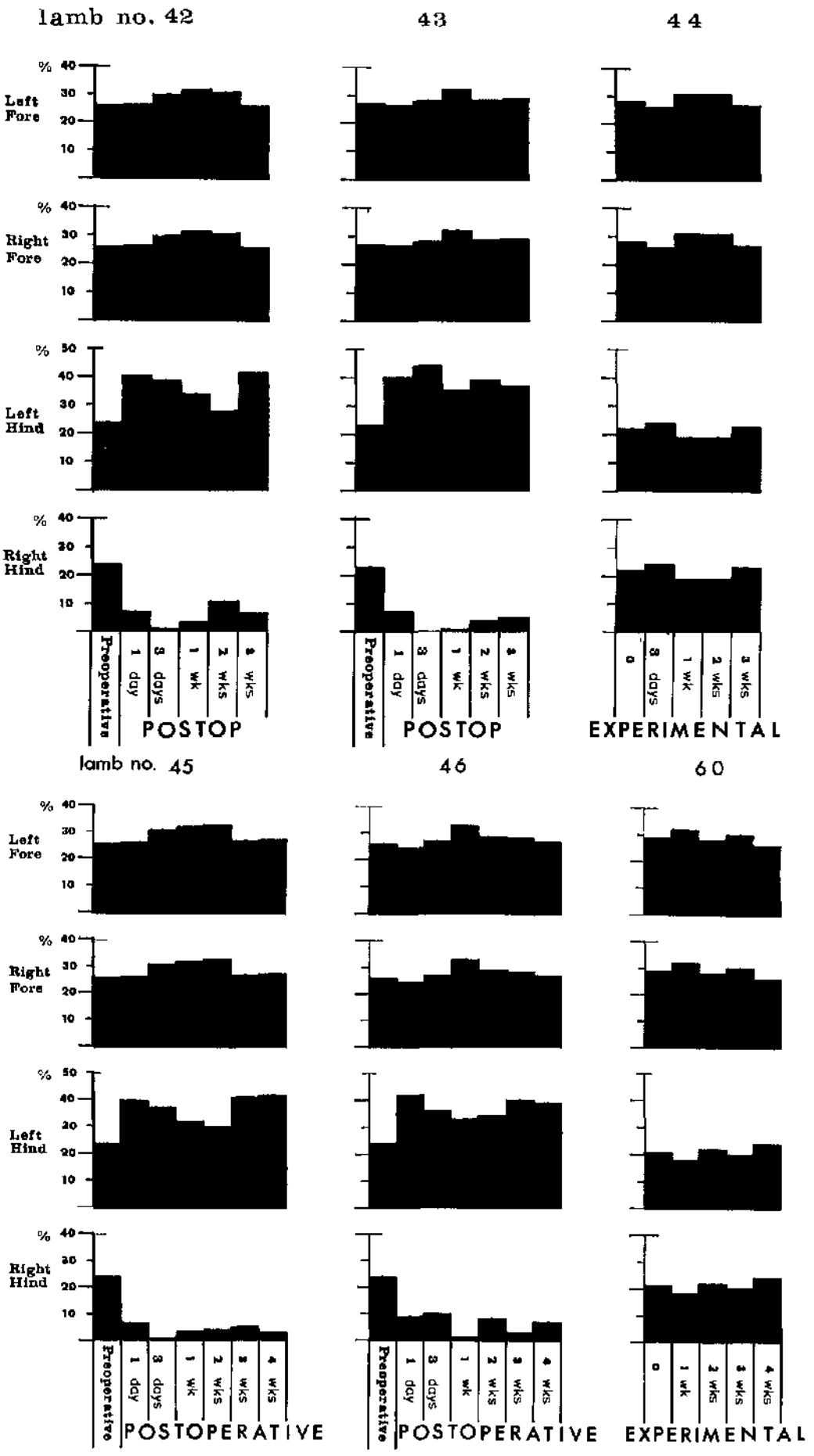
EVALUATION OF LIMB TORSION - EXPERIMENTAL LAMBS

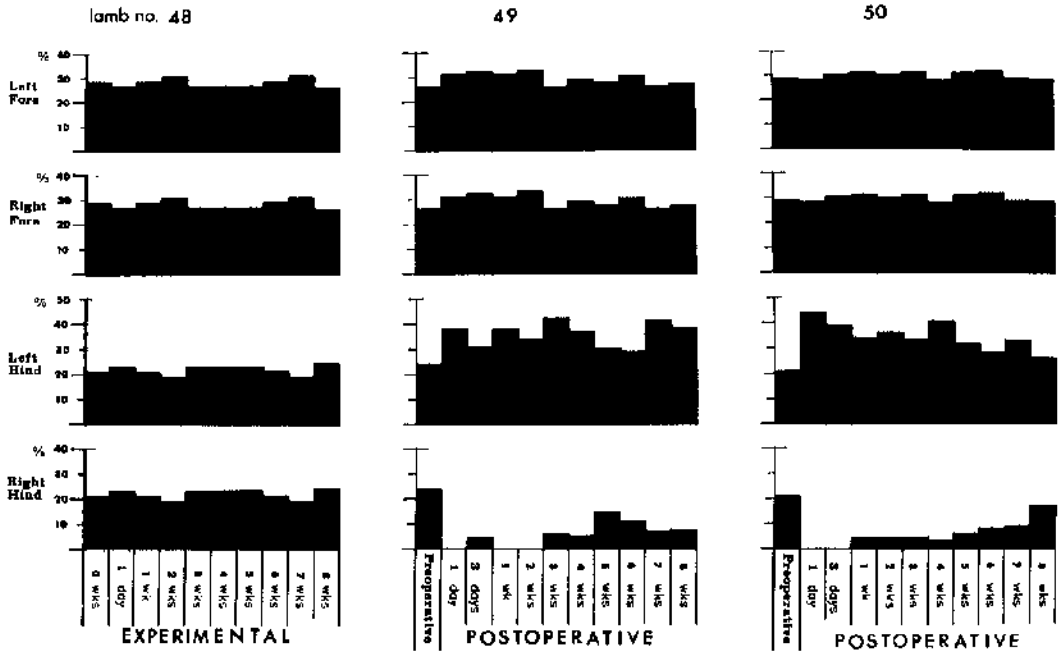
Lamb Number	Metatarsal Torsion	Tibial Torsion	Total Internal Torsion between stifle & fetlock joint	Anteversion Angle	Post Operative Survival Time
21	2.0 (I)	9.0 (I)	11.0	20.0	1 week
58	3.0 (I)	10.0 (I)	13.0	28.5	1 week
56	2.0 (I)	-	-	27.0	1 week
24	1.0 (I)	16.0 (I)	17.0	25.0	2 weeks
33	2.0 (I)	12.0 (I)	14.0	23.0	2 weeks
40	3.0 (I)	-	-	25.0	2 weeks
41	4.0 (I)	-	-	25.5	2 weeks
42	2.0 (I)	-	-	24.5	3 weeks
43	2.0 (I)	-	-	17.5	3 weeks
45	6.0 (I)	-	-	17.0	1 month
46	3.0 (I)	-	-	27.5	1 month
49	8.0 (I)	15.0 (I)	23.0	14.0	2 months
50	3.0 (I)	10.0 (I)	13.0	19.0	2 months
51	14.0 (I)	14.0 (I)	28.0	24.0	3 months
52	13.0 (I)	18.0 (I)	31.0	16.0	3 months
38	2.0 (I)	17.0 (I)	19.0	22.5	4 months
54	13.0 (I)	15.0 (I)	28.0	25.0	4 months
29	18.0 (I)	15.0 (I)	33.0	16.0	5 months
36	14.0 (I)	37.0 (I)	51.0	18.0	5 months
37	5.0 (I)	23.0 (I)	28.0	27.5	6 months
39	18.0 (I)	29.0 (I)	47.0	25.0	6 months
55	18.0 (I)	25.0 (I)	43.0	21.0	6 months
17	14.0 (I)	16.0 (I)	30.0	18.5	8 months
18	7.0 (I)	47.0 (I)	54.0	20.5	8 months
14	21.0 (I)	30.0 (I)	51.0	19.0	10 months
15	9.0 (I)	18.0 (I)	27.0	22.0	10 months
12	28.0 (I)	49.9 (I)	77.0	30.5	12 months
13	5.0 (I)	11.0 (I)	16.0	20.0	12 months

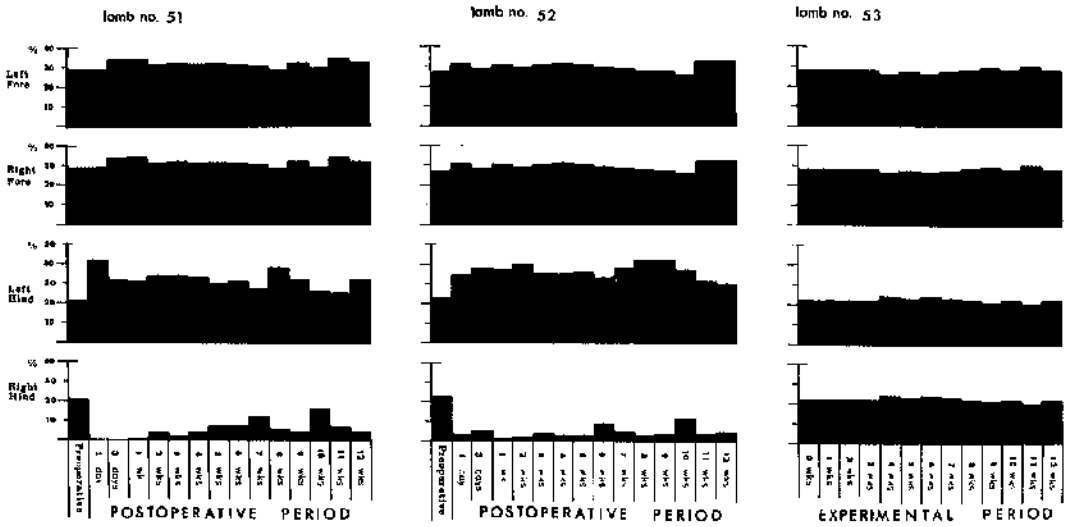
(I) = Internal Torsion


lamb no. 20
21
56
59
58
%
Left Fore
Right Fore
Left Hind
Right Hind
40
30
20
10
50
1 wk
Preoperative
1 day
3 days
1 wk
0
EXP
POSTOP

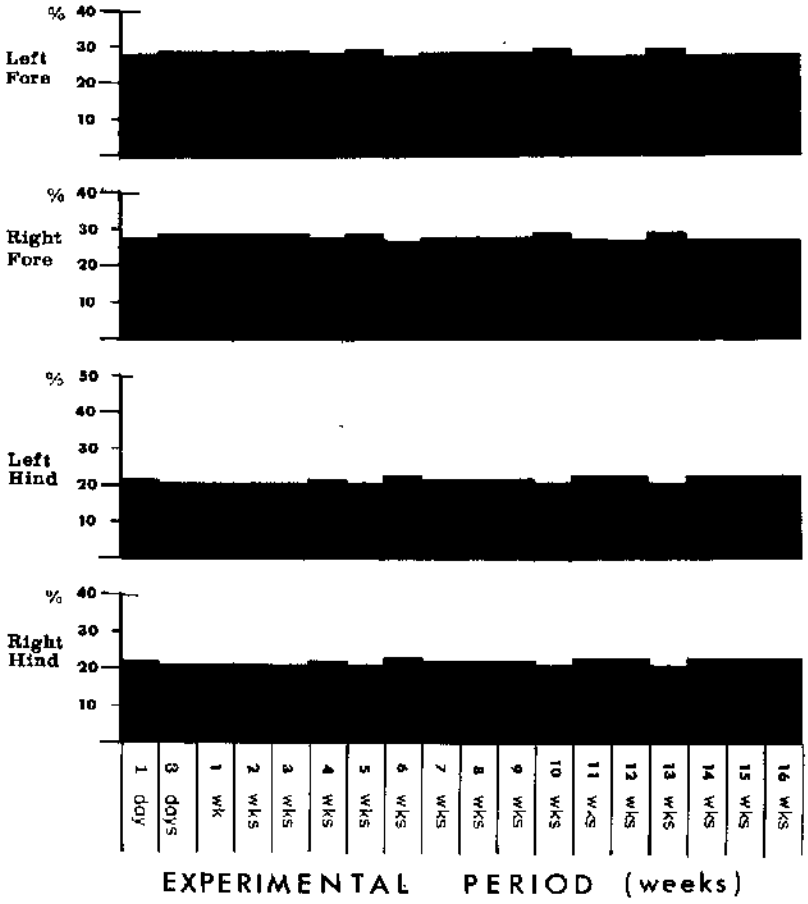

lamb no. 24
25
33
40
41
57
%
Left Fore
Right Fore
Left Hind
Right Hind
Preoperative
1 day
3 days
1 wk
2 wks
EXP
POSTOP


lamb no. 42
43
44
%
Left Fore
Right Fore
Left Hind
Right Hind
Preoperative
1 day
3 days
1 wk
2 wks
3 wks
POSTOP
0
EXPERIMENTAL
lamb no. 45
46
60
4 wks
POSTOPERATIVE

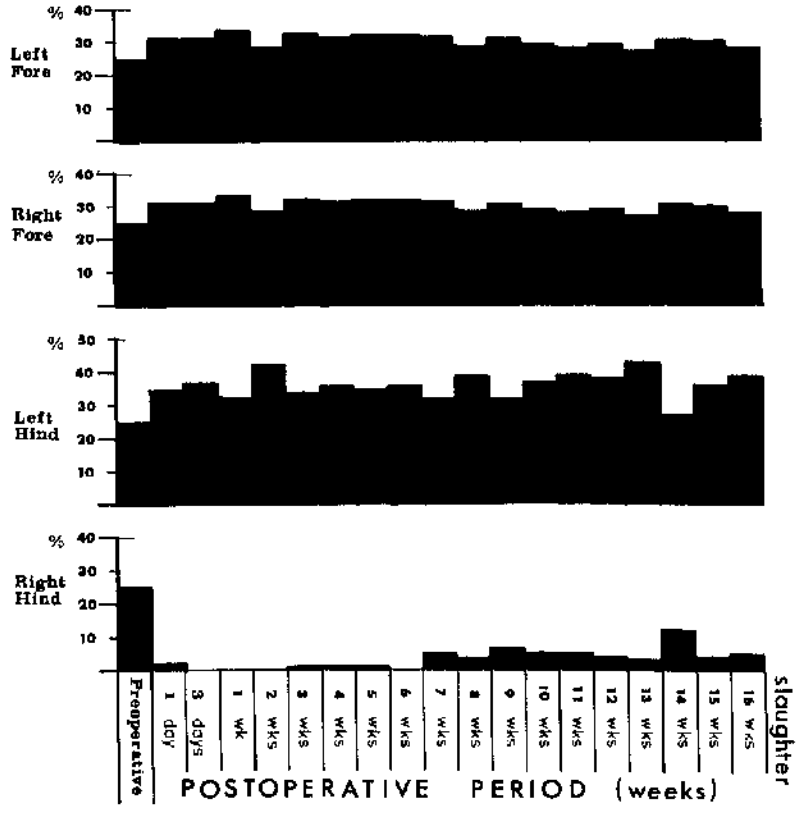

lamb no. 48
49
50
Left Fore
Right Fore
Left Hind
Right Hind
%
EXPERIMENTAL
POSTOPERATIVE
POSTOPERATIVE

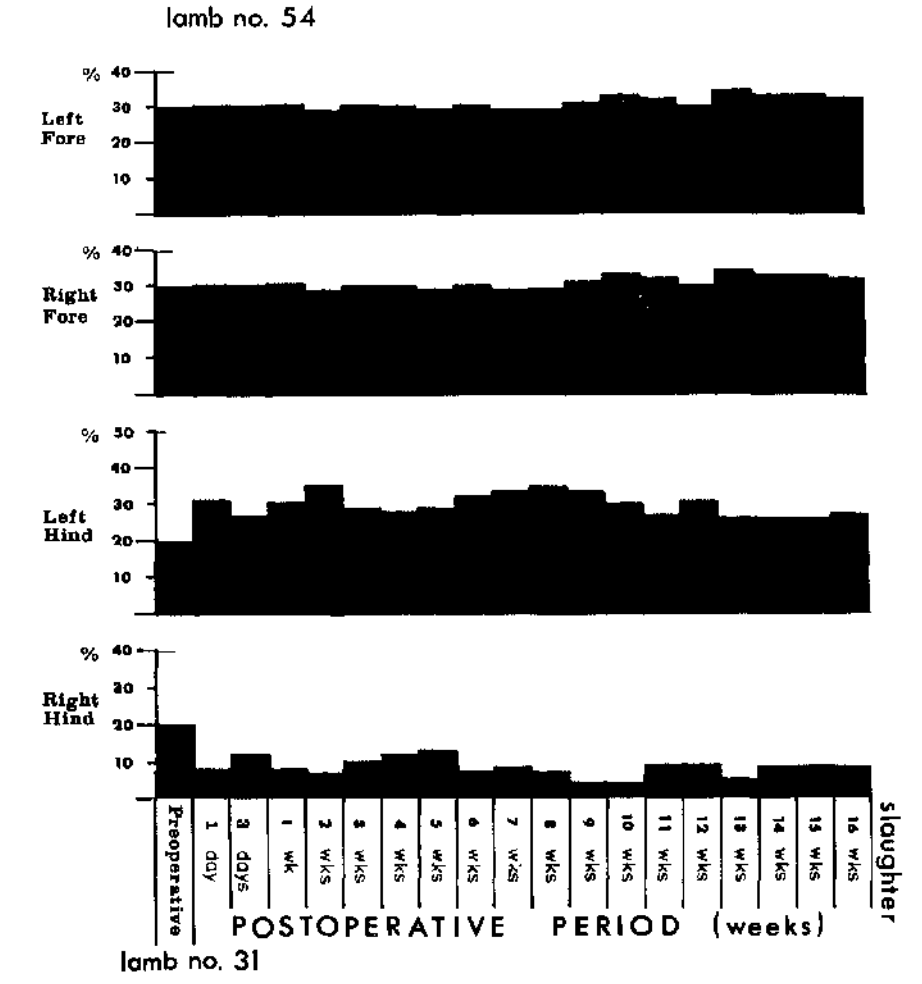

lamb no. 51
lamb no. 52
lamb no. 53
Left Fore
Right Fore
Left Hind
Right Hind
%
POSTOPERATIVE PERIOD
POSTOPERATIVE PERIOD
EXPERIMENTAL PERIOD

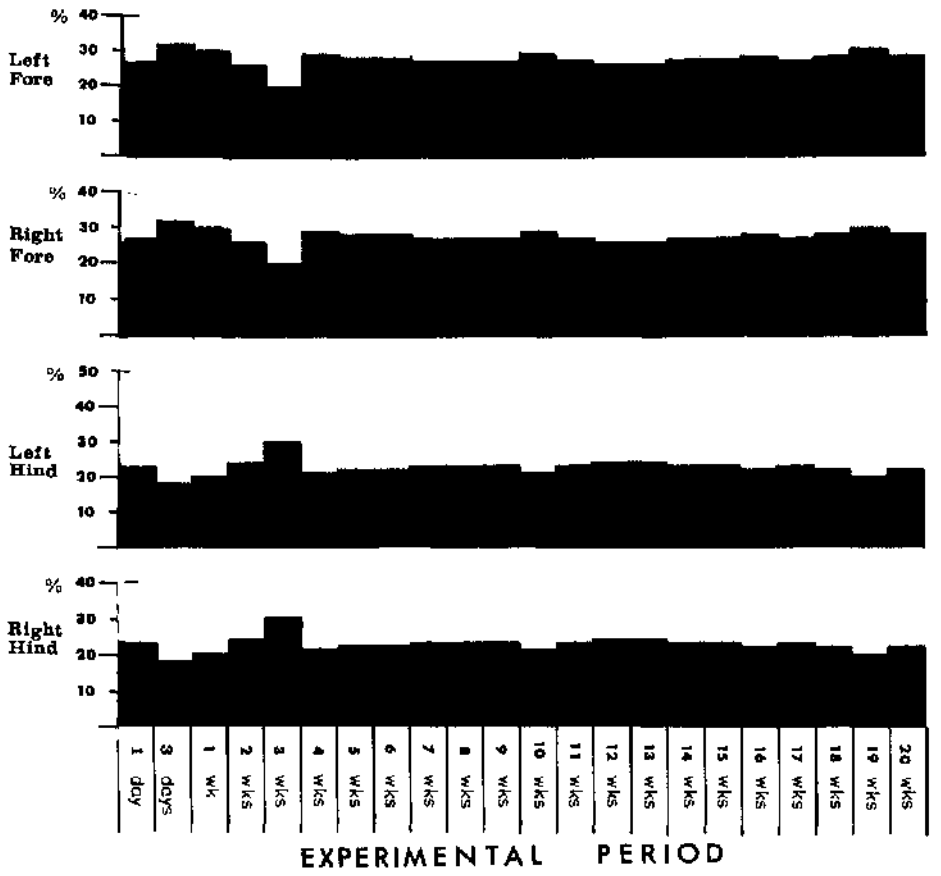
lamb no. 30



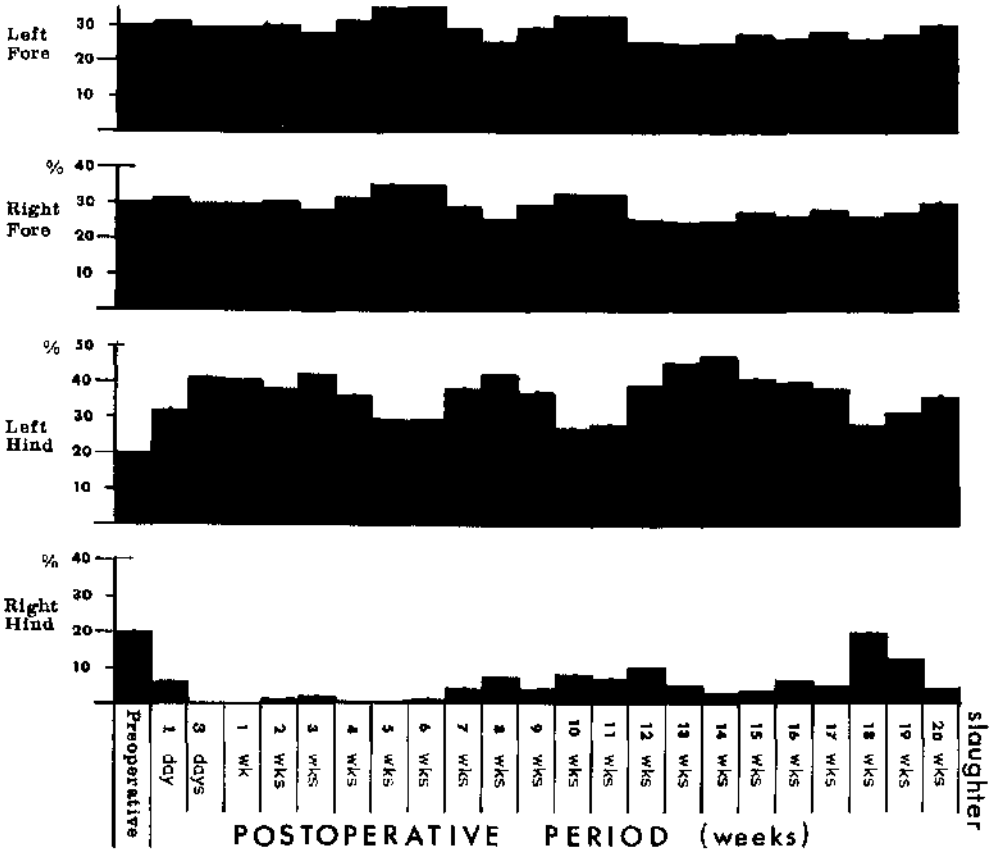
lamb no. 38



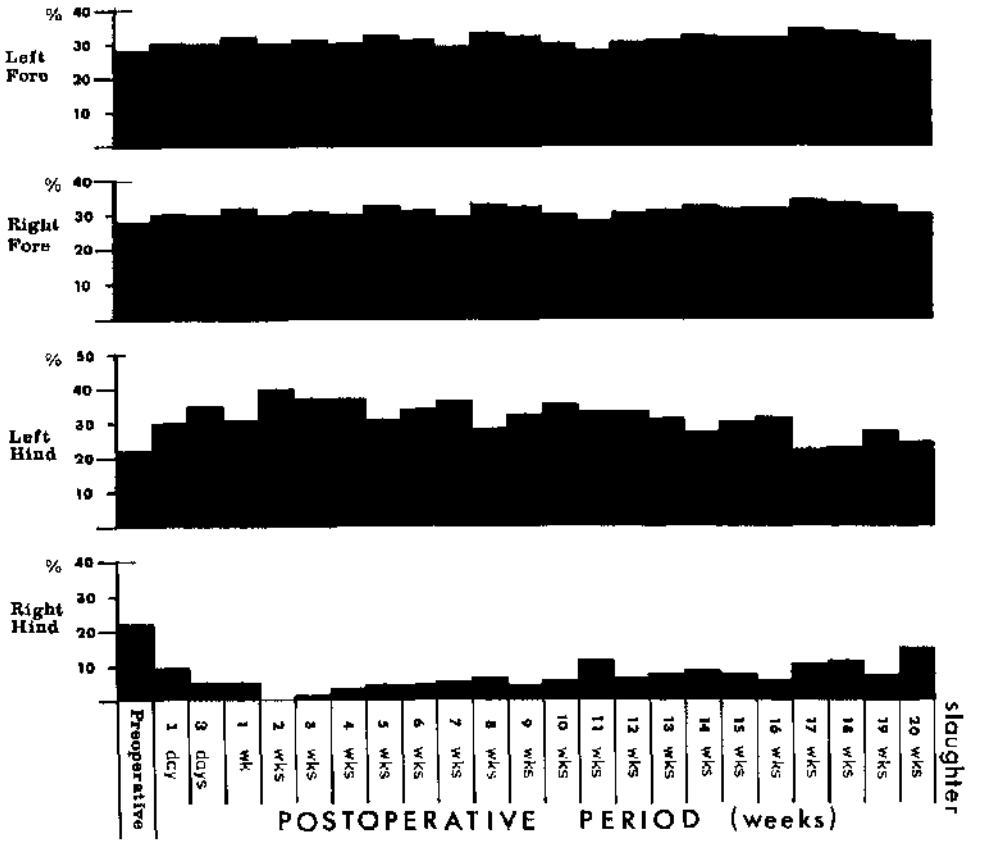

lamb no. 54
%
Left Fore
Right Fore
Left Hind
Right Hind
Preoperative
1 day
3 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
slaughter
POSTOPERATIVE PERIOD (weeks)


lamb no. 31
%
Left Fore
Right Fore
Left Hind
Right Hind
1 day
3 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
EXPERIMENTAL PERIOD

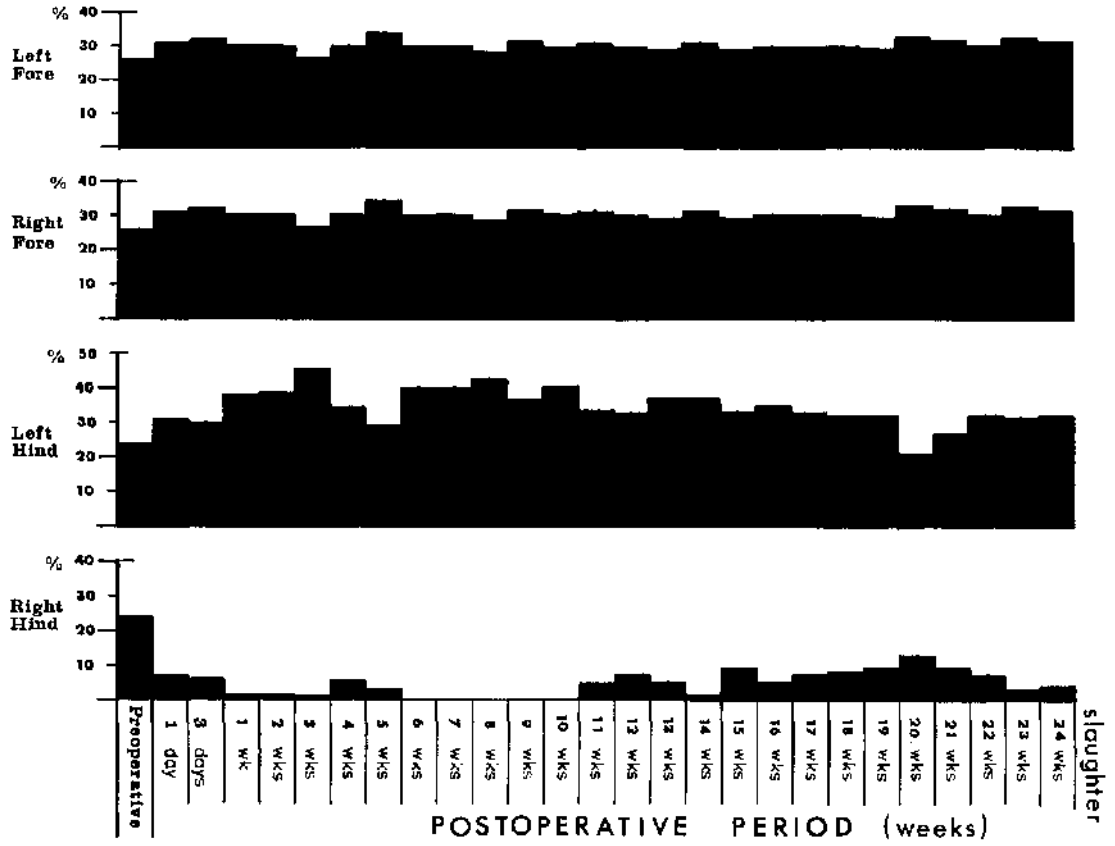
lamb no. 29



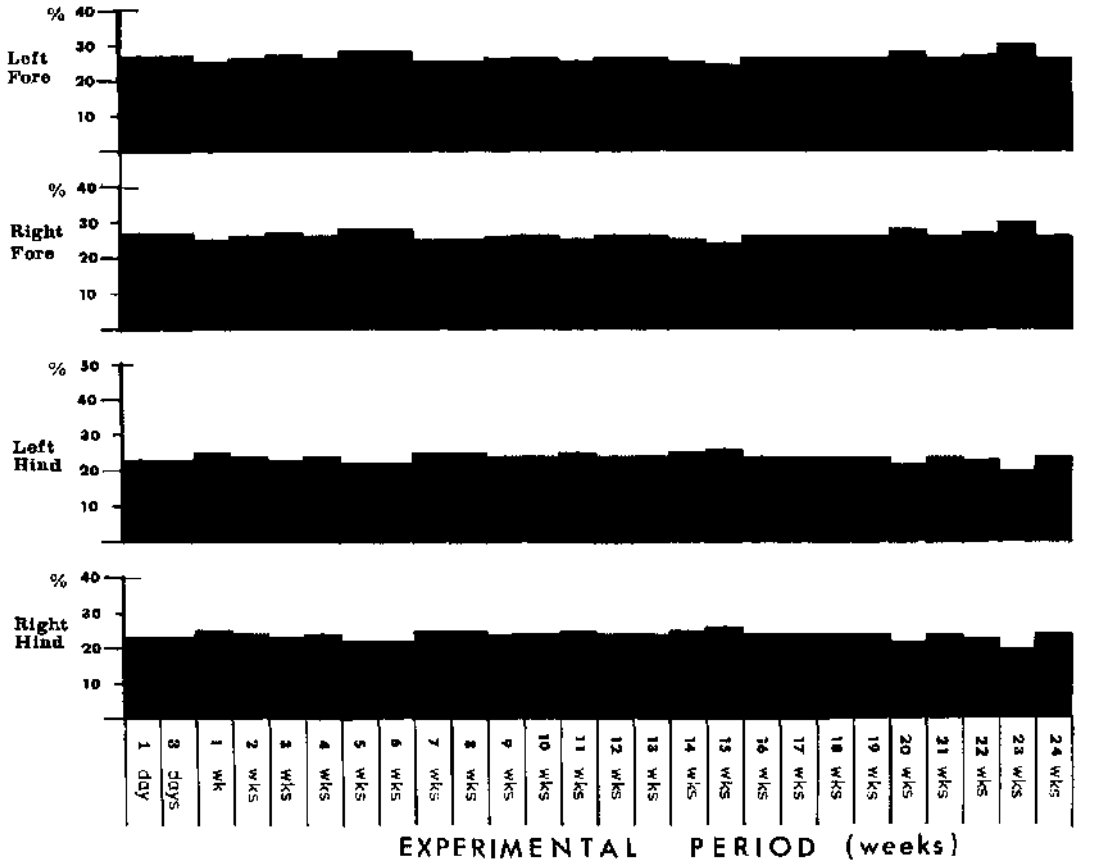
lamb no. 36



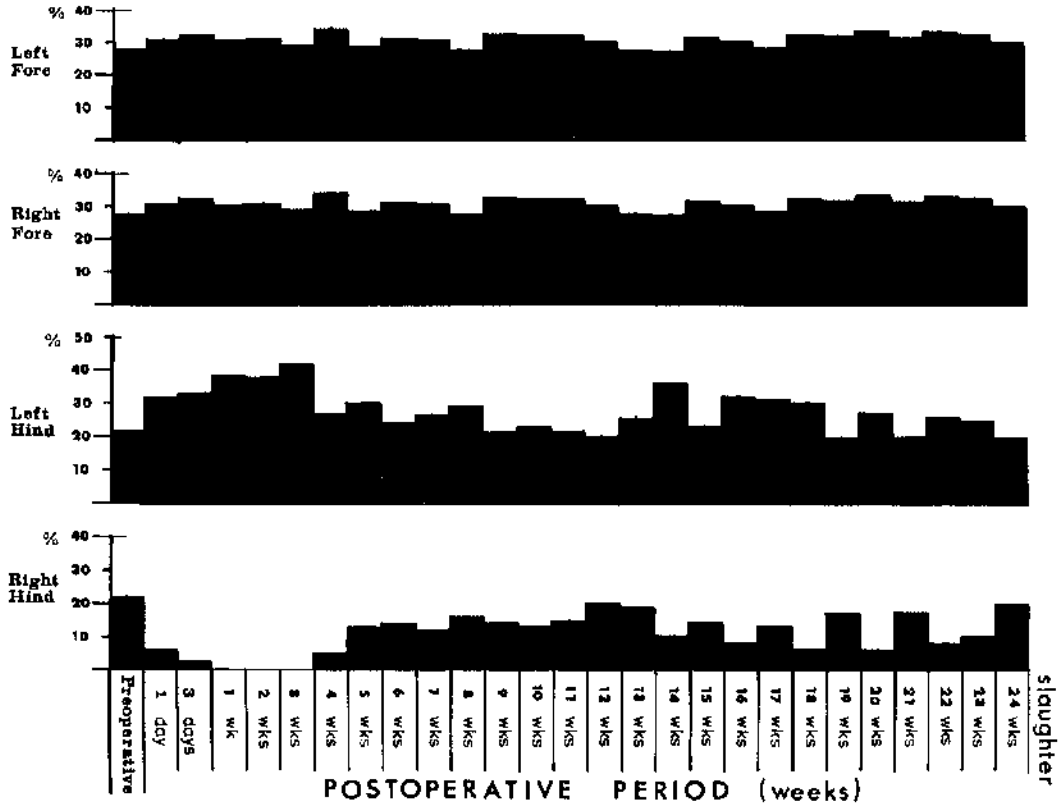
lamb no. 37



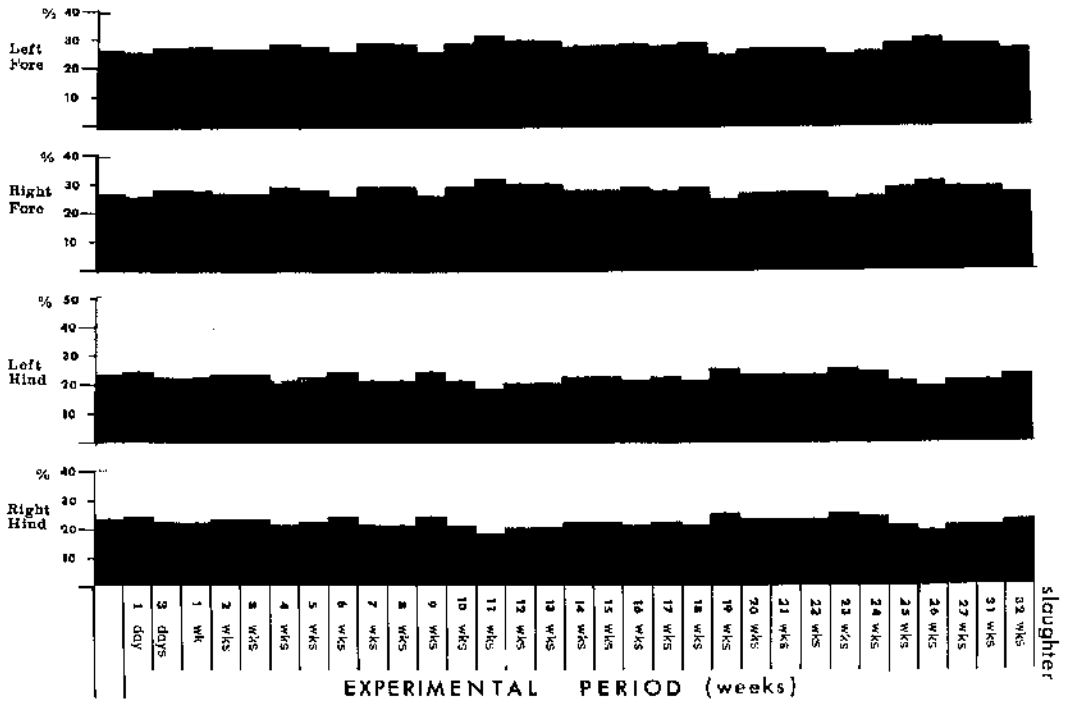
lamb no. 35



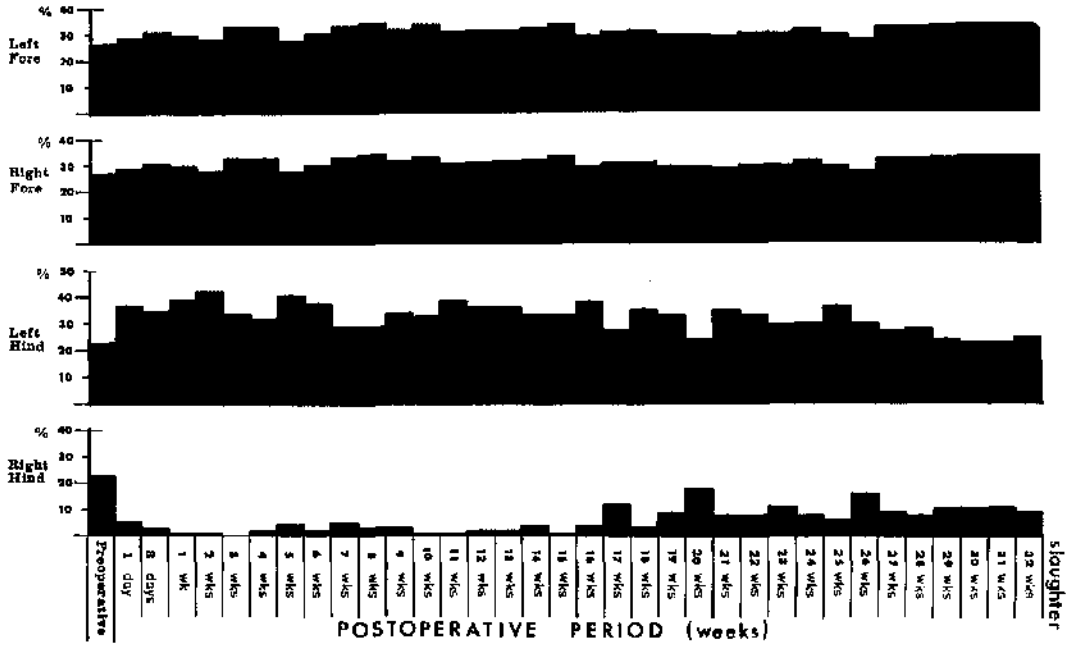
lamb no. 39



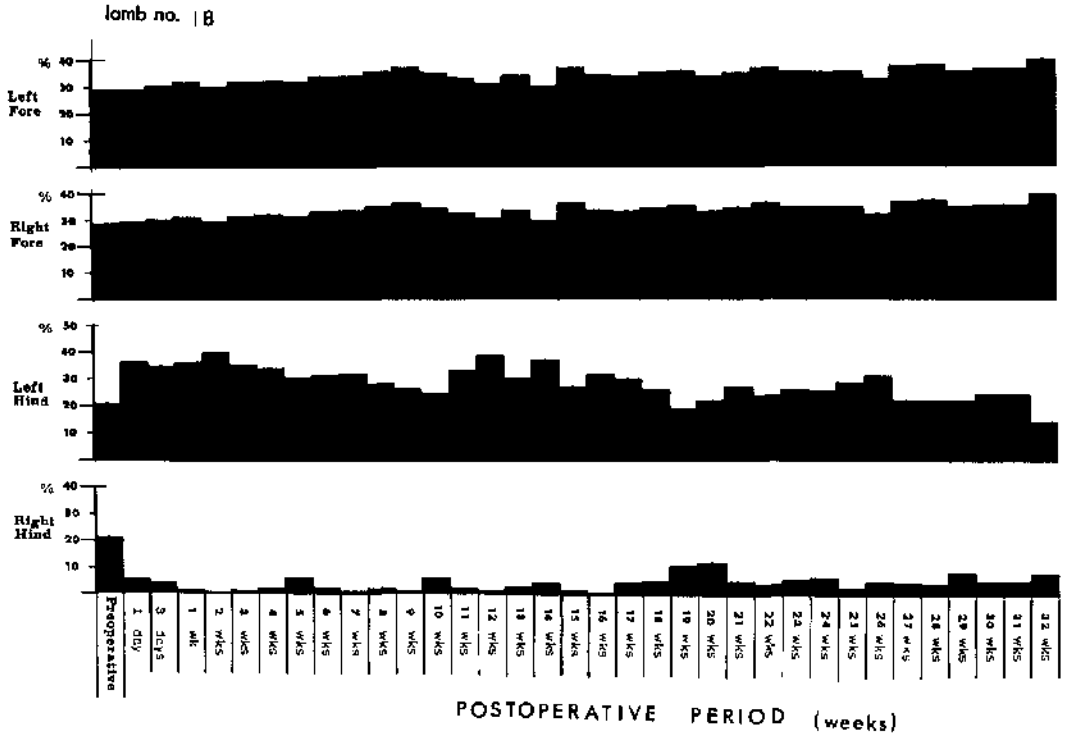
lamb no. 19



lamb no 17



lamb no. 18



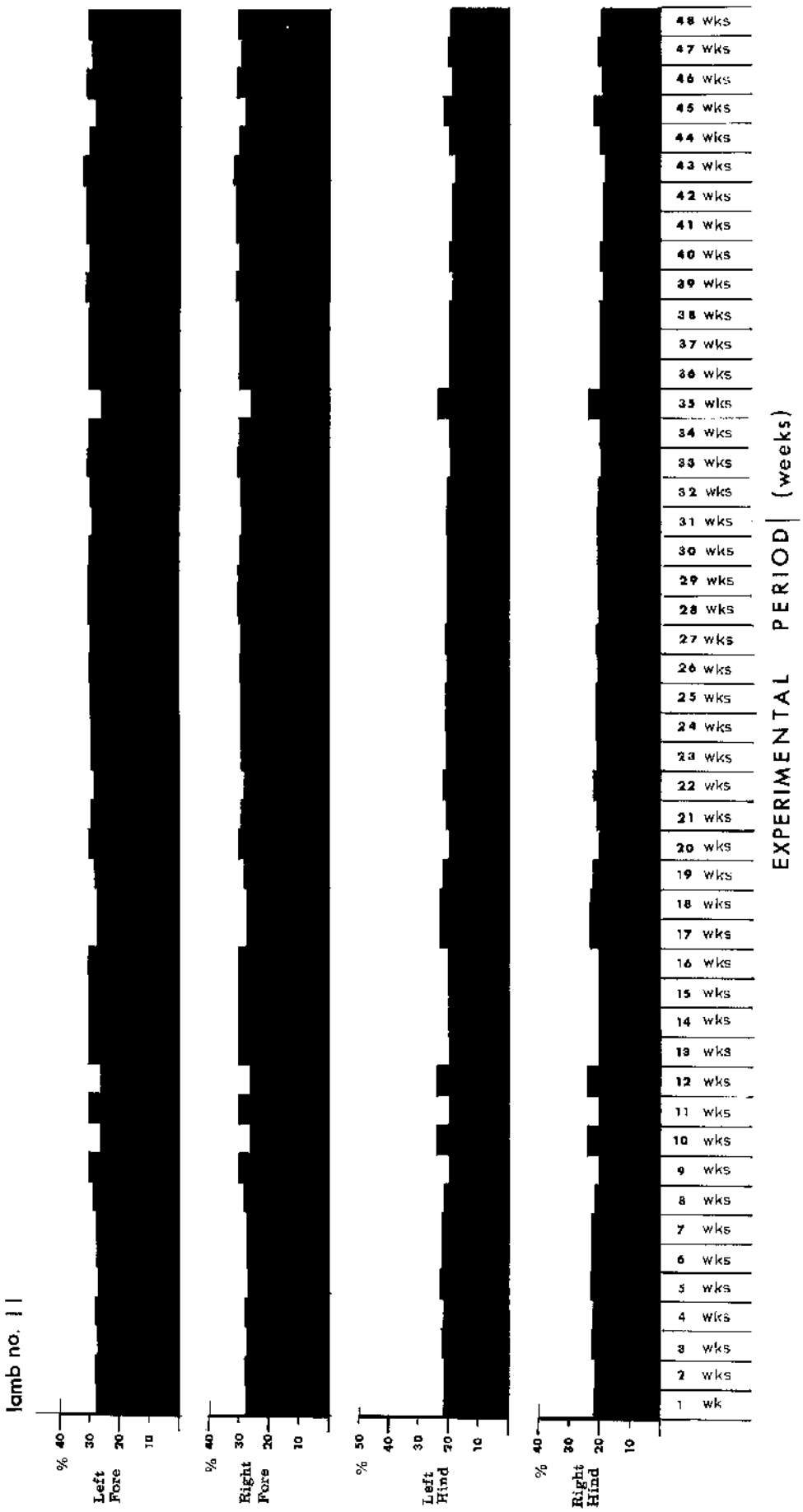

lamb no. 11
% 40 30 20 10
Left Fore
% 40 30 20 10
Right Fore
% 50 40 30 20 10
Left Hind
% 40 30 20 10
Right Hind
48 wks
47 wks
46 wks
45 wks
44 wks
43 wks
42 wks
41 wks
40 wks
39 wks
38 wks
37 wks
36 wks
35 wks
34 wks
33 wks
32 wks
31 wks
30 wks
29 wks
28 wks
27 wks
26 wks
25 wks
24 wks
23 wks
22 wks
21 wks
20 wks
19 wks
18 wks
17 wks
16 wks
15 wks
14 wks
13 wks
12 wks
11 wks
10 wks
9 wks
8 wks
7 wks
6 wks
5 wks
4 wks
3 wks
2 wks
1 wk
EXPERIMENTAL PERIOD (weeks)

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fig 1

DISTANCE BETWEEN ACETABULI PLOTTED AGAINST AGE

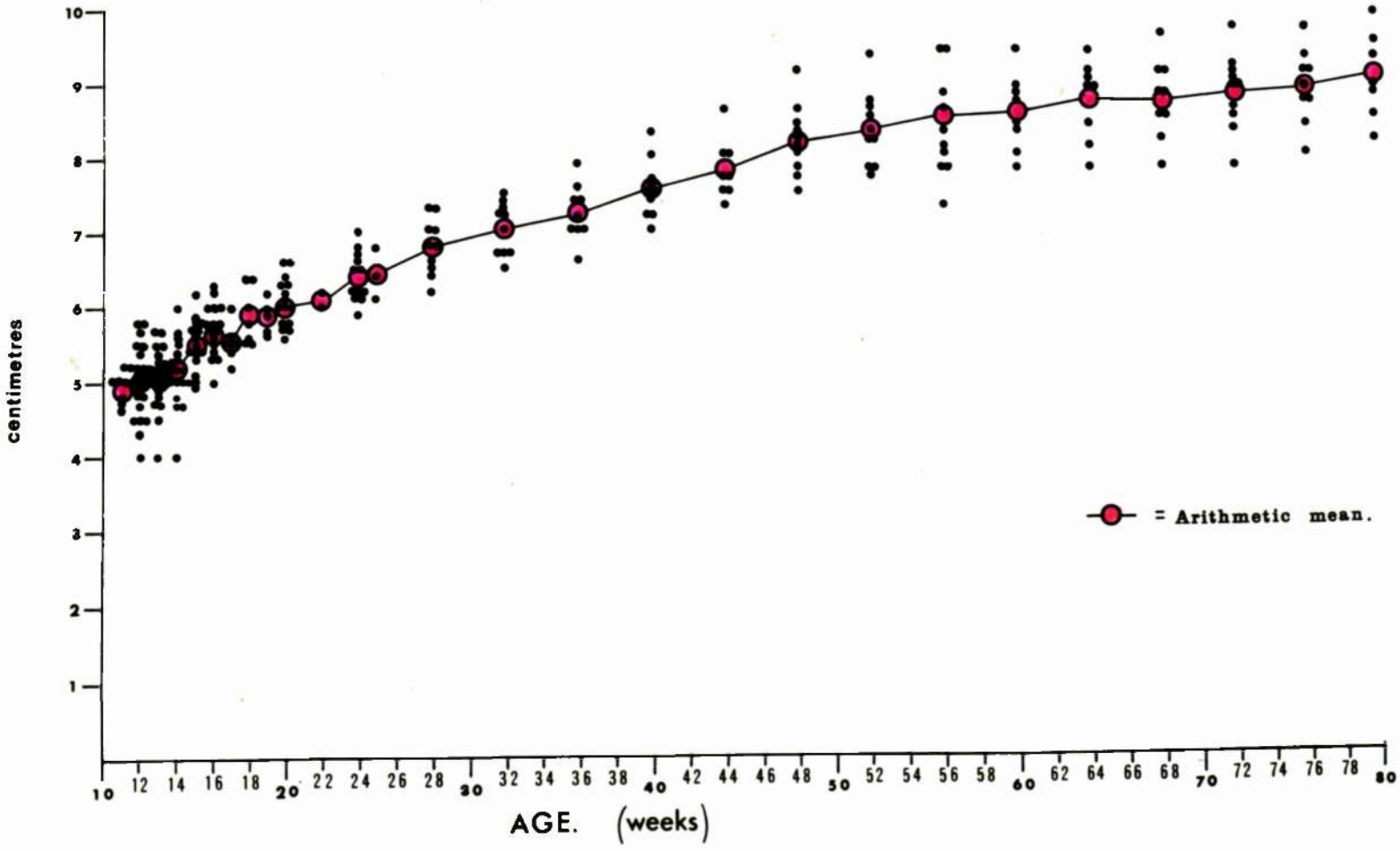
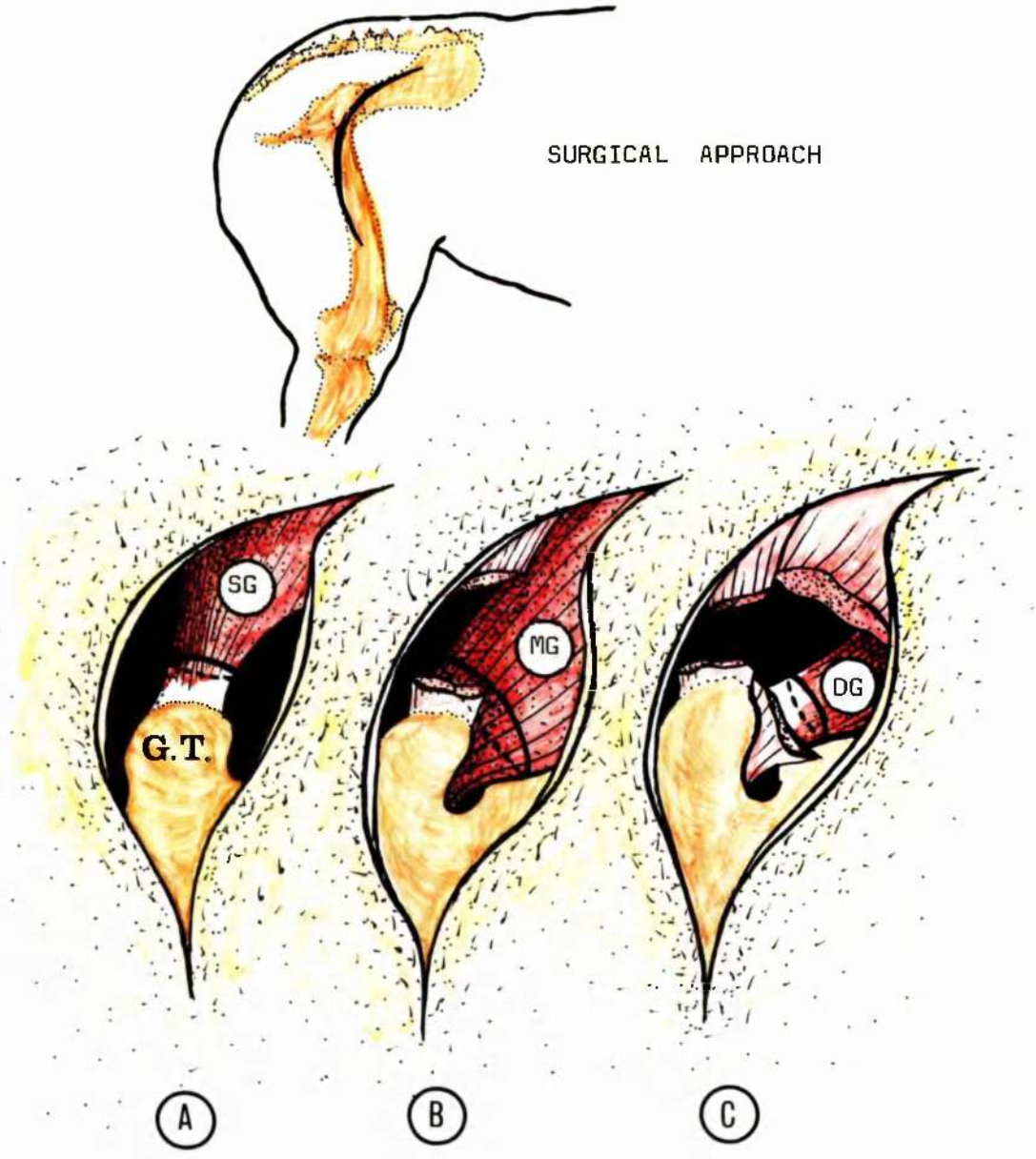


FIG 2 FEMORAL HEAD AND NECK EXCISION - APPROACH TO HIP JOINT BY GLUTEAL TENOTOMY



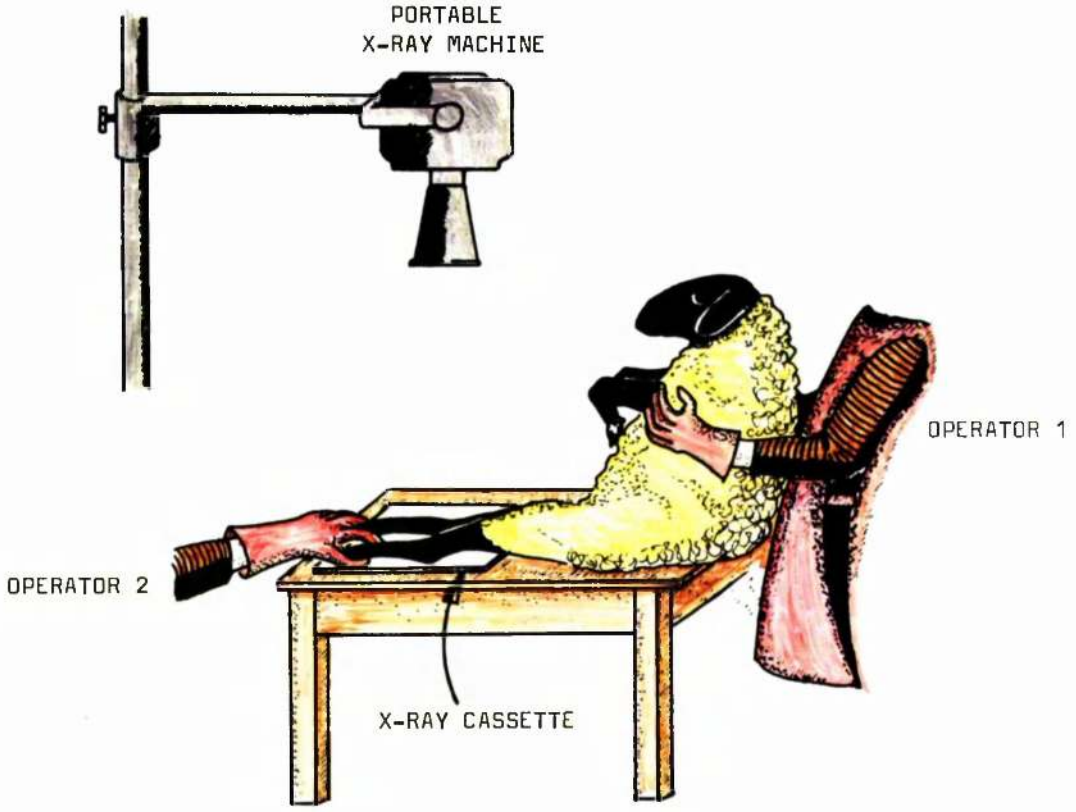
A = Tenotomy of superficial gluteal muscle (SG)

B = Tenotomy of middle gluteal muscle (MG)

C = Tenotomy of deep gluteal muscle (DG)

G.T. = greater trochanter

FIG. 3 Method of limb radiography



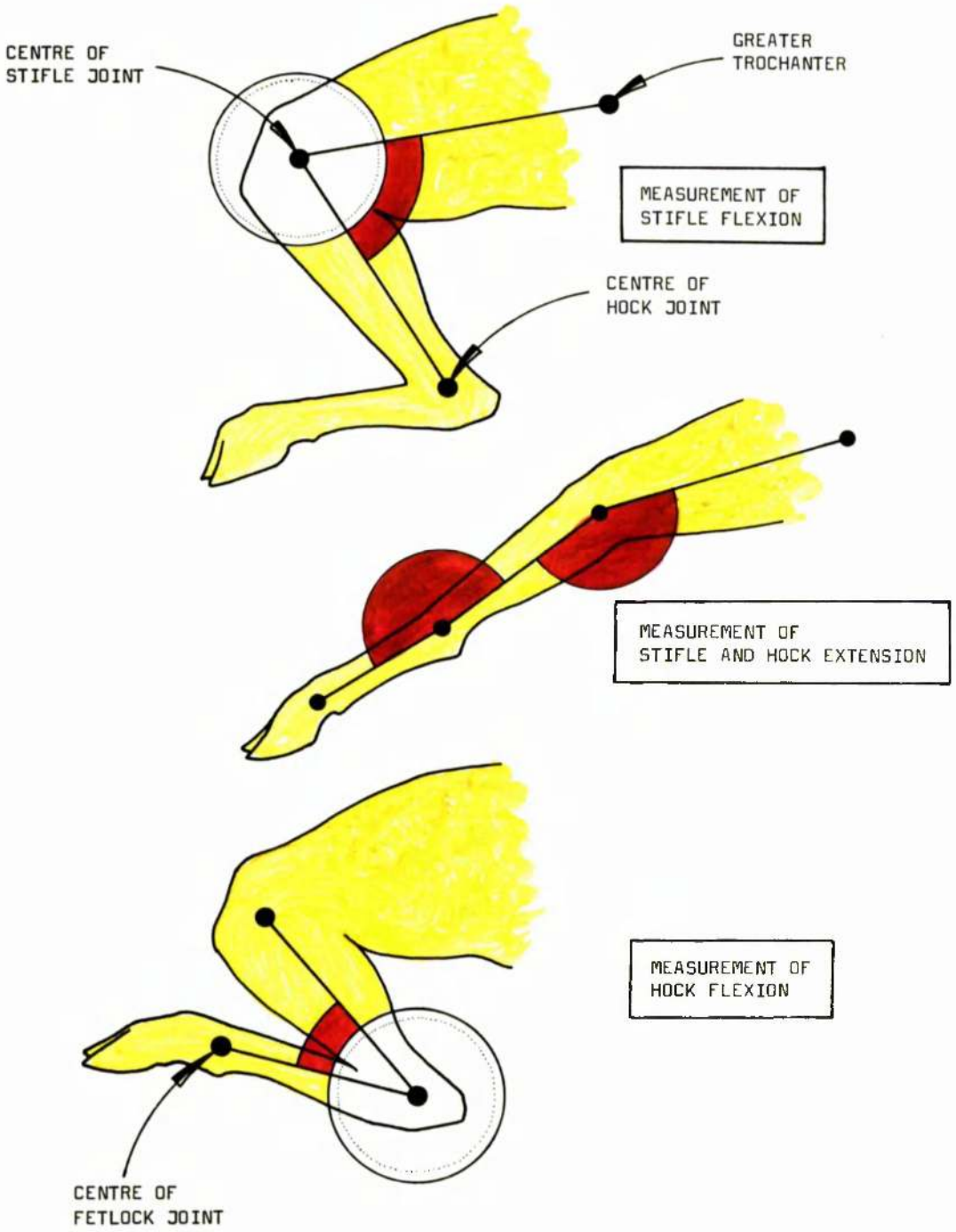
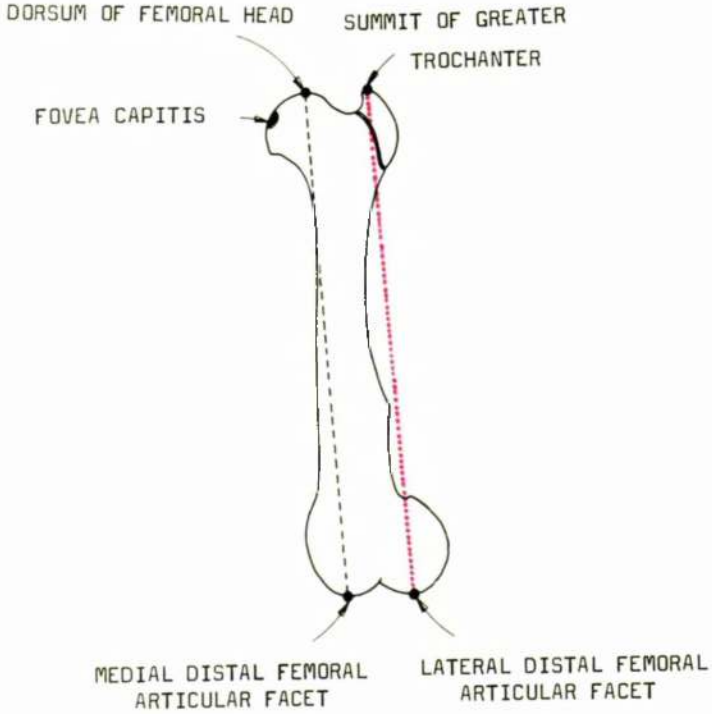


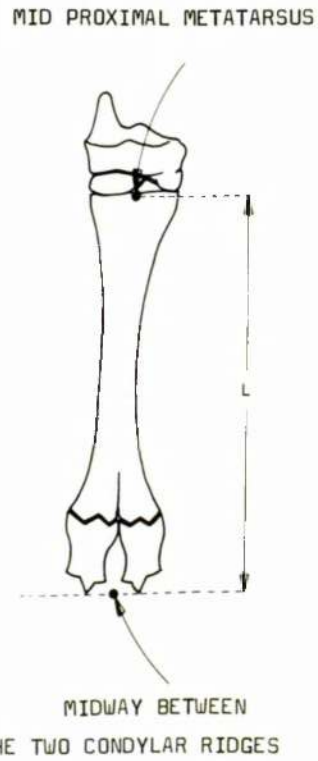
FIG. 4 Estimation of hock and stifle joint mobility

fig 5

a FIG. Femoral length



b FIG. Metatarsal length



c FIG. Tibial length

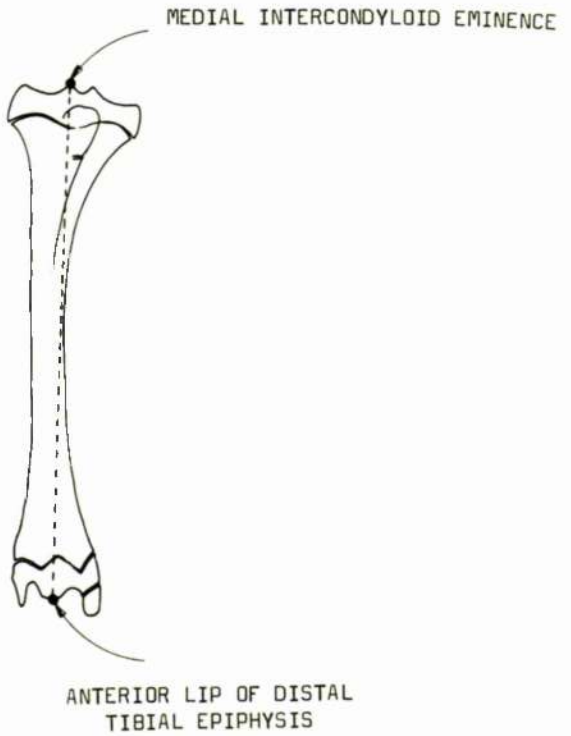
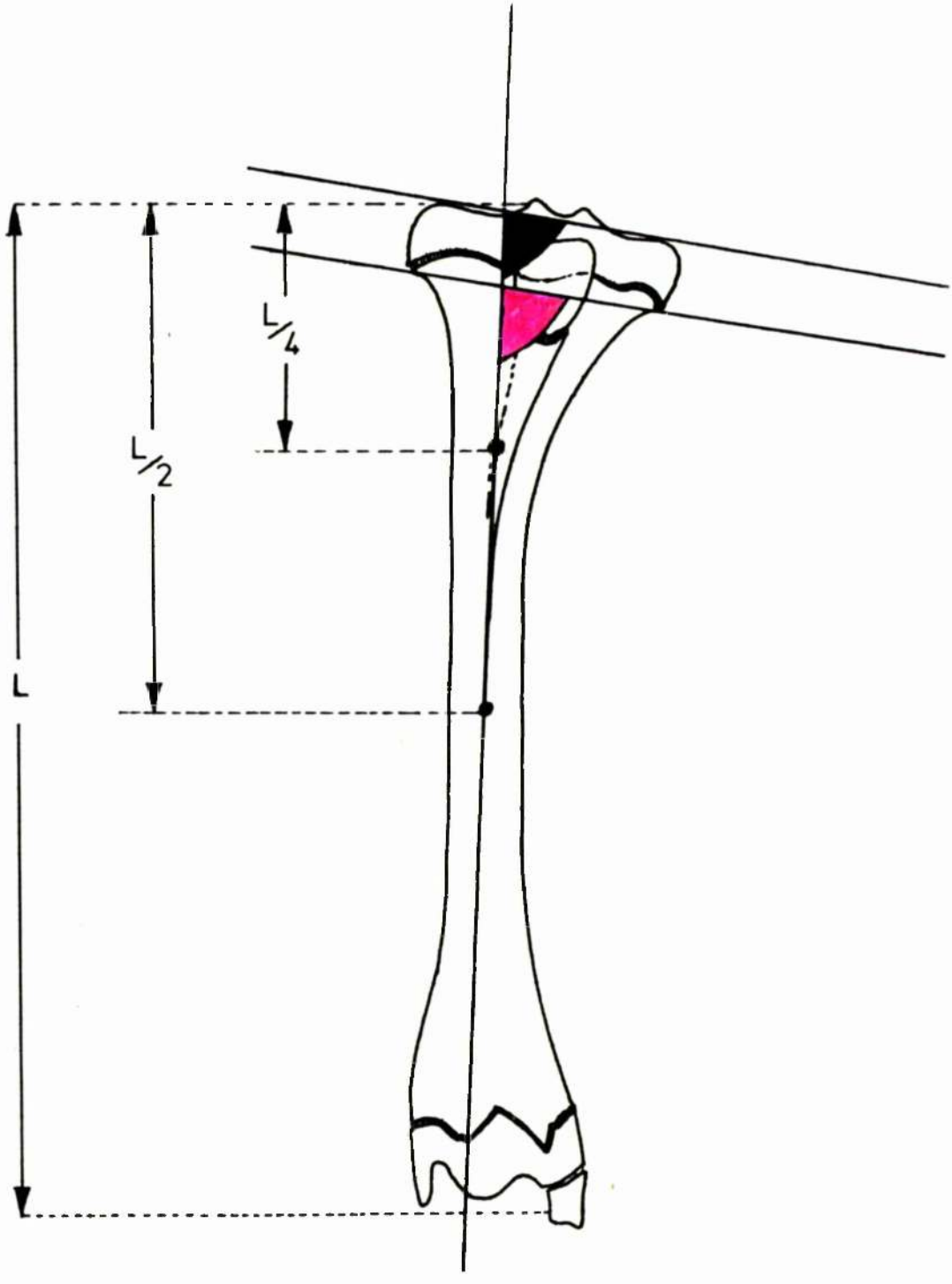


FIG. 6 Tibial plateau angle (on anteroposterior radiographs)



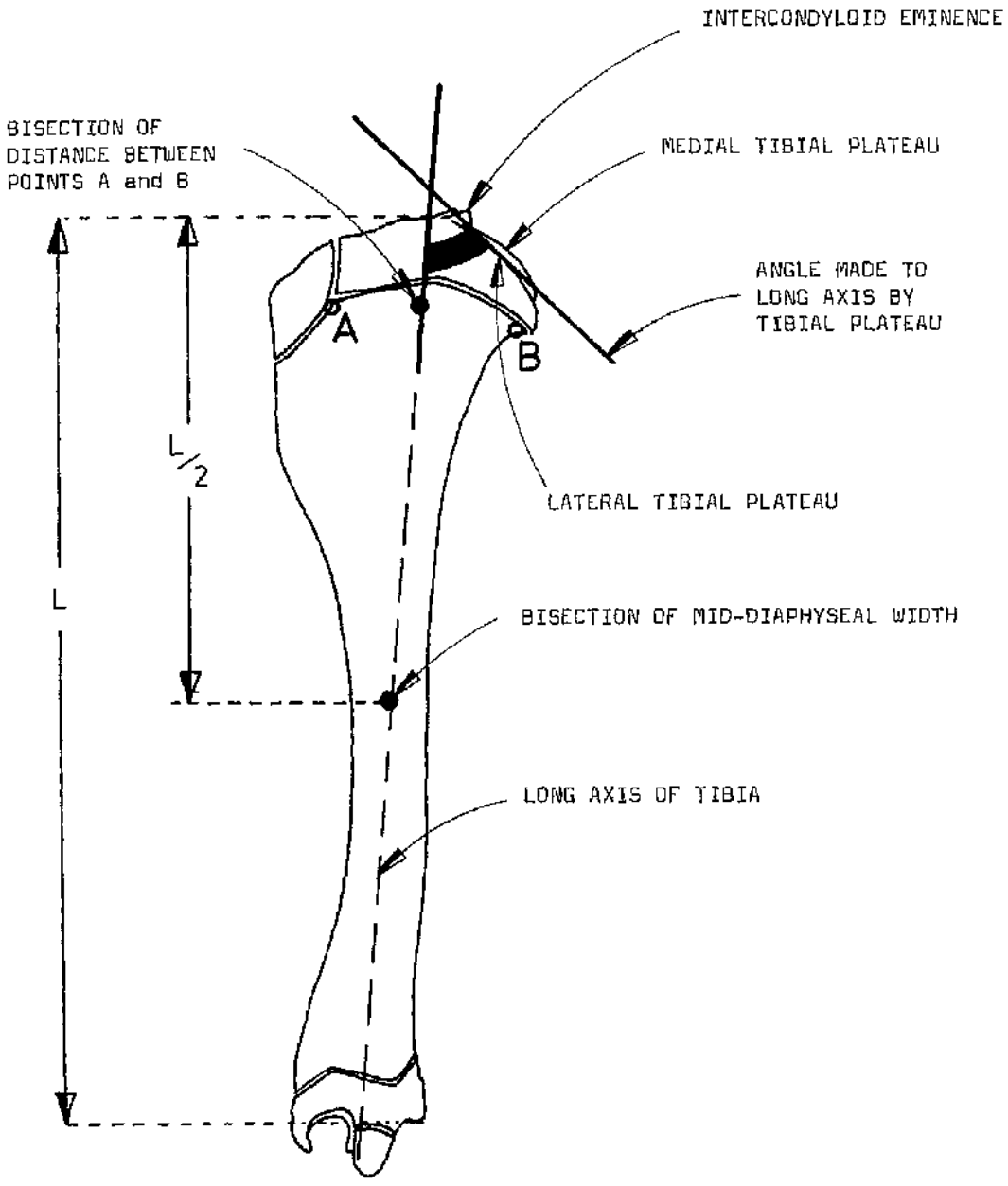


FIG. 7 Estimation of tibial plateau angle
(on lateral radiographs)

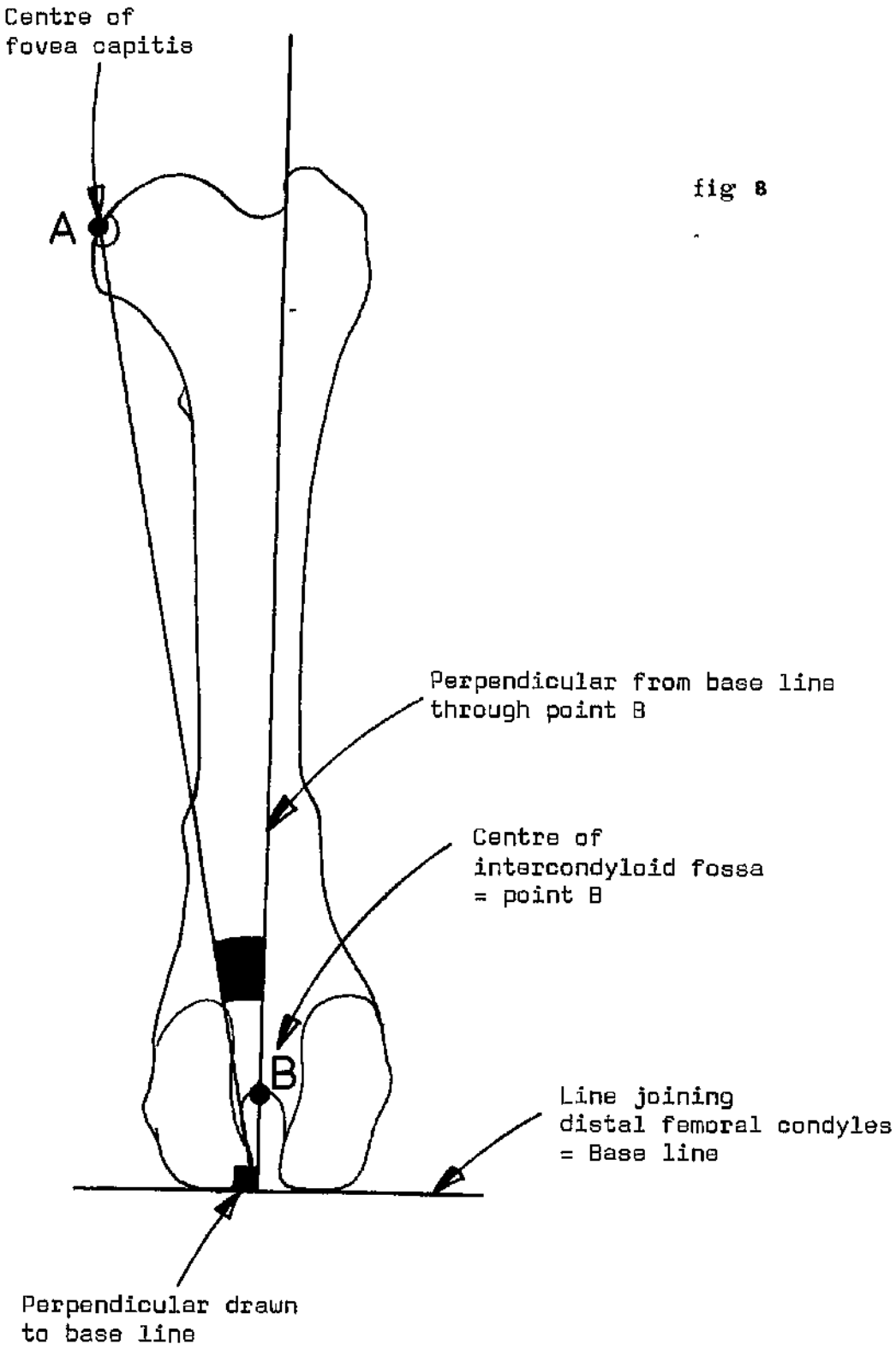


fig 8

Angle of fovea capitis relative to femoral condylar plateau

fig 9 Estimation of Growth Contribution from each Bone Extremity.

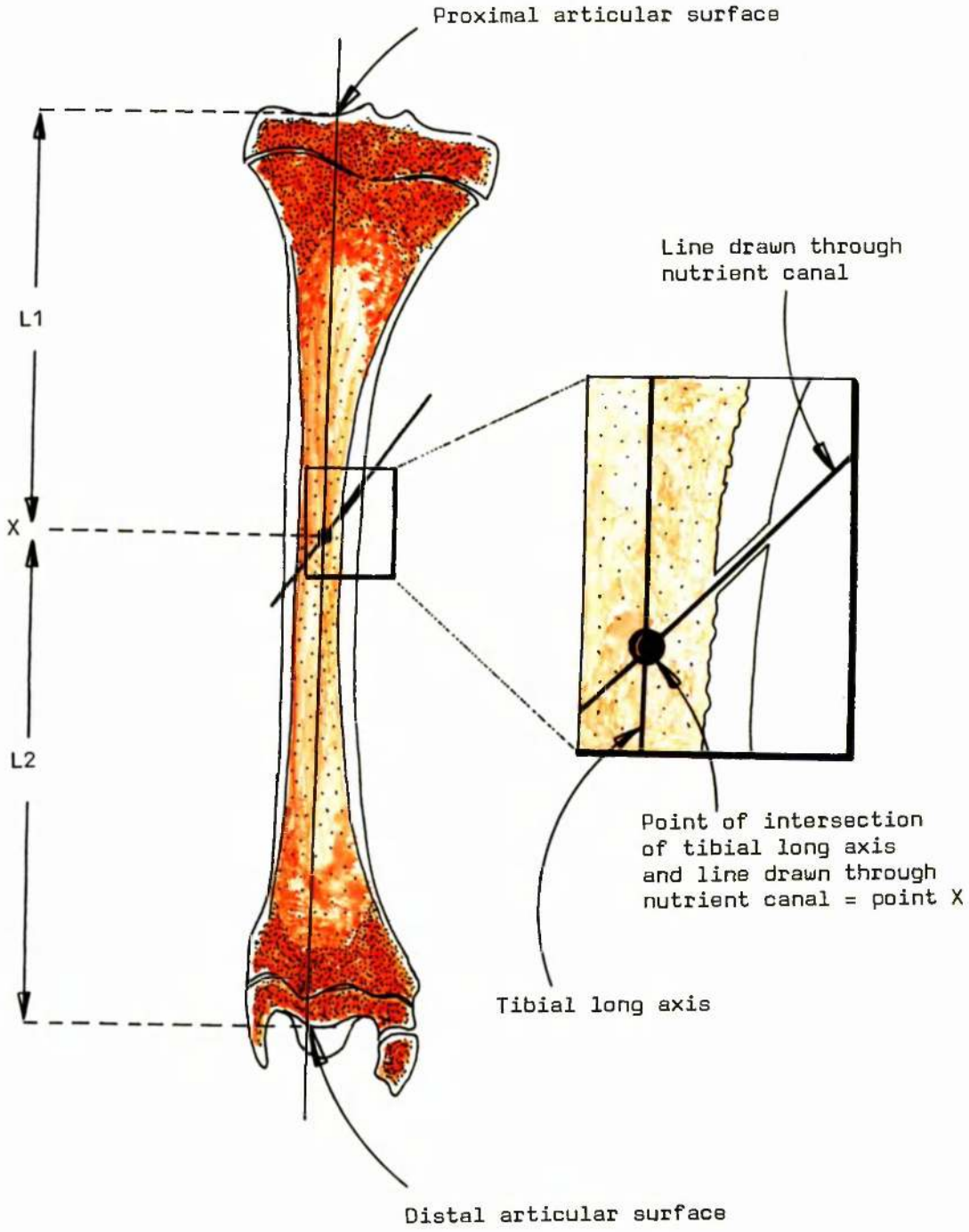


FIG. 10a Greater Trochanter Base Angle

(On Anteroposterior Radiographs)

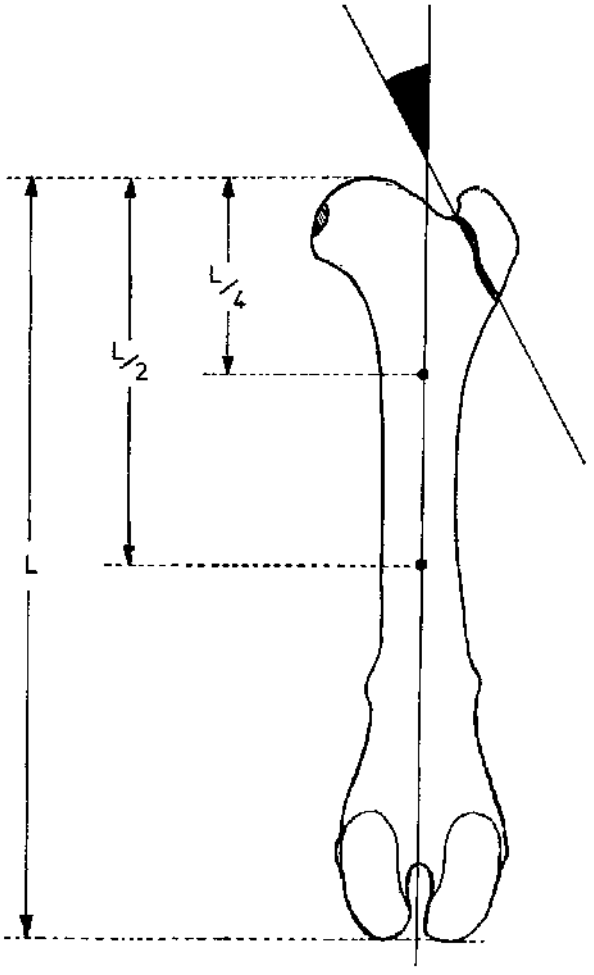


FIG. 10b Femoral Neck Angle

(On Anteroposterior Radiographs)

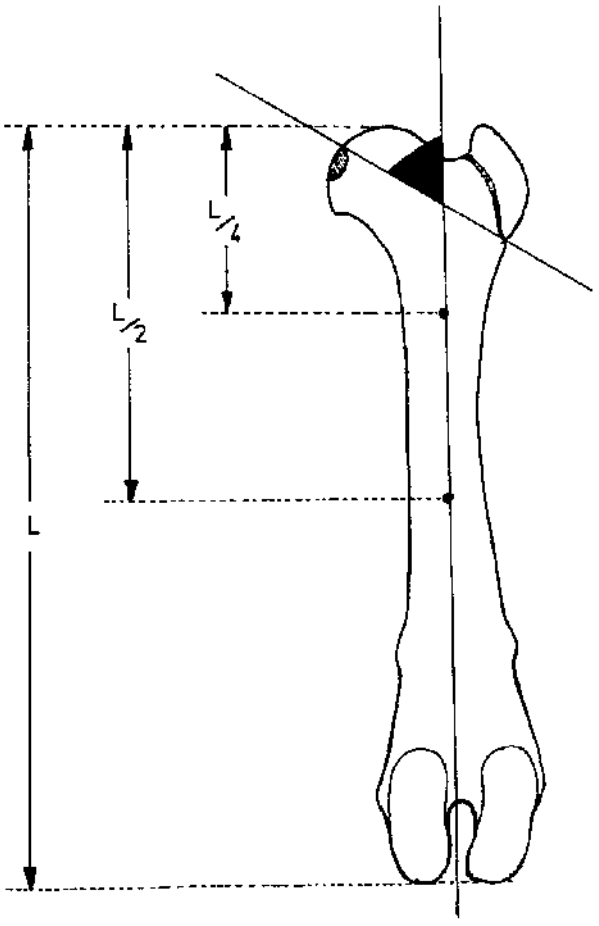


fig 11 Estimation of tibial angular deformity

(on anteroposterior radiographs)

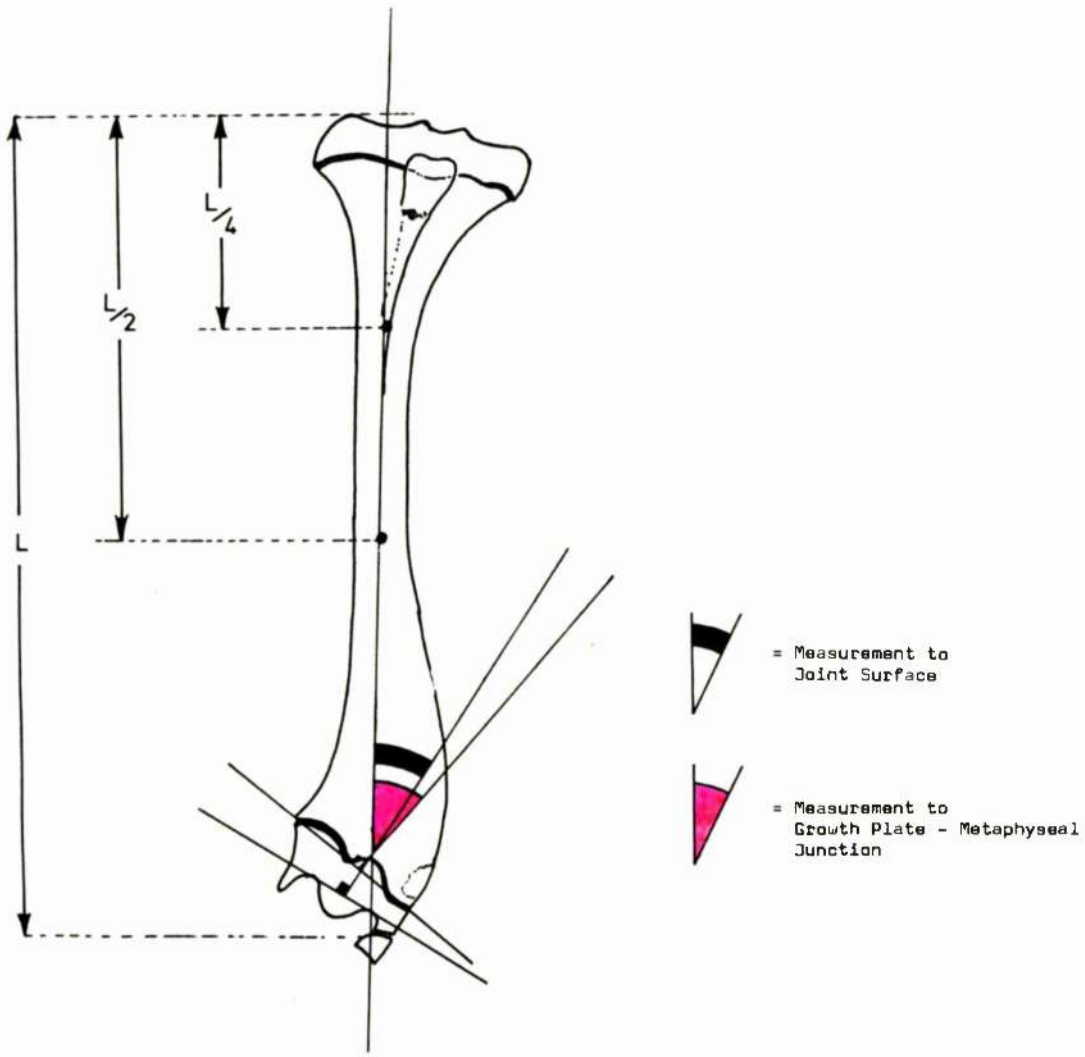


FIG.12 Estimation of tibial angular deformity

(on lateral radiographs)

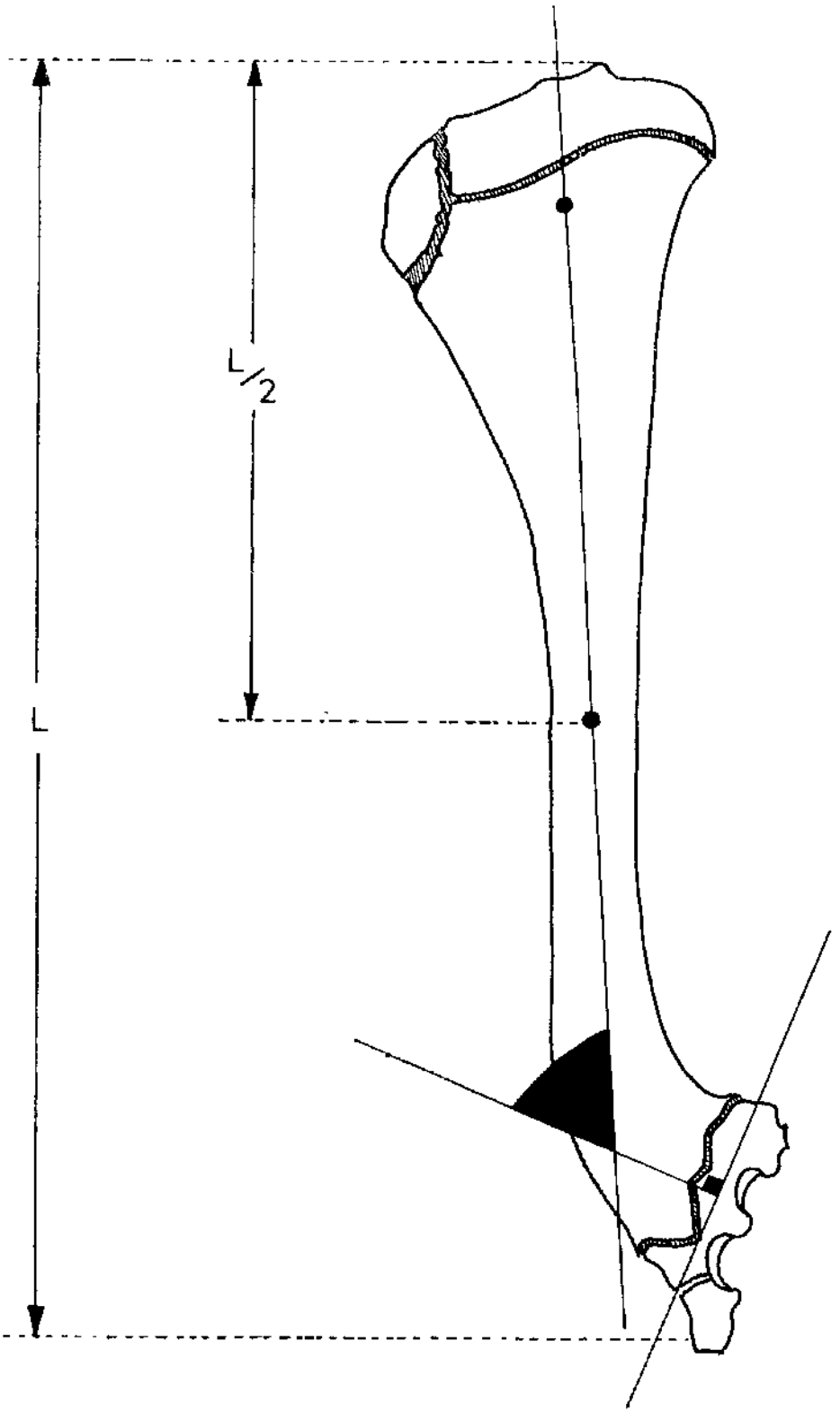
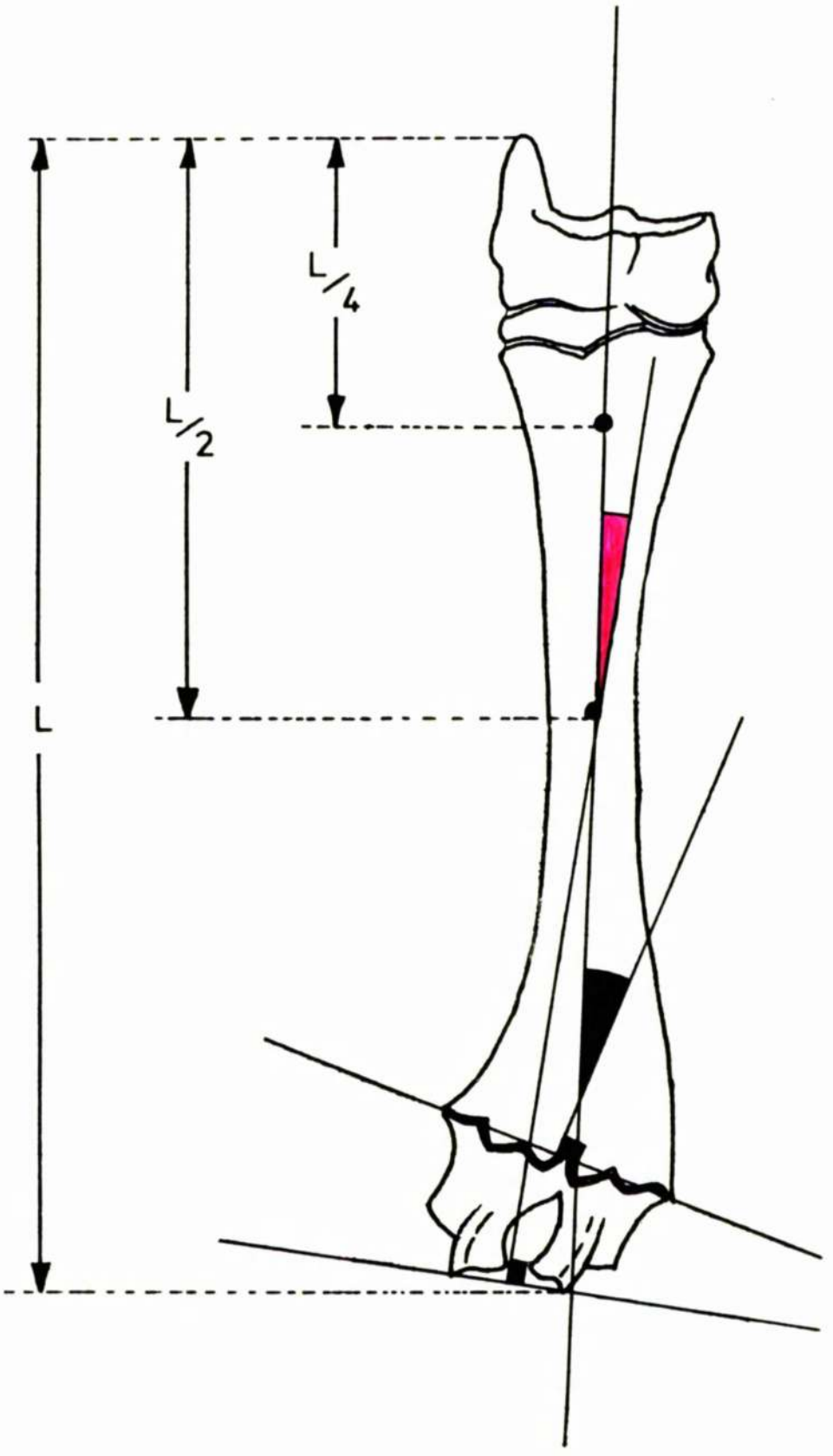


FIG. 13 Estimation of metatarsal angular deformity

(on anteroposterior radiographs)



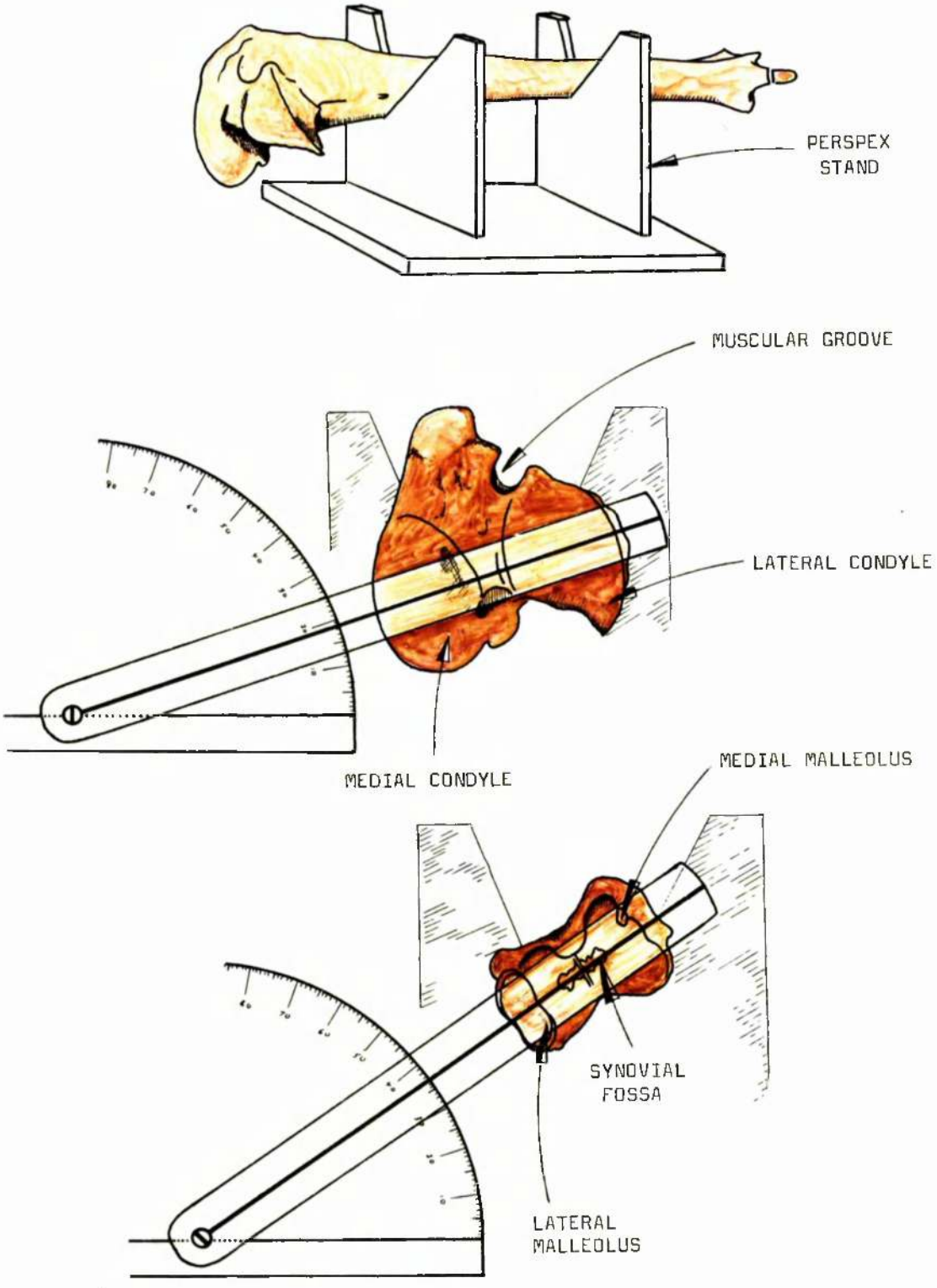


FIG. 14 Estimation of tibial torsion

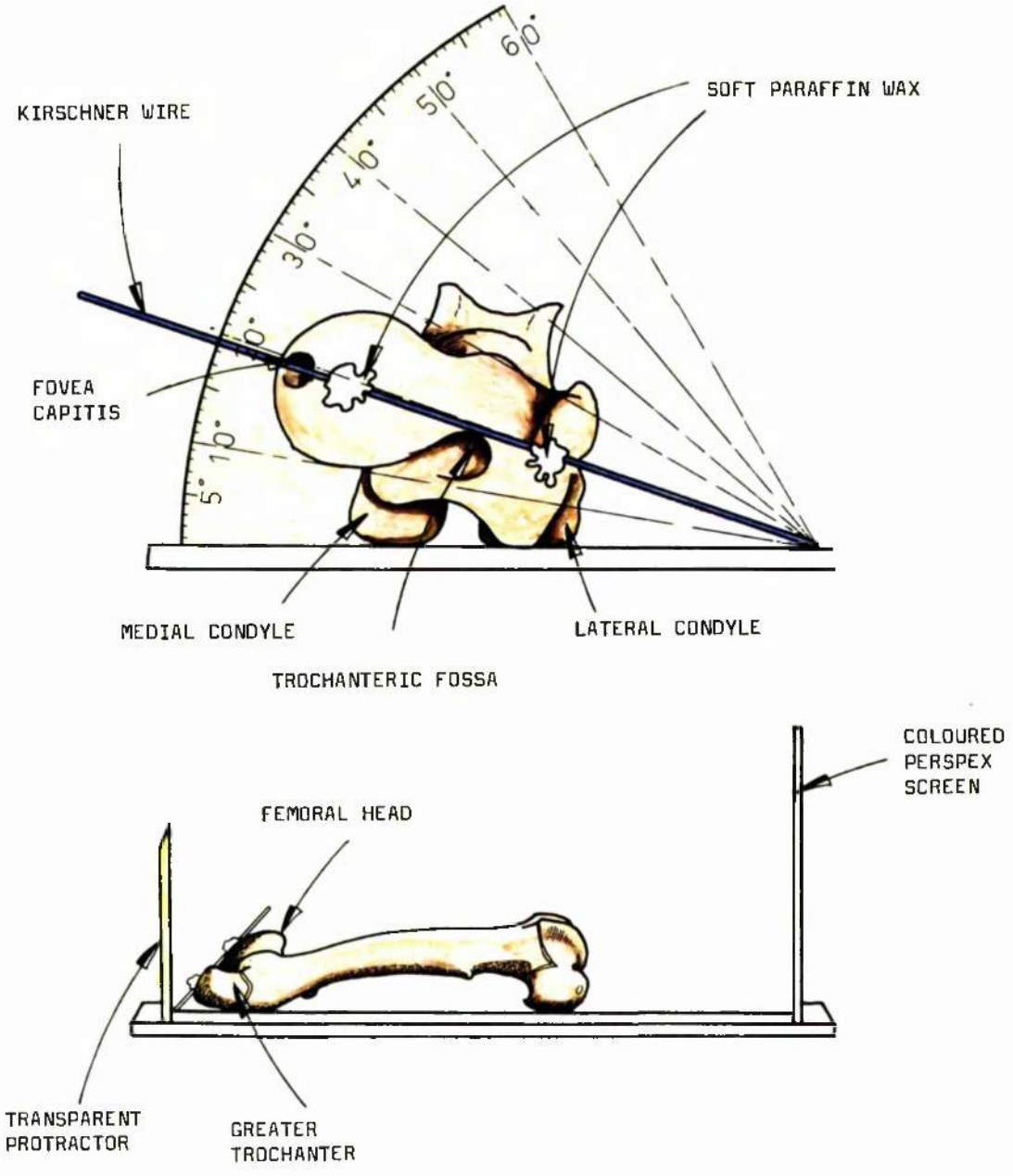
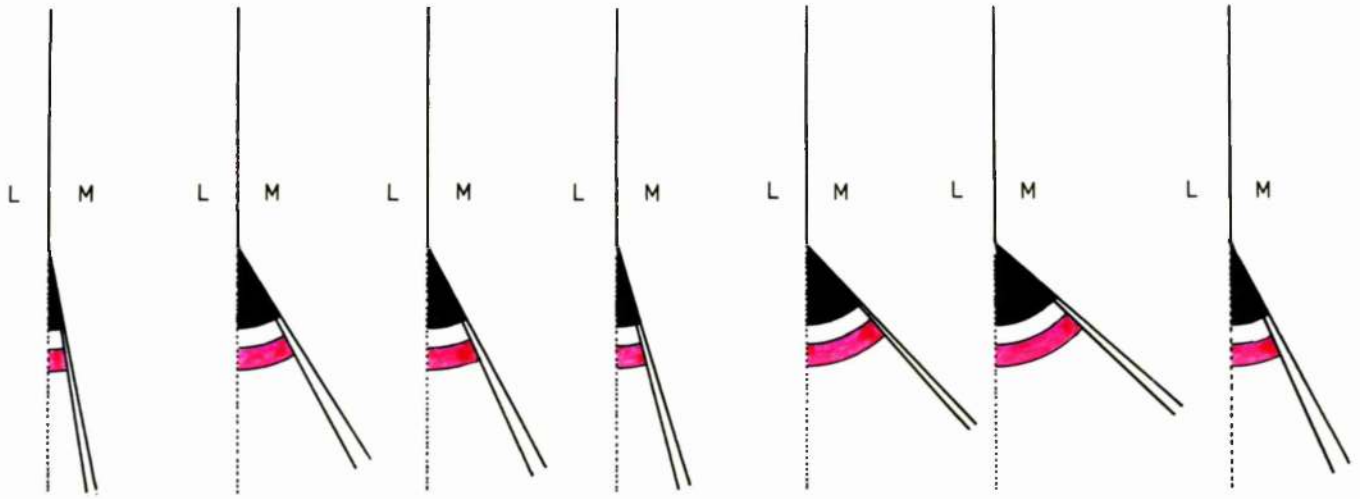

KIRSCHNER WIRE
SOFT PARAFFIN WAX
FOVEA CAPITIS
MEDIAL CONDYLE
LATERAL CONDYLE
TROCHANTERIC FOSSA
COLOURED PERSPEX SCREEN
FEMORAL HEAD
TRANSPARENT PROTRACTOR
GREATER TROCHANTER

fig 15

FIG 16 Distal Tibial Deformity (left hind limb)
(on A P radiographs)

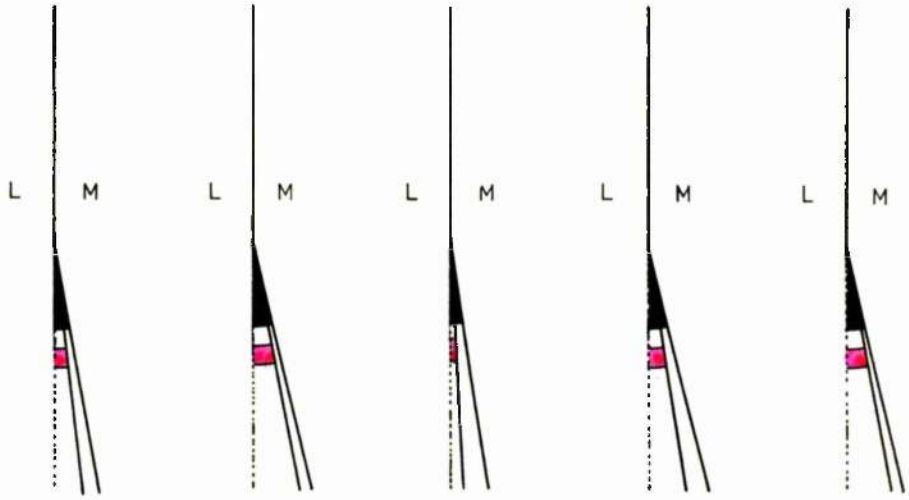


lamb no. 52 36 39 55 18 14 12

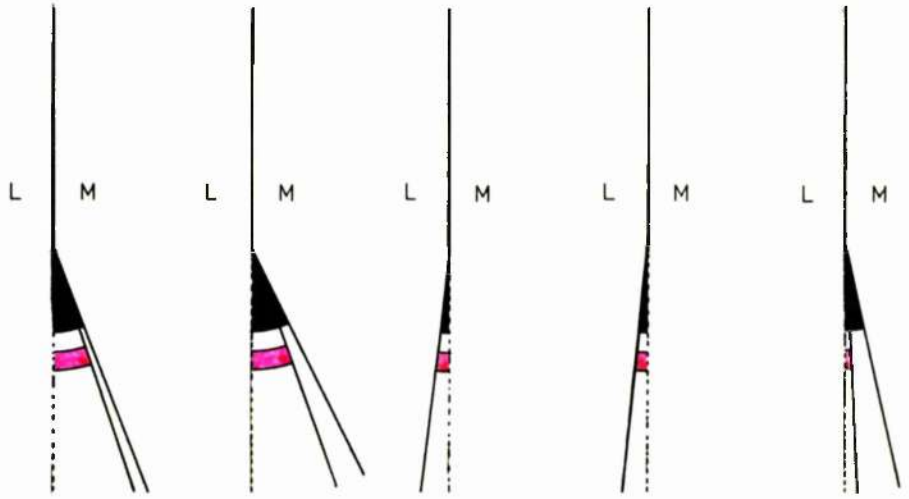
= measurement to metaphyseal-growth plate junction

= " " " joint surface

fig 17 Distal Metatarsal Deformity (left hind limb)
(on AP radiographs)



lamb no. 50 38 54 29 36



lamb no. 37 17 14 12 13

= measurement to metaphyseal-growth plate junction

= " " " joint surface

FIG 18 TIBIAL TORSION

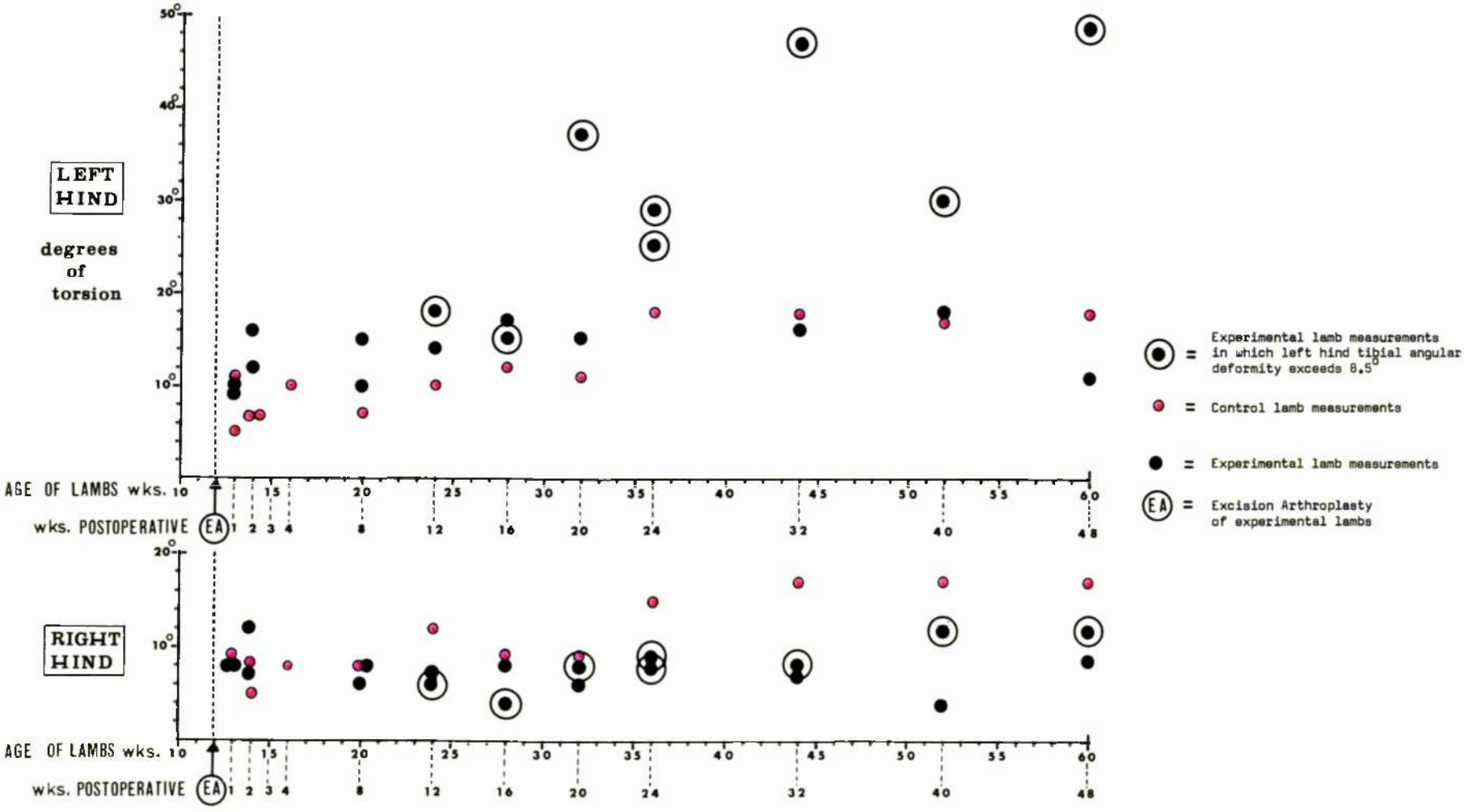


fig 19 TIBIAL TORSION BOILED OUT SPECIMENS

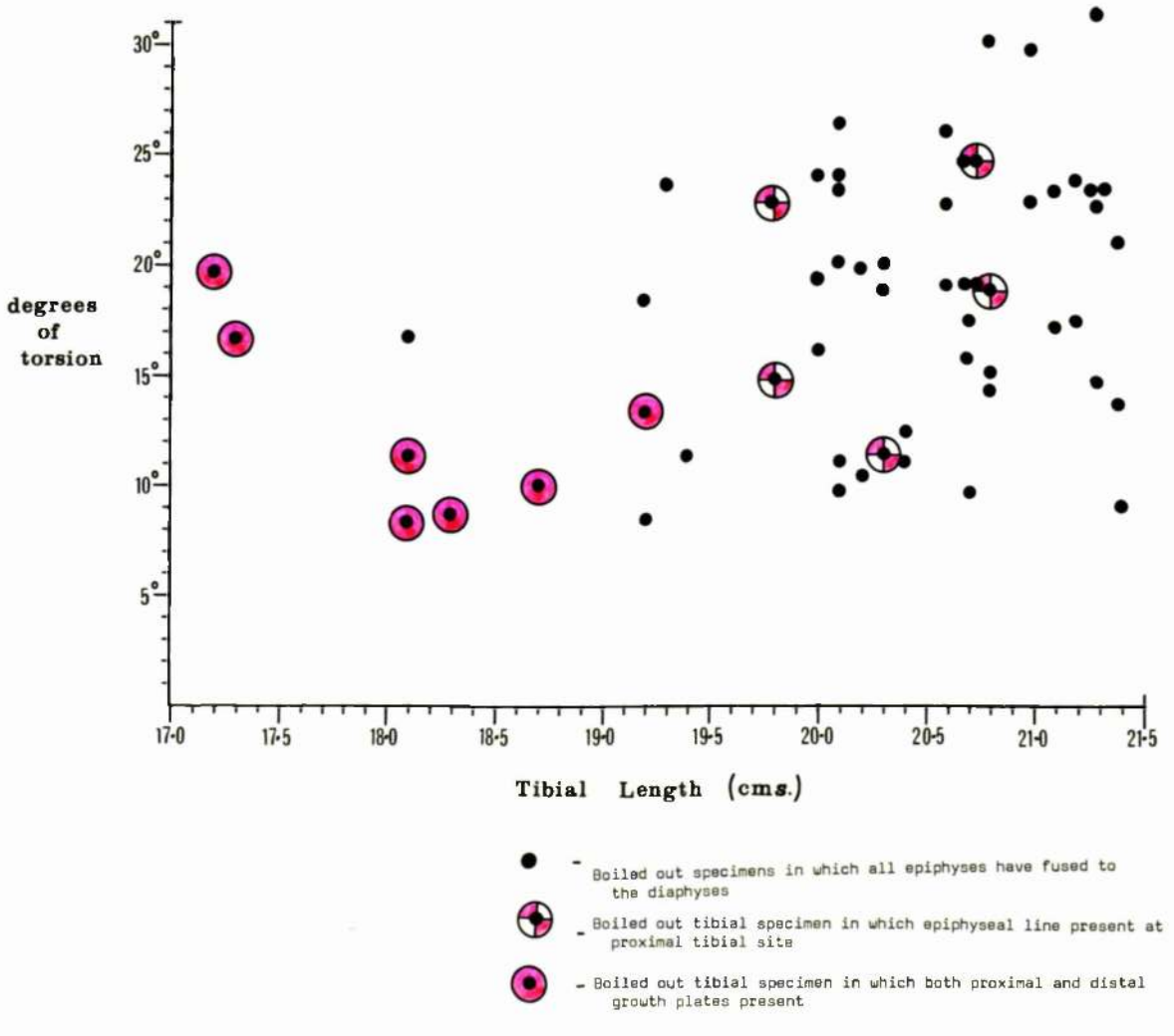


FIG 20 METATARSAL TORSION

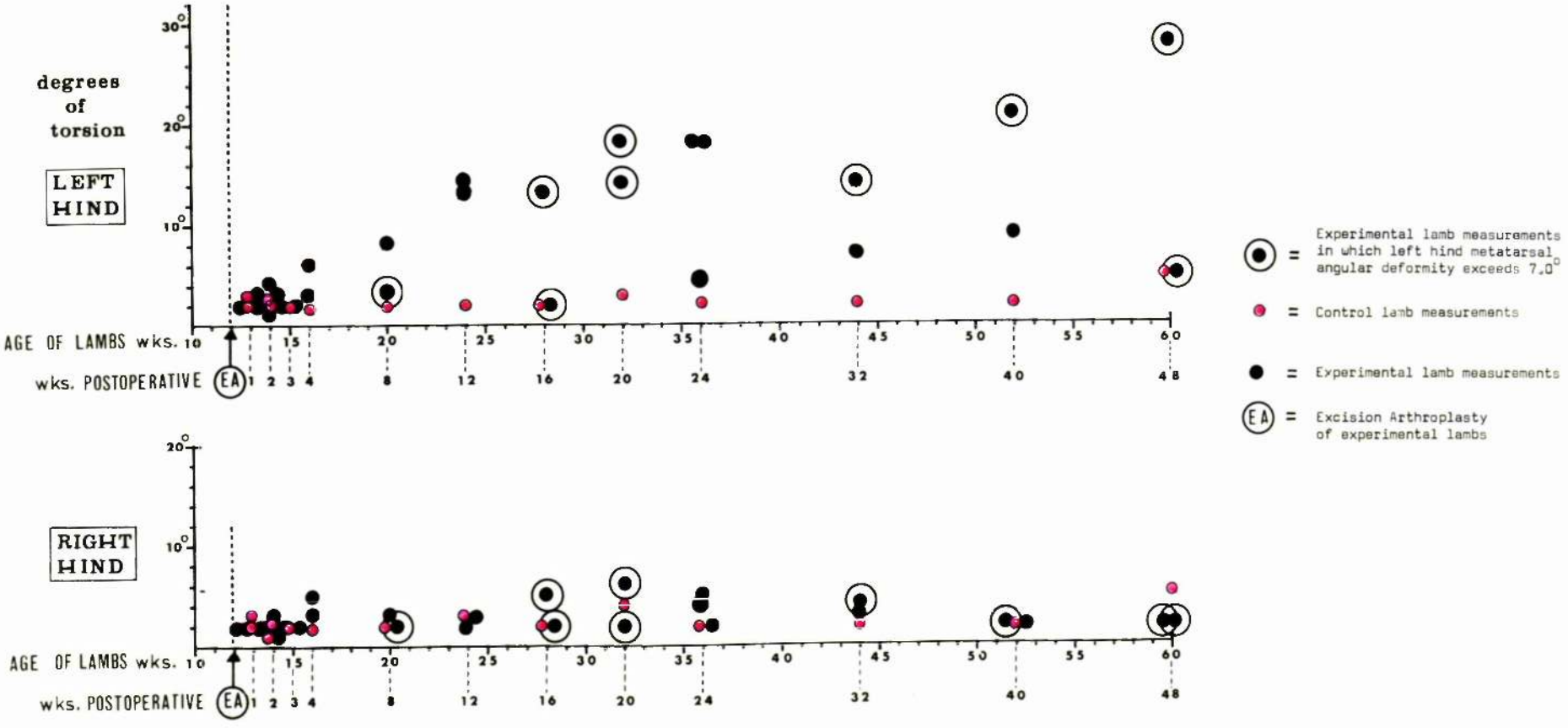


FIG 21 Joint mobility (left hock joint)

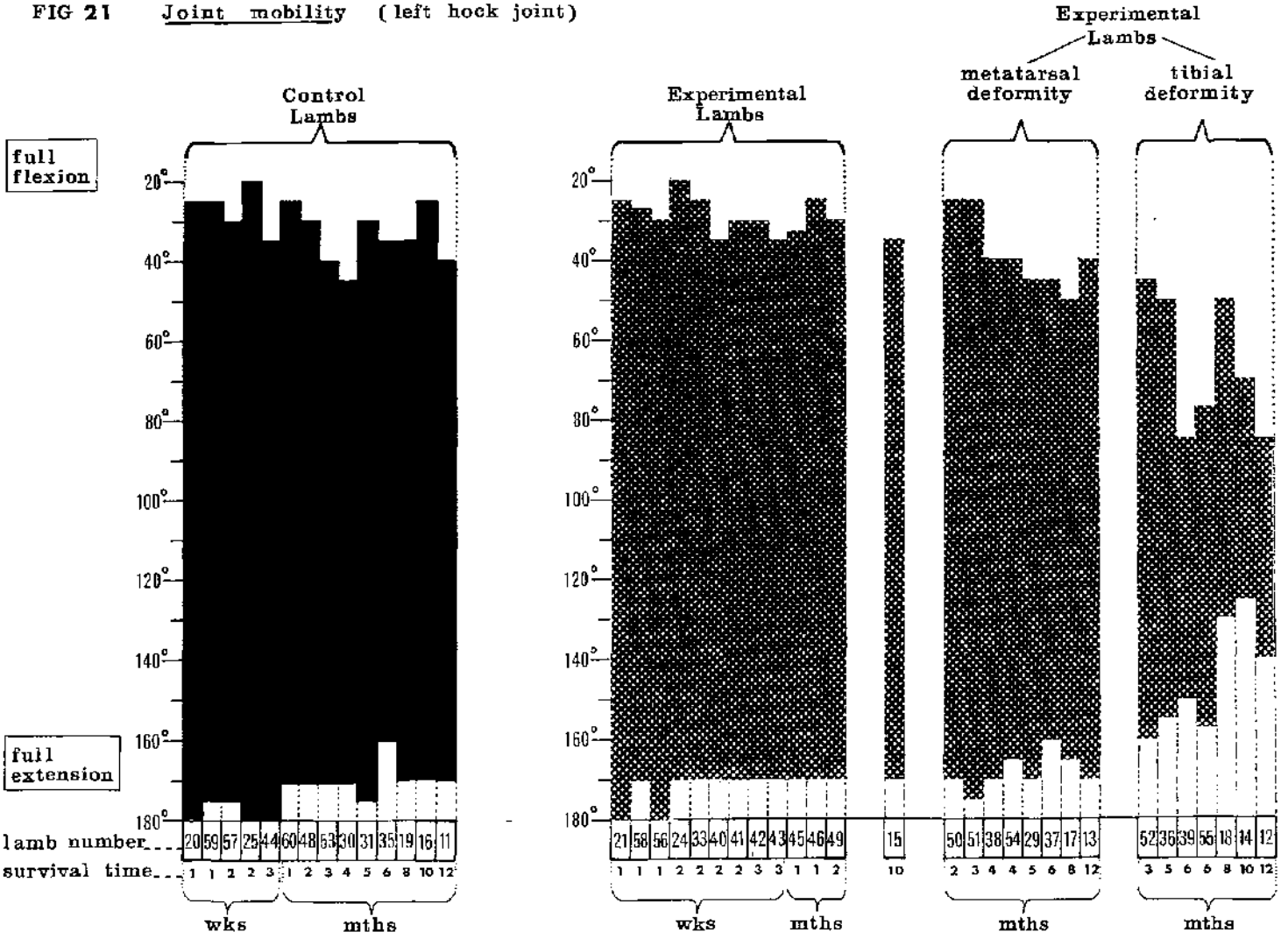


FIG 22a TIBIAL PLATEAU ANGLE measurement to joint surface

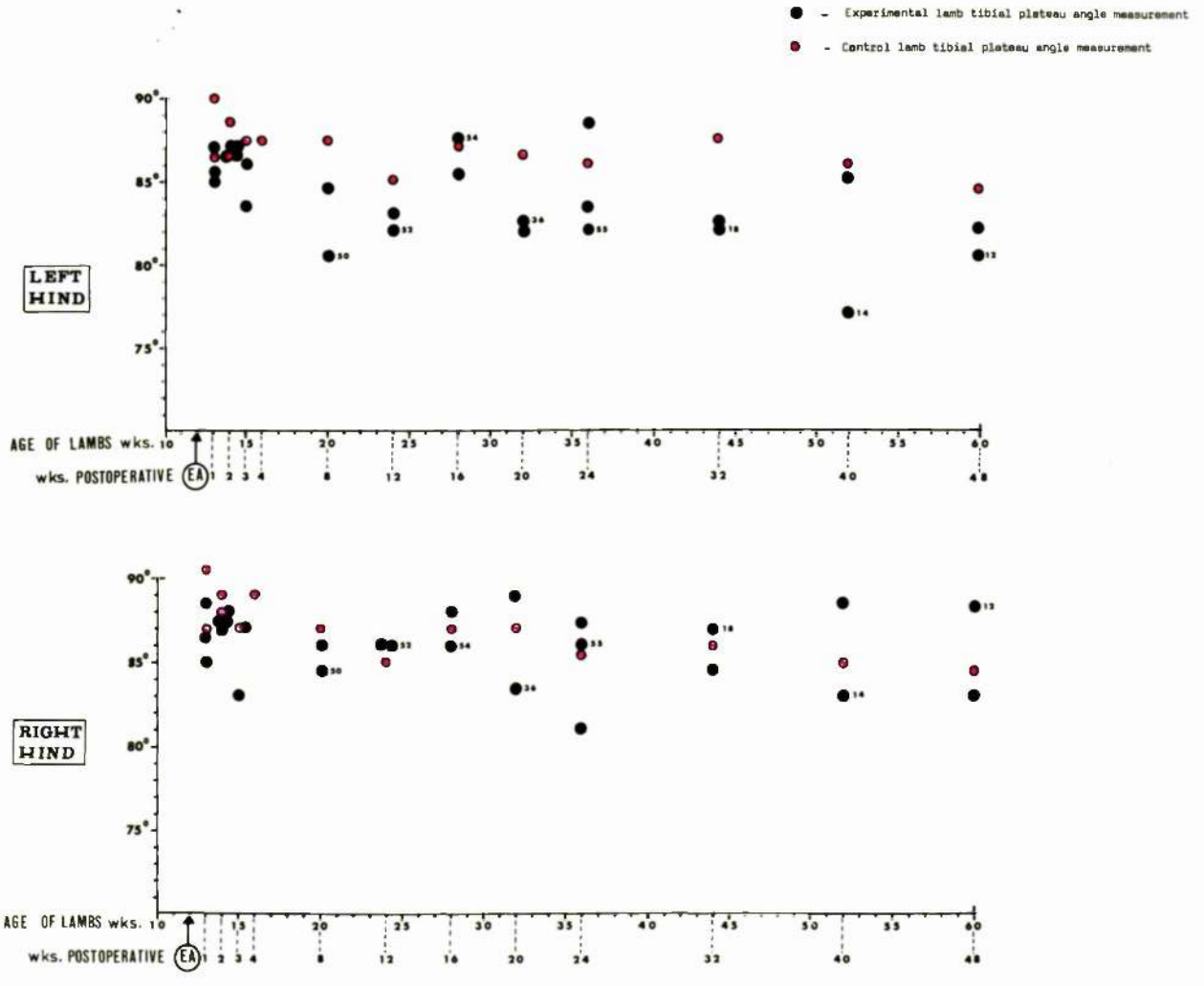


FIG 22b TIBIAL PLATEAU ANGLE measurement to metaphyseal-growth plate junction

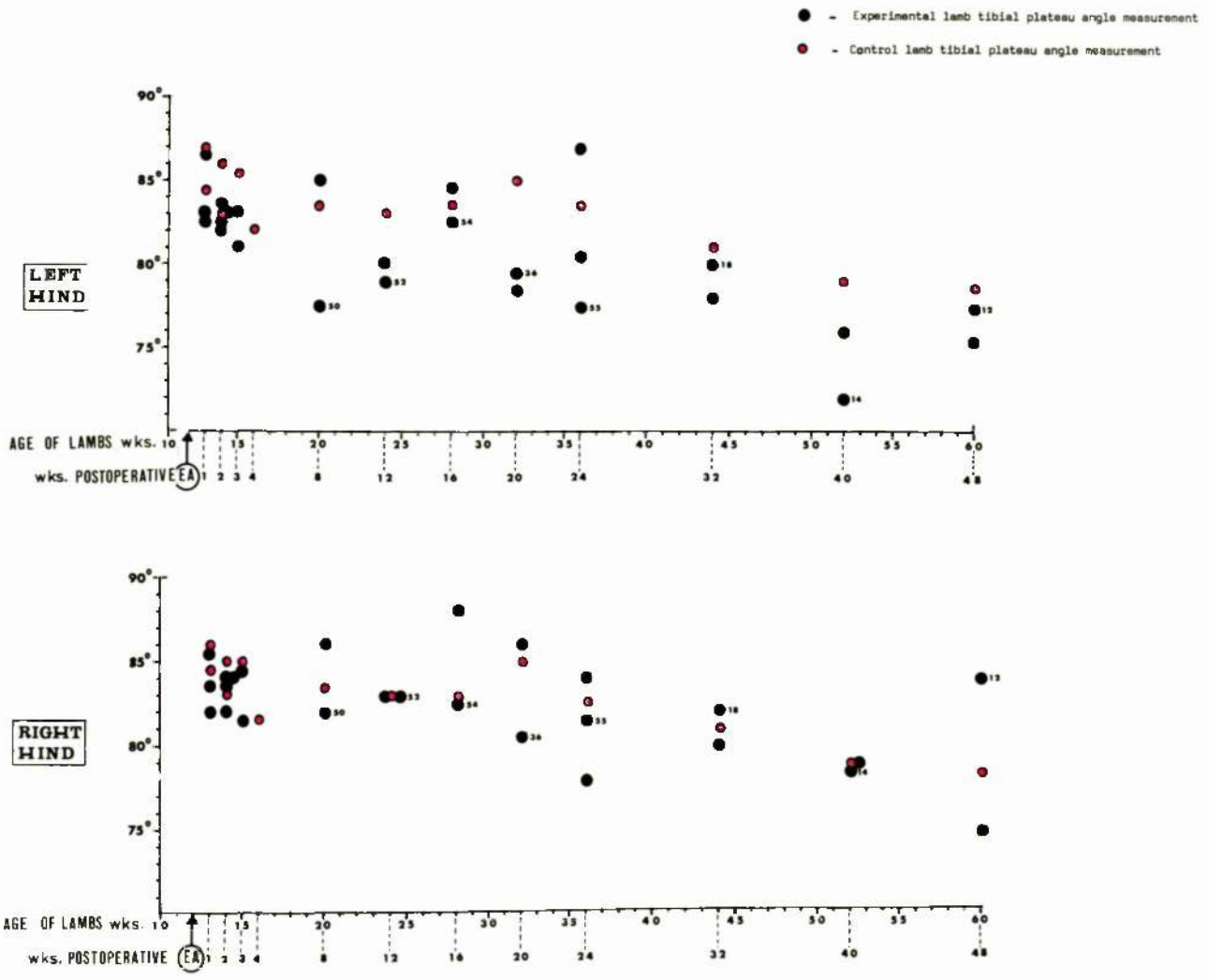


FIG 23 ANTEVERSION ANGLE

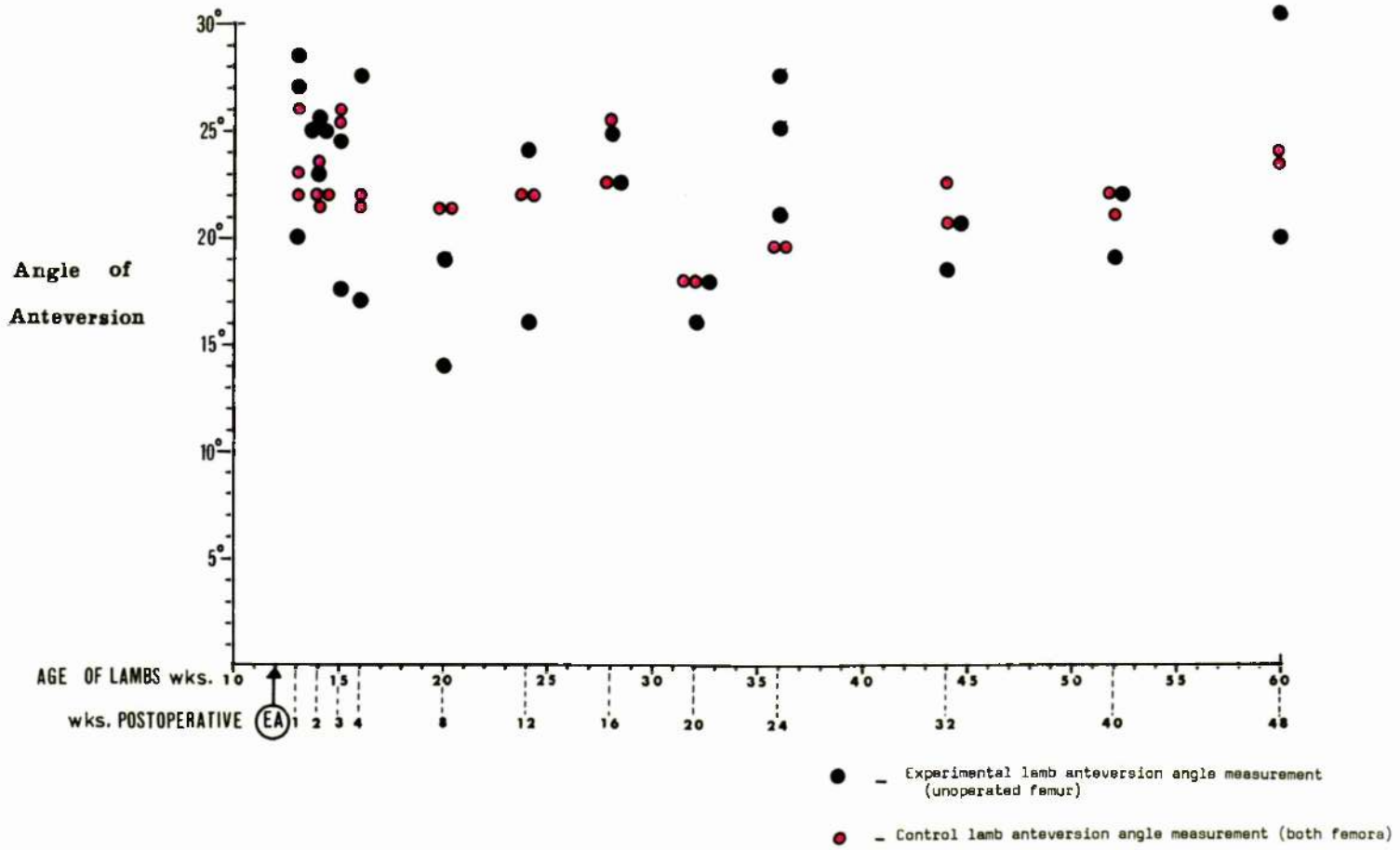


FIG 24 **ANTEVERSION ANGLE** - BOILED OUT SPECIMENS

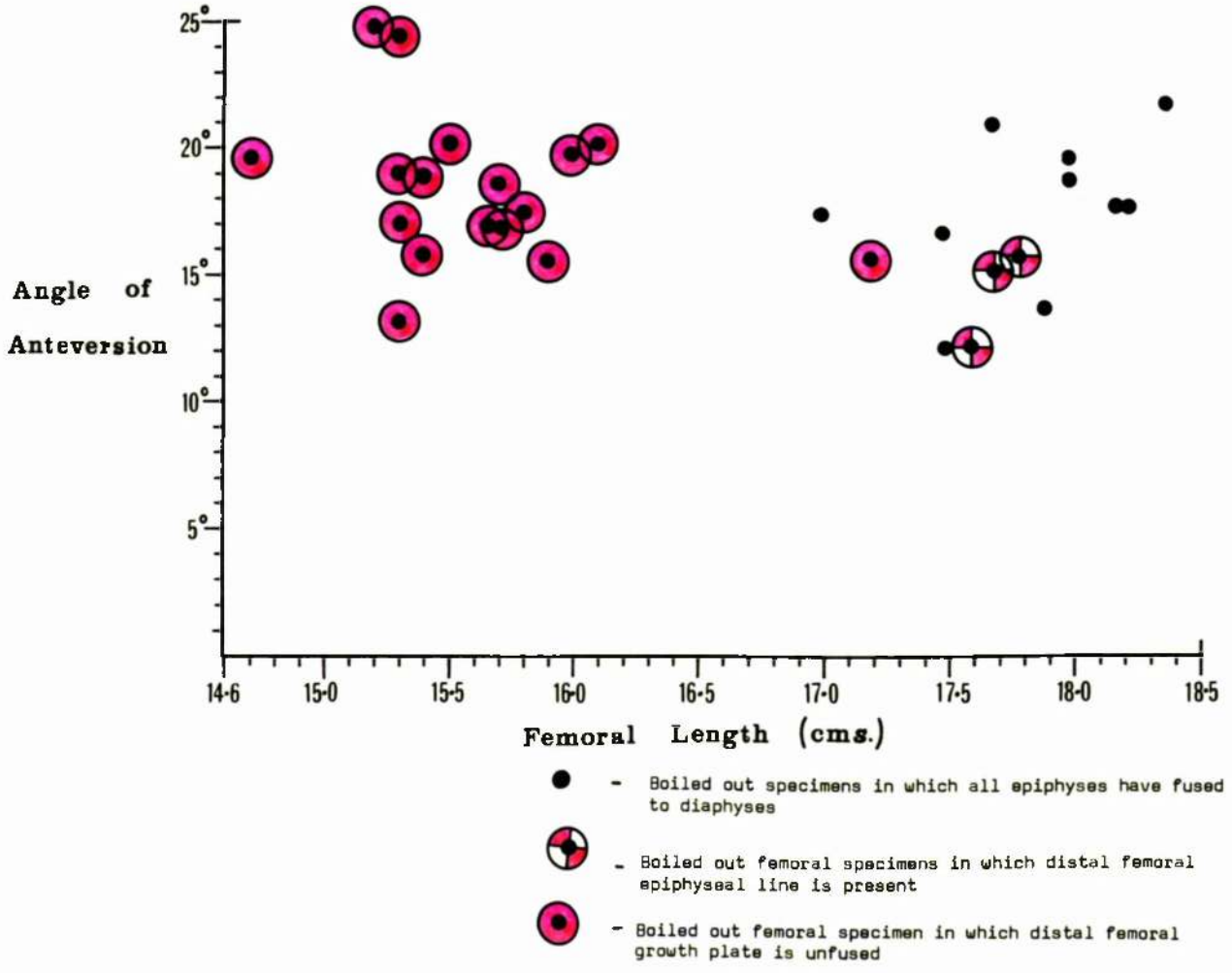
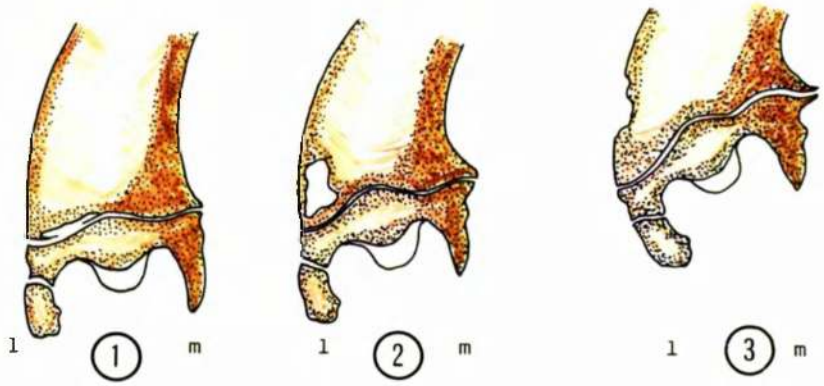
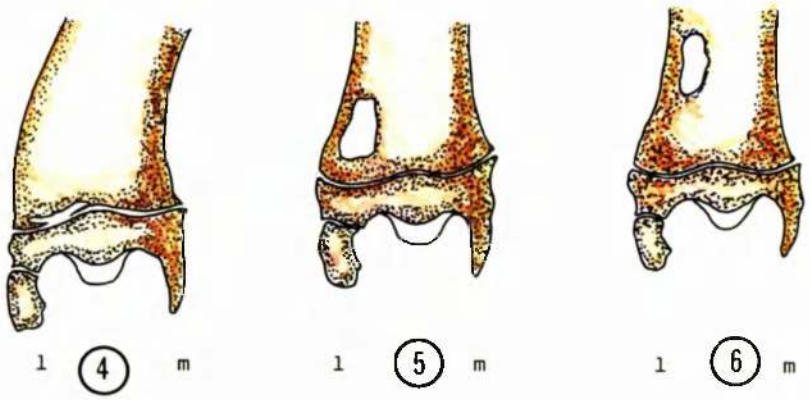


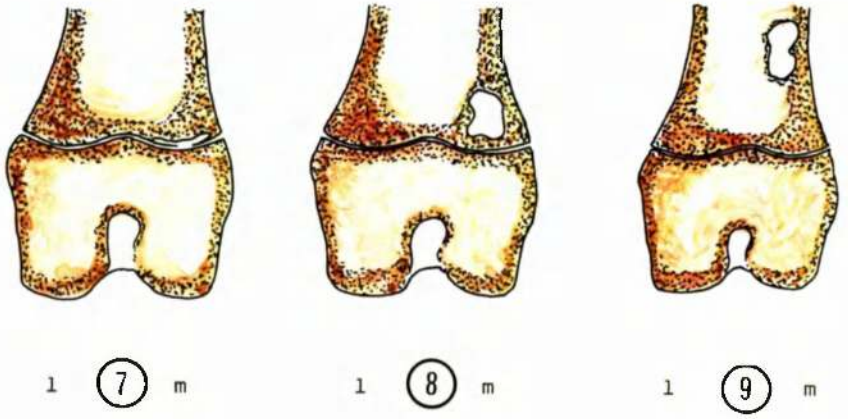
FIG. **25** METAPHYSEAL DEFECTS



(1)—(3) - Suggested aetiology of metaphyseal defects in experimental lambs



(4)—(6) - Suggested outcome of varus deformity correction in experimental lambs

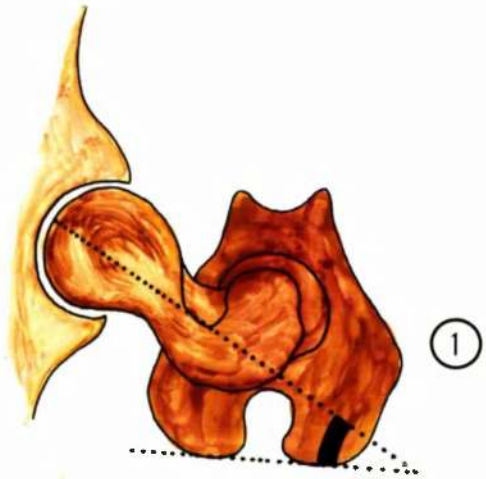


(7)—(9) - Suggested aetiology of metaphyseal fibrous defects in distal femur of growing children

l = lateral aspect

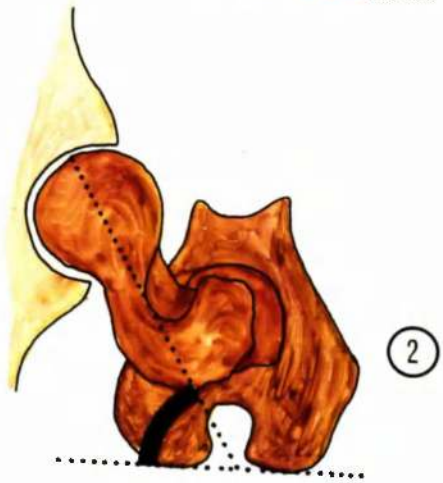
m = medial aspect

FIG. **26** ANTEVERSION ANGLE



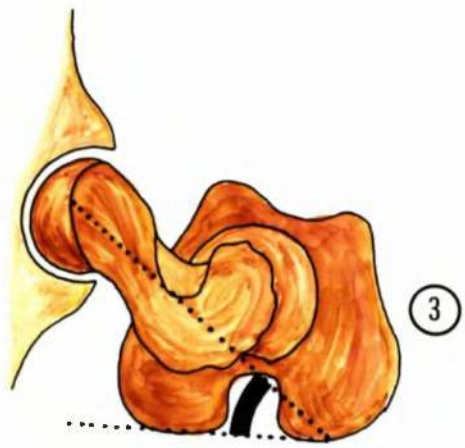
(1) PHYSIOLOGICAL ANTEVERSION

Anteversion of this magnitude is not manifested by axial rotational abnormality at the knee (stifle) joint. In such cases anteversion is accounted for by the normal position of the femoral head in the acetabulum.



(2) INCREASED ANTEVERSION

Exaggerated diagrammatic representation of the manner by which a degree of increased anteversion could be accommodated by the hip joint without axial rotation occurring at the knee. A slight change in the normal position of the femoral head in the acetabulum could absorb an equal amount of increased anteversion.



(3) EPIPHYSIS OF FEMORAL HEAD IN MAN

In a large number of human femora it is reported that the femoral head is not centred over the femoral neck. If there is a degree of increased anteversion but the child is voluntarily placing the limb in a neutral rather than in a medially rotated position there will be opposing forces established in the proximal femur. Postural correction will be tending to outwardly rotate the femur whereas the anchorage of the femoral head by the teres ligament will be opposing such an action. Possibly such a mechanical environment results in asymmetrical positioning of the femoral head on the femoral neck. Similarly retroversion could result in a mirror. image asymmetry.

fig 27

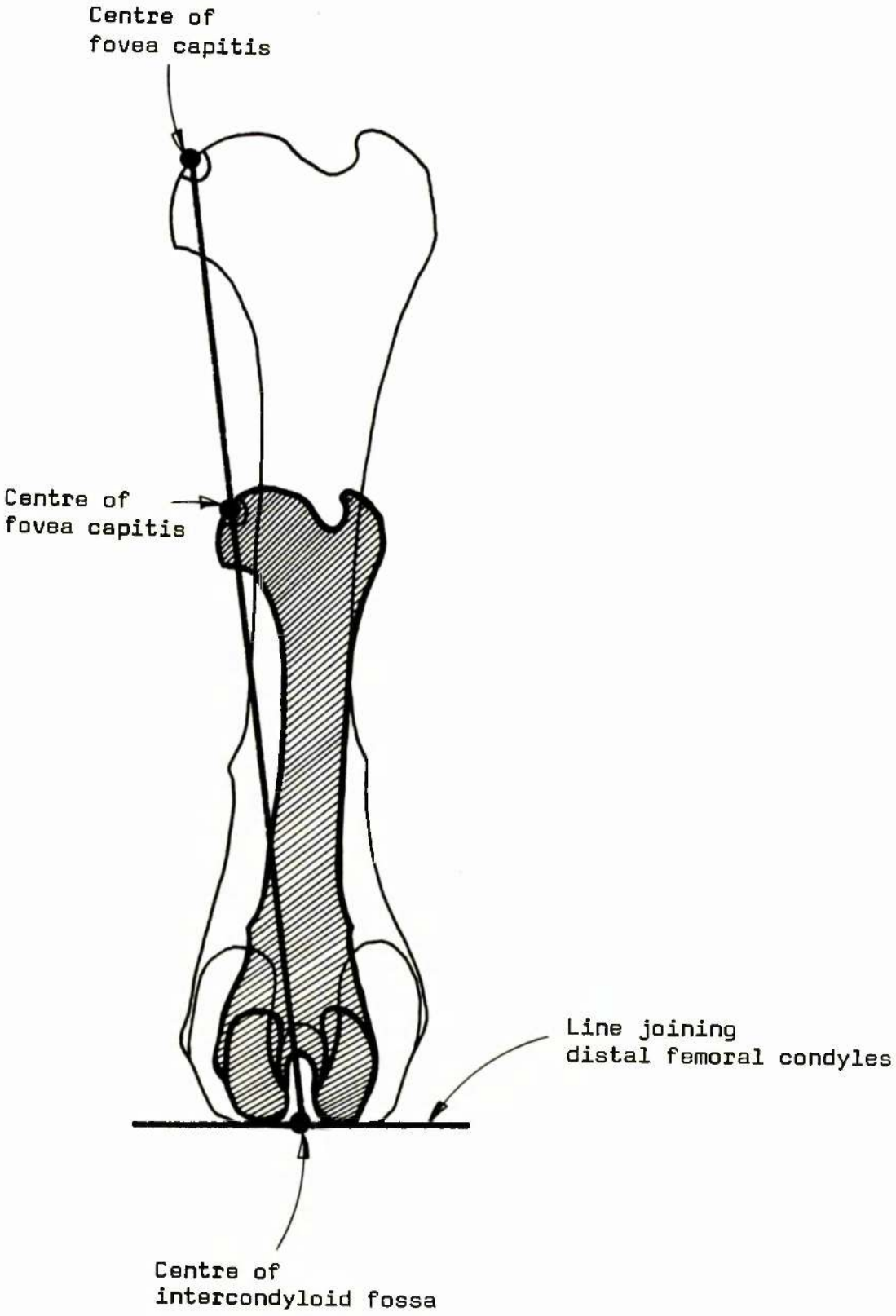

Centre of
fovea capitis
Centre of
fovea capitis
Line joining
distal femoral condyles
Centre of
intercondyloid fossa

FIG 28 REPRESENTATION OF GROWTH CARTILAGE ANATOMY

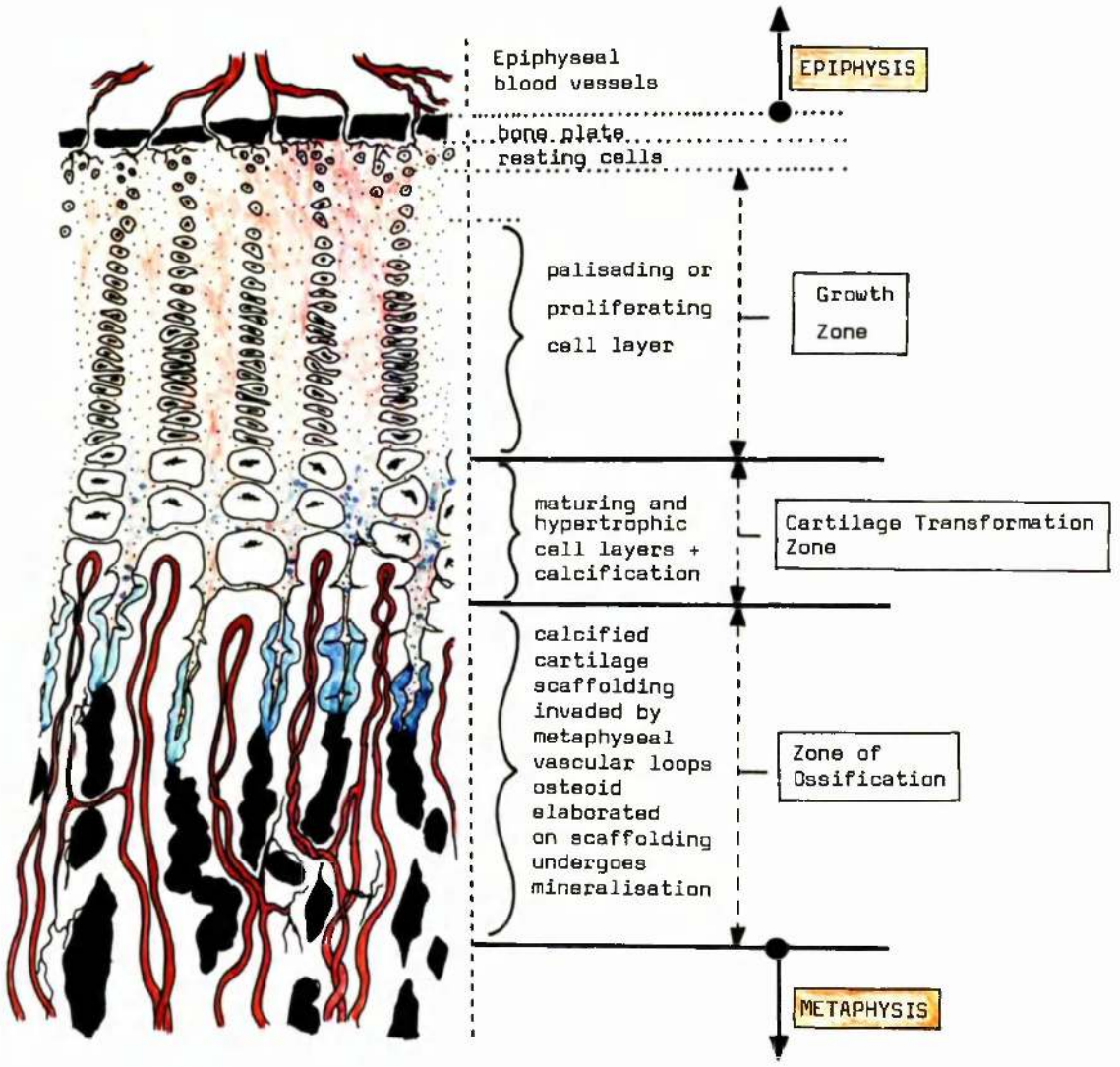
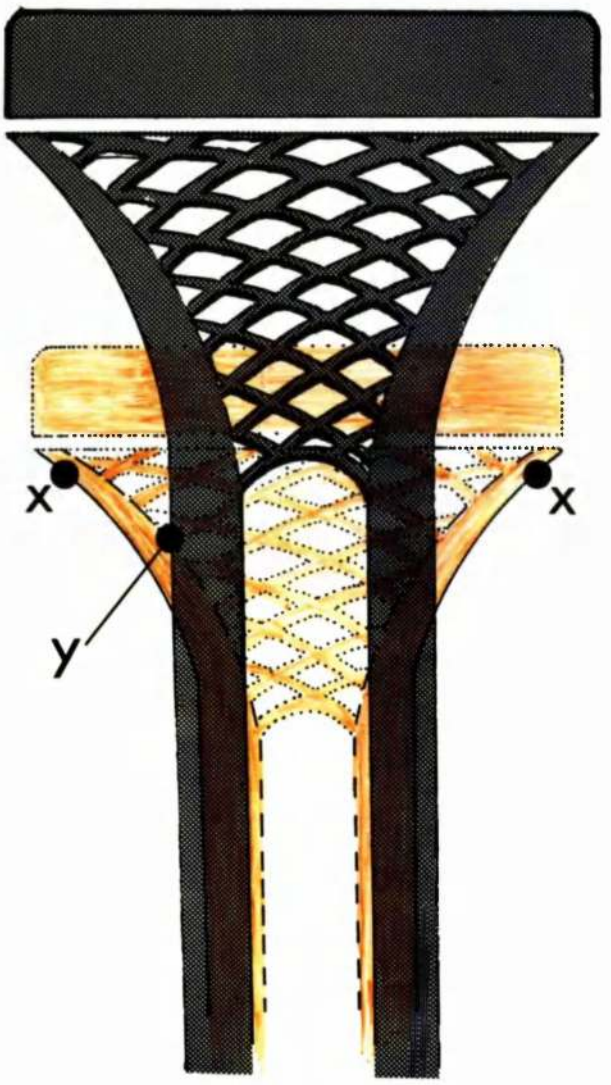


FIG 29 METAPHYSEAL REMODELLING



Metaphyseal remodelling - In order that bone growth can occur without abnormal diaphyseal contour reduction of metaphyseal width (points X) is essential and occurs by surface remodelling

Area relocation - Such remodelling of the metaphysis is in essence one of area relocation. For example, whereas point Y is endosteal initially with growth it comes to lie at a periosteal site on the diaphysis

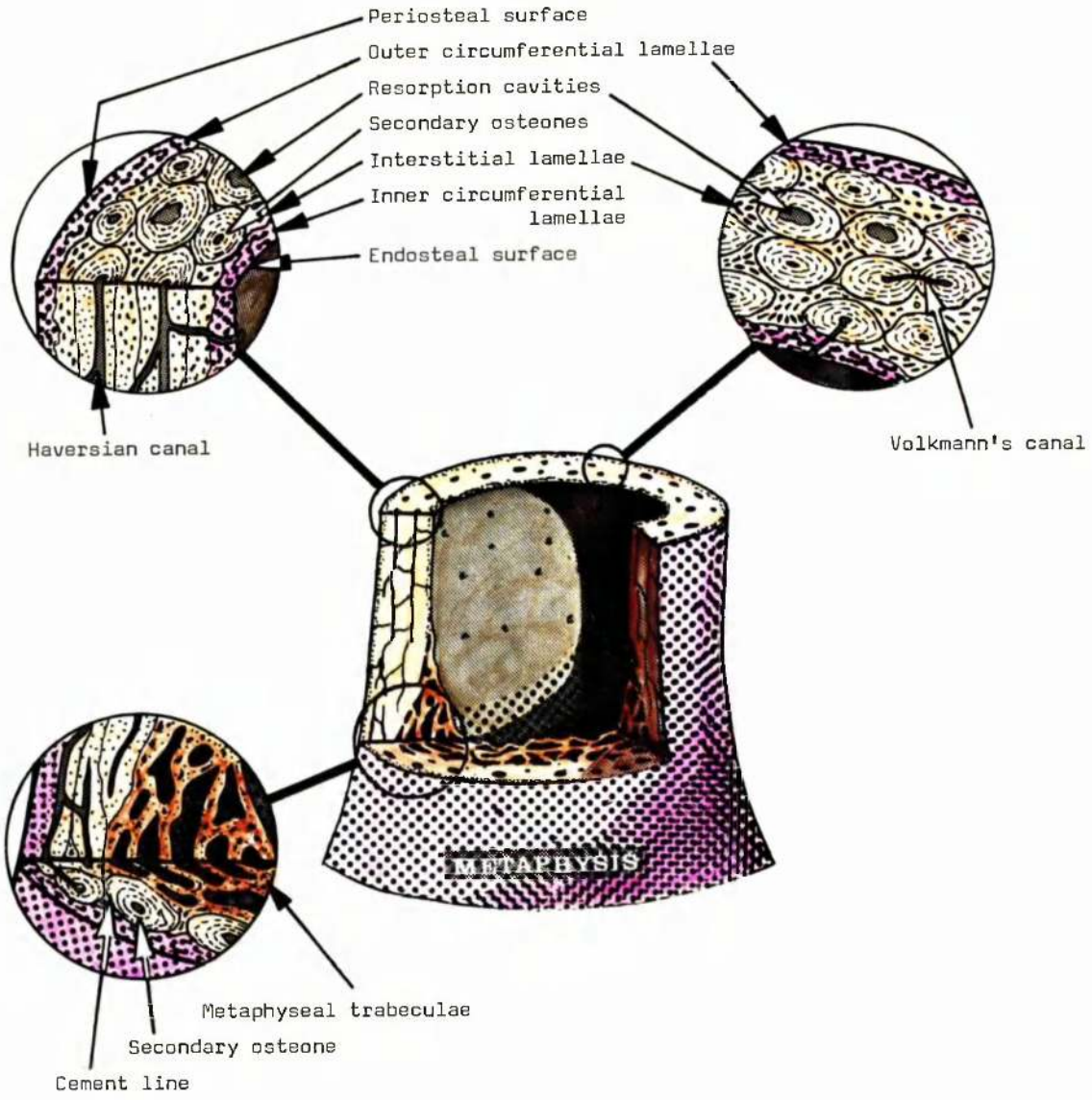
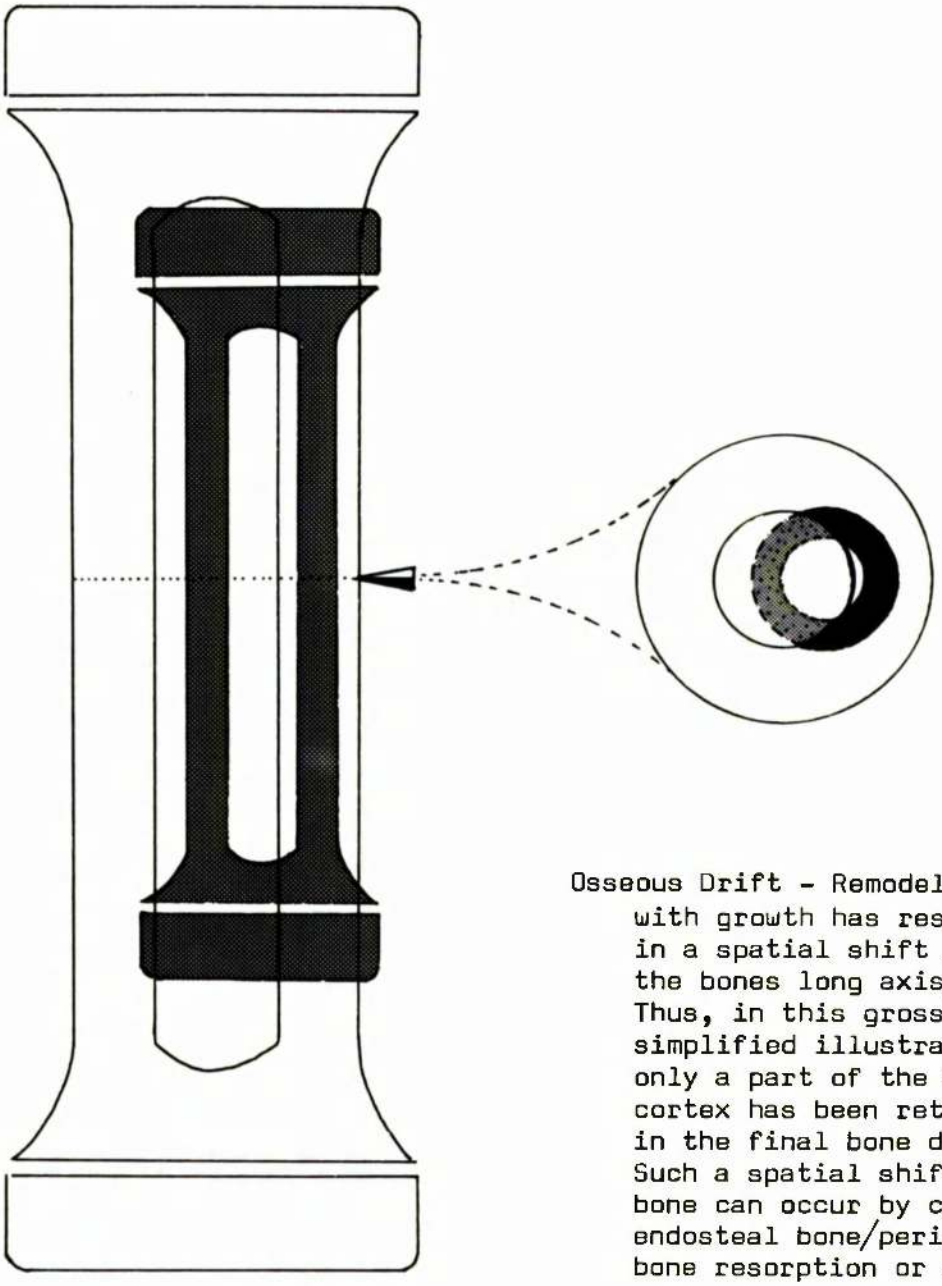


FIG. 30 BASIC CONCEPT OF CORTICAL BONE ANATOMY.

FIG 31 OSSEOUS DRIFT



Osseous Drift - Remodelling with growth has resulted in a spatial shift in the bones long axis. Thus, in this grossly simplified illustration, only a part of the original cortex has been retained in the final bone diaphysis. Such a spatial shift in a bone can occur by controlled endosteal bone/periosteal bone resorption or deposition

FIG.32 Apparatus used for limb perfusion

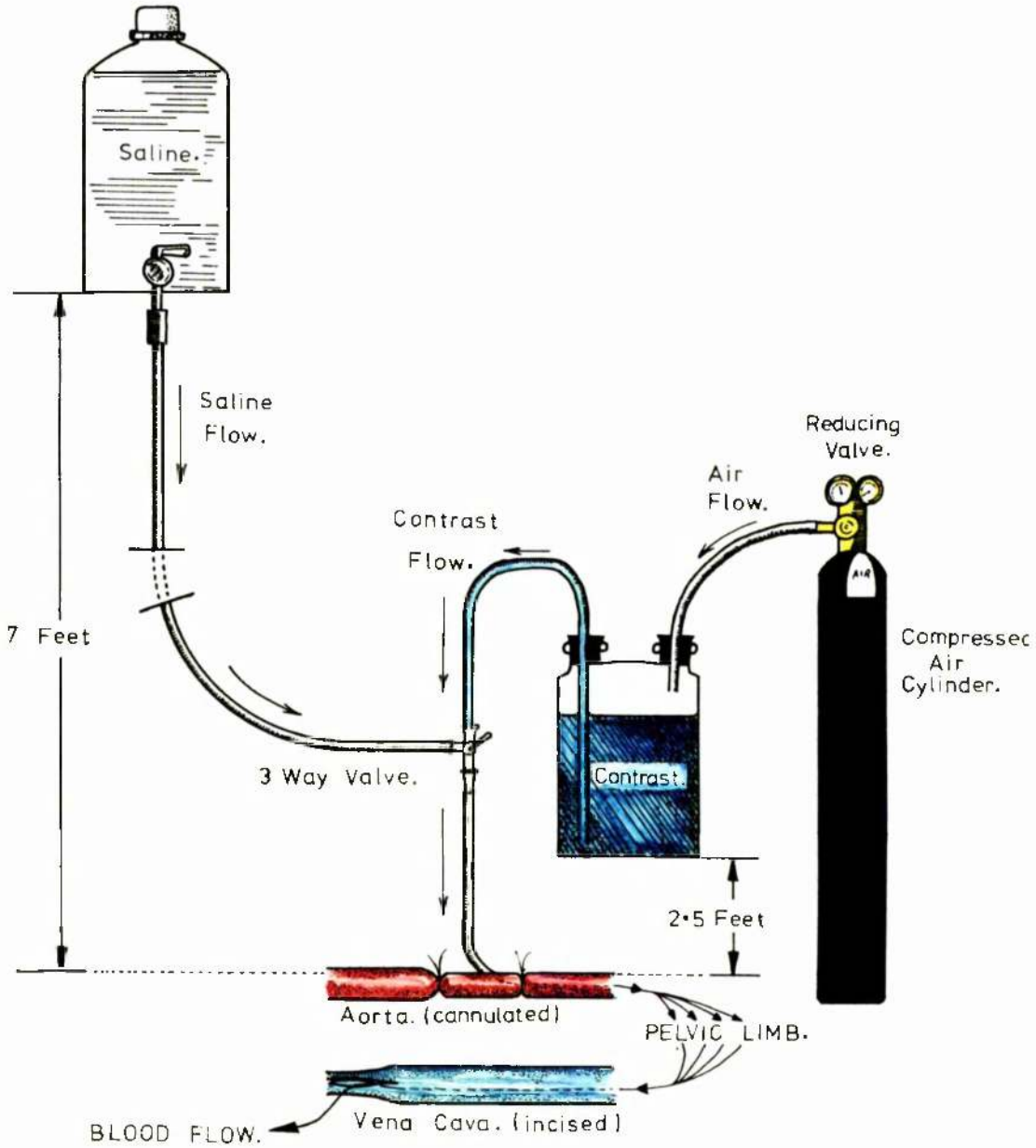


FIG. 33 Samples taken from dissected bones

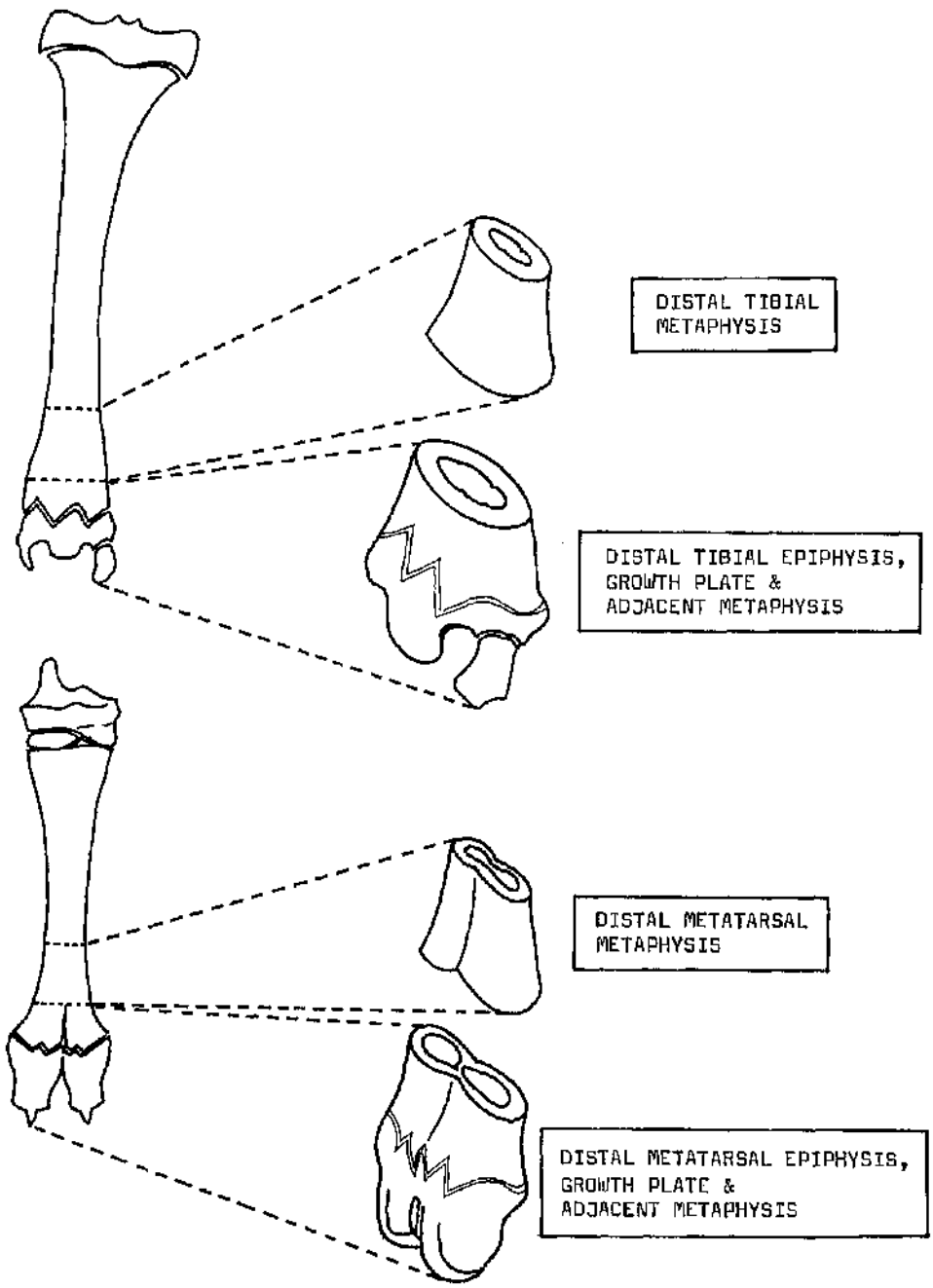
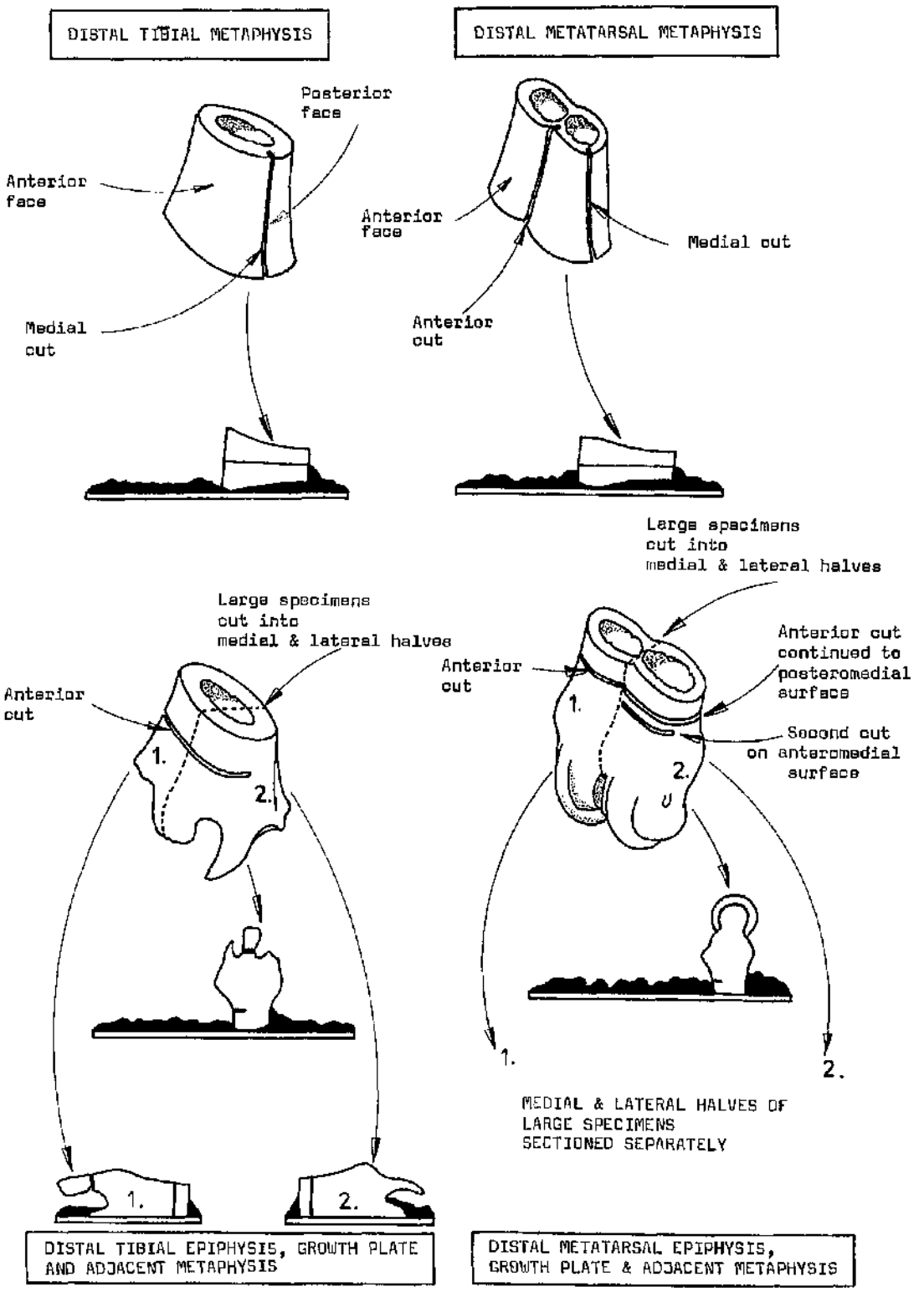


FIG. 34 Method of mounting bone specimens on glass slide



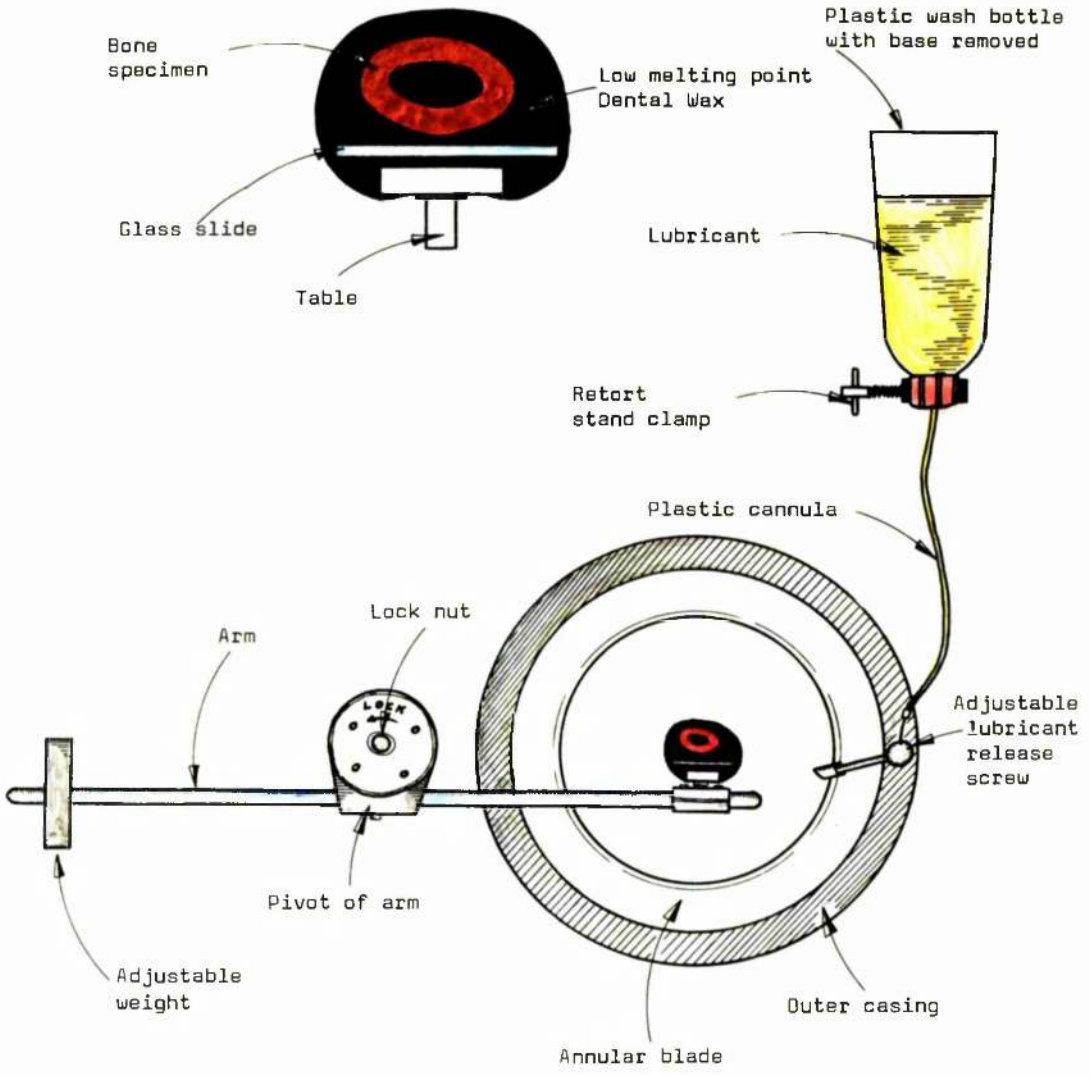


FIG. **35** Method of mounting bone specimens prior to sectioning

FIG. 36 Method of bone sectioning

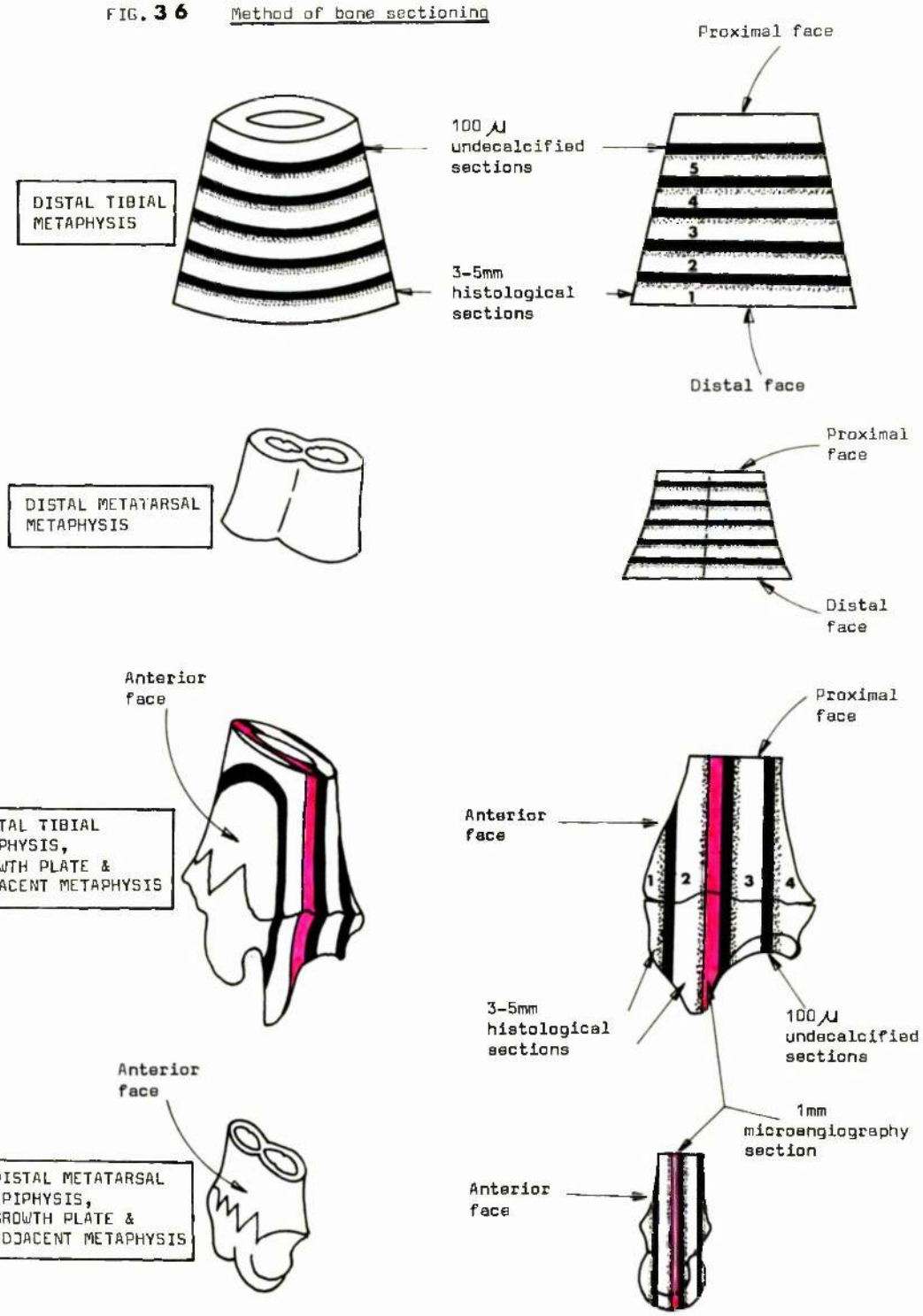


FIG 37 **Predominant Vessel Orientation in Distal Metaphyseal Cortices.**

57 LH DME
60 RH DME
52 RH DTE
14 RH DME
13 LH DTE

57 RH DME	54 RH DTE	19 RH DME
43 LH DME	31 LH DTE	17 LH DTE
60 LH DME	31 LH DME	17 RH DME
50 LH DME	31 RH DME	14 LH DME
50 RH DME	29 LH DTE	15 LH DME
53 LH DTE	29 RH DME	13 RH DME
53 LH DME	36 RH DME	
53 RH DME	35 RH DME	
51 LH DTE	55 RH DME	
52 LH DME	19 LH DTE	

51 LH DME
52 LH DTE
54 LH DME
29 LH DME
36 LH DME
55 LH DME

51 LH DME	39 LH DTE	
52 LH DTE	55 LH DME	
54 LH DME	55 LH DTE	
29 LH DME	17 LH DME	(Ant)
36 LH DME	18 LH DTE	(Ant)
36 LH DTE		
37 LH DME		

36 LH DTE
37 LH DME
39 LH DTE
55 LH DTE
17 LH DME (Ant)
18 LH DTE (Ant)
14 LH DTE

14 LH DTE
12 LH DTE

KEY

(Ant) = Anterior section
DTE = Distal tibial epiphysis and metaphysis
DME = Distal metatarsal epiphysis and metaphysis

FIG.38a OSTEONAL REMODELLING Distal Tibial Metaphyses.

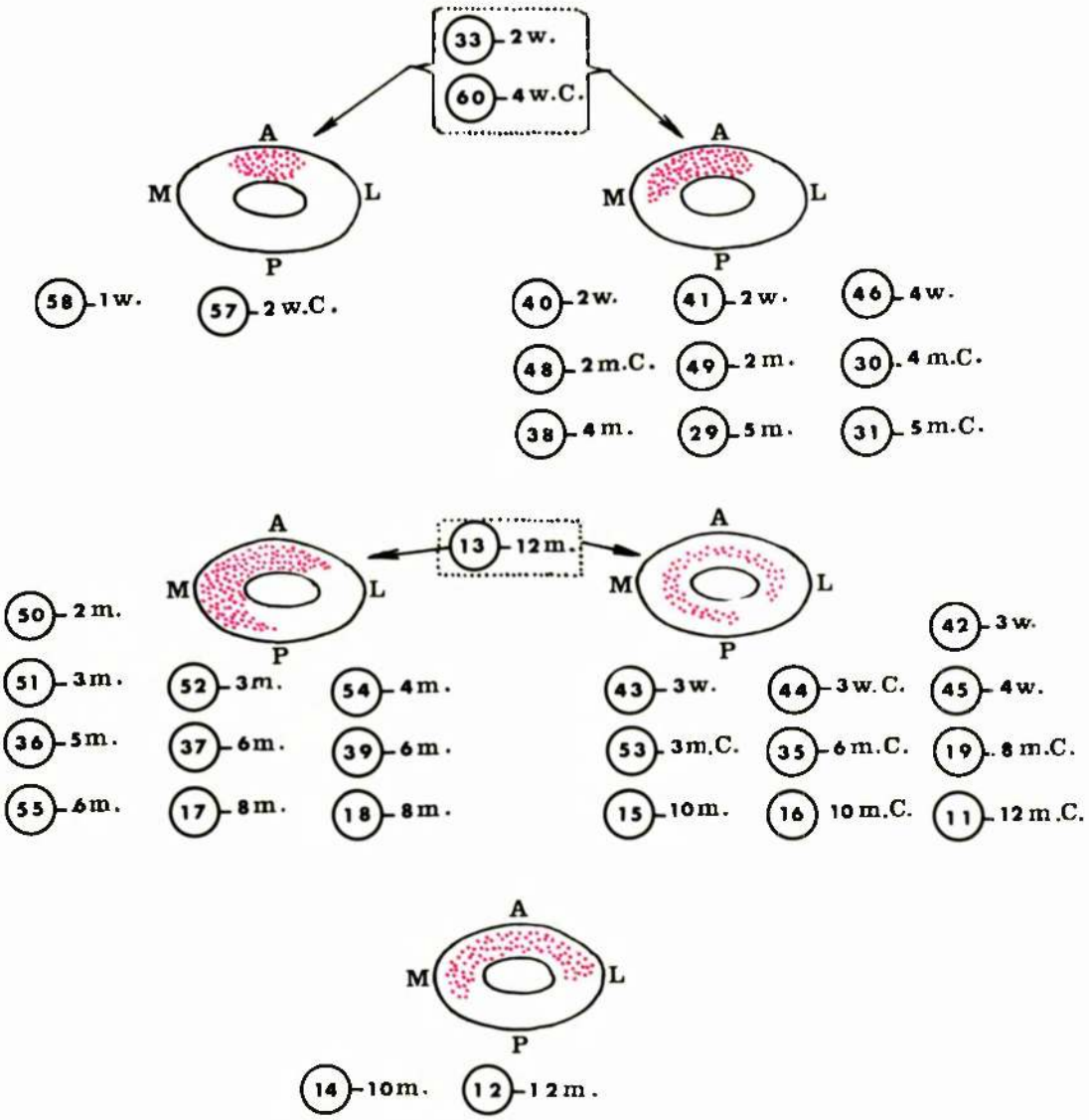


FIG. 38b OSTEONAL REMODELLING Distal Metatarsal Metaphyses.

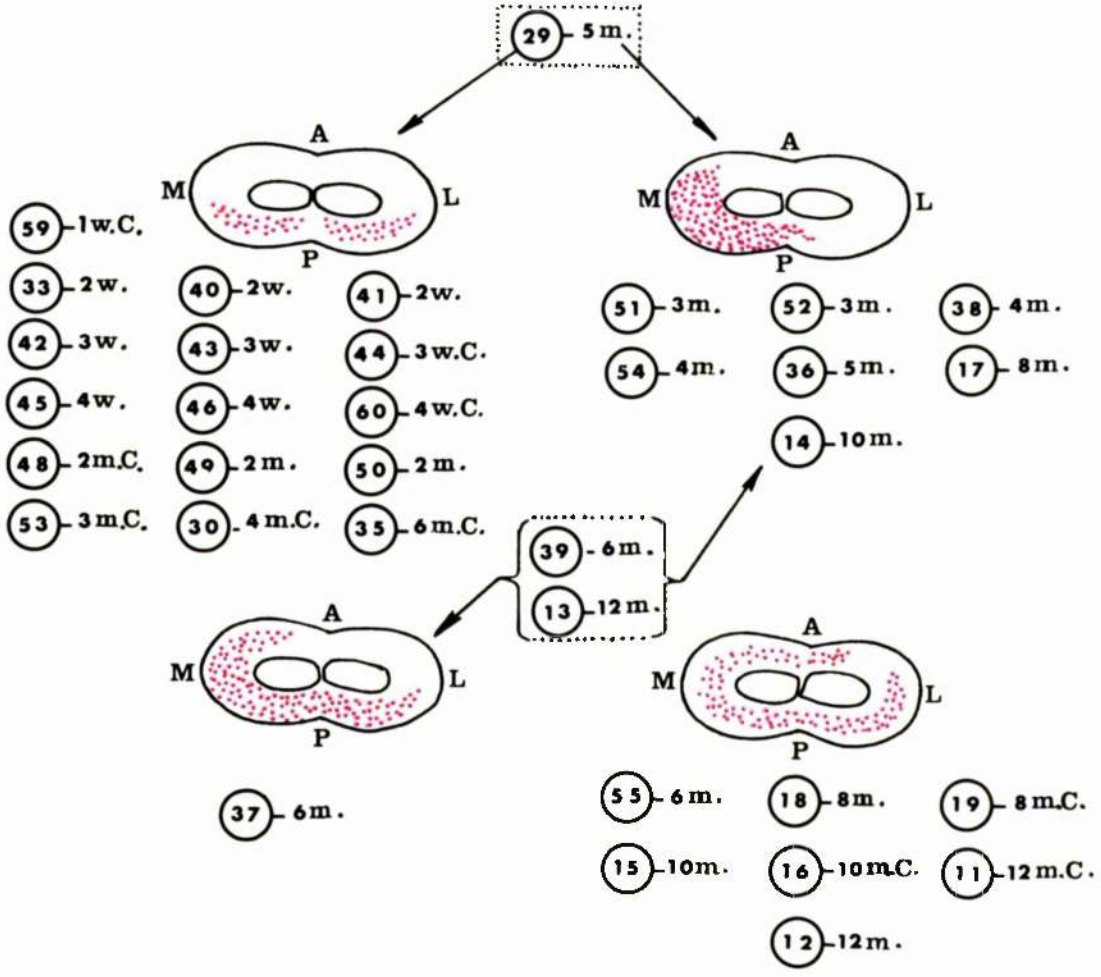
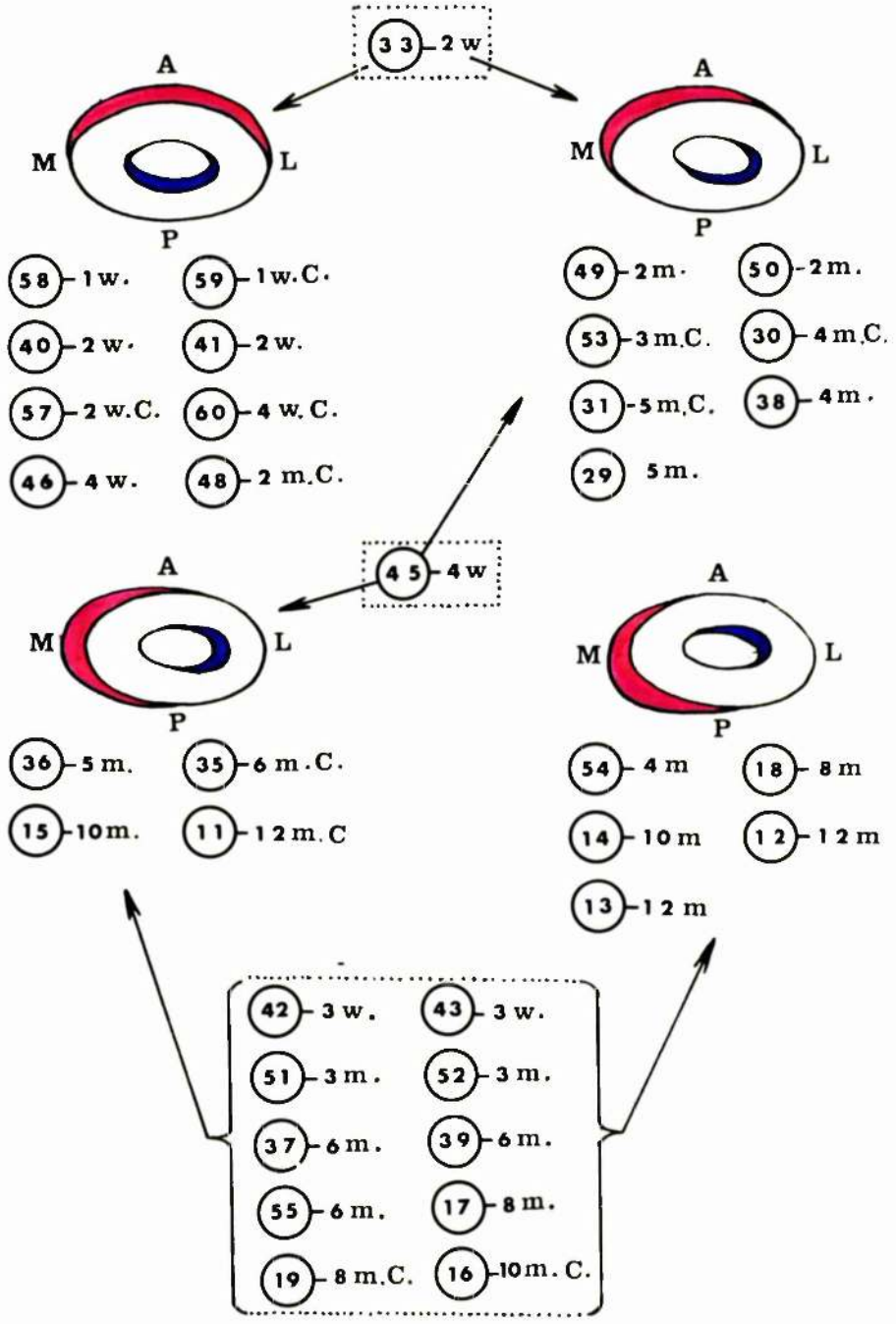


FIG.39a CORTICAL DRIFT Distal Tibial Metaphyses.



A = Anterior.
P = Posterior.
M = Medial.
L = Lateral.

C = Control lamb.
(-) = lamb number.
- w. or - m = postoperative survival period..weeks or months

FIG. 39b CORTICAL DRIFT Distal Metatarsal Metaphyses.

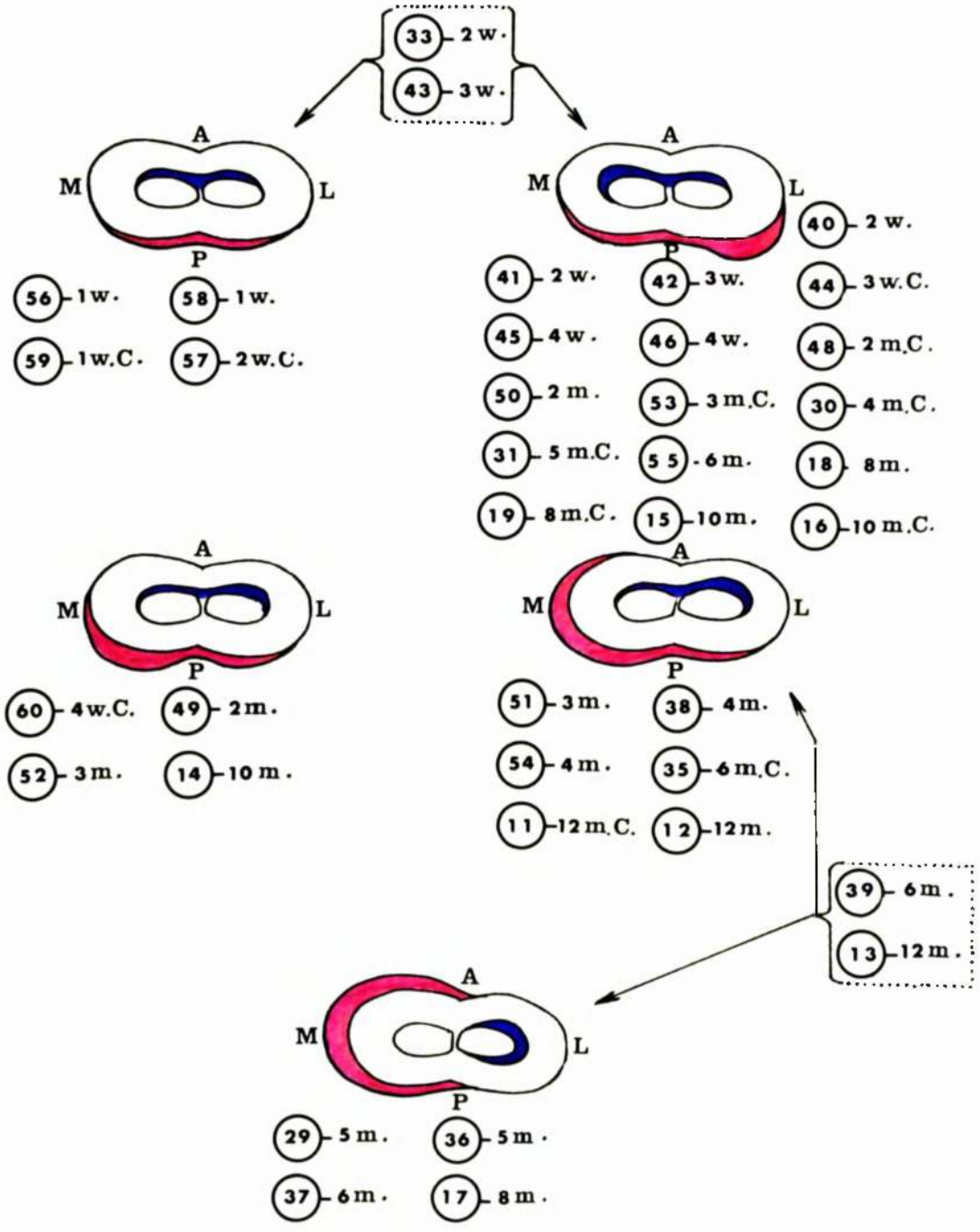


fig 40 a

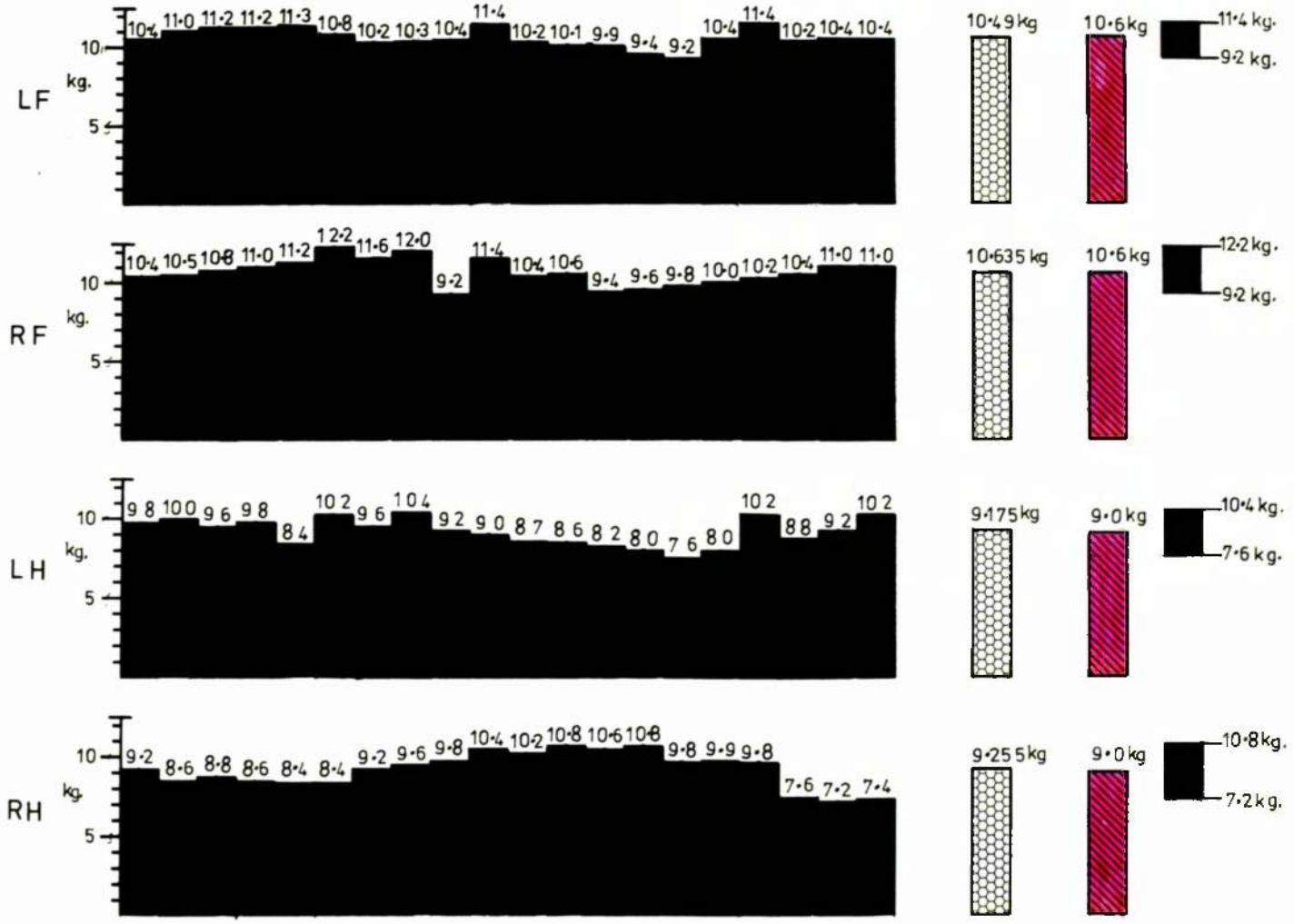

LF
RF
LH
RH
kg.
10
5
10·4 11·0 11·2 11·2 11·3 10·8 10·2 10·3 10·4 11·4 10·2 10·1 9·9 9·4 9·2 10·4 11·4 10·2 10·4 10·4
10·4 10·5 10·8 11·0 11·2 12·2 11·6 12·0 9·2 11·4 10·4 10·6 9·4 9·6 9·8 10·0 10·2 10·4 11·0 11·0
9 8 10 0 9 6 9 8 8 4 10 2 9 6 10 4 9 2 9 0 8 7 8 6 8 2 8 0 7 6 8 0 10 2 8 8 9 2 10 2
9·2 8·6 8·8 8·6 8·4 8·4 9·2 9·6 9·8 10·4 10·2 10·8 10·6 10·8 9·8 9·9 9·8 7·6 7·2 7·4
10·49kg
10·6kg
11·4 kg.
9·2 kg.
10·635 kg
10·6kg
12·2kg.
9·2 kg.
9·175kg
9·0kg
10·4 kg.
7·6 kg.
9·255kg
9·0kg
10·8kg.
7·2kg.

fig 40 b

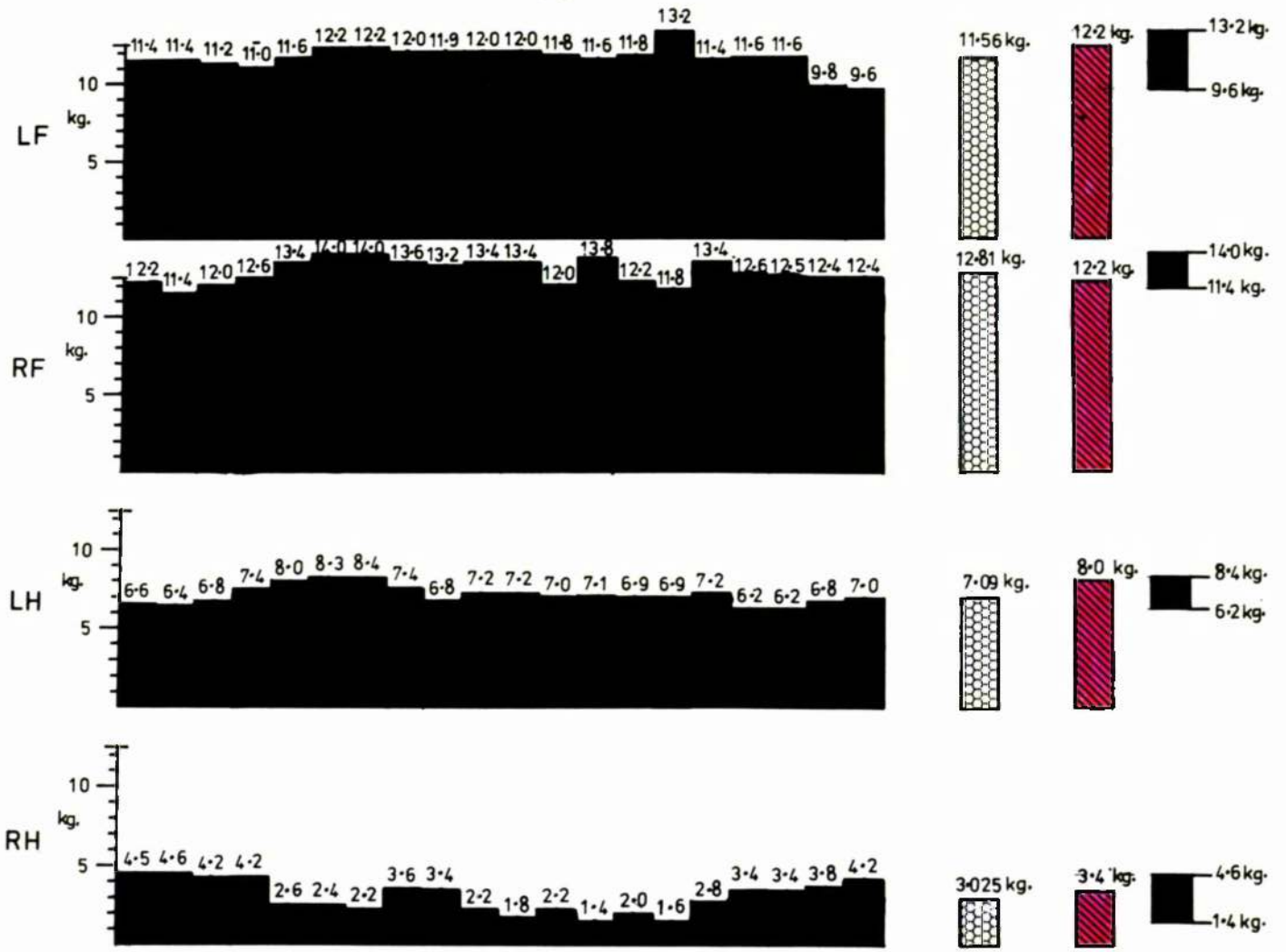

LF
RF
LH
RH
kg.
10
5
11·4 11·4 11·2 11·0 11·6 12·2 12·2 12·0 11·9 12·0 12·0 11·8 11·6 11·8 13·2 11·4 11·6 11·6 9·8 9·6
12·2 11·4 12·0 12·6 13·4 13·6 13·2 13·4 13·4 12·0 13·8 12·2 11·8 13·4 12·6 12·5 12·4 12·4
6·6 6·4 6·8 7·4 8·0 8·3 8·4 7·4 6·8 7·2 7·2 7·0 7·1 6·9 6·9 7·2 6·2 6·2 6·8 7·0
4·5 4·6 4·2 4·2 2·6 2·4 2·2 3·6 3·4 2·2 1·8 2·2 1·4 2·0 1·6 2·8 3·4 3·4 3·8 4·2
11·56 kg.
12·2 kg.
13·2 kg.
9·6 kg.
12·81 kg.
12·2 kg.
14·0 kg.
11·4 kg.
7·09 kg.
8·0 kg.
8·4 kg.
6·2 kg.
3·025 kg.
3·4 kg.
4·6 kg.
1·4 kg.

fig 40c

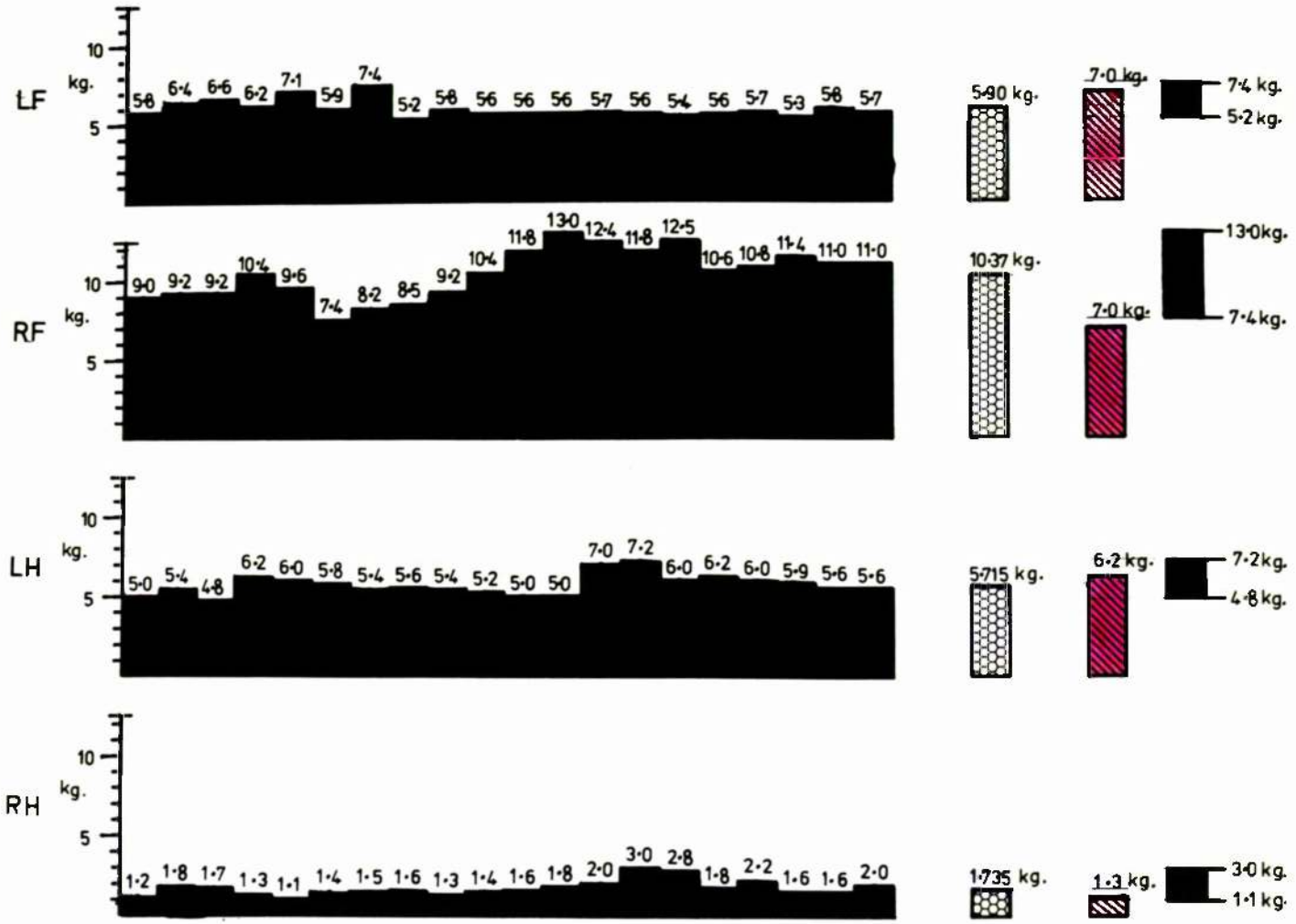

LF
kg.
10
5
5·8 6·4 6·6 6·2 7·1 5·9 7·4 5·2 5·8 5·6 5·6 5·6 5·7 5·6 5·4 5·6 5·7 5·3 5·8 5·7
5·90 kg.
7·0 kg.
7·4 kg.
5·2 kg.
RF
kg.
10
5
9·0 9·2 9·2 10·4 9·6 7·4 8·2 8·5 9·2 10·4 11·8 13·0 12·4 11·8 12·5 10·6 10·8 11·4 11·0 11·0
10·37 kg.
7·0 kg.
13·0kg.
7·4kg.
LH
kg.
10
5
5·0 5·4 4·8 6·2 6·0 5·8 5·4 5·6 5·4 5·2 5·0 5·0 7·0 7·2 6·0 6·2 6·0 5·9 5·6 5·6
5·715 kg.
6·2 kg.
7·2kg.
4·8 kg.
RH
kg.
10
5
1·2 1·8 1·7 1·3 1·1 1·4 1·5 1·6 1·3 1·4 1·6 1·8 2·0 3·0 2·8 1·8 2·2 1·6 1·6 2·0
1·735 kg.
1·3 kg.
3·0 kg.
1·1 kg.

fig 40d

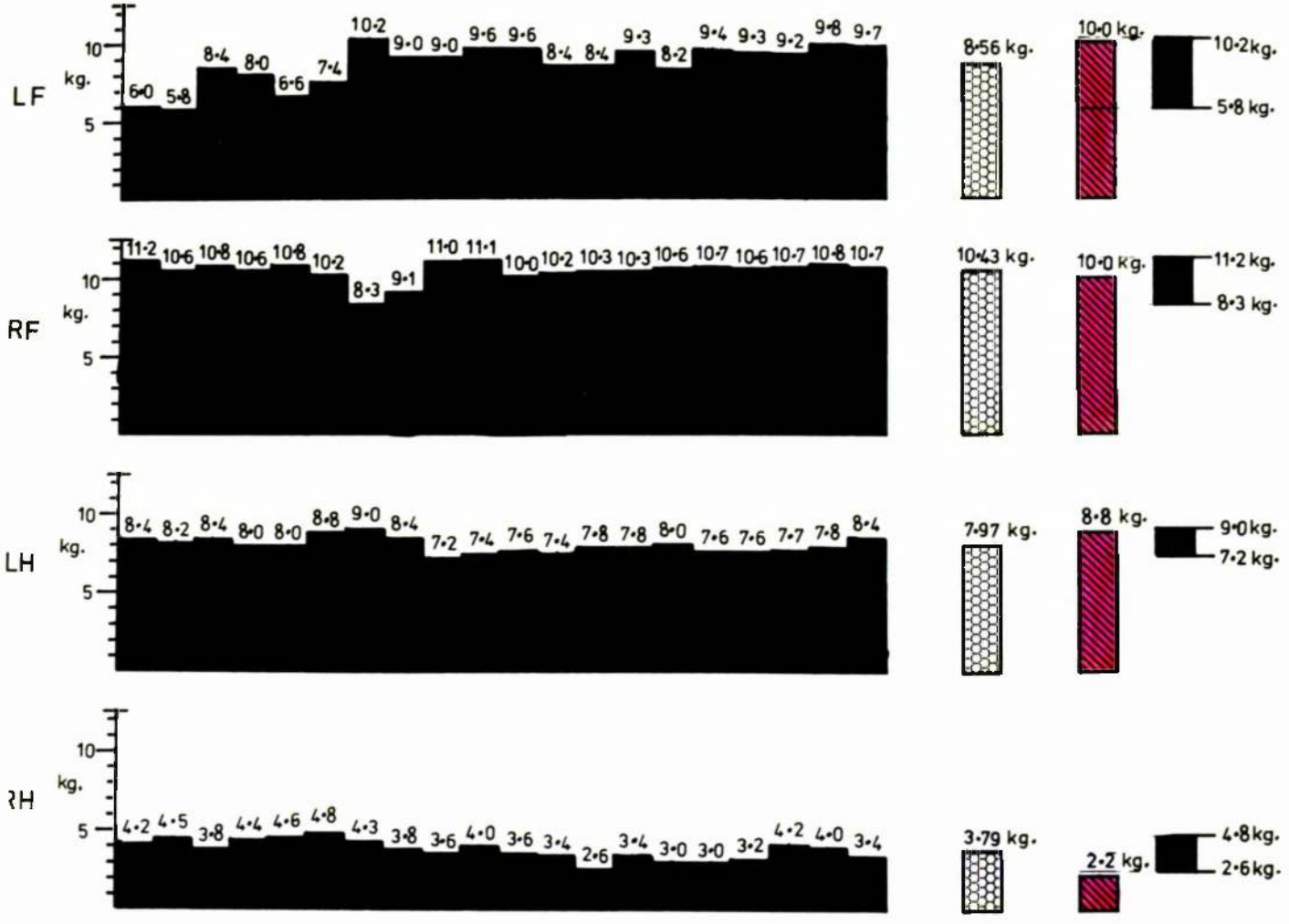

LF
kg.
10
5
6·0 5·8 8·4 8·0 6·6 7·4 10·2 9·0 9·0 9·6 9·6 8·4 8·4 9·3 8·2 9·4 9·3 9·2 9·8 9·7
8·56 kg.
10·0 kg.
10·2 kg.
5·8 kg.
RF
kg.
10
5
11·2 10·6 10·8 10·6 10·8 10·2 8·3 9·1 11·0 11·1 10·0 10·2 10·3 10·3 10·6 10·7 10·6 10·7 10·8 10·7
10·43 kg.
10·0 kg.
11·2 kg.
8·3 kg.
LH
kg.
10
5
8·4 8·2 8·4 8·0 8·0 8·8 9·0 8·4 7·2 7·4 7·6 7·4 7·8 7·8 8·0 7·6 7·6 7·7 7·8 8·4
7·97 kg.
8·8 kg.
9·0 kg.
7·2 kg.
RH
kg.
10
5
4·2 4·5 3·8 4·4 4·6 4·8 4·3 3·8 3·6 4·0 3·6 3·4 2·6 3·4 3·0 3·0 3·2 4·2 4·0 3·4
3·79 kg.
2·2 kg.
4·8 kg.
2·6 kg.

fig 40e

12

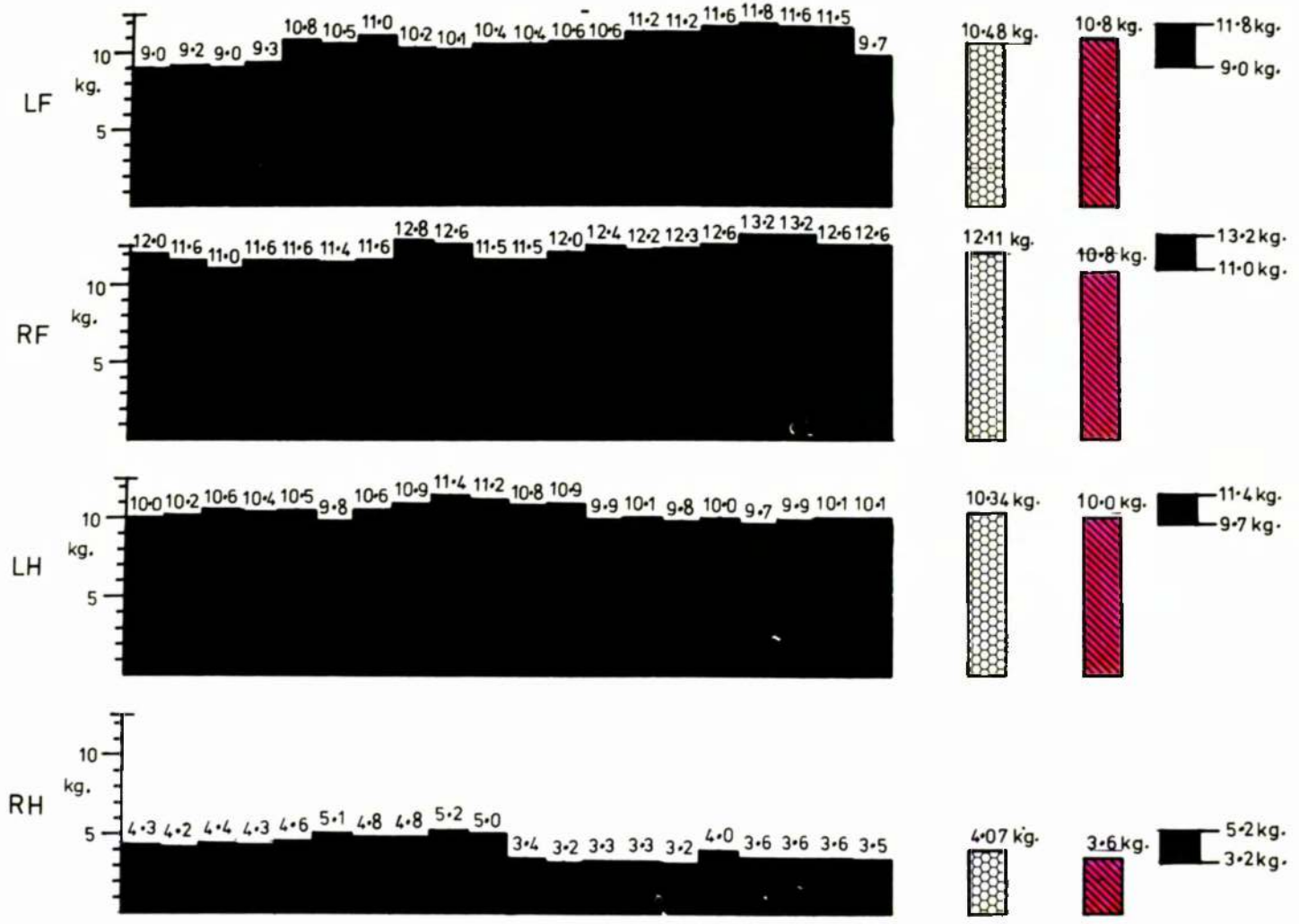

LF
RF
LH
RH
kg.
10
5
9·0 9·2 9·0 9·3 10·8 10·5 11·0 10·2 10·1 10·4 10·4 10·6 10·6 11·2 11·2 11·6 11·8 11·6 11·5 9·7
12·0 11·6 11·0 11·6 11·6 11·4 11·6 12·8 12·6 11·5 11·5 12·0 12·4 12·2 12·3 12·6 13·2 13·2 12·6 12·6
10·0 10·2 10·6 10·4 10·5 9·8 10·6 10·9 11·4 11·2 10·8 10·9 9·9 10·1 9·8 10·0 9·7 9·9 10·1 10·1
4·3 4·2 4·4 4·3 4·6 5·1 4·8 4·8 5·2 5·0 3·4 3·2 3·3 3·3 3·2 4·0 3·6 3·6 3·6 3·5
10·48 kg.
10·8 kg.
11·8 kg.
9·0 kg.
12·11 kg.
10·8 kg.
13·2 kg.
11·0 kg.
10·34 kg.
10·0 kg.
11·4 kg.
9·7 kg.
4·07 kg.
3·6 kg.
5·2 kg.
3·2 kg.

fig 4 of

13

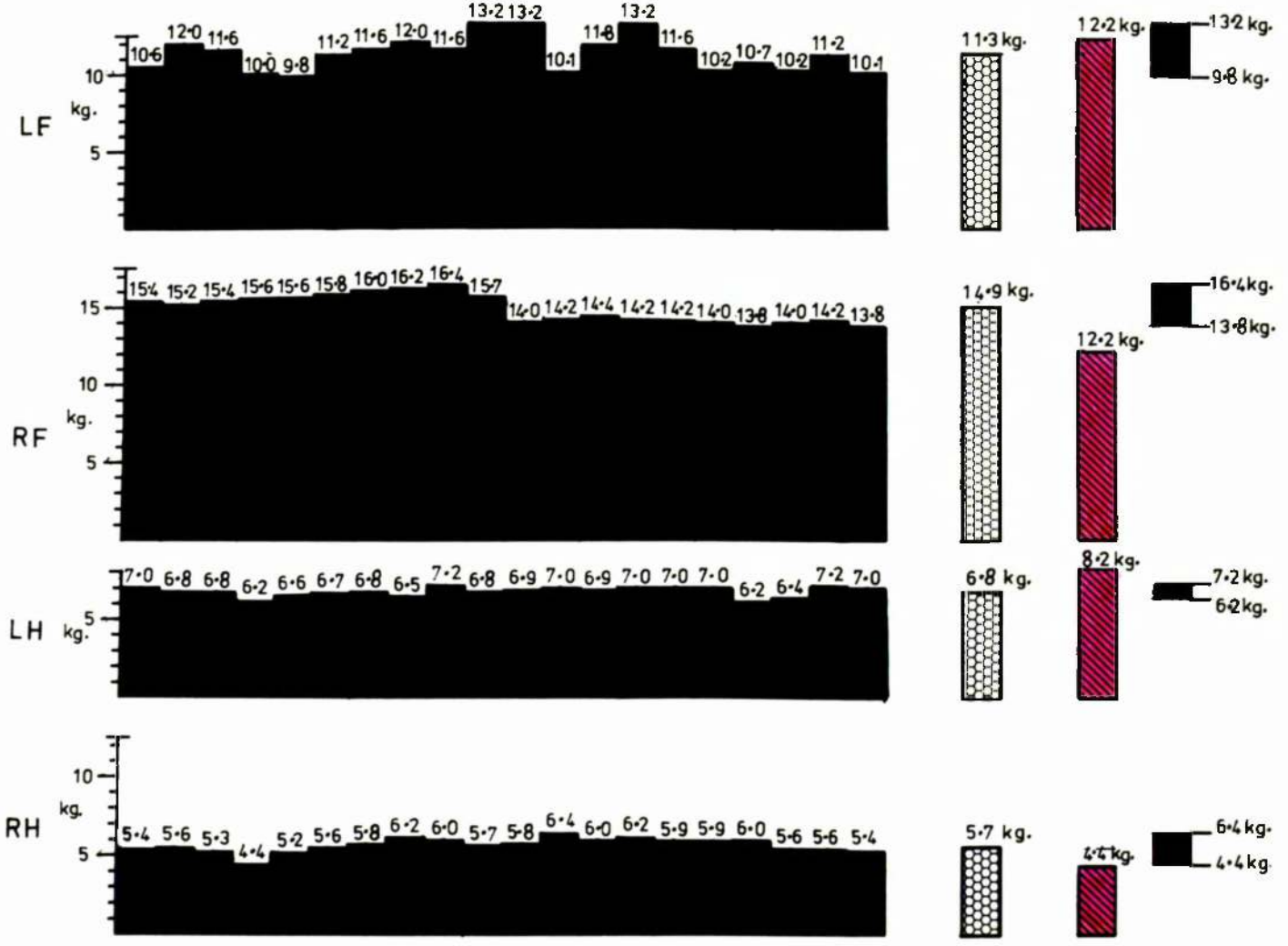

LF
kg.
10
5
10·6 12·0 11·6 10·0 9·8 11·2 11·6 12·0 11·6 13·2 13·2 10·1 11·8 13·2 11·6 10·2 10·7 10·2 11·2 10·1
11·3 kg.
12·2 kg.
13·2 kg.
9·8 kg.
RF
kg.
15
10
5
15·4 15·2 15·4 15·6 15·6 15·8 16·0 16·2 16·4 15·7 14·0 14·2 14·4 14·2 14·2 14·0 13·8 14·0 14·2 13·8
14·9 kg.
12·2 kg.
16·4 kg.
13·8 kg.
LH
kg.
5
7·0 6·8 6·8 6·2 6·6 6·7 6·8 6·5 7·2 6·8 6·9 7·0 6·9 7·0 7·0 7·0 6·2 6·4 7·2 7·0
6·8 kg.
8·2 kg.
7·2 kg.
6·2 kg.
RH
kg.
10
5
5·4 5·6 5·3 4·4 5·2 5·6 5·8 6·2 6·0 5·7 5·8 6·4 6·0 6·2 5·9 5·9 6·0 5·6 5·6 5·4
5·7 kg.
4·4 kg.
6·4 kg.
4·4 kg.

fig 41a

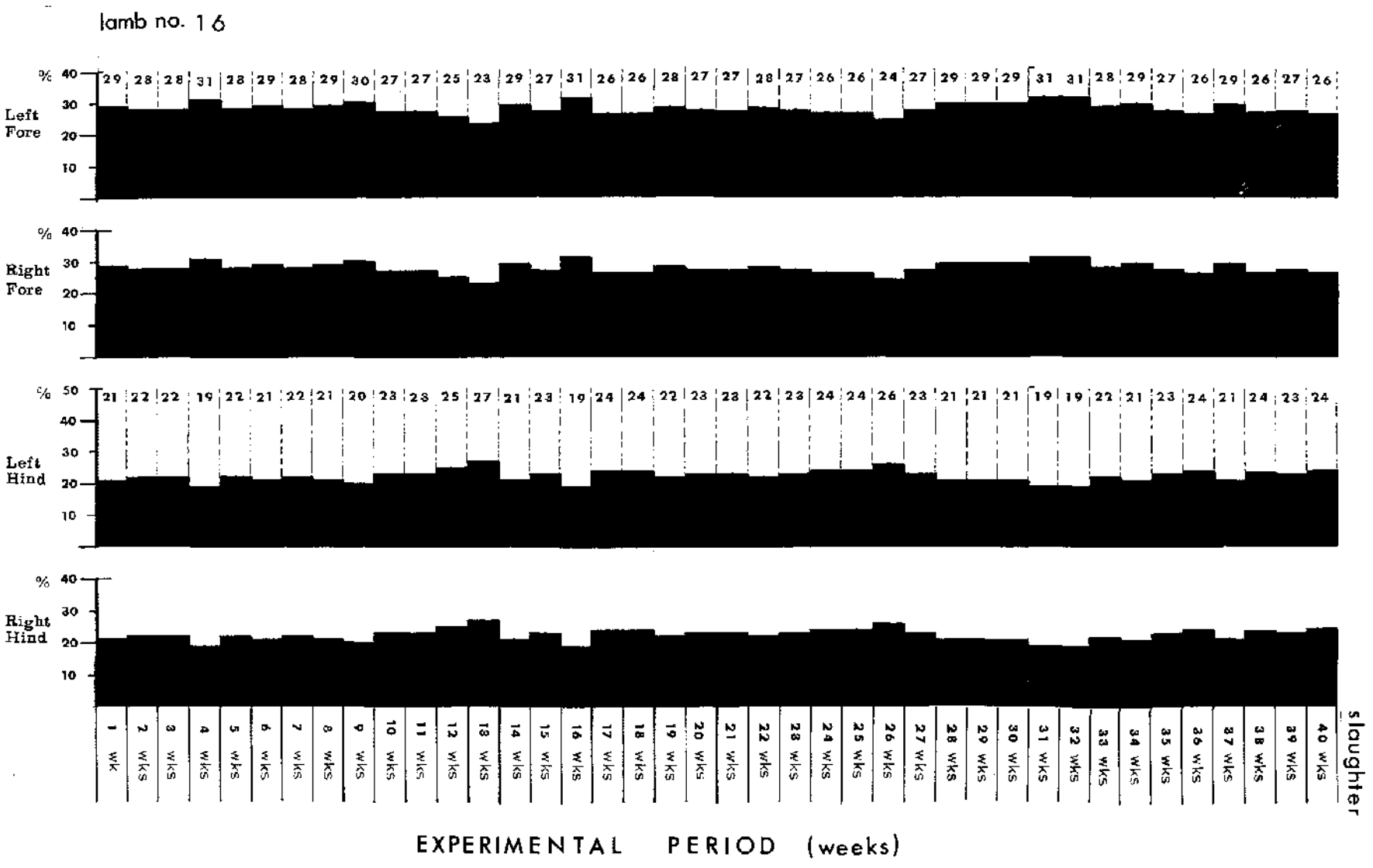

lamb no. 16
Left Fore
Right Fore
Left Hind
Right Hind
%
29 28 28 31 28 29 28 29 30 27 27 25 23 29 27 31 26 26 28 27 27 28 27 26 26 24 27 29 29 29 31 31 28 29 27 26 29 26 27 26
21 22 22 19 22 21 22 21 20 23 23 25 27 21 23 19 24 24 22 23 23 22 23 24 24 26 23 21 21 21 19 19 22 21 23 24 21 24 23 24
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
21 wks
22 wks
23 wks
24 wks
25 wks
26 wks
27 wks
28 wks
29 wks
30 wks
31 wks
32 wks
33 wks
34 wks
35 wks
36 wks
37 wks
38 wks
39 wks
40 wks
slaughter
EXPERIMENTAL PERIOD (weeks)

fig 41b

lamb no. 55

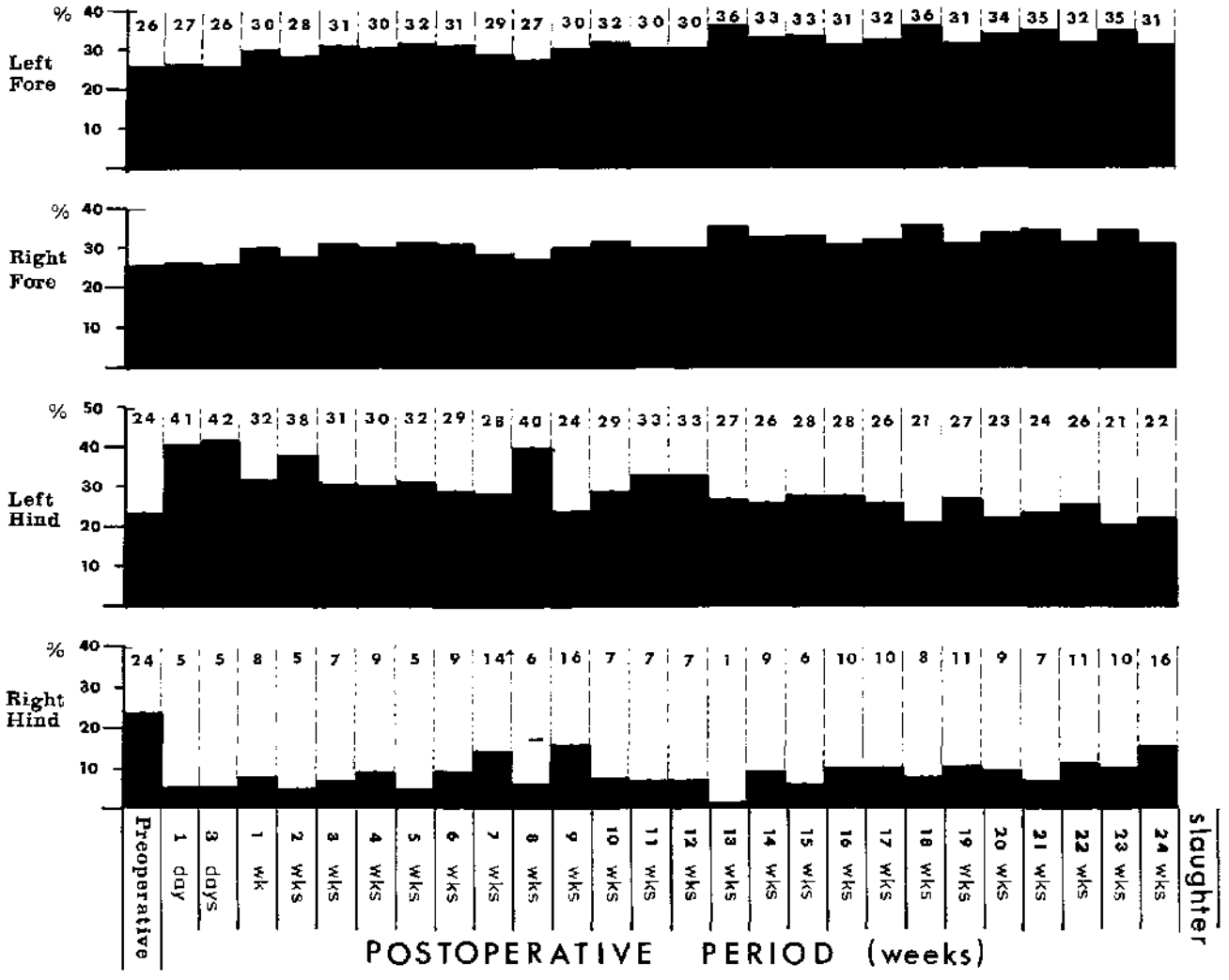


fig 41c

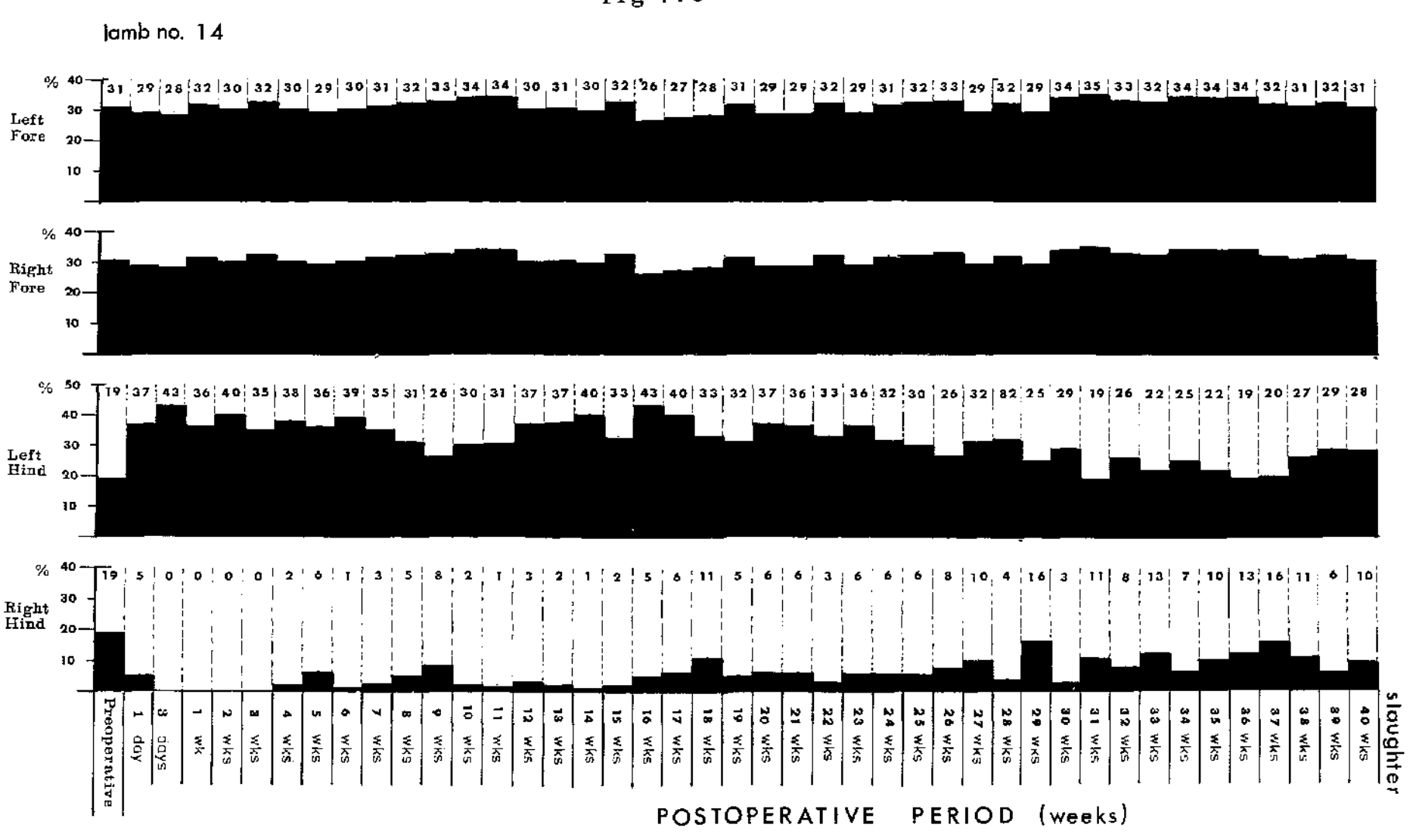

lamb no. 14
%
Left Fore
Right Fore
Left Hind
Right Hind
Preoperative
1 day
3 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
21 wks
22 wks
23 wks
24 wks
25 wks
26 wks
27 wks
28 wks
29 wks
30 wks
31 wks
32 wks
33 wks
34 wks
35 wks
36 wks
37 wks
38 wks
39 wks
40 wks
slaughter
POSTOPERATIVE PERIOD (weeks)

fig 41d

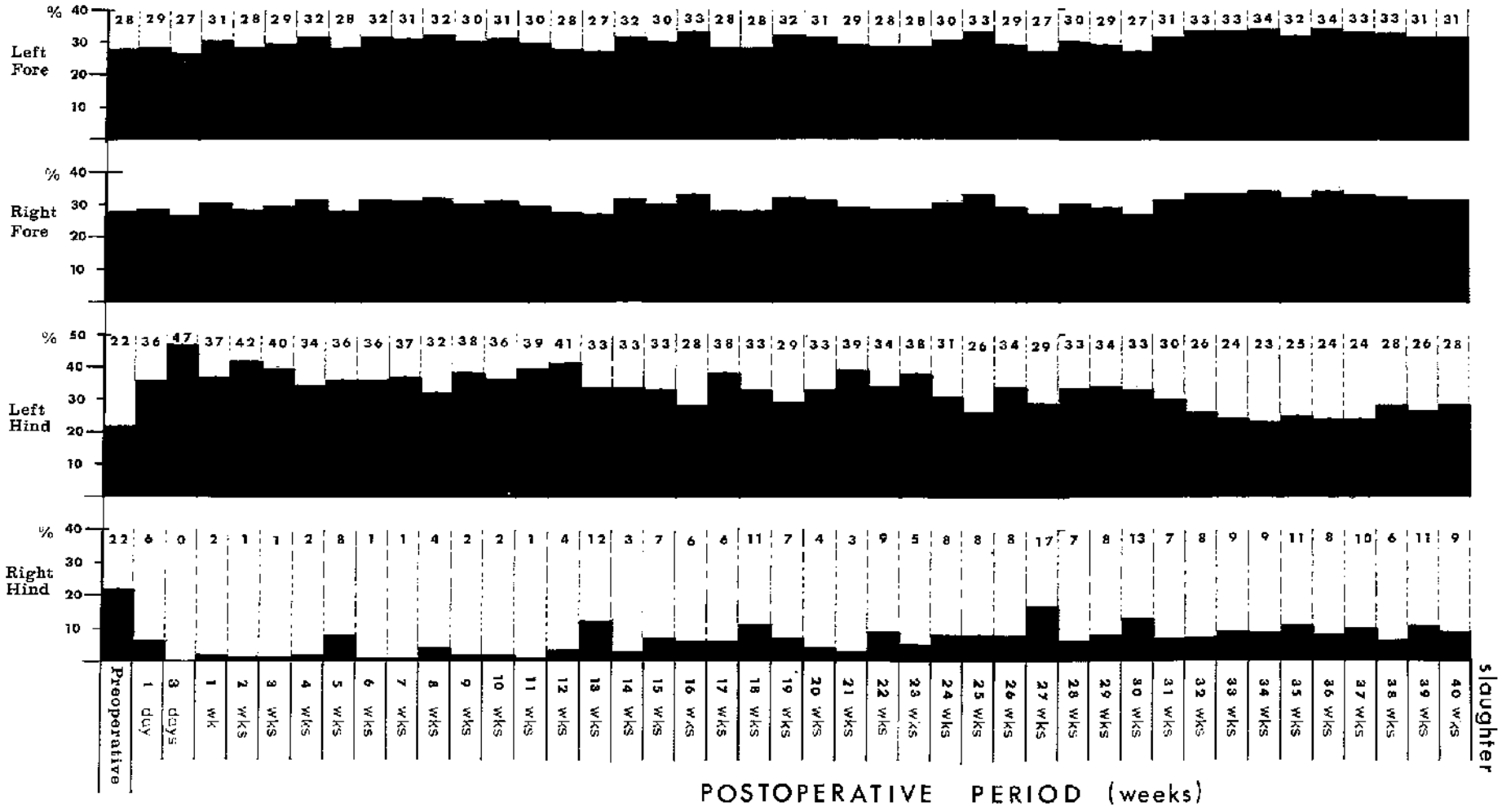

lamb no. 15
Left Fore
Right Fore
Left Hind
Right Hind
%
Preoperative
1 day
3 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
21 wks
22 wks
23 wks
24 wks
25 wks
26 wks
27 wks
28 wks
29 wks
30 wks
31 wks
32 wks
33 wks
34 wks
35 wks
36 wks
37 wks
38 wks
39 wks
40 wks
slaughter
POSTOPERATIVE PERIOD (weeks)

fig 41e

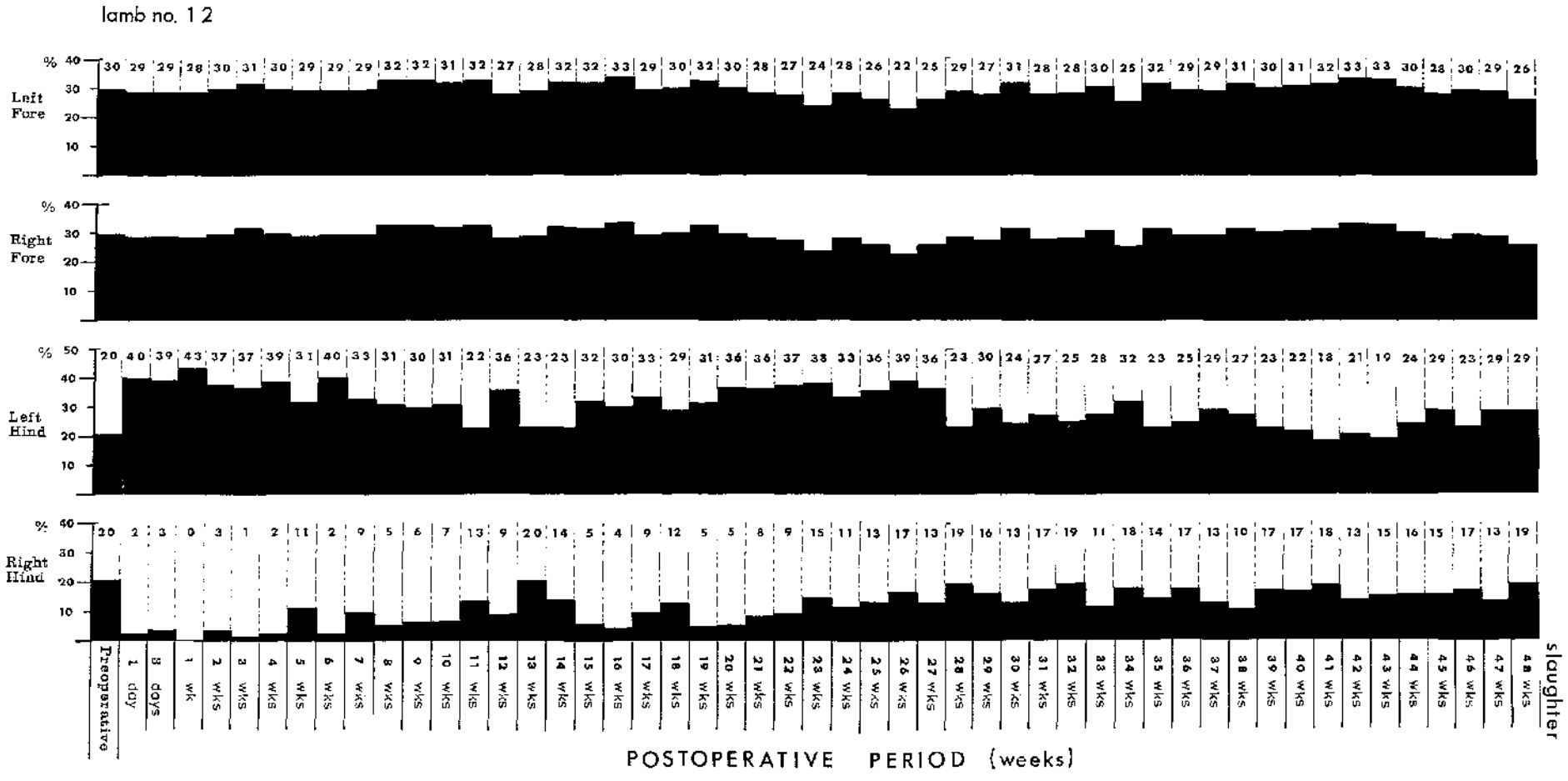

lamb no. 12
Left Fore
Right Fore
Left Hind
Right Hind
%
Preoperative
1 day
8 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
21 wks
22 wks
23 wks
24 wks
25 wks
26 wks
27 wks
28 wks
29 wks
30 wks
31 wks
32 wks
33 wks
34 wks
35 wks
36 wks
37 wks
38 wks
39 wks
40 wks
41 wks
42 wks
43 wks
44 wks
45 wks
46 wks
47 wks
48 wks
slaughter
POSTOPERATIVE PERIOD (weeks)

fig 41 f

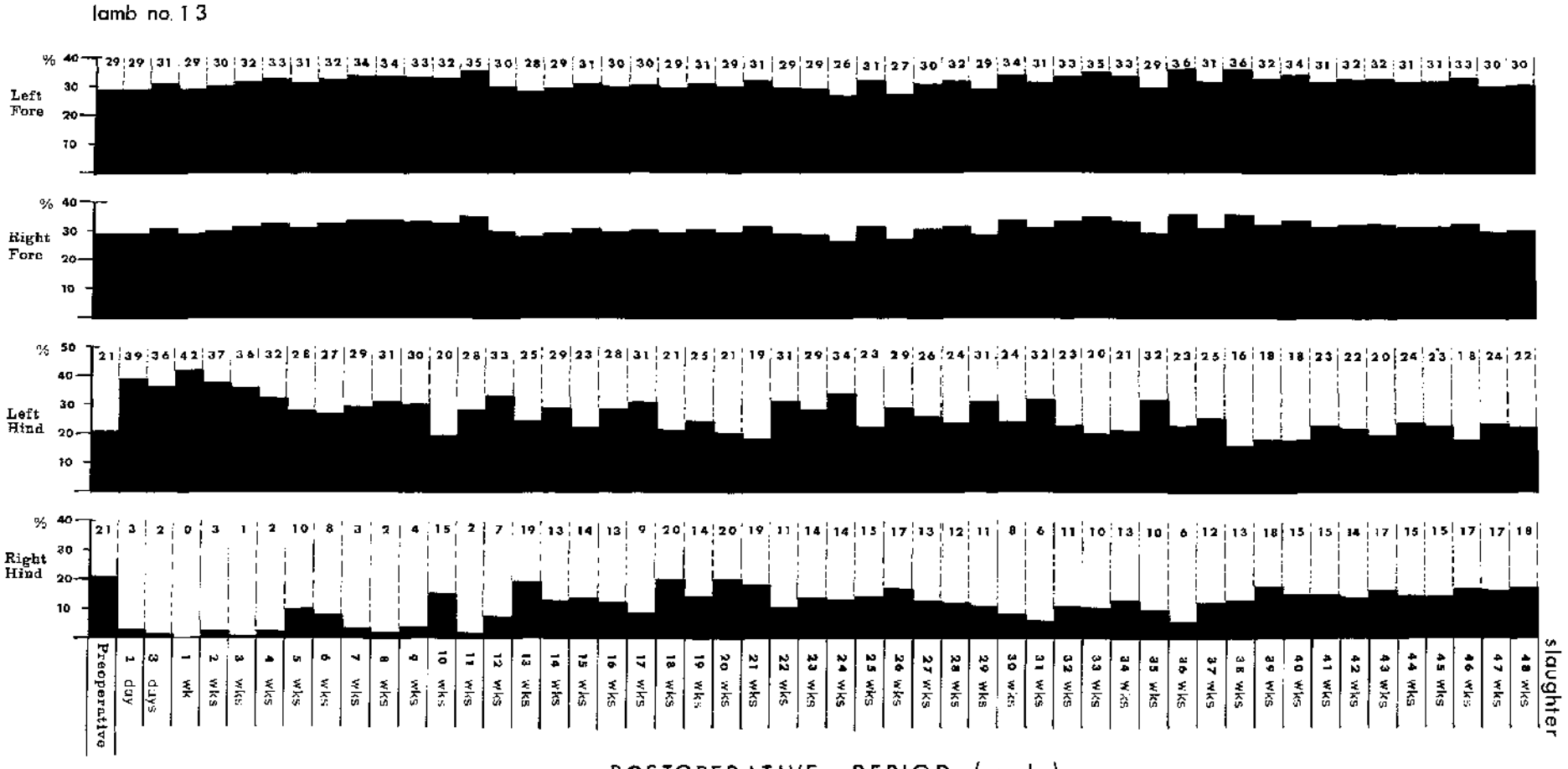

lamb no. 13
Left Fore
Right Fore
Left Hind
Right Hind
%
Preoperative
1 day
3 days
1 wk
2 wks
3 wks
4 wks
5 wks
6 wks
7 wks
8 wks
9 wks
10 wks
11 wks
12 wks
13 wks
14 wks
15 wks
16 wks
17 wks
18 wks
19 wks
20 wks
21 wks
22 wks
23 wks
24 wks
25 wks
26 wks
27 wks
28 wks
29 wks
30 wks
31 wks
32 wks
33 wks
34 wks
35 wks
36 wks
37 wks
38 wks
39 wks
40 wks
41 wks
42 wks
43 wks
44 wks
45 wks
46 wks
47 wks
48 wks
slaughter
POSTOPERATIVE PERIOD (weeks)

fig 42

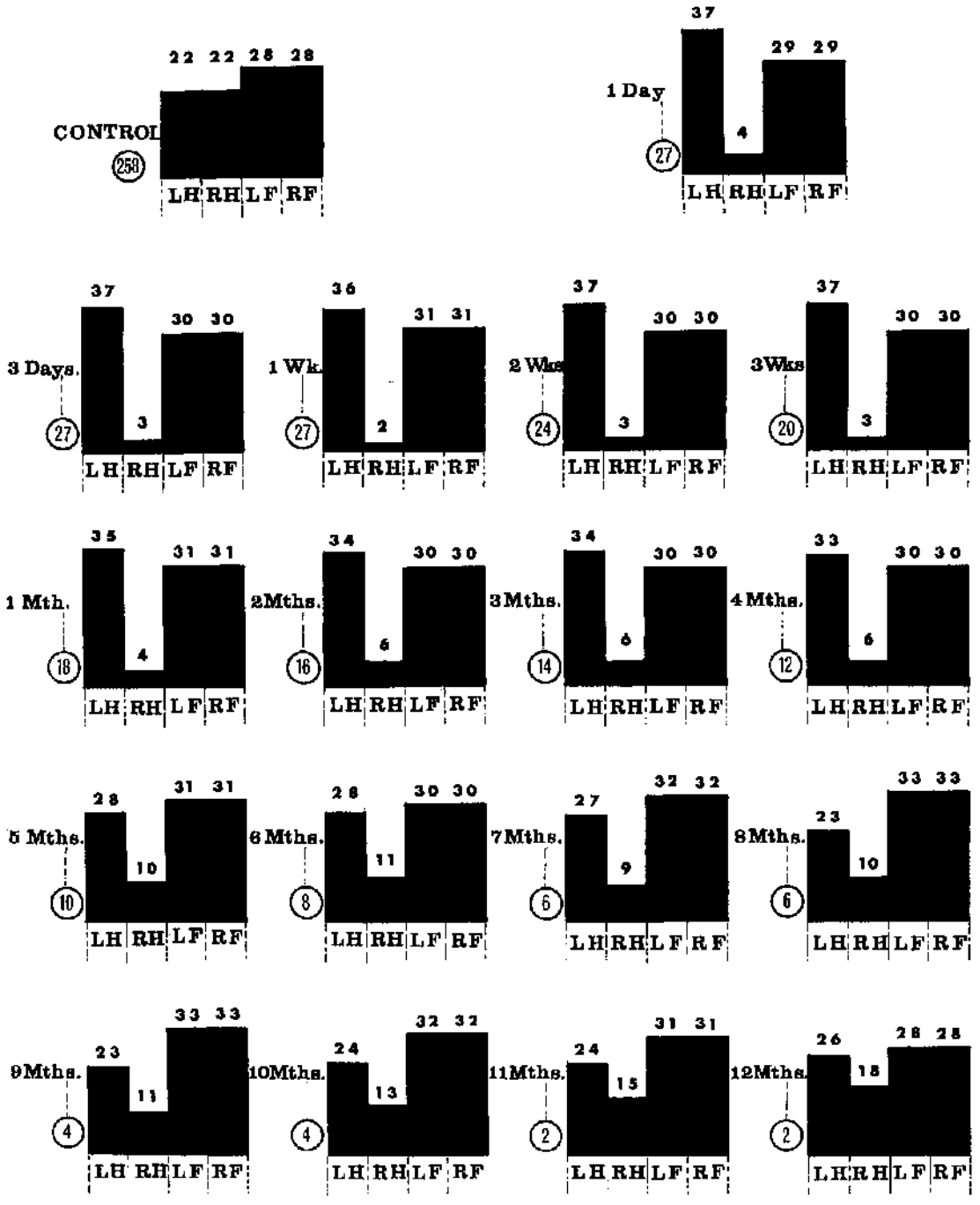
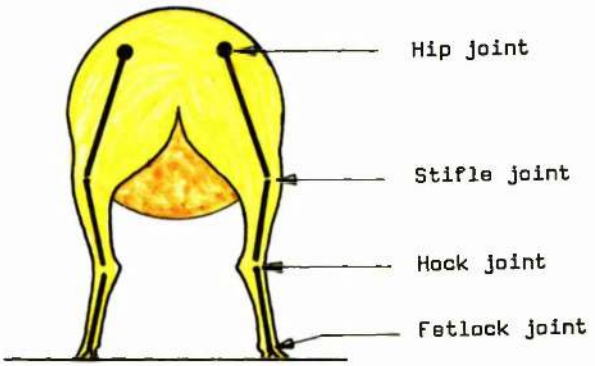

CONTROL
22 22 28 28
258
LH RH LF RF
1 Day
37
29 29
4
27
3 Days.
37
30 30
3
27
1 Wk.
36
31 31
2
27
2 Wks
37
30 30
3
24
3Wks
37
30 30
3
20
1 Mth.
35
31 31
4
18
2Mths.
34
30 30
6
16
3Mths.
34
30 30
6
14
4Mths.
33
30 30
6
12
5 Mths.
28
31 31
10
10
6Mths.
28
30 30
11
8
7Mths.
27
32 32
9
6
8Mths.
23
33 33
10
6
9Mths.
23
33 33
11
4
10Mths.
24
32 32
13
4
11Mths.
24
31 31
15
2
12Mths.
26
28 28
15
2

FIG. 4 3 Pre-mortem posture



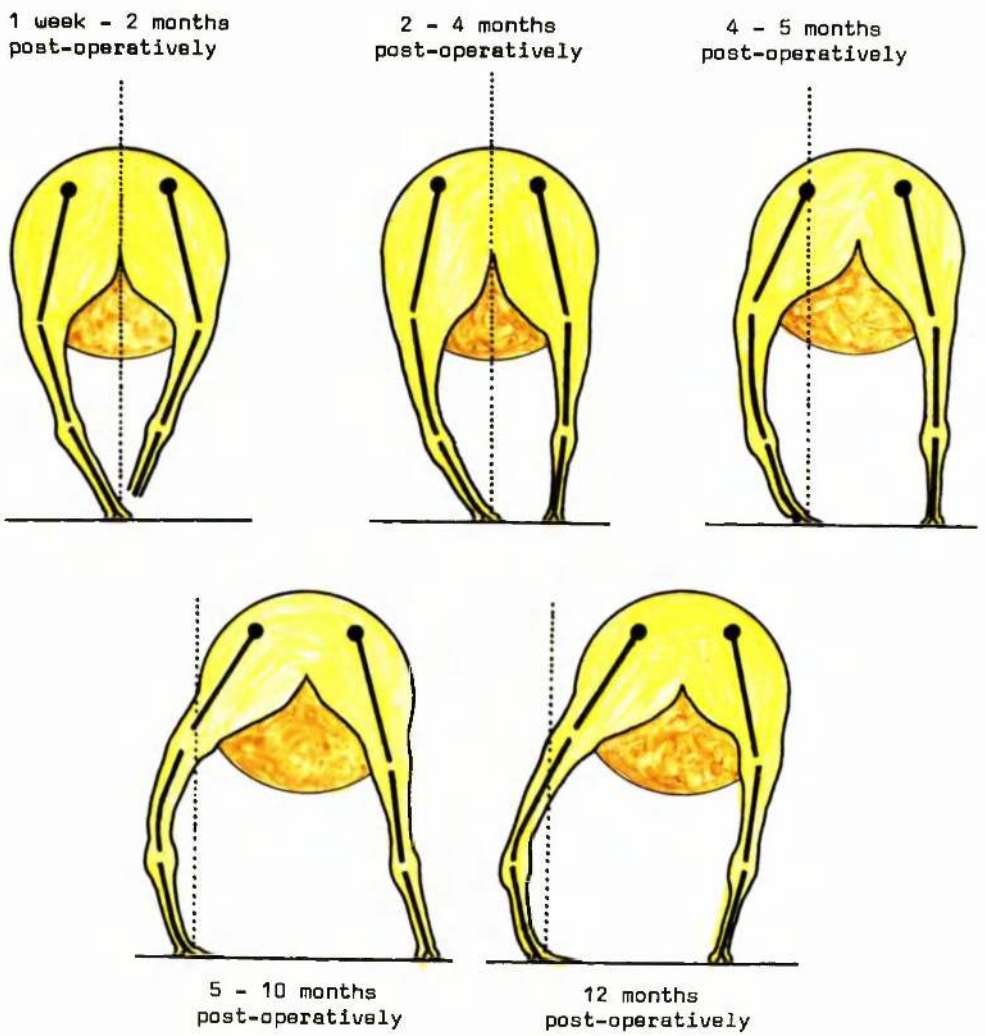
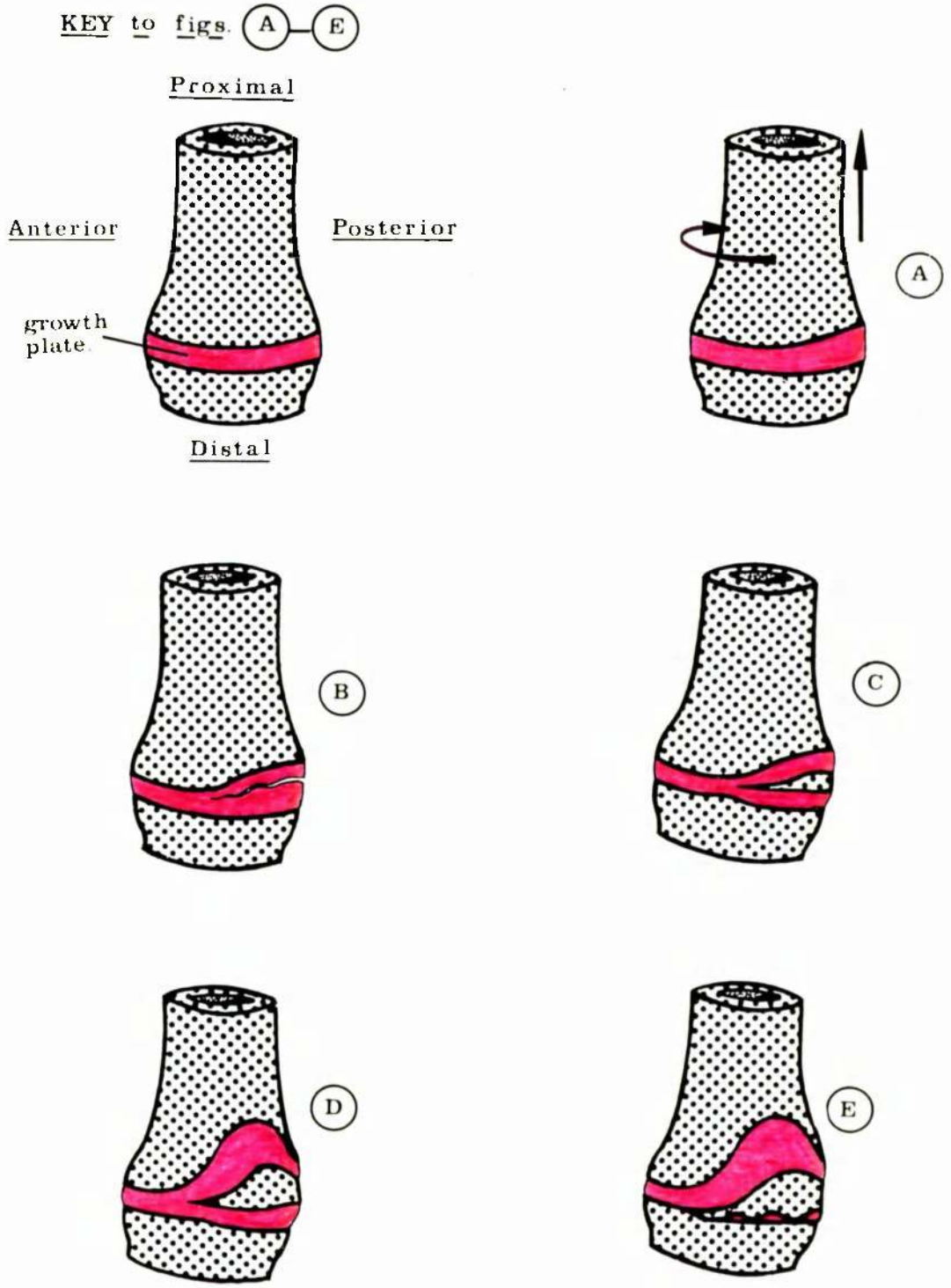


FIG. 44 DEVELOPMENT OF LATERAL GROWTH PLATE DEFECT.



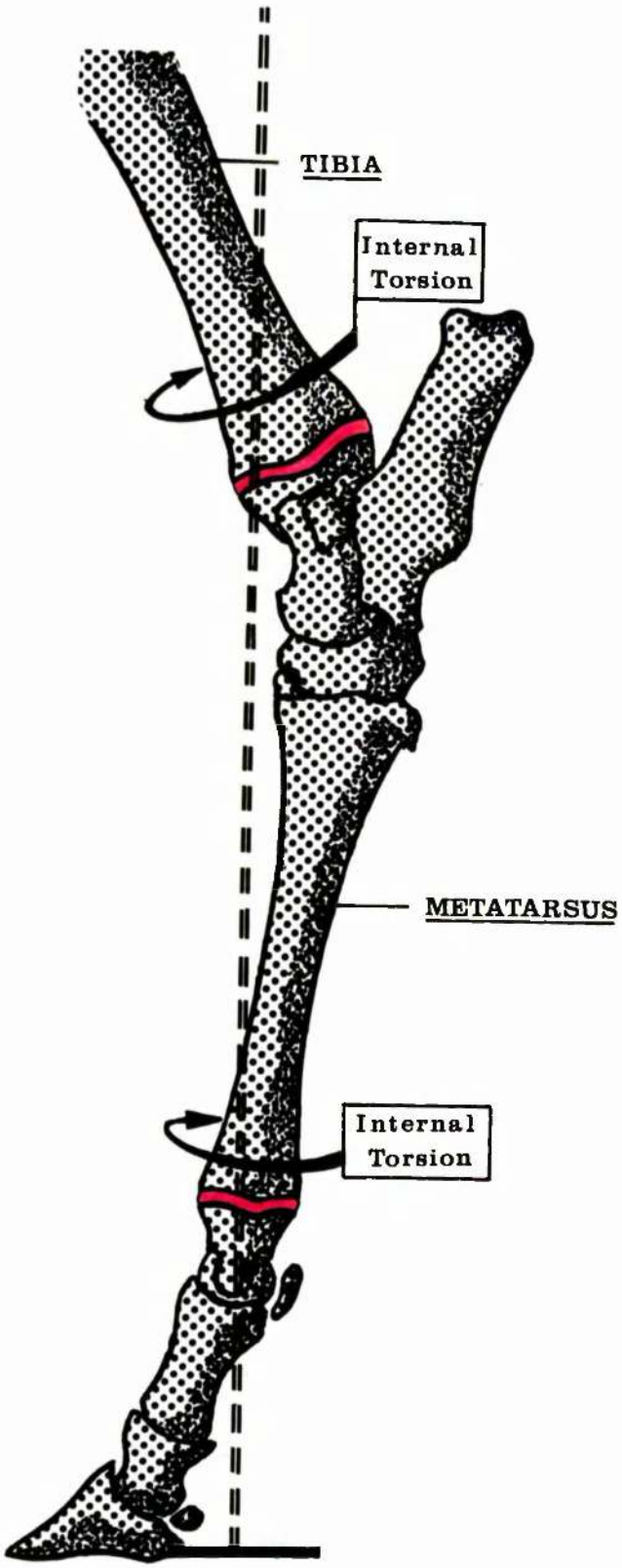
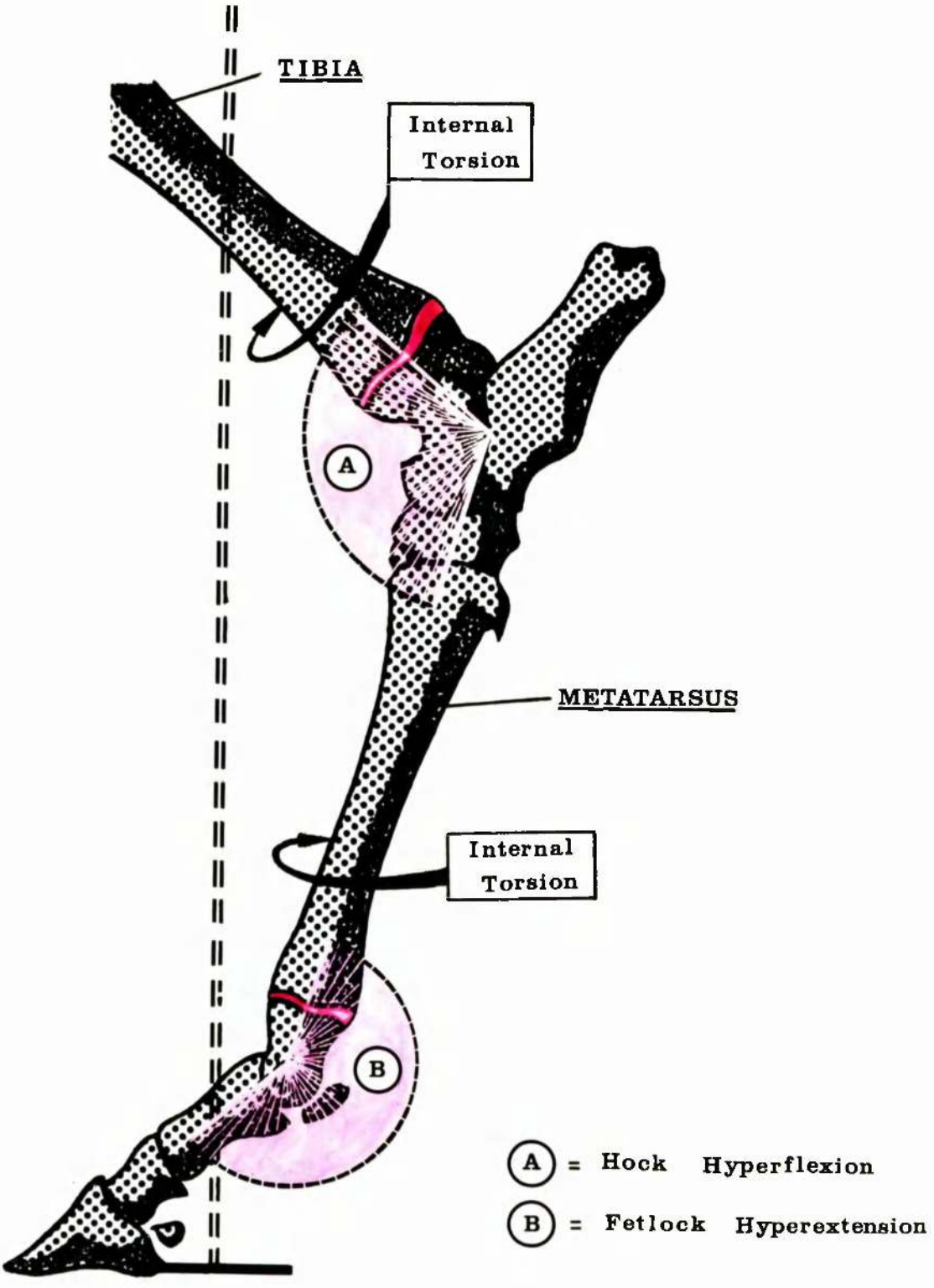


FIG.45a THE INITIAL EFFECT OF ABNORMAL HINDLIMB POSTURE — LATERAL VIEW.

FIG. 45b THE EFFECT OF PROTRACTED ABNORMAL HINDLIMB POSTURE — LATERAL VIEW.



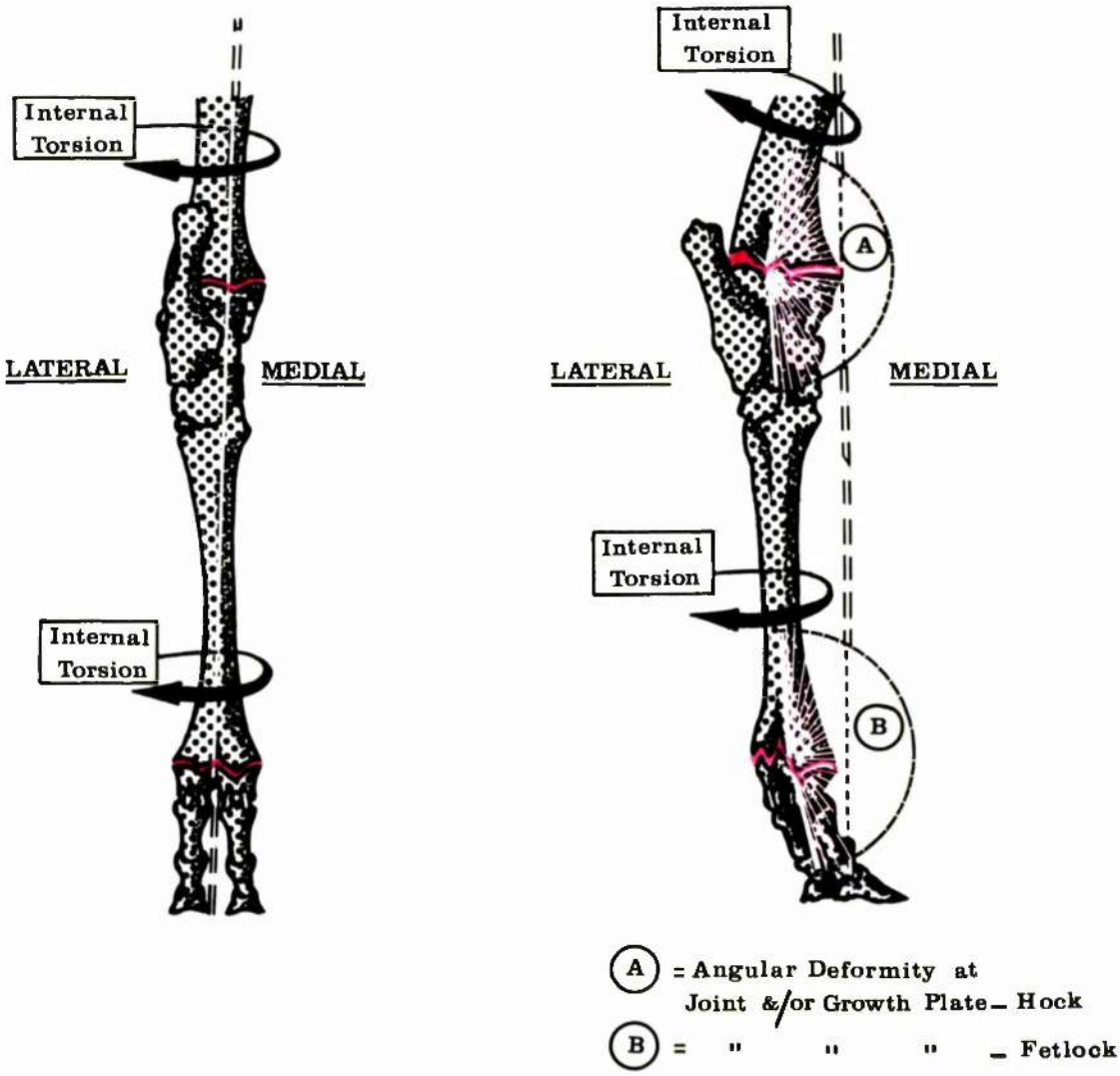


FIG.45c THE EFFECT OF INITIAL & PROTRACTED ABNORMAL HINDLIMB POSTURE — ANTEROPOSTERIOR VIEW.

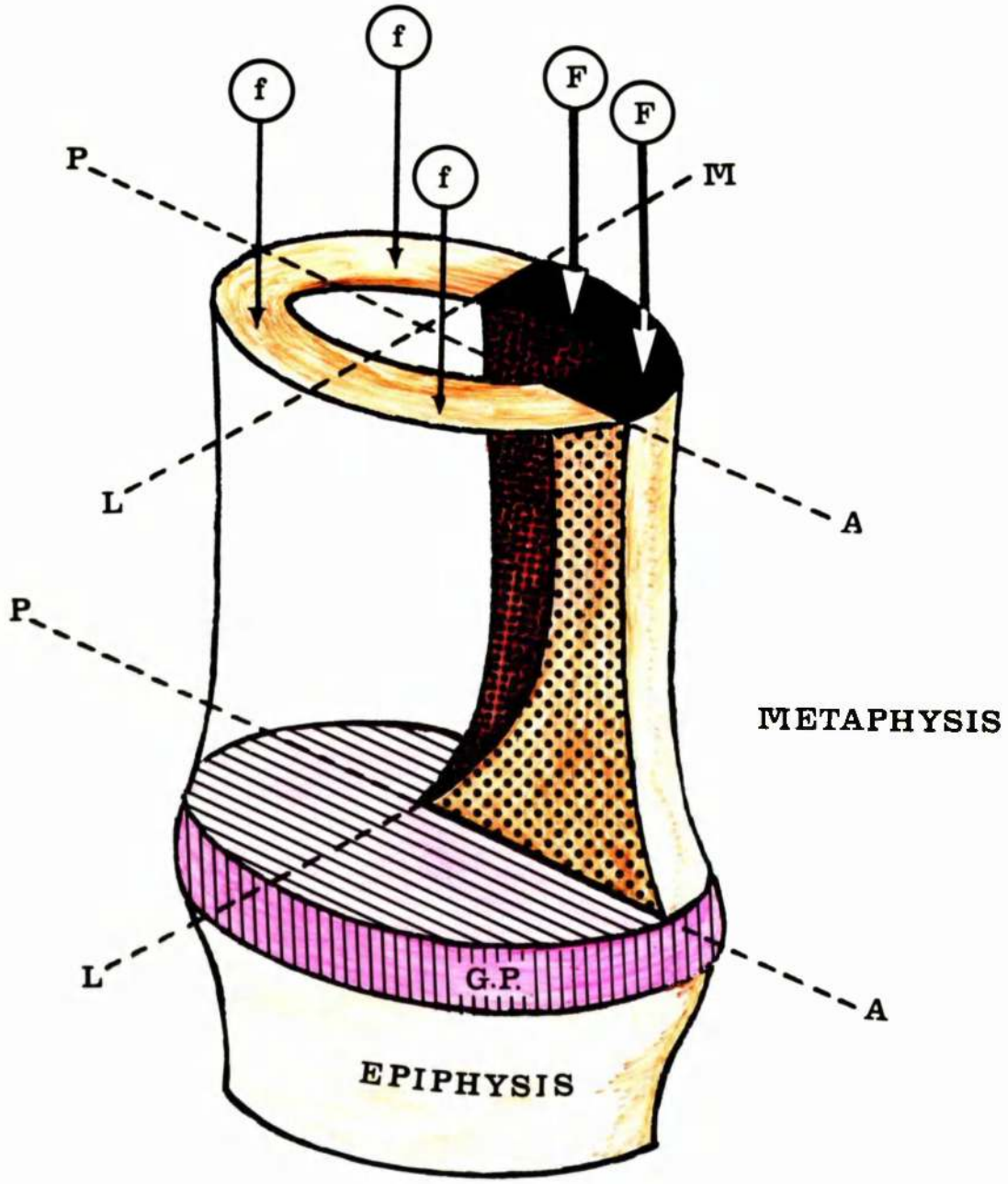


Fig 46

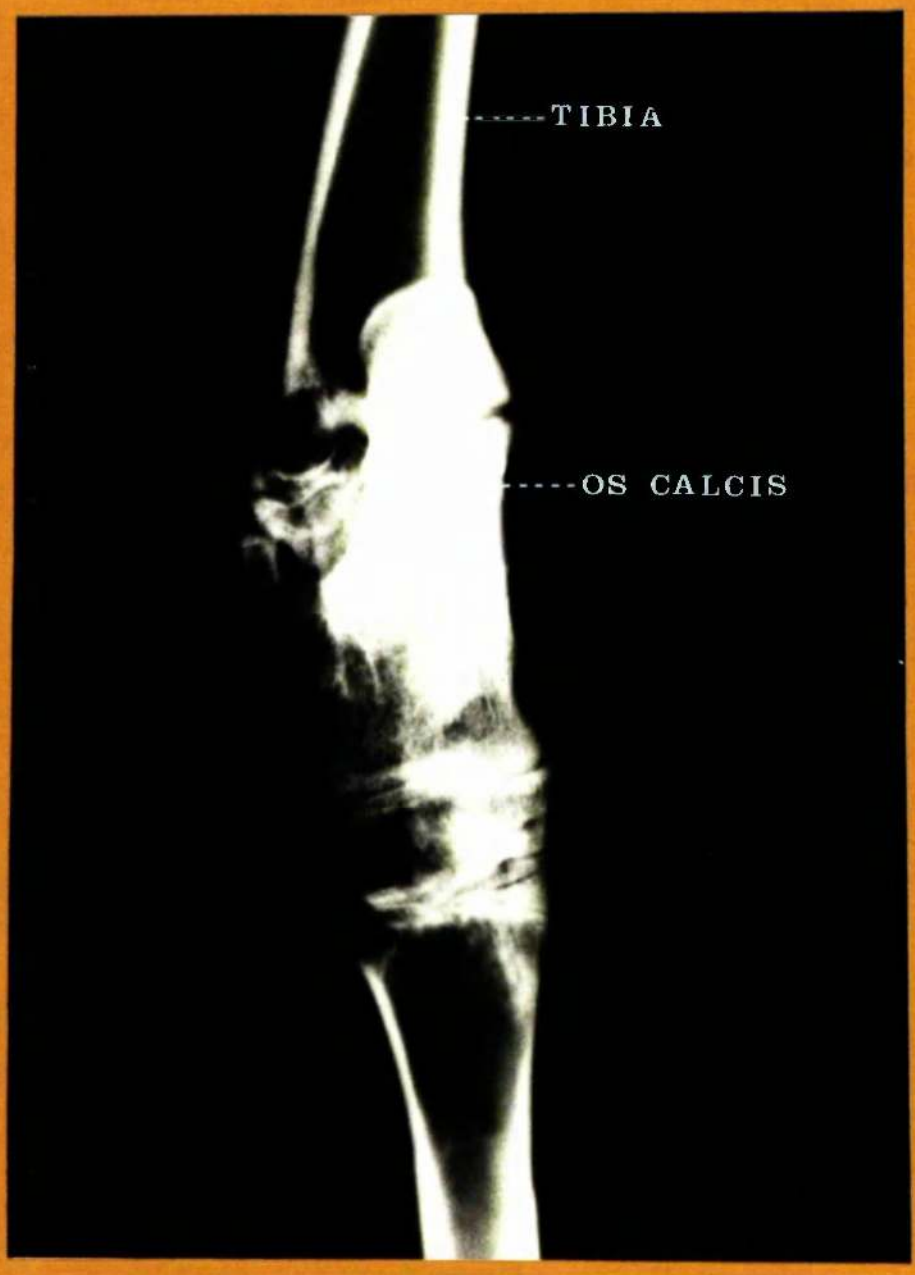


PLATE I Anteroposterior radiograph of ovine tarsus demonstrating angular deformity of distal tibia. Such deformities occurred following hip excision arthroplasty of the contralateral limb in skeletally immature lambs.(DUFF 1975)

A

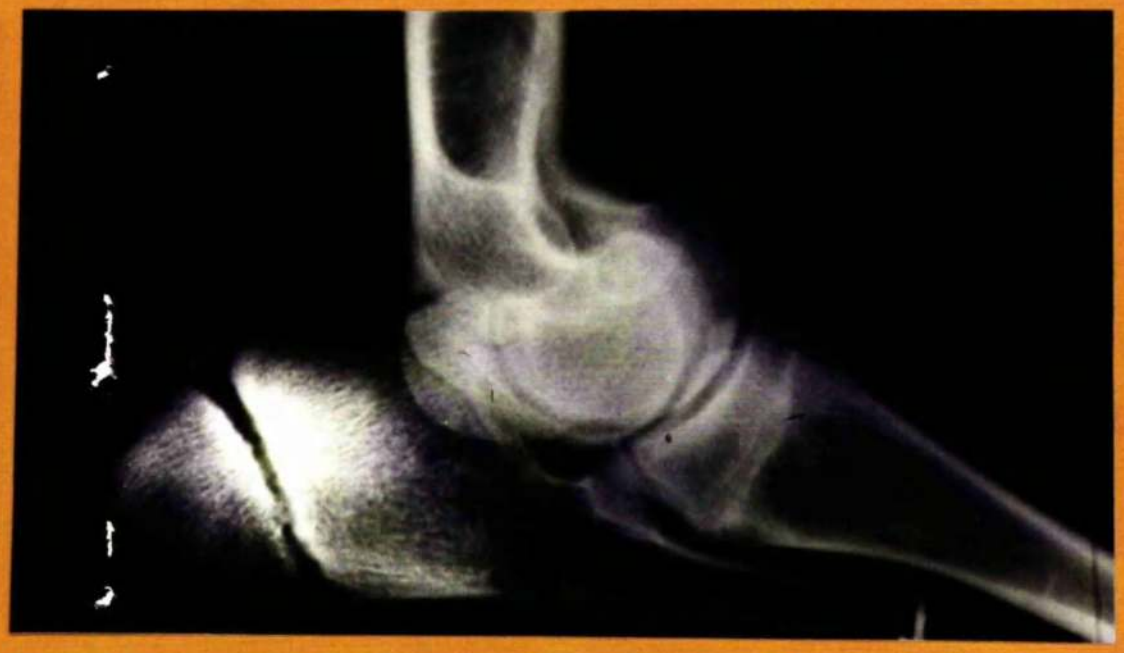
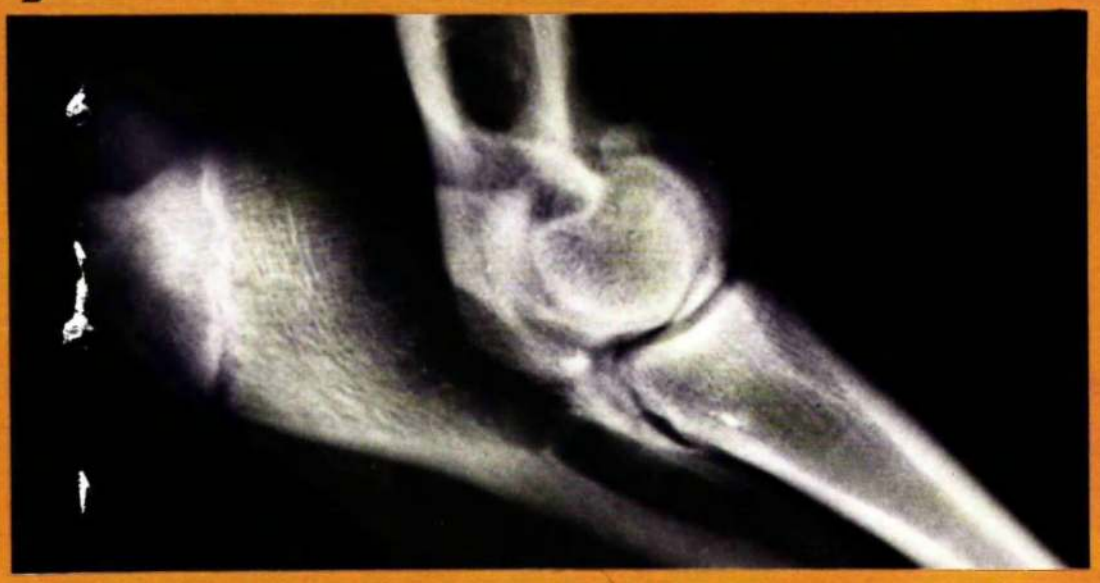


PLATE 2 A&B Lateral radiographs of normal ovine elbow joint demonstrating stages of proximal ulnar epiphyseal union.

B



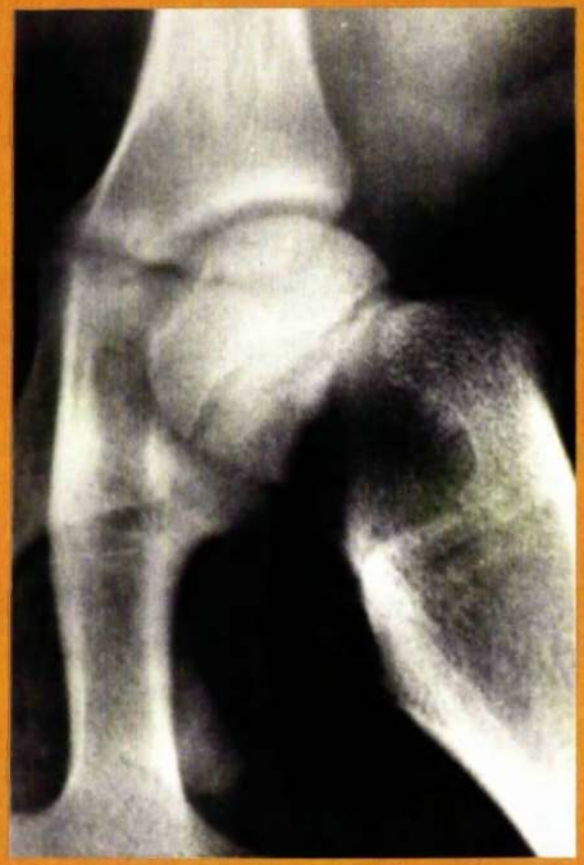


PLATE 3 Ventrodorsal radiograph of normal ovine hip joint.

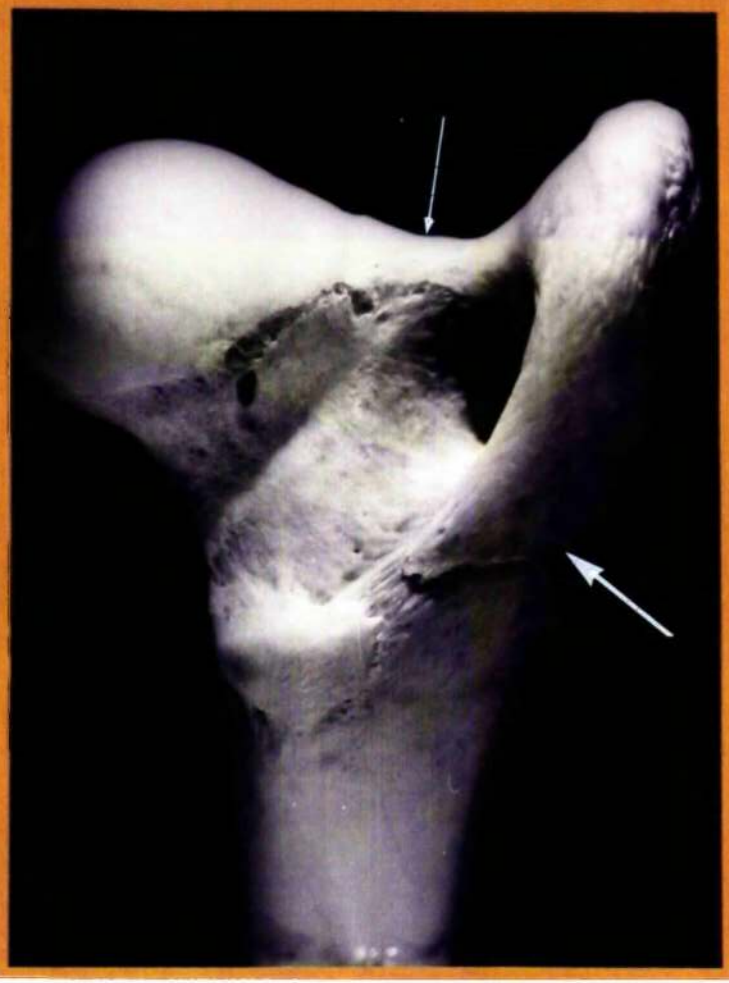


PLATE 4 Boiled-out ovine femur demonstrating fusion of greater trochanteric epiphysis. Initially fusion occurred dorsally (small arrow). The distal aspect of the epiphysis (large arrow) was last to fuse.

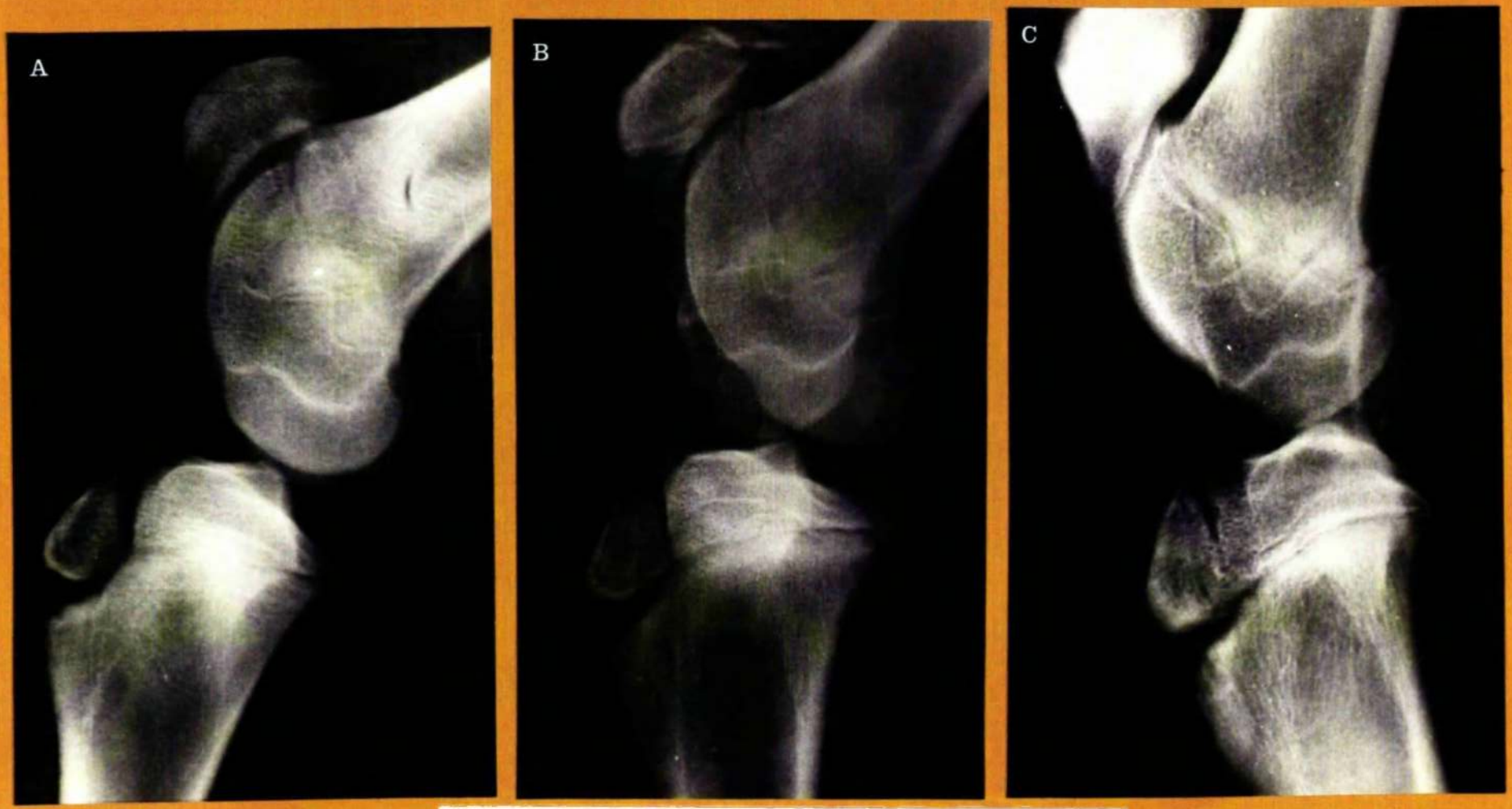


PLATE 5 A,B&C Lateral radiographs of normal ovine tibia demonstrating stages of tibial tuberosity epiphyseal union.

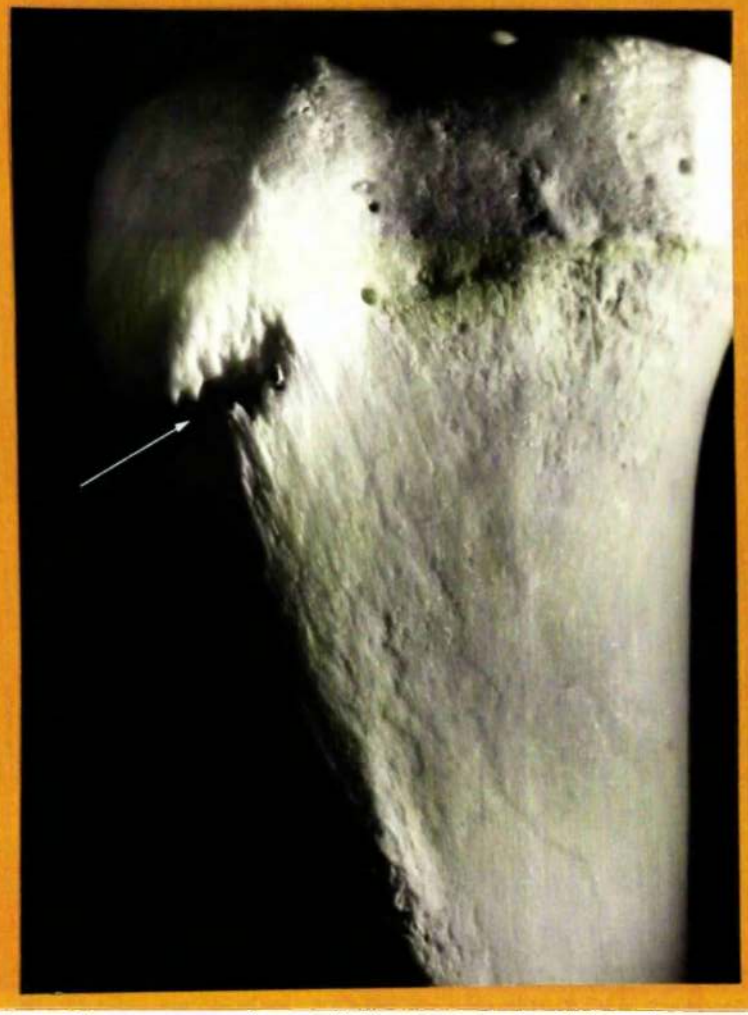


PLATE 6 Boiled-out ovine tibia demonstrating incomplete union of distal tibial tuberosity (arrowed).

PLATE 7 Sectioned boiled-out ovine tibia demonstrating incomplete union of distal tibial tuberosity epiphysis (arrowed).

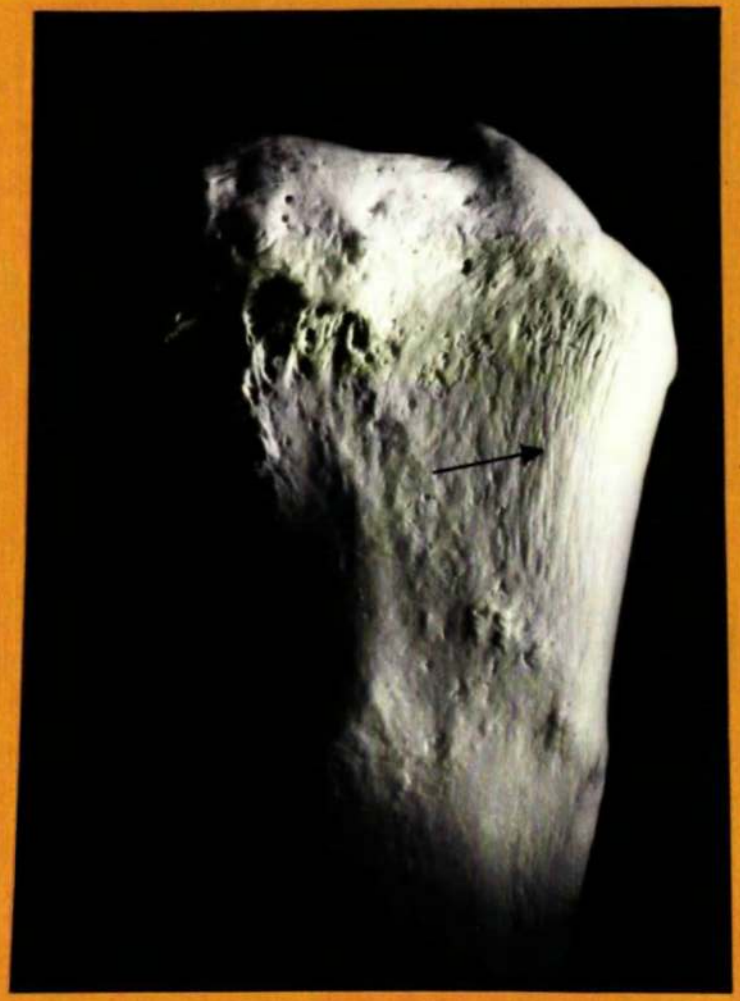


PLATE 8 Boiled-out ovine tibia demonstrating striated cortical bone surface anteriorly.

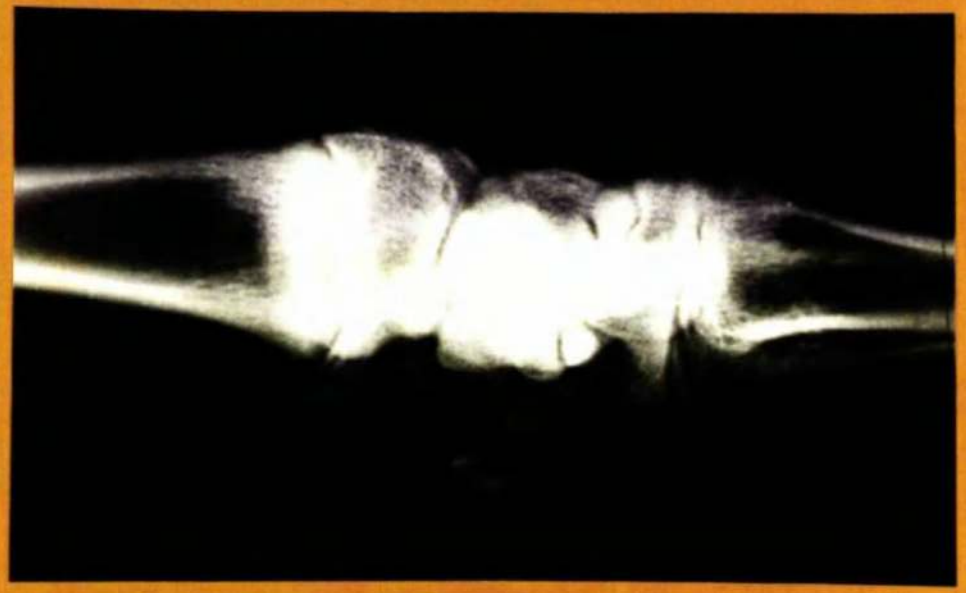


PLATE 9 Lateral radiograph of normal ovine carpus demonstrating posterior peaking of distal radial epiphysis and metaphysis.

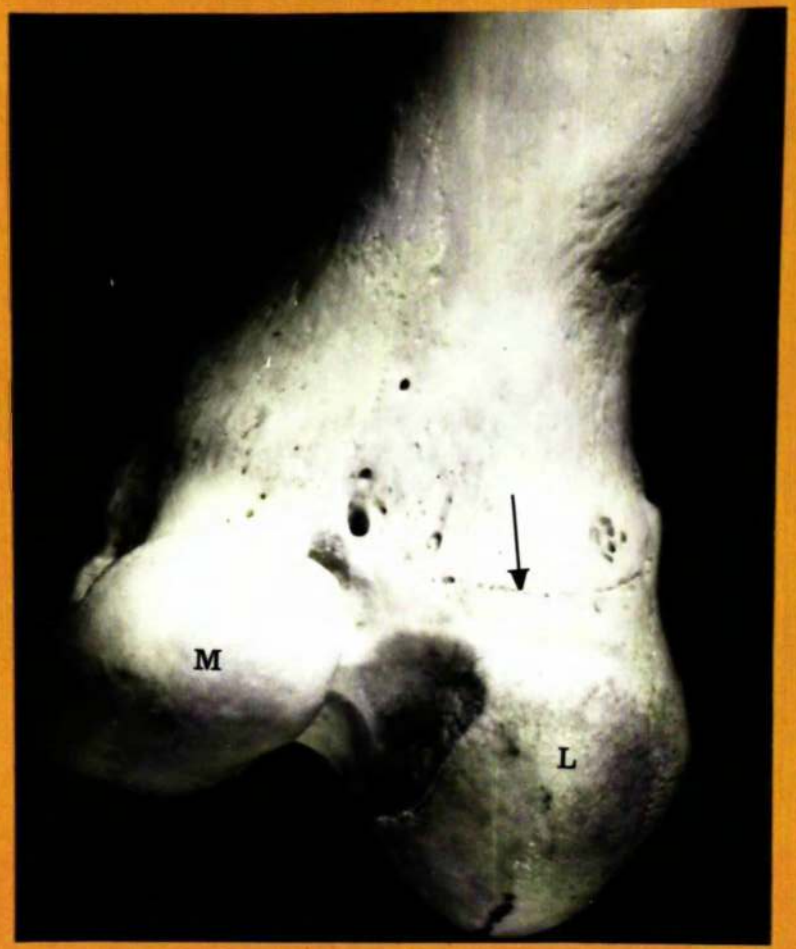


PLATE 10 Posterior aspect of boiled-out ovine femoral condyles demonstrating pronounced epiphyseal line posterolaterally (arrowed).

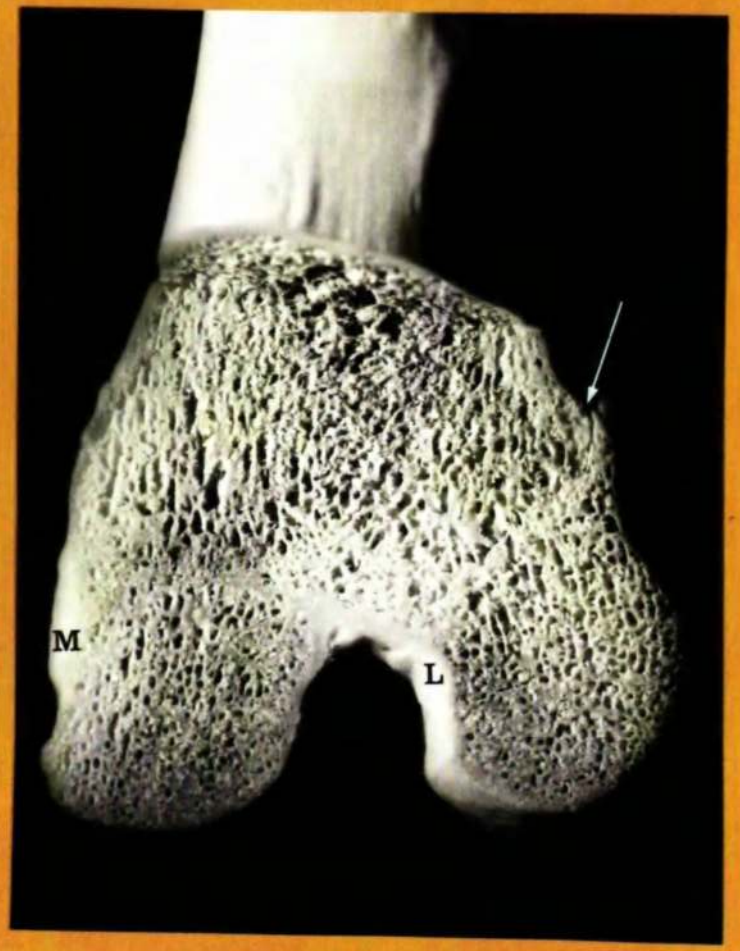


PLATE 11 Sectioned boiled-out ovine femur demonstrating incomplete union of lateral femoral condyle (arrowed).

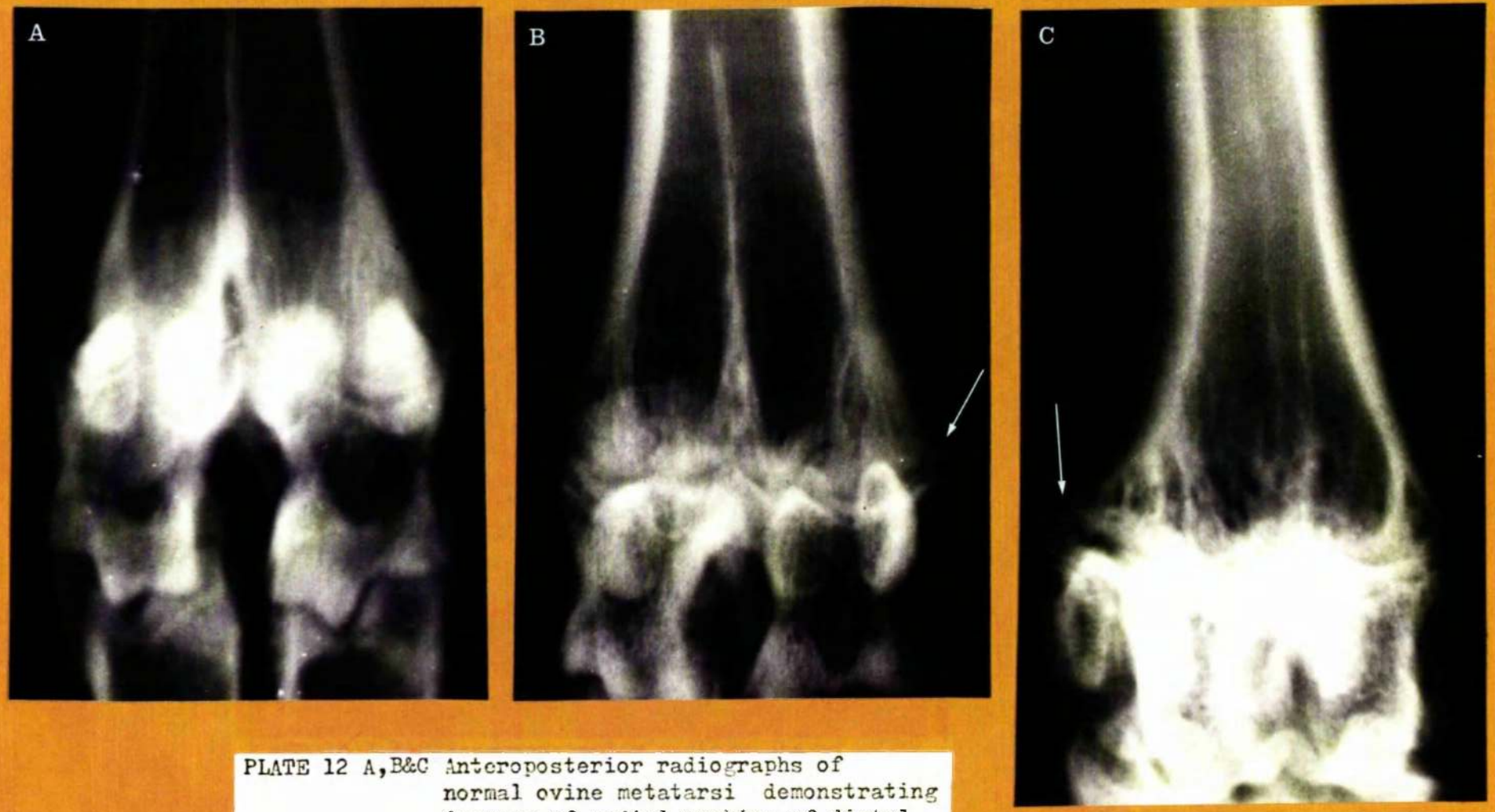


PLATE 12 A,B&C Anteroposterior radiographs of normal ovine metatarsi demonstrating degrees of medial peaking of distal metaphysis & epiphysis encountered.

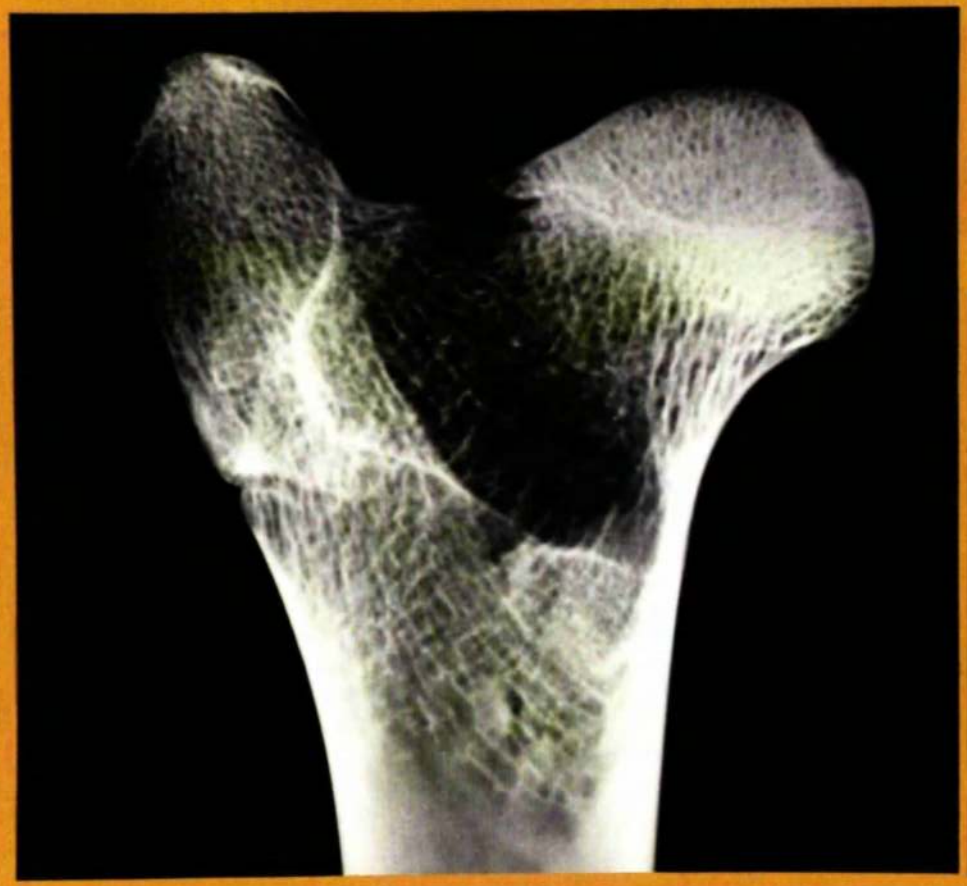


PLATE 13 Anteroposterior radiograph of boiled-out ovine proximal femur.

PLATE 14 Lateral radiograph of normal ovine carpus demonstrating distal radial epiphyseal scar.

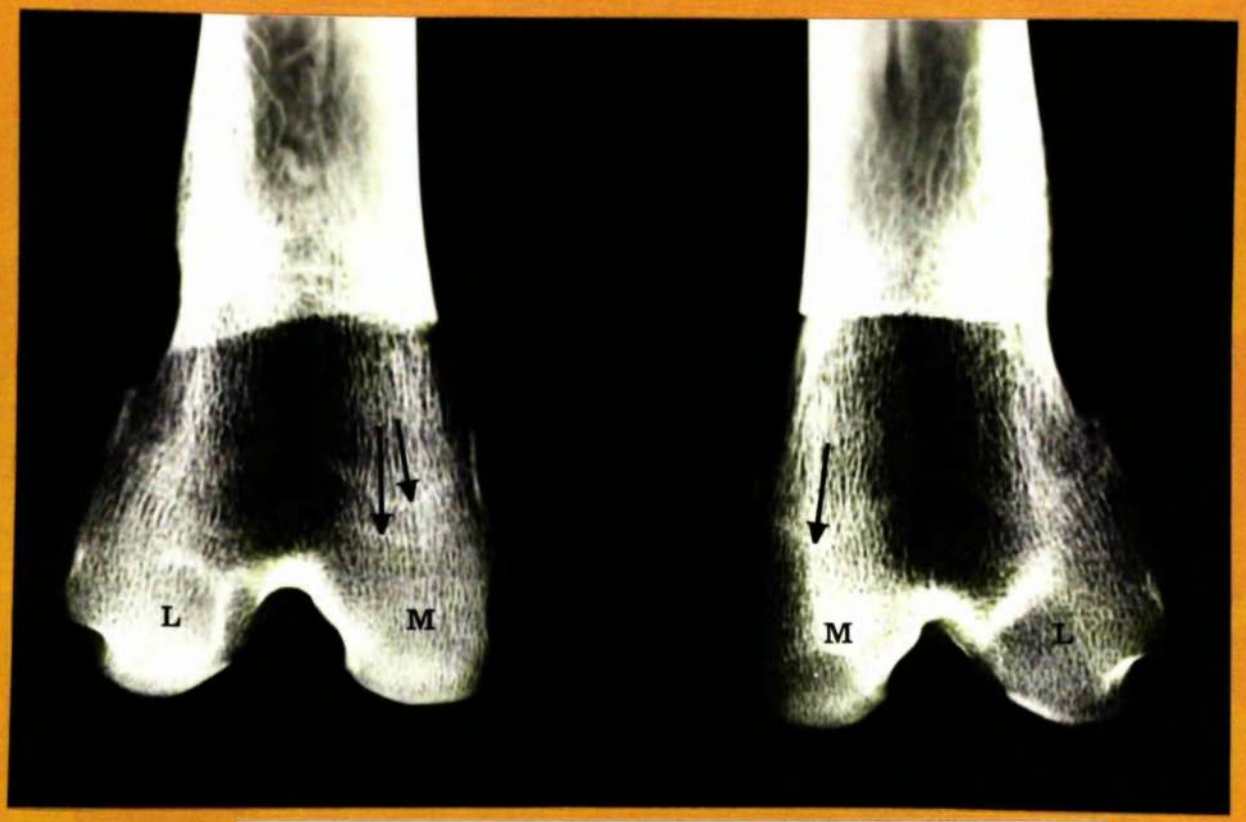


PLATE 15 Anteroposterior radiograph of sectioned boiled-out ovine femora demonstrating more pronounced distal epiphyseal scars medially (arrowed).

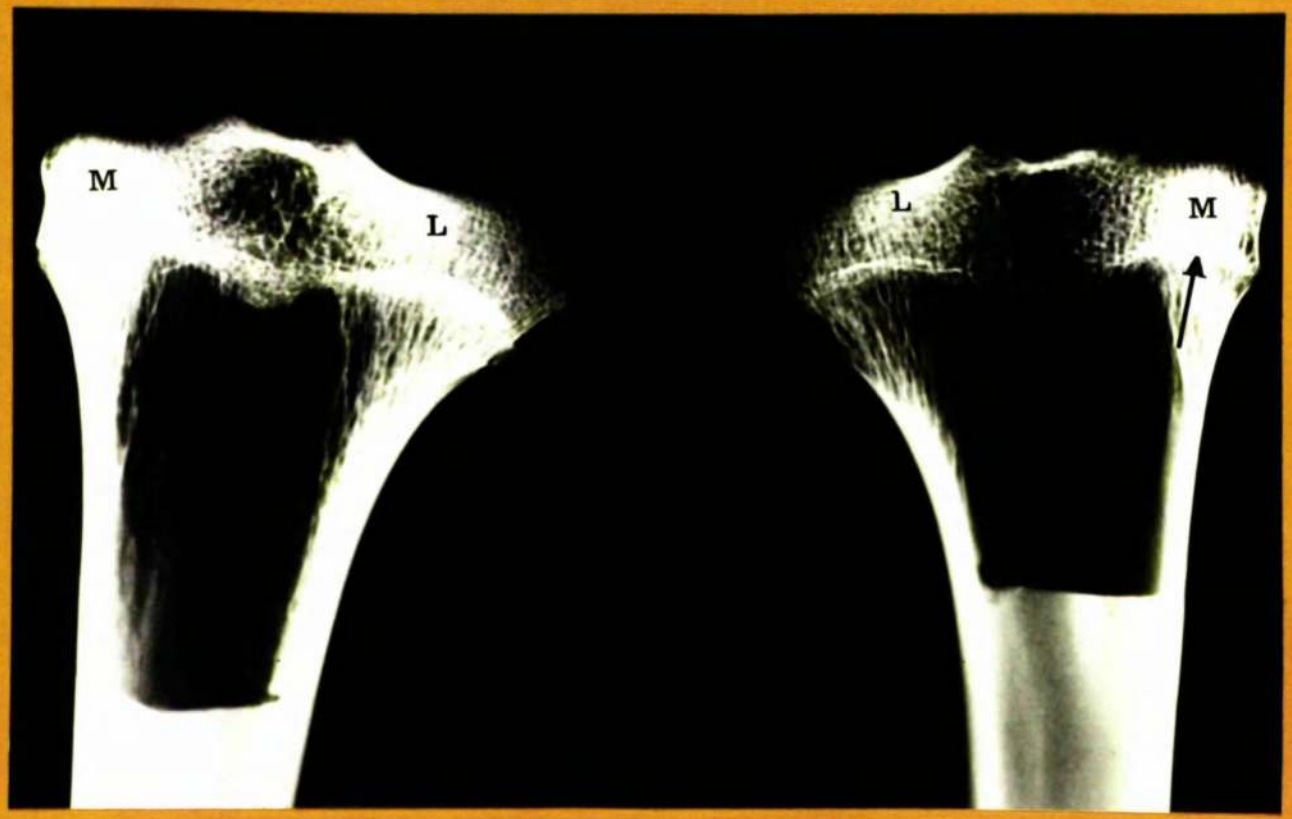


PLATE 16 Anteroposterior radiograph of sectioned boiled-out ovine tibiae demonstrating more pronounced proximal epiphyseal scars medially (arrowed).

PLATE 17 Lateral radiograph of ovine antebrachium demonstrating normal bone curvature.

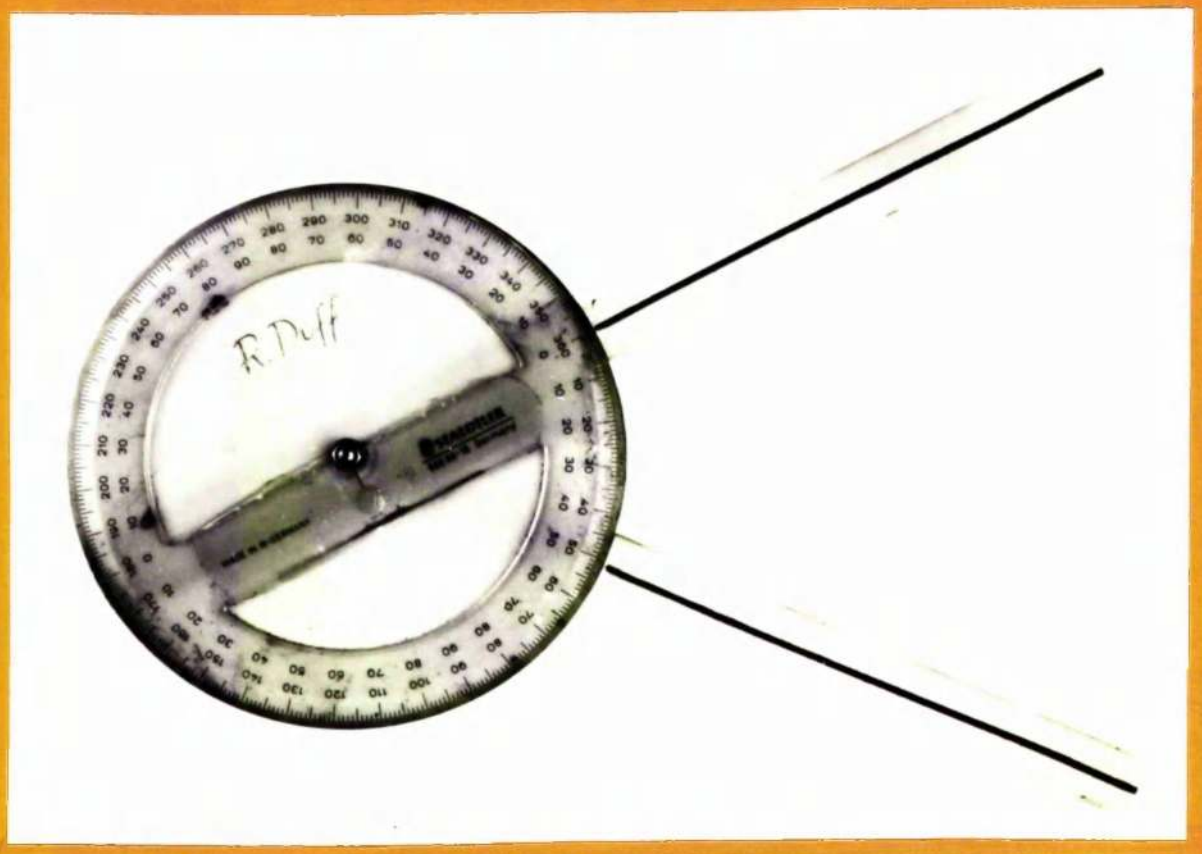


PLATE 18 Goniometer used for joint mobility estimations.

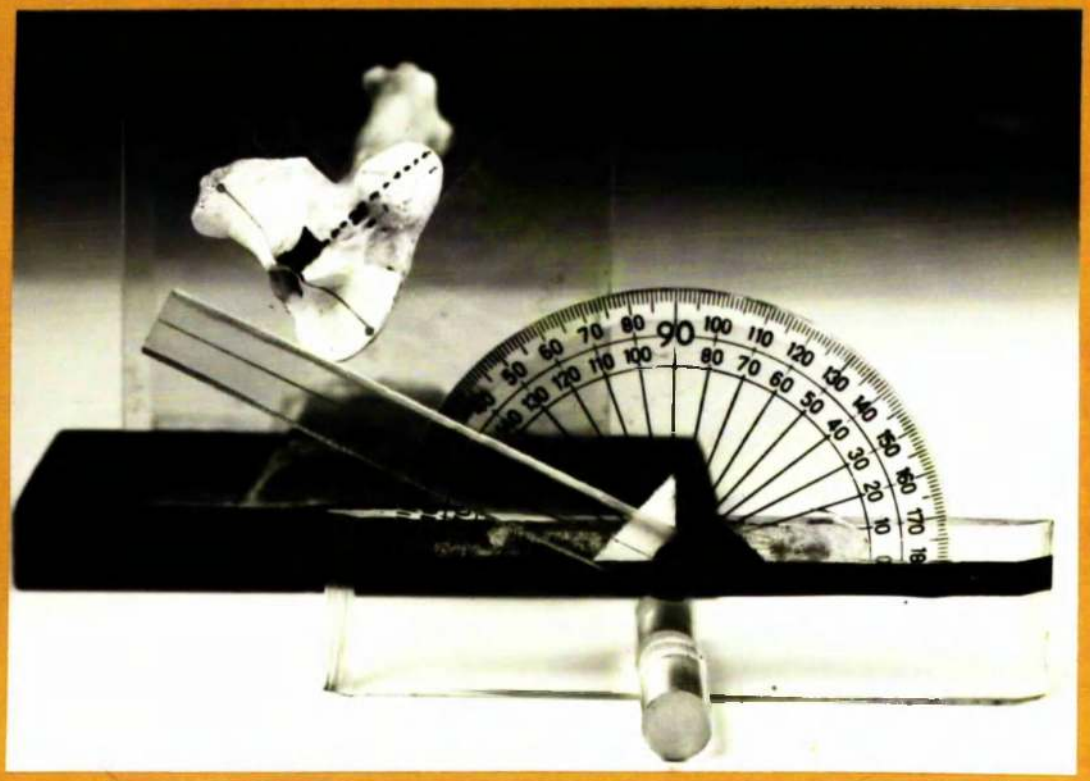
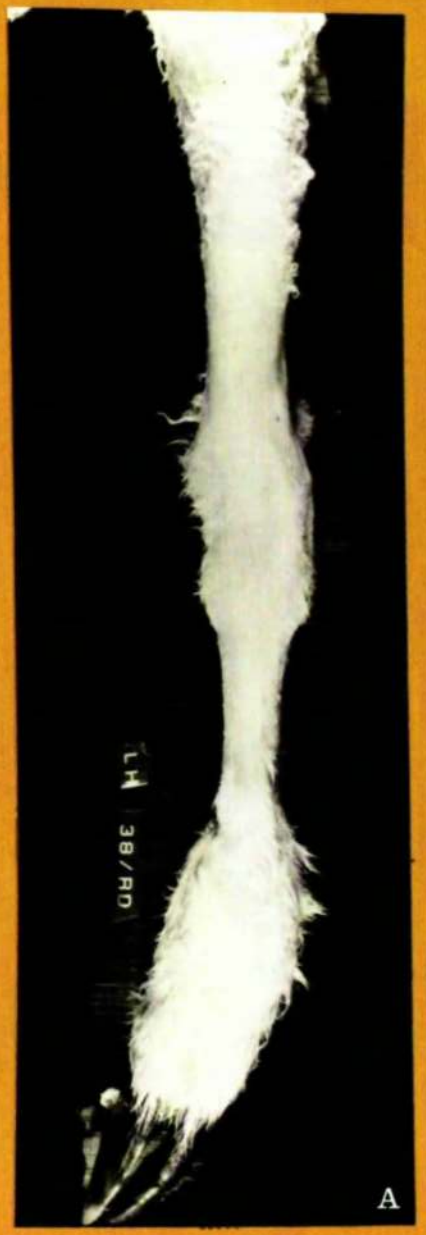
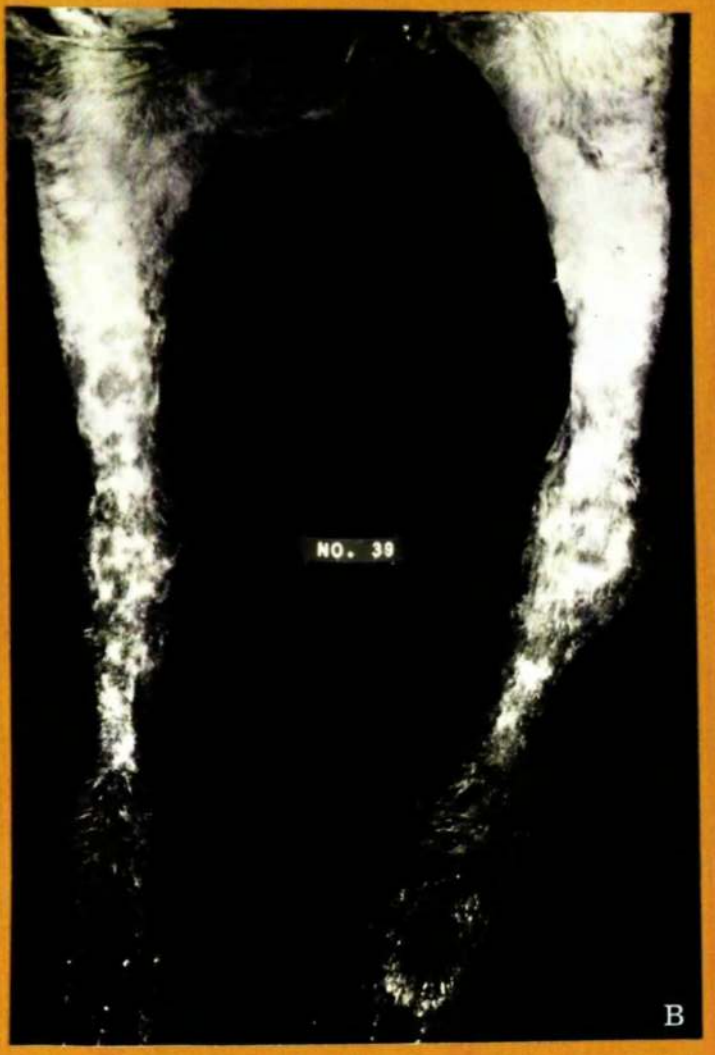


PLATE 19 Apparatus used for estimation of axial torsion of tibiae and metatarsi.



A



B

C

PLATE 20 A,B&C Examples of post mortem limb deformities encountered in the nonoperated LH pelvic limb. (No.38=4months, No.39=6months & No.14=10months postoperatively).

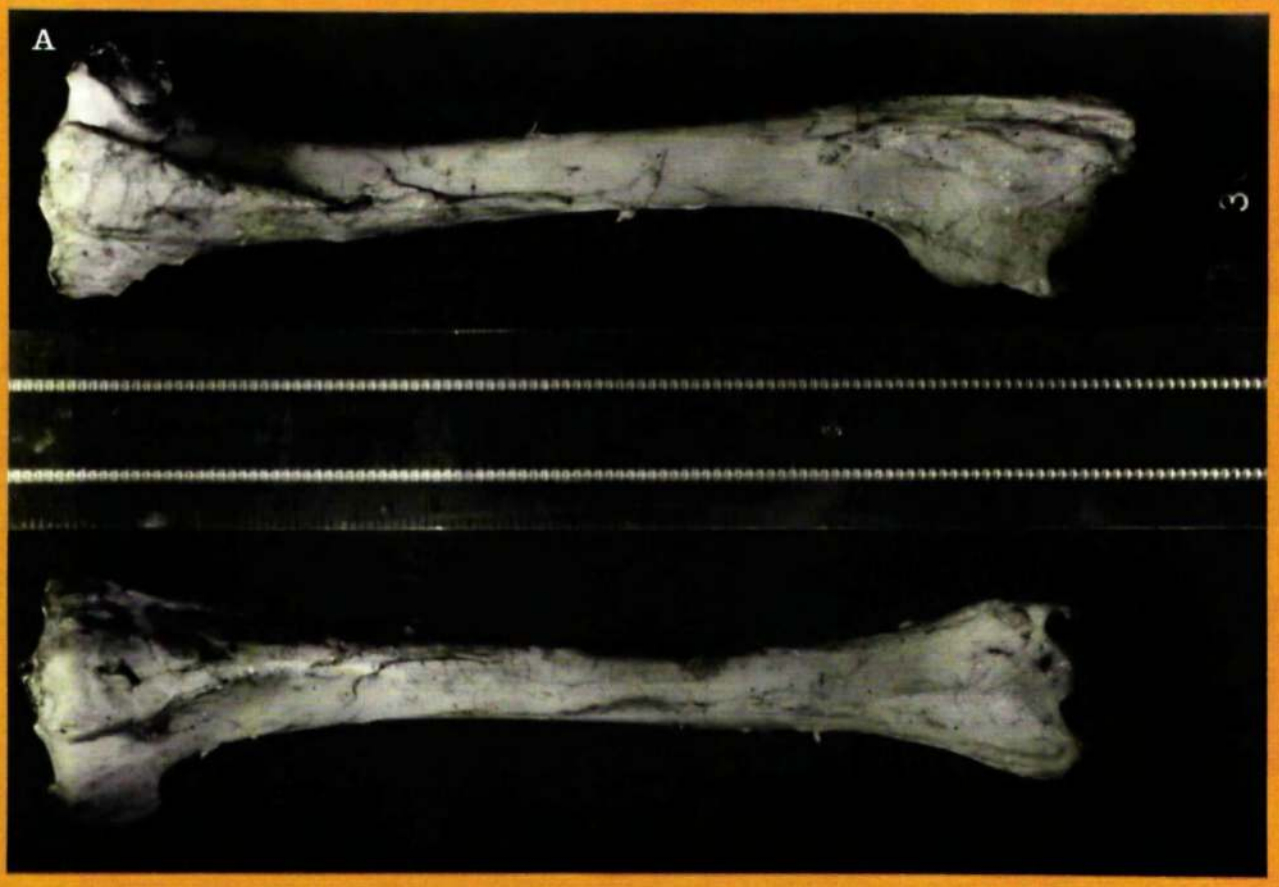
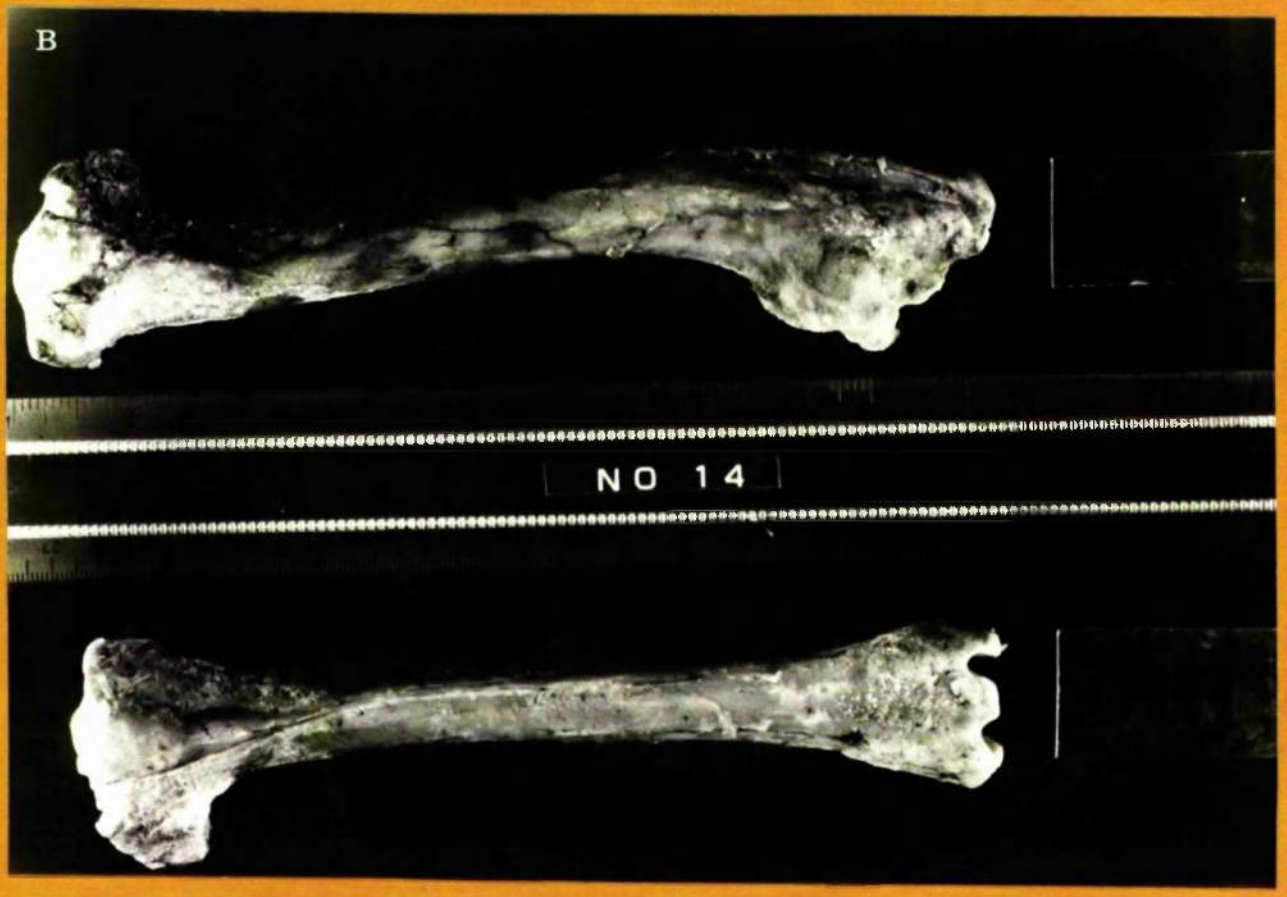


PLATE 21 A&B Examples of distal tibial deformity.
(No.36=5months & No.14=10months postoperatively).



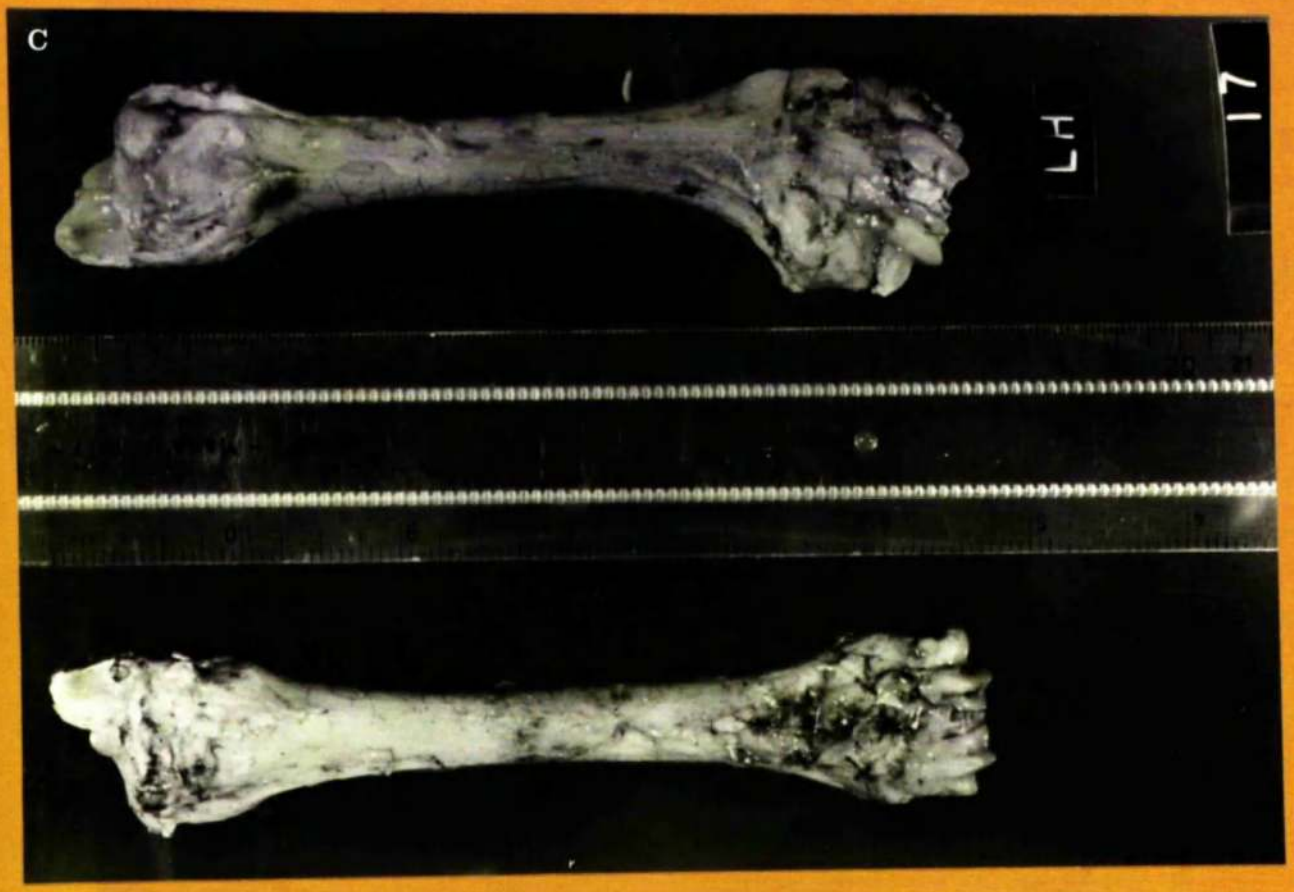
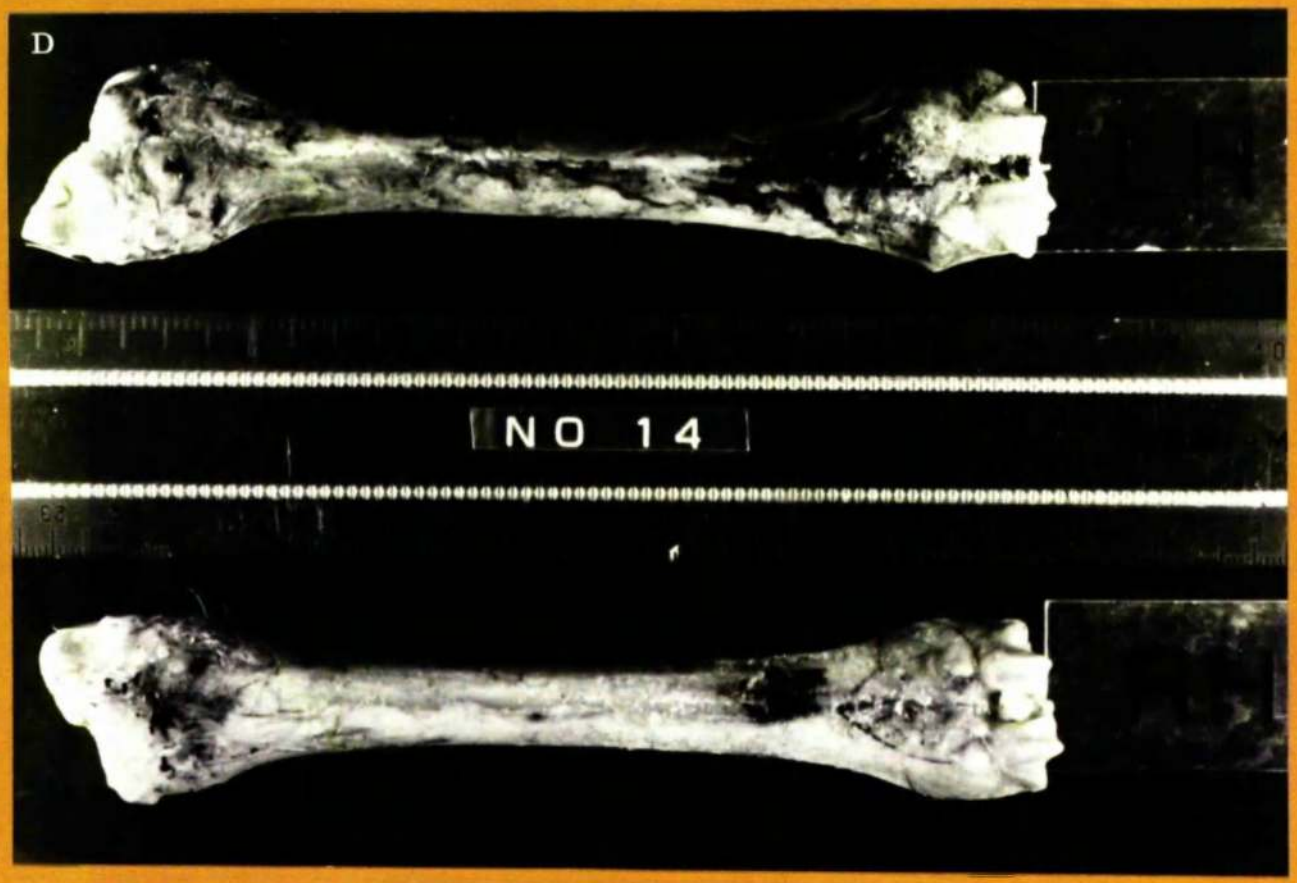


PLATE 21 C&D Examples of distal metatarsal deformity. N.B. Mild LH metatarsal valgus in lamb no.14. (No.17=8months & No.14=10months postoperatively).



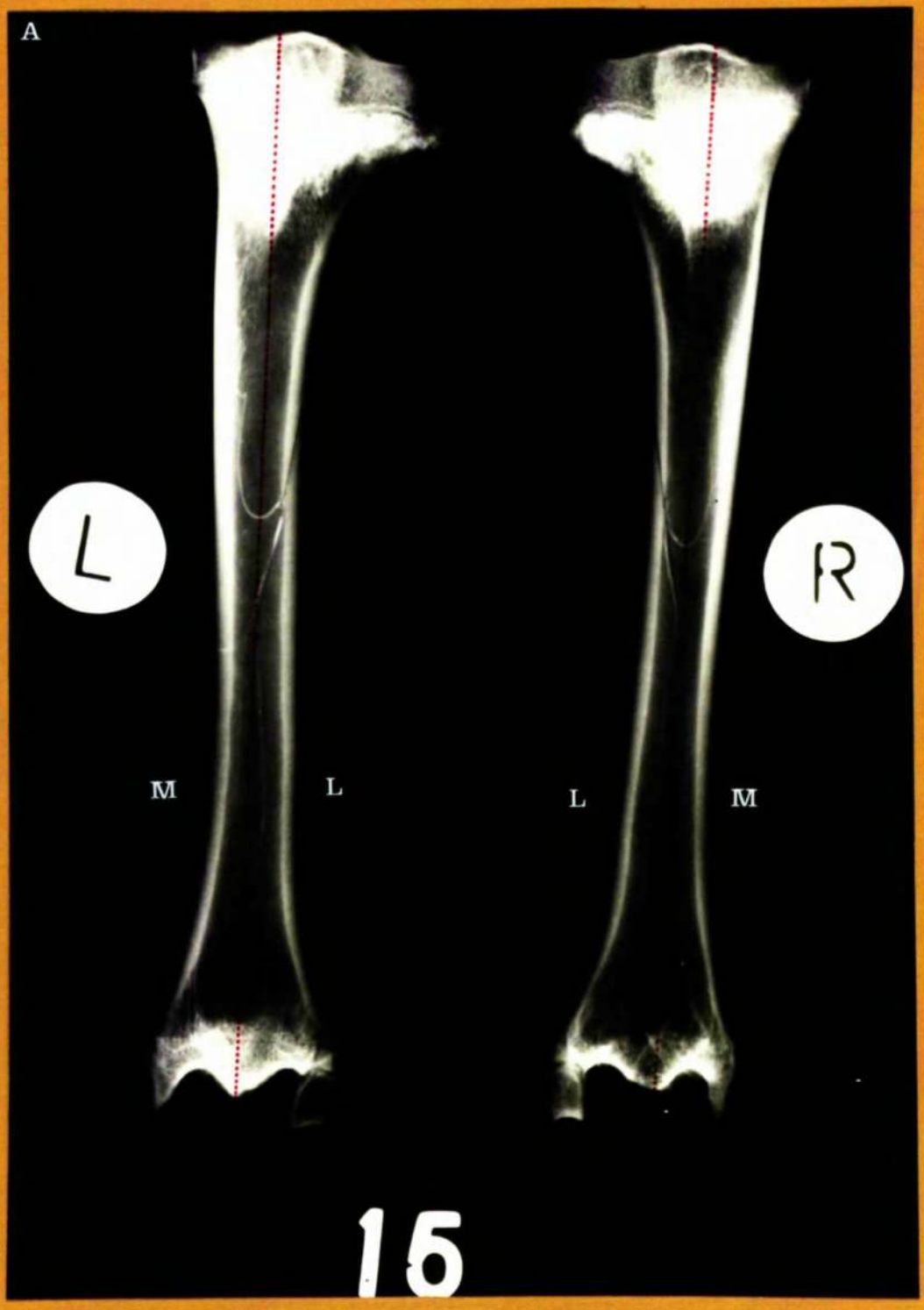


PLATE 22A Anteroposterior radiograph of dissected tibiae, lamb no.15 (10months postoperatively). N.B. Grossly normal appearance of unoperated (LH) limb distal tibia. (L=Lateral cortex, M=Medial cortex, red line=approximate long axis of bone).

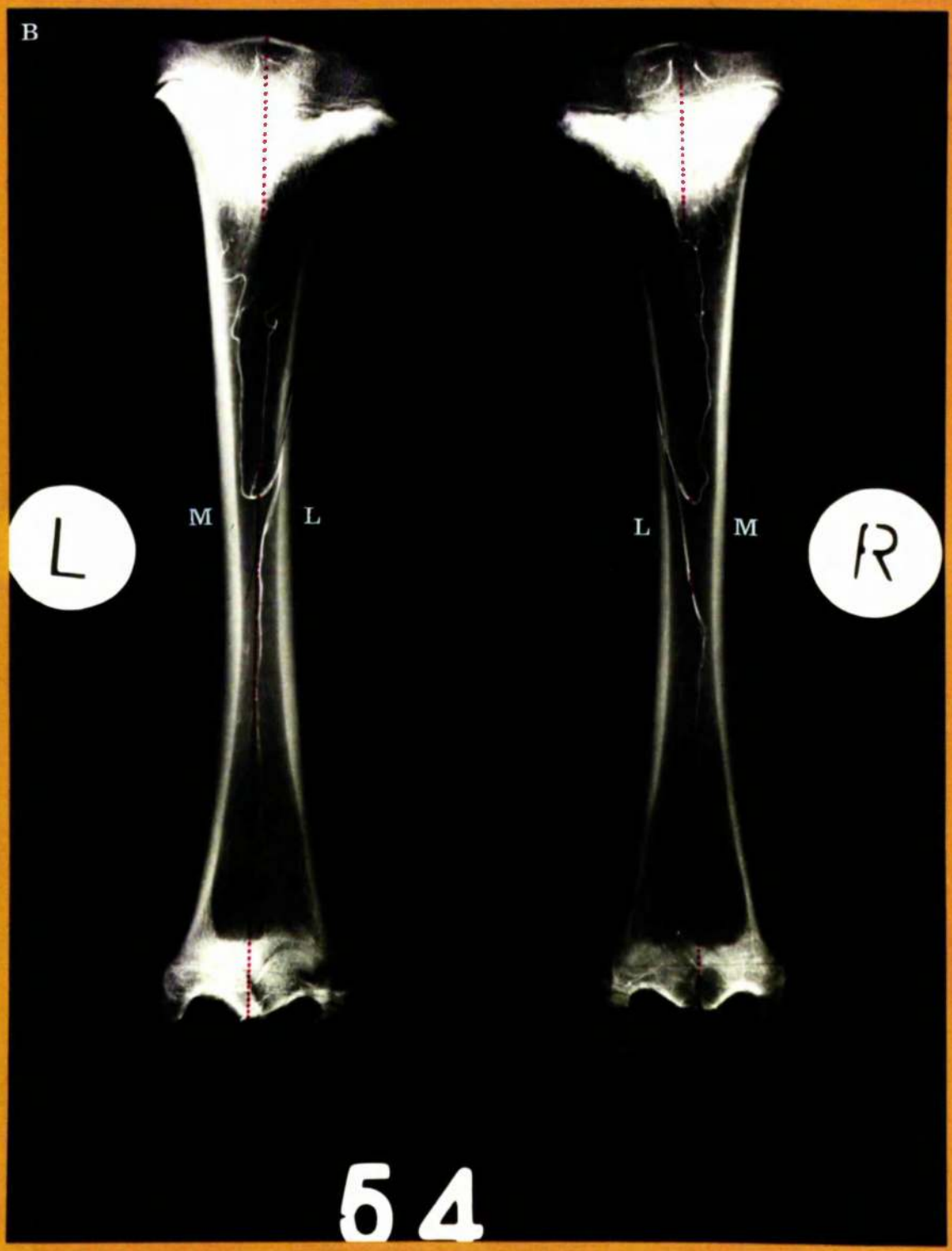


PLATE 22B Anteroposterior radiograph of dissected tibiae, lamb no.54 (4months postoperatively). N.B.radiolucent defect in lateral metaphysis of unoperated (LH) limb distal tibia.

C

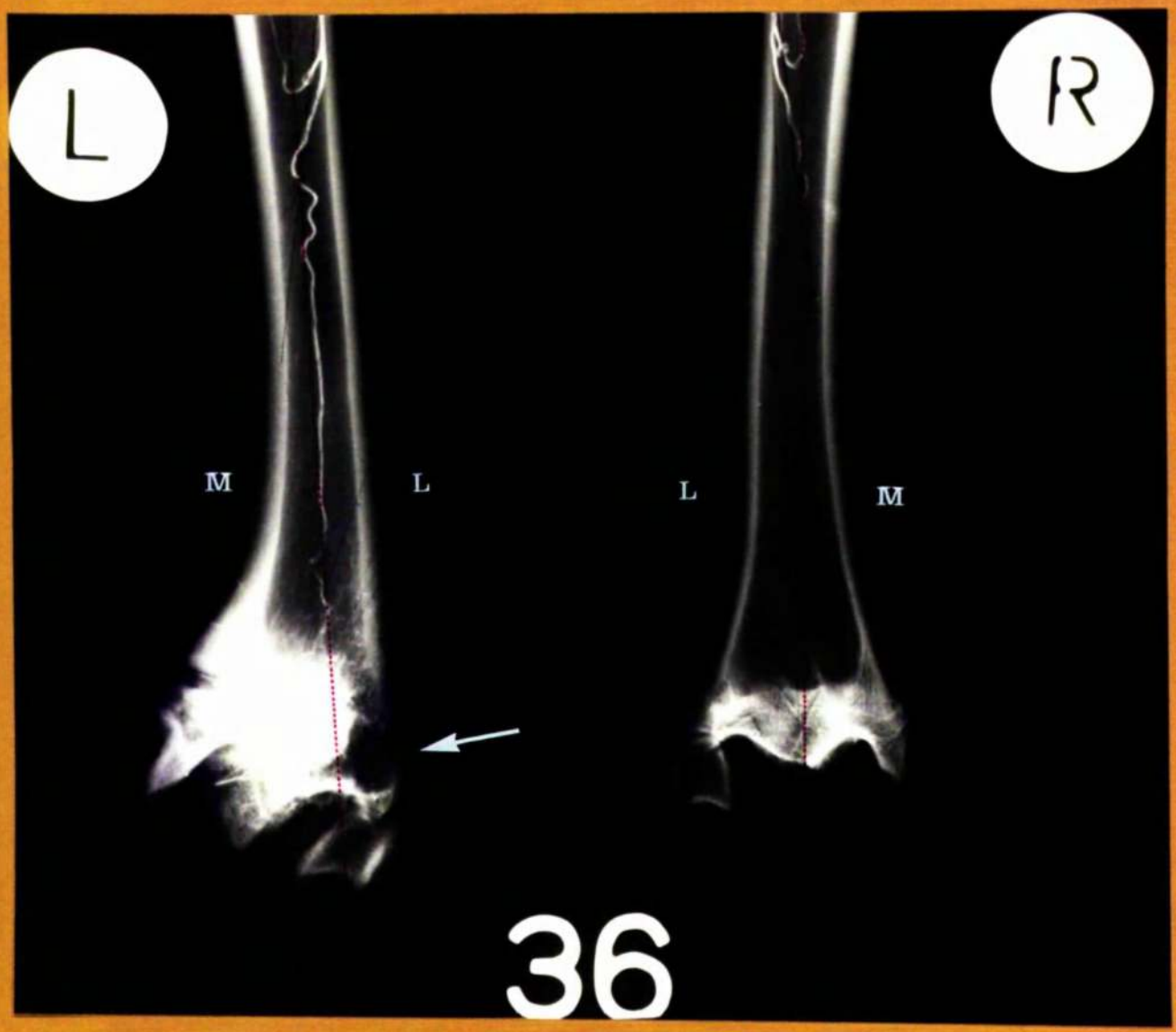


PLATE 22C Anteroposterior radiograph of dissected tibiae, lamb no.36 (5months postoperatively).
N.B. More extensive lateral metaphyseal defect (arrowed) and increased distance between epiphysis and metaphysis medially. Slightly greater medial cortical thickness is present.

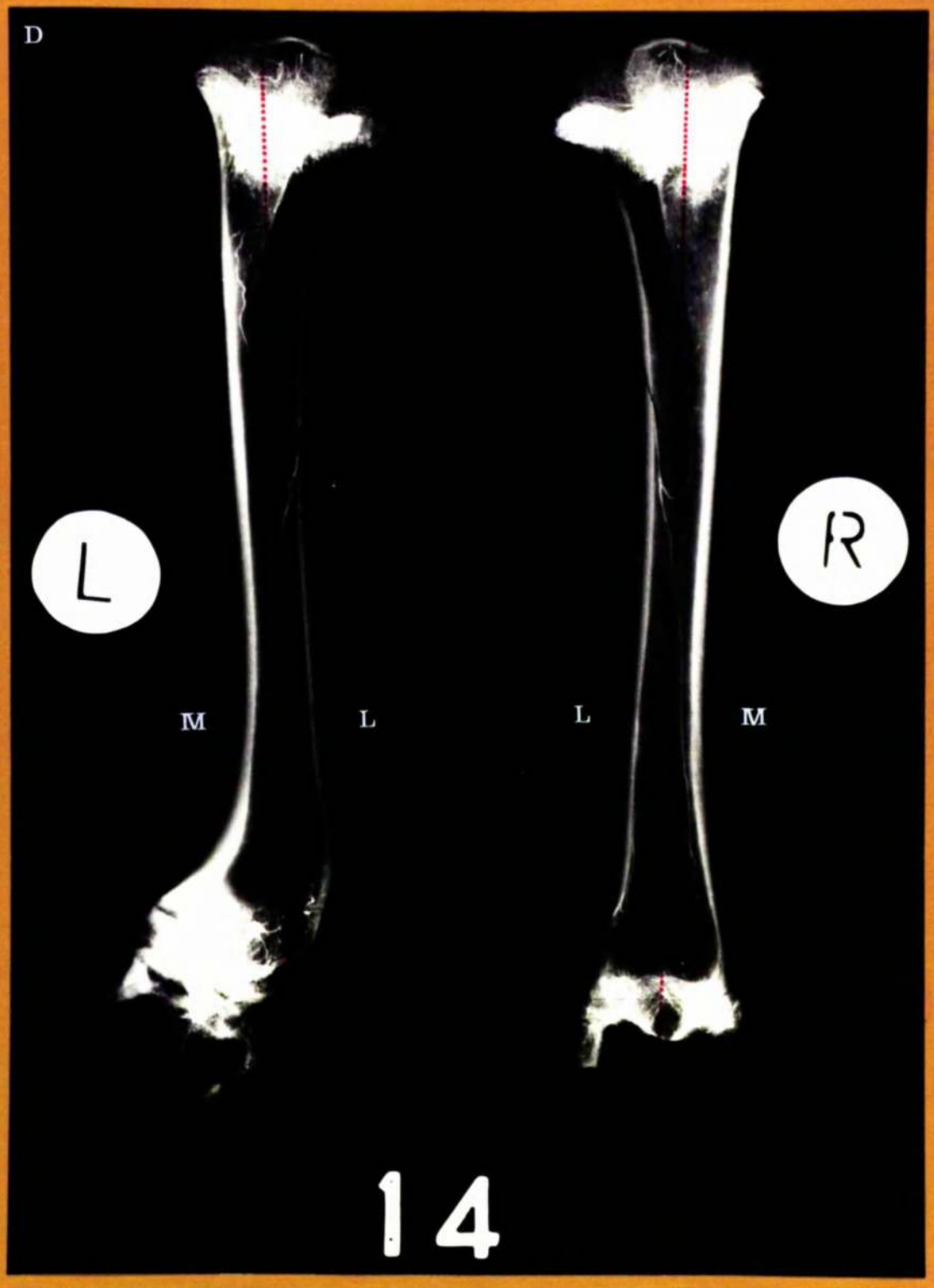


PLATE 22D Anteroposterior radiograph of dissected tibiae, lamb no.14,(10months postoperatively). N.B. Gross lateral metaphyseal defect, increased distance between epiphysis and metaphysis medially and increased medial cortical density both in diaphysis and metaphysis.

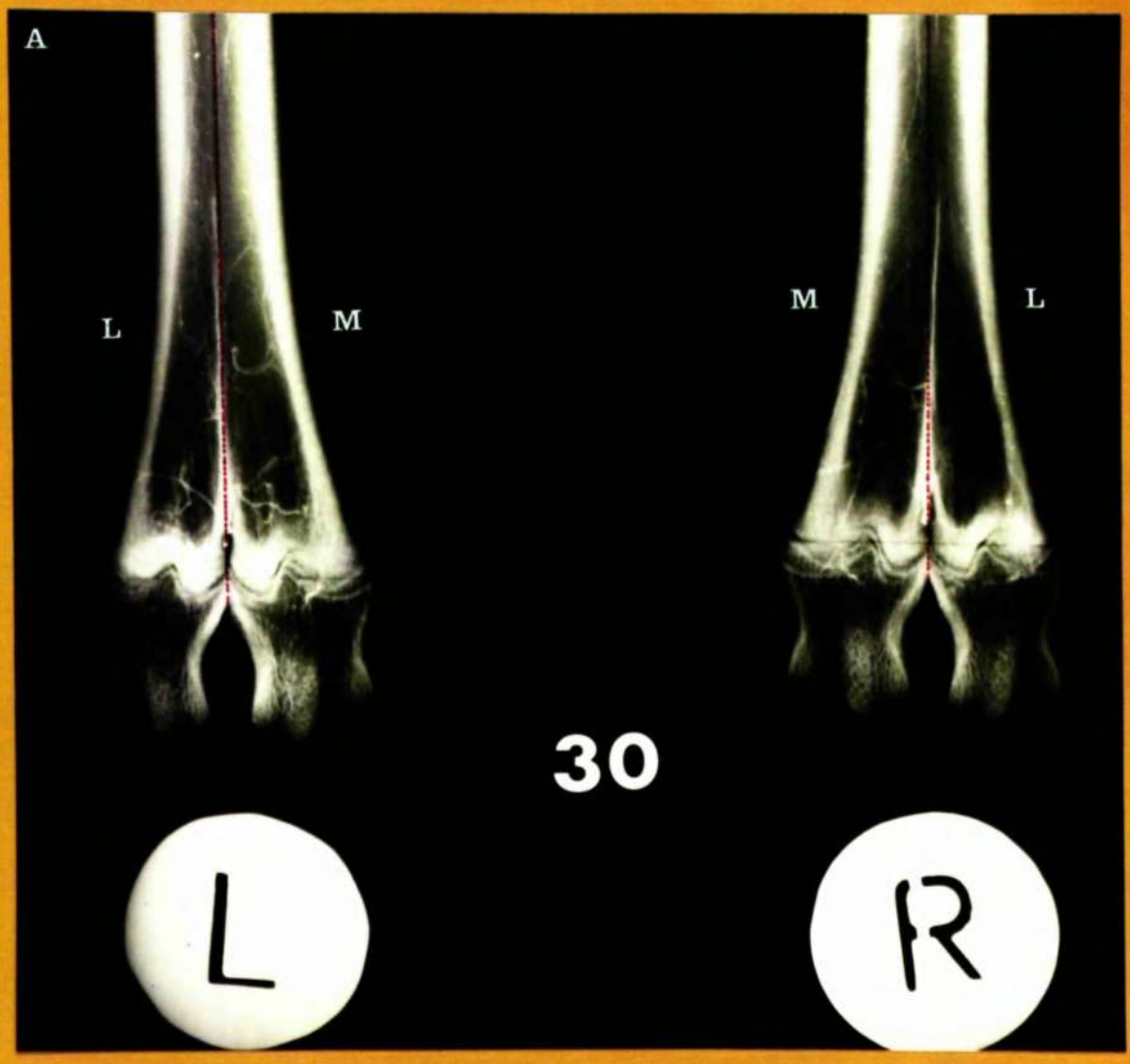


PLATE 23A Anteroposterior radiograph of dissected metatarsi, lamb no.30 (4months control). N.B. Slightly thicker medial cortex and slightly increased distance between epiphysis and metaphysis medially.

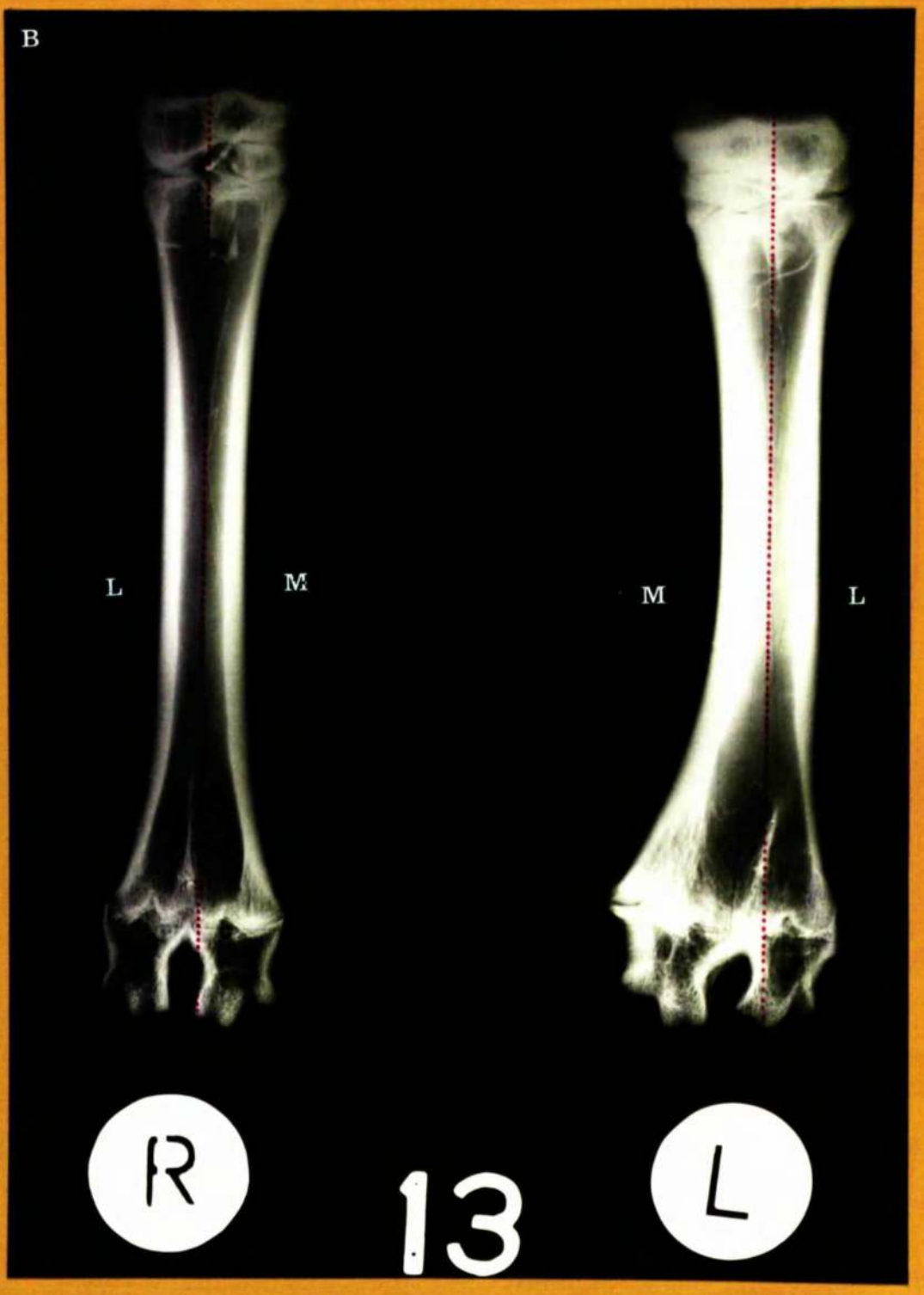


PLATE 23B Anteroposterior radiograph of dissected metatarsi, lamb no.13 (12months postoperatively). N.B. Obvious disparity in dimensions of LH lateral and medial epiphysis. Also, increased distance between epiphysis and metaphysis medially, denser metaphyseal trabecular pattern medially and more pronounced peaking of medial epiphysis and metaphysis.

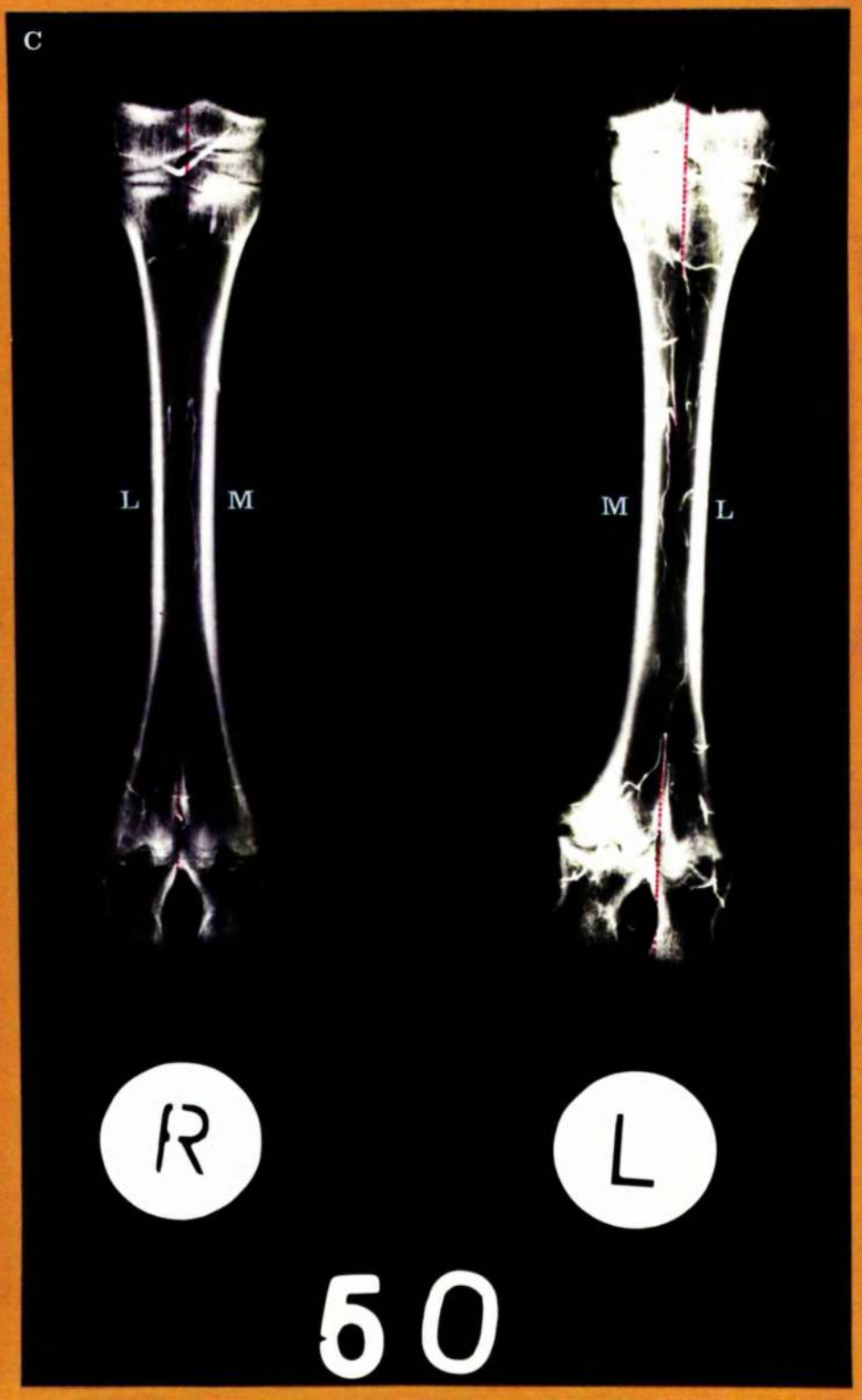


PLATE 23C Anteroposterior radiograph of dissected metatarsi, lamb no.50 (2months postoperatively). N.B. Lateral metaphyseal radiolucency, increased distance between epiphysis and metaphysis medially and dense medial metaphyseal trabeculae.

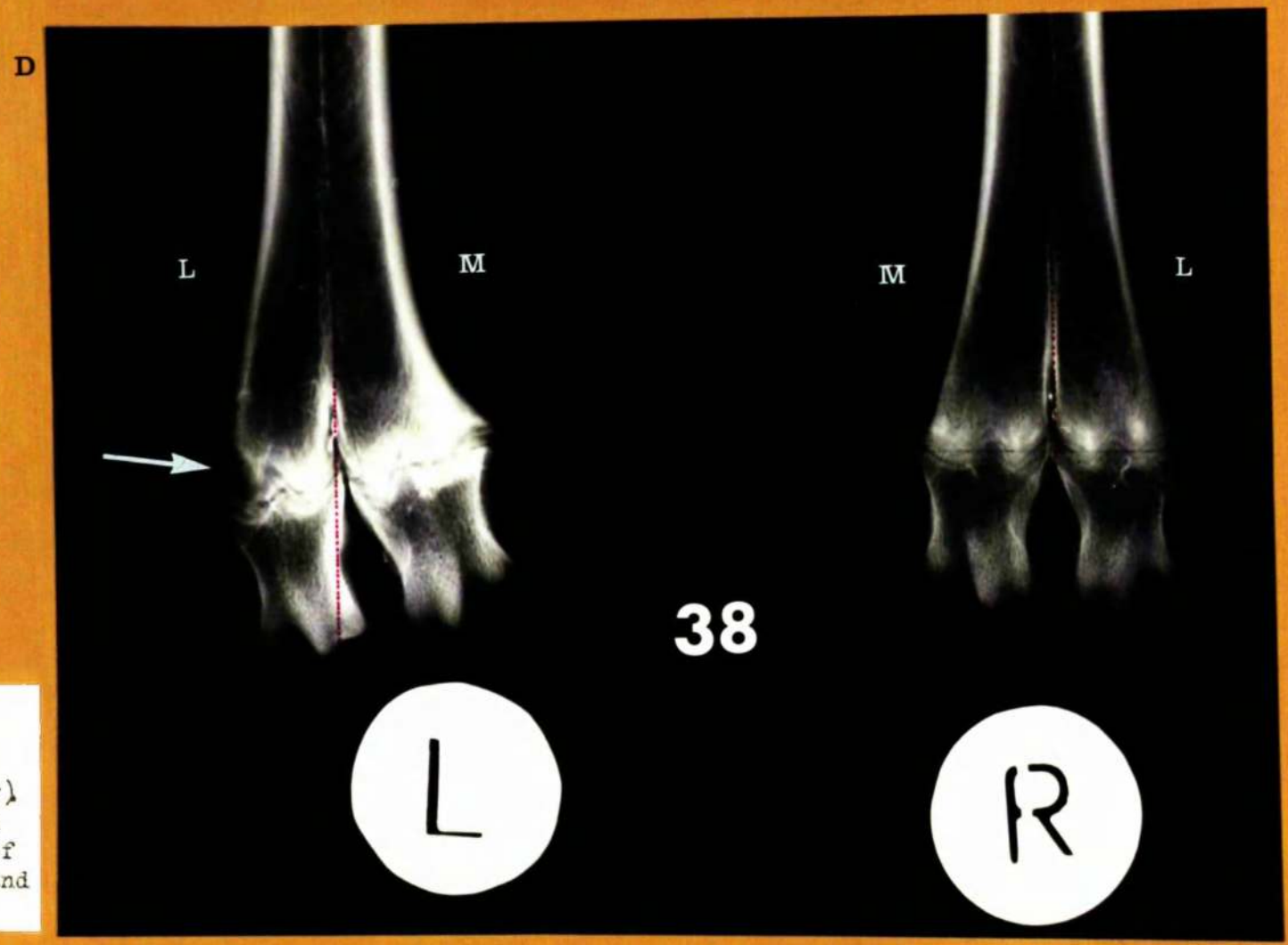


PLATE 23D Anteroposterior radiograph of dissected metatarsi, lamb no.38 (4months postoperatively) N.B. Lateral metaphyseal defect with small area of bone separating defect and growth plate.

E

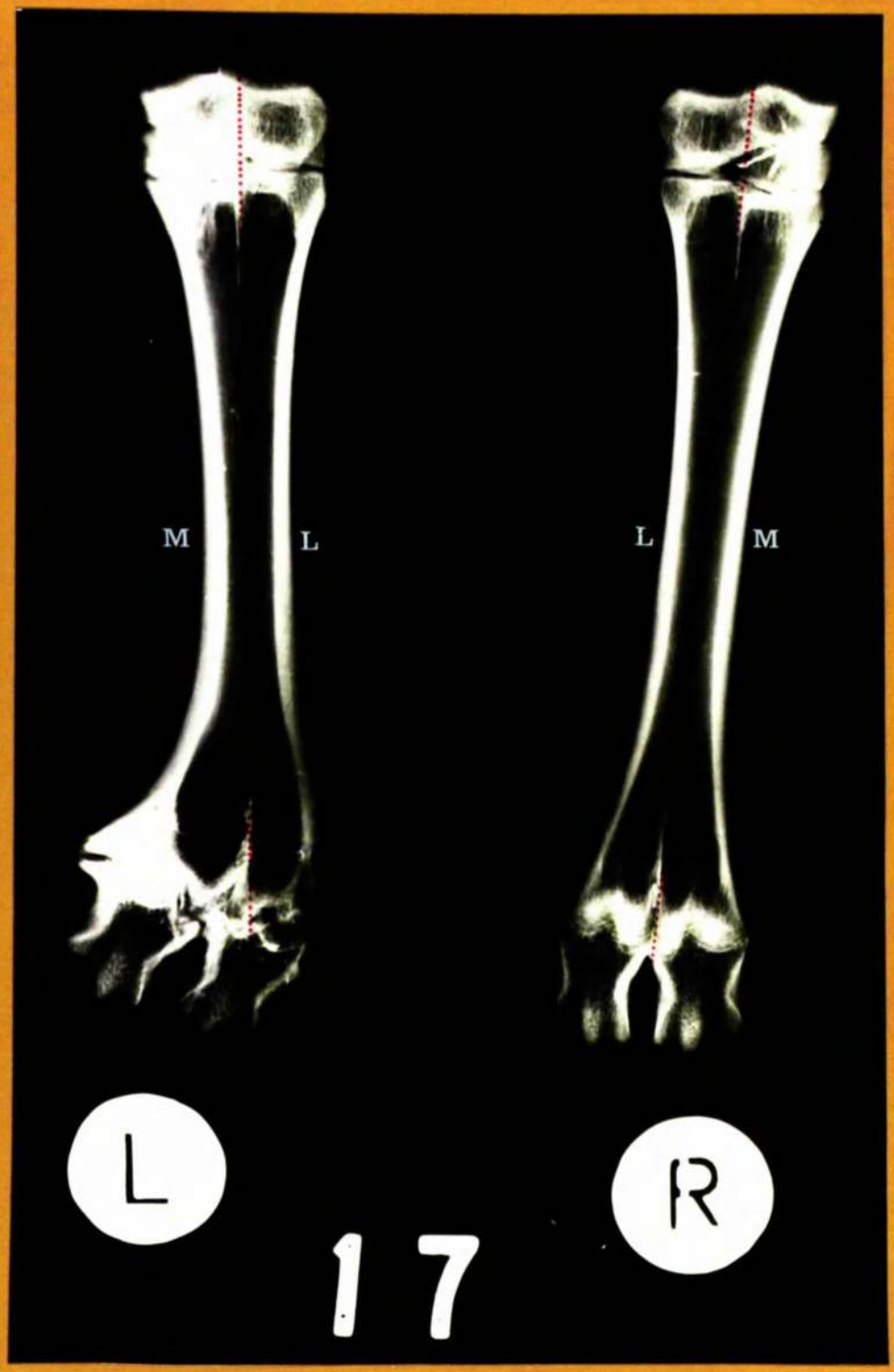


PLATE 23E Anteroposterior radiograph of dissected metatarsi, lamb no.17 (8months postoperatively). N.B. New bone separating lateral metaphyseal defect and growth plate. Increased distance between medial metaphysis and epiphysis is marked. Medial metaphyseal trabecular density is grossly increased and medial metaphyseal/epiphyseal peaking is pronounced.

F

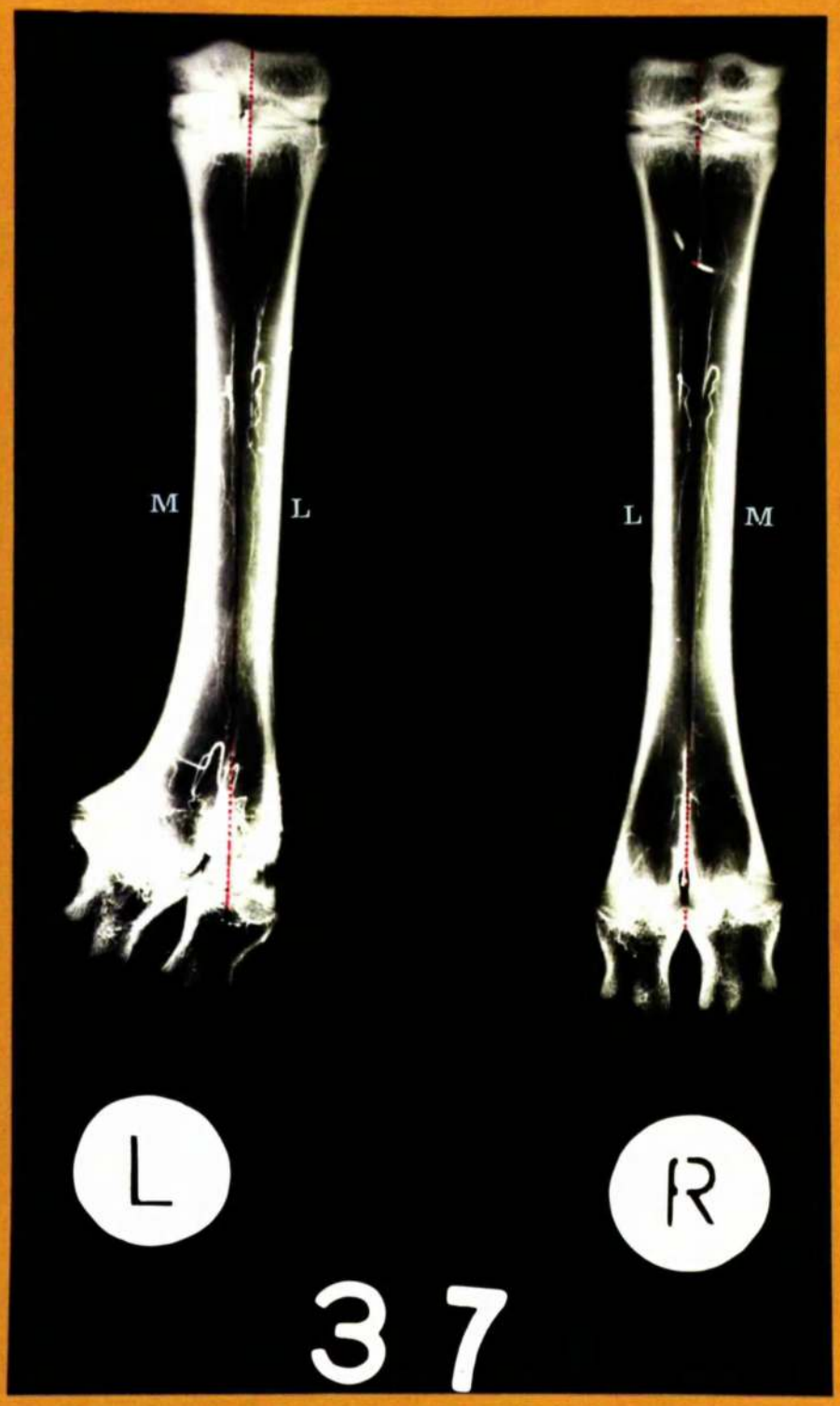


PLATE 23F Anteroposterior radiograph of dissected metatarsi, lamb no.37 (6months postoperatively). N.B. More obvious new bone separating lateral metaphyseal defect and growth plate. Medial diaphyseal cortical thickness is relatively increased.

G

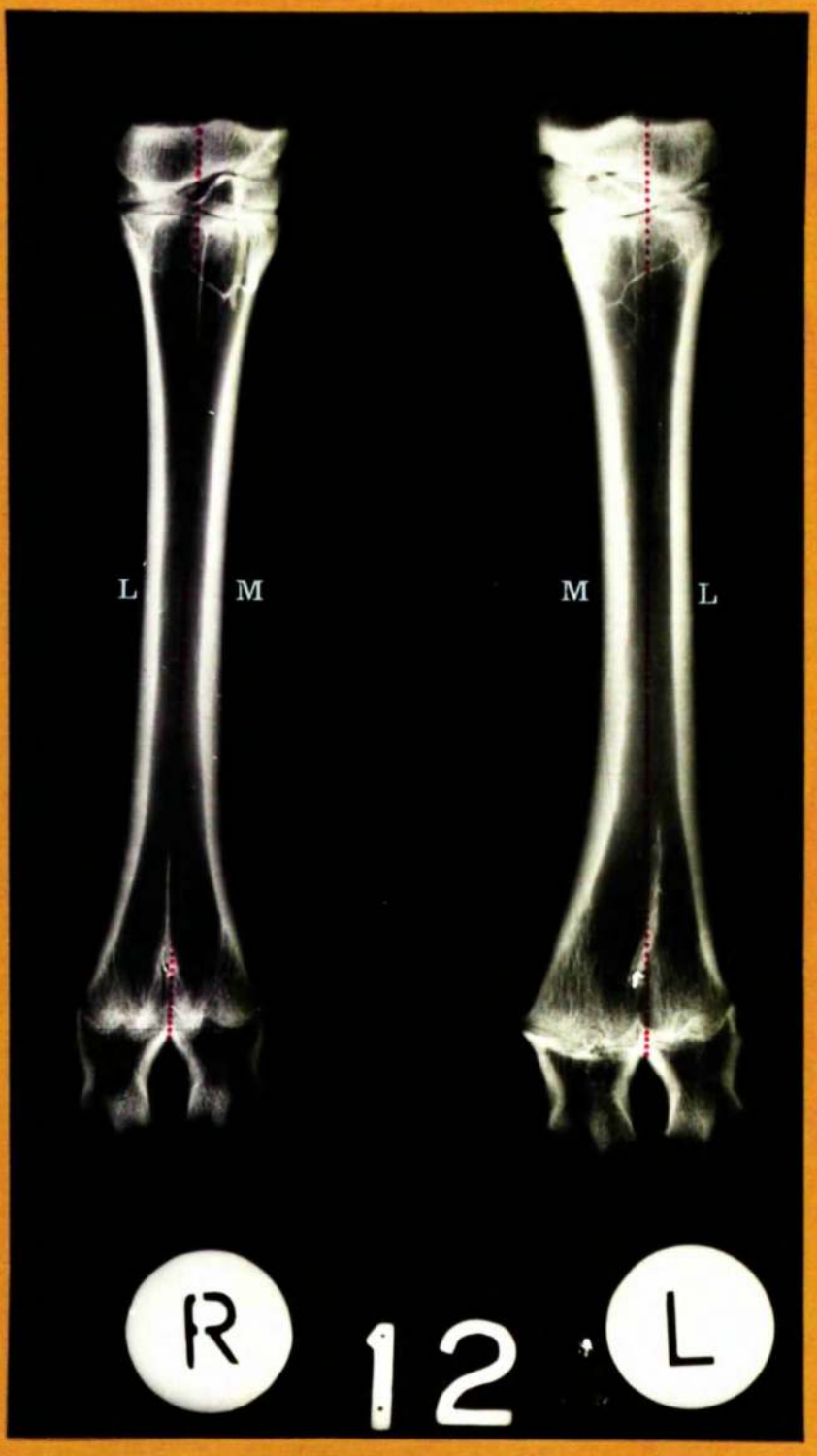


PLATE 23G Anteroposterior radiograph of dissected metatarsi, lamb no.12 (12months postoperatively). N.B. Mild valgus of LH nonoperated limb metatarsus. Medial diaphyseal cortical thickness is however greater in both metatarsi and a degree of medial metaphyseal/epiphyseal peaking is present in LH metatarsus.

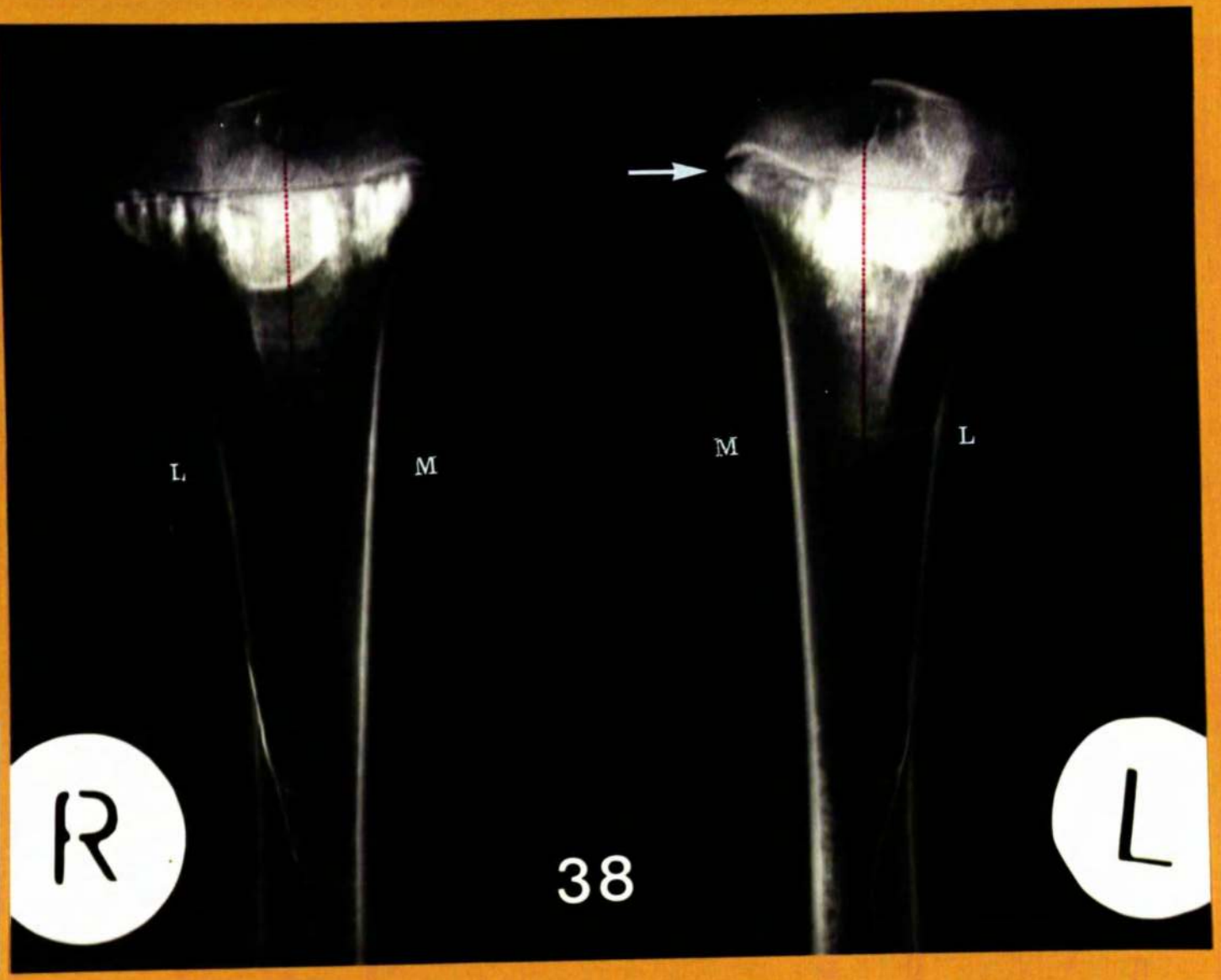


PLATE 24 Anteroposterior radiograph of lamb no.38 proximal tibiae, (4months post-operatively). N.B. Increased space between epiphysis and metaphysis medially in LH nonoperated limb (arrowed).

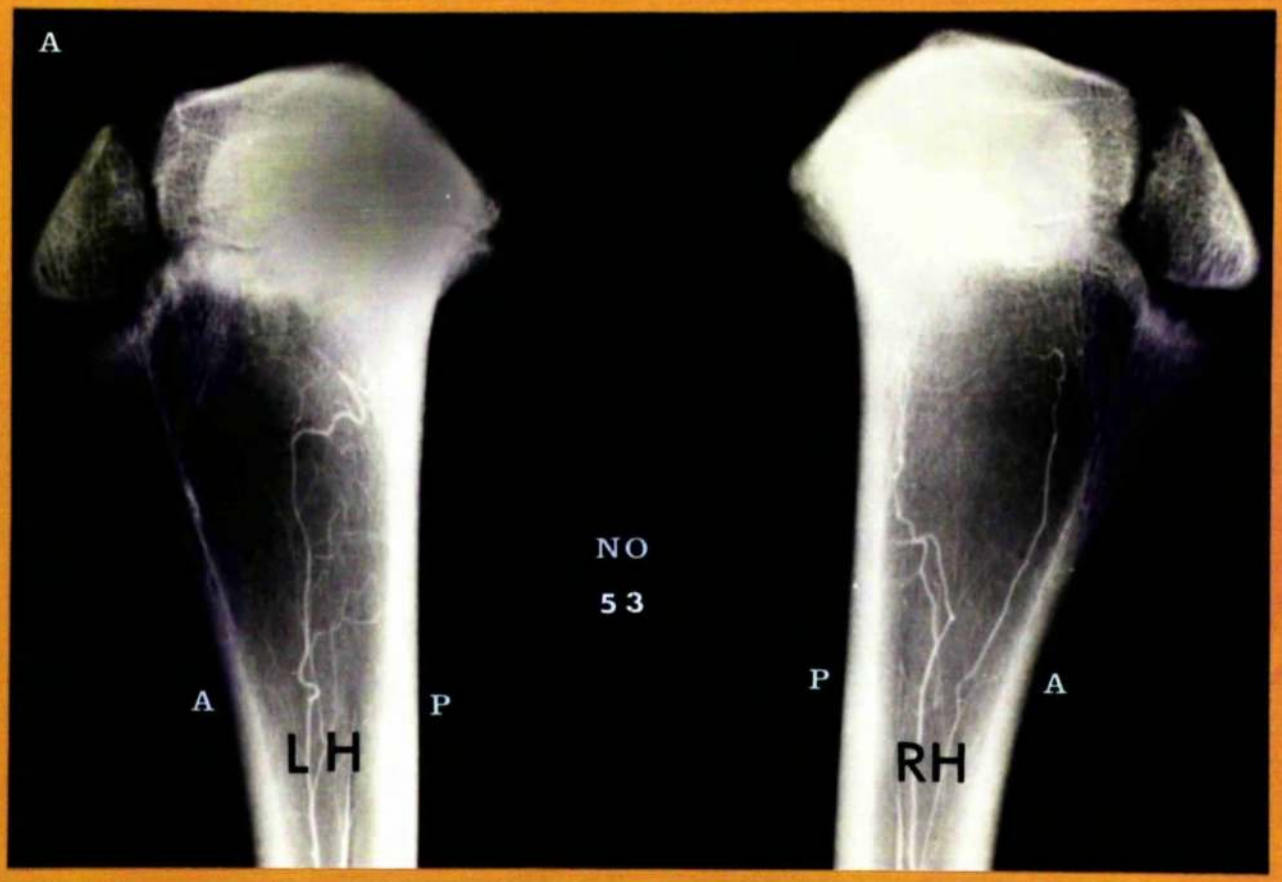
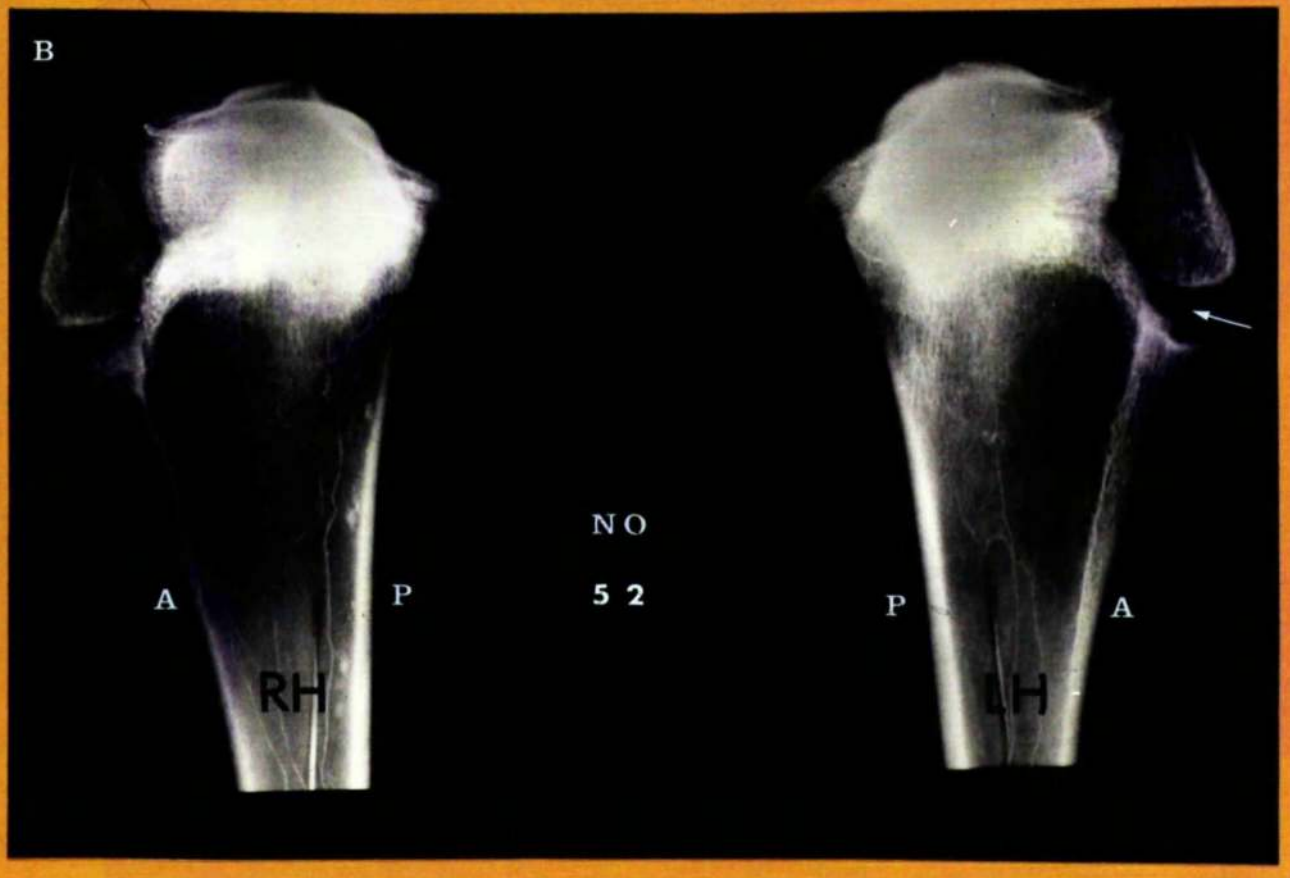


PLATE 25 A&B Lateral radiographs of proximal tibiae, lambs no.53 (3months control) and 52 (3months postoperatively). N.B. Relatively greater space between tibial tuberosity and tibial crest, lamb no.52 LH.



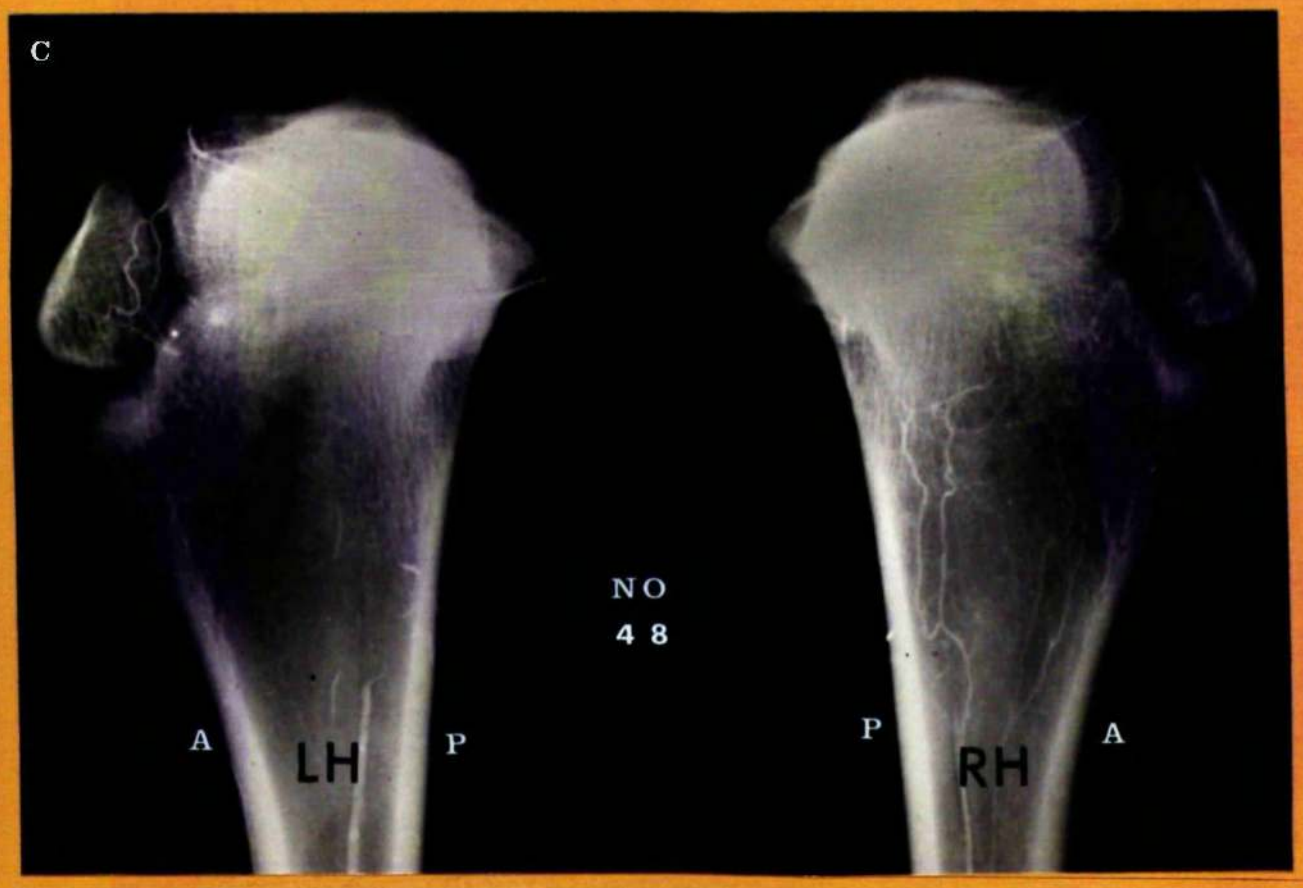
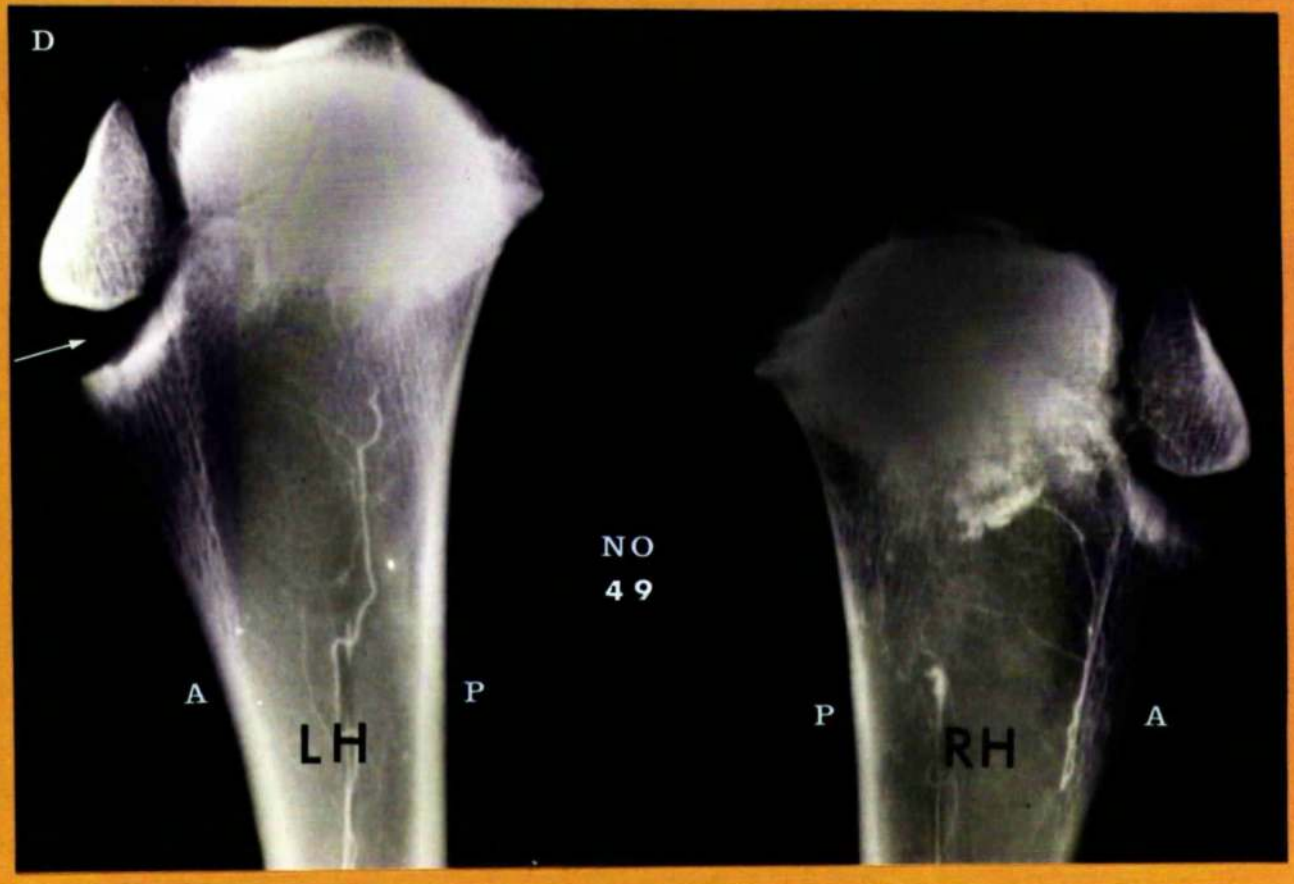


PLATE 25 C&D Lateral radiographs of proximal tibiae, lambs no.48 (2months control) and 49 (2months postoperatively). N.B. Relatively greater space between tibial tuberosity and tibial crest, lamb no.49 LH.



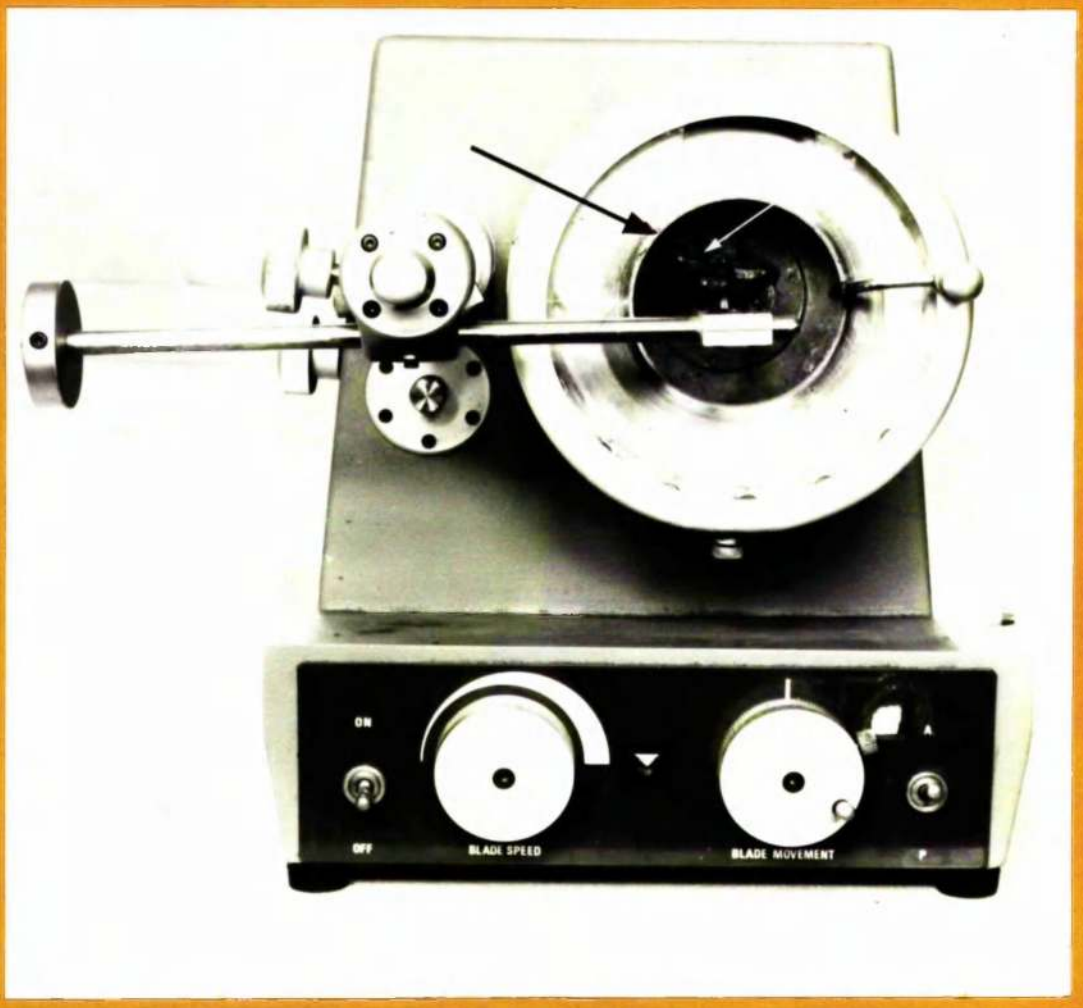


PLATE 26 The Metals Research Microslice 2 precision saw. The rotating annular blade (large arrow) is brought into contact with the wax-encased bone specimen (small arrow) by release of a locking mechanism on the counterbalanced arm. The speed of sectioning is adjusted by means of the variable speed control for the annular blade and the counterbalance weight on the end of the arm.

A

PLATE 27A Spalteholz prepared section of lamb no.44, LH distal metatarsus. (3week control). m=metaphysis, e=epiphysis. N.B. Step like defect in metaphysis.

B

PLATE 27B Spalteholz prepared section of lamb no.43 lateral LH distal metatarsus (3week post-operatively). m=metaphysis, e=epiphysis. N.B. Fracture line traversing metaphyseal trabeculae/spongiosa.

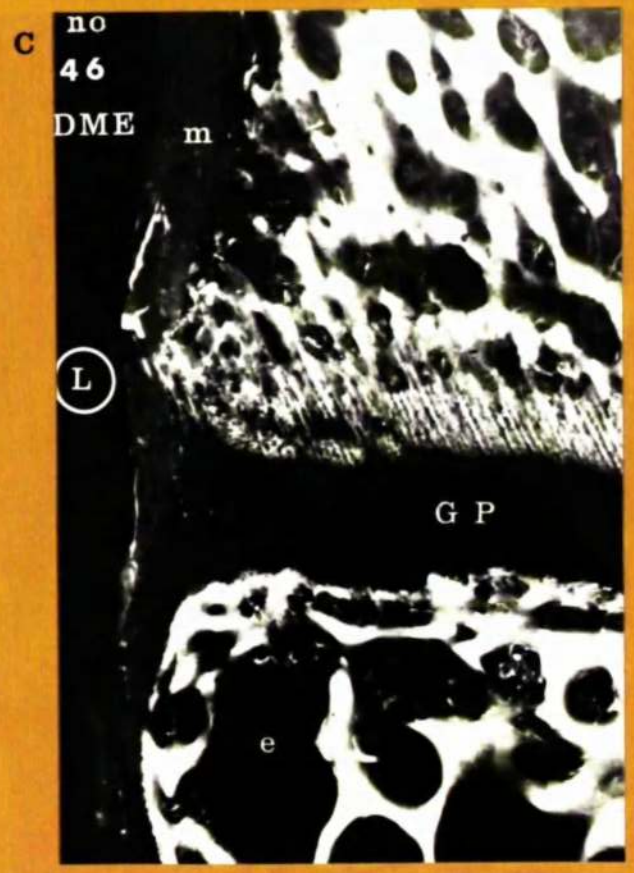


PLATE 27C Microradiograph of lateral distal LH metatarsus, lamb no.46 (1month postoperatively). m=metaphysis, e=epiphysis, G.P.= growth plate. N.B. Fracture line traversing lateral primary spongiosa.

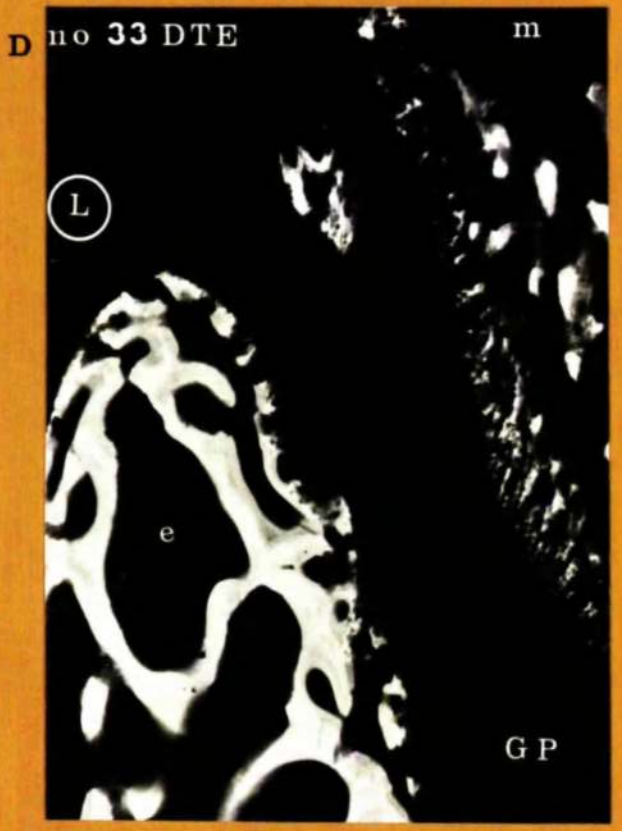
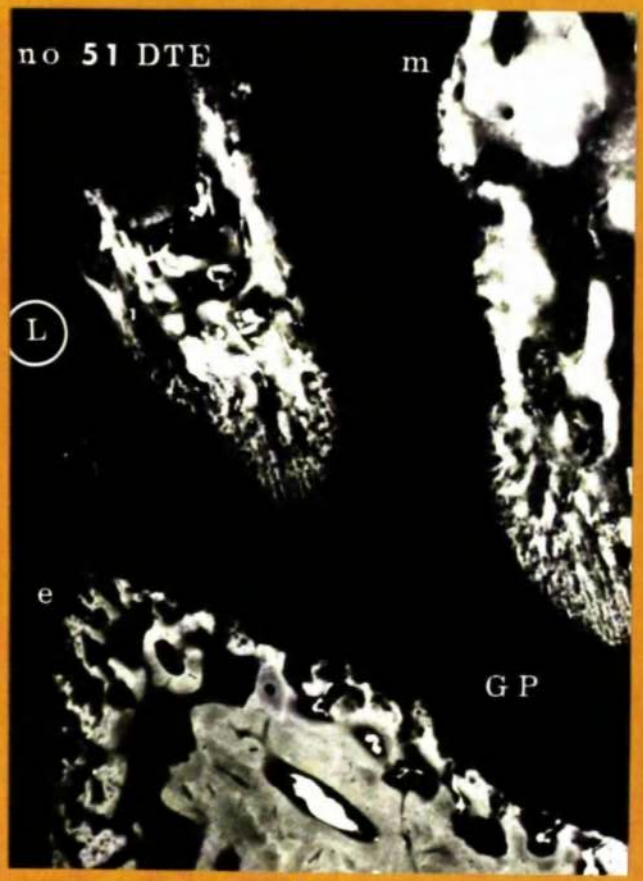
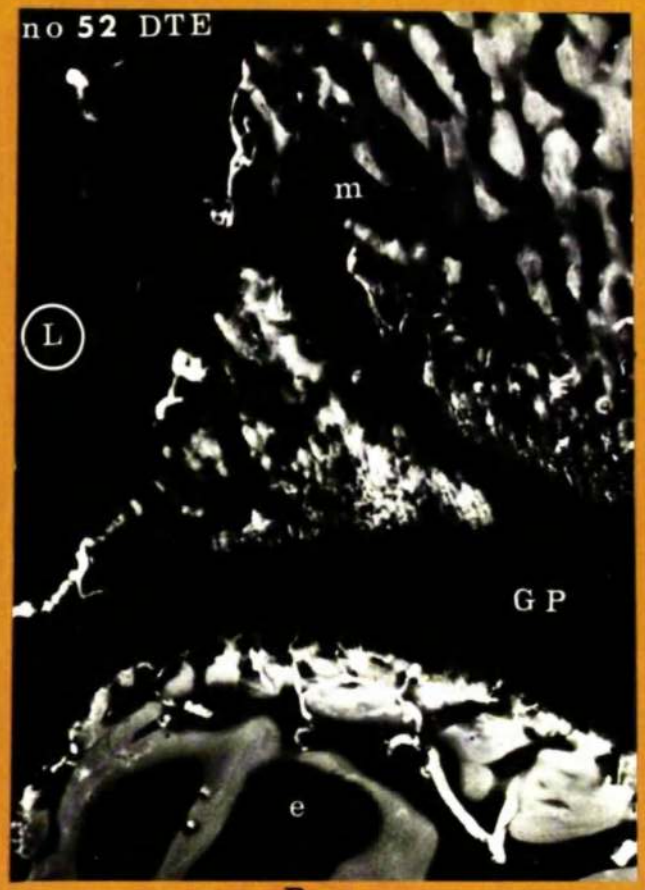


PLATE 27D Microradiograph of lateral distal LH tibia, lamb no.33 (2weeks postoperatively). N.B. Island of trabecular bone in growth plate periphery.



A



B

C

PLATE 28A,B&C Microradiographs of lateral distal tibiae, lambs no. 51 & 52 (3months postoperatively) and lateral distal metatarsus, lamb no.29 (5months postoperatively). N.B. Bone islands in lateral peripheral growth plate.

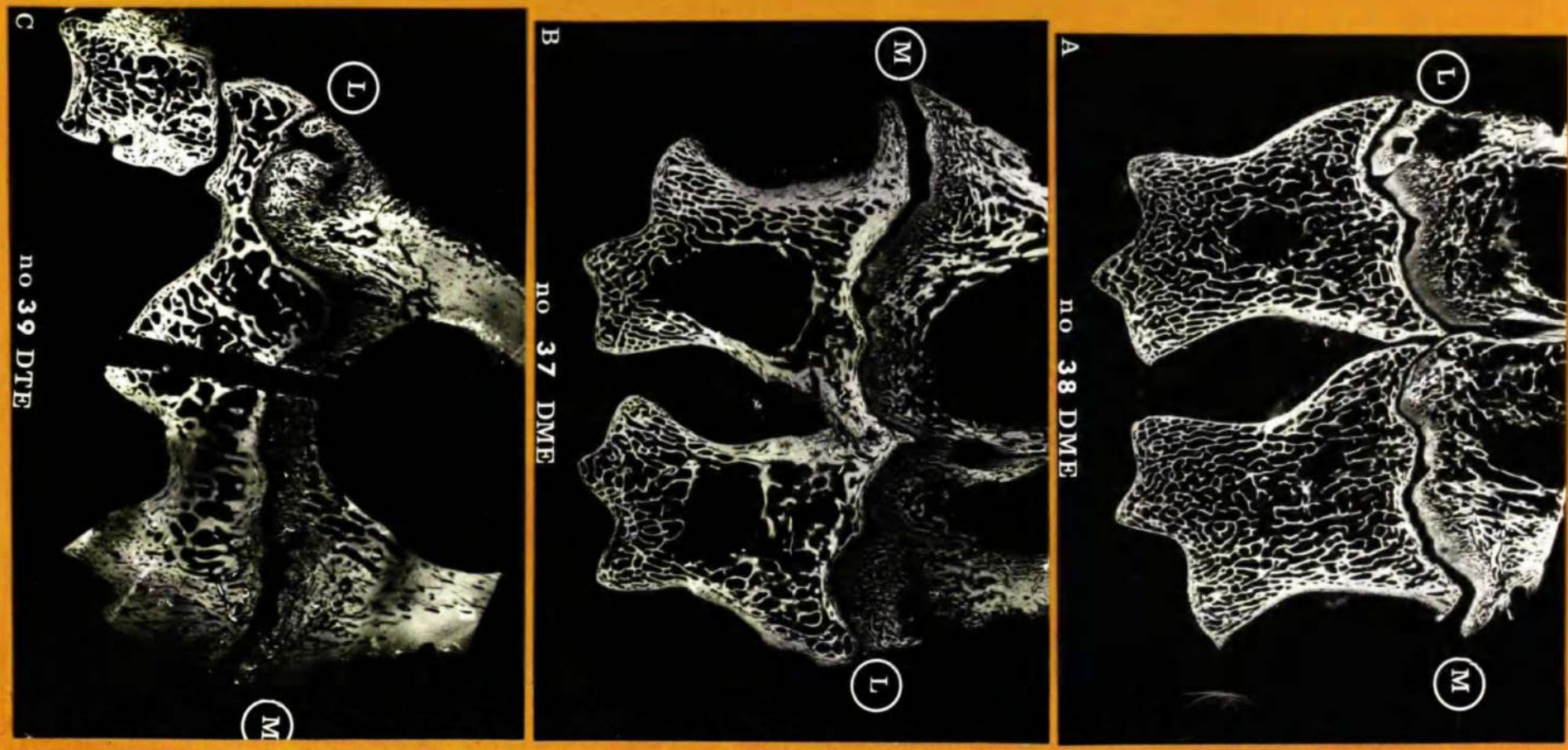


PLATE 29A,B&C Microradiographs of distal metatarsae, lambs 38 (4months postoperatively) and 37 (6months postoperatively) and distal tibia, lamb 39 (6months postoperatively). L=lateral, M=medial. N.B. Increased growth plate space medially and undulated medial metaphyseal contour. Also metaphyseal defects laterally with varying degrees of endochondral bone separating them from growth cartilage.

A

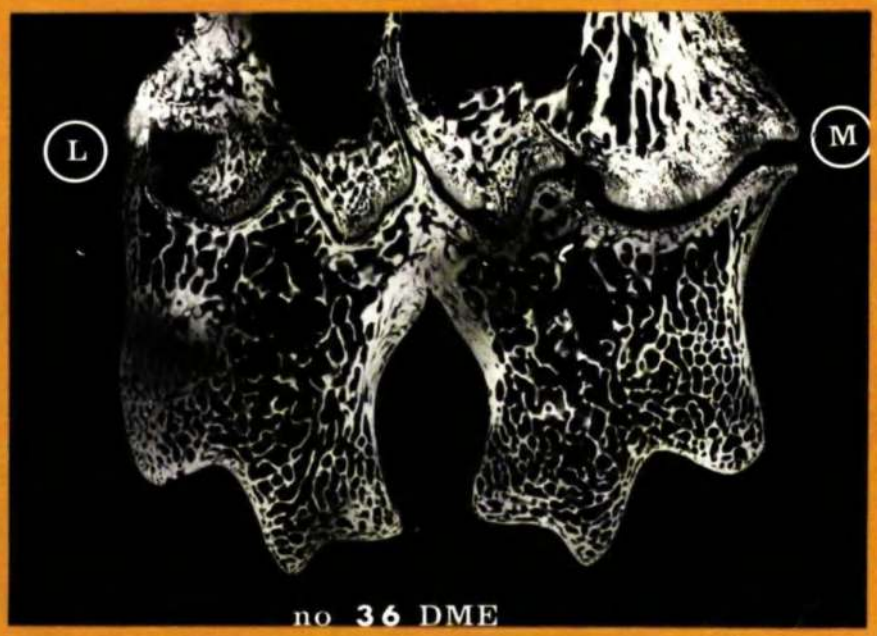


PLATE 30A Microradiograph of distal metatarsus, lamb no.36 (5months postoperatively). N.B. loss of peripheral growth cartilage laterally and protrusion of medial growth plate space into metaphysis.

B

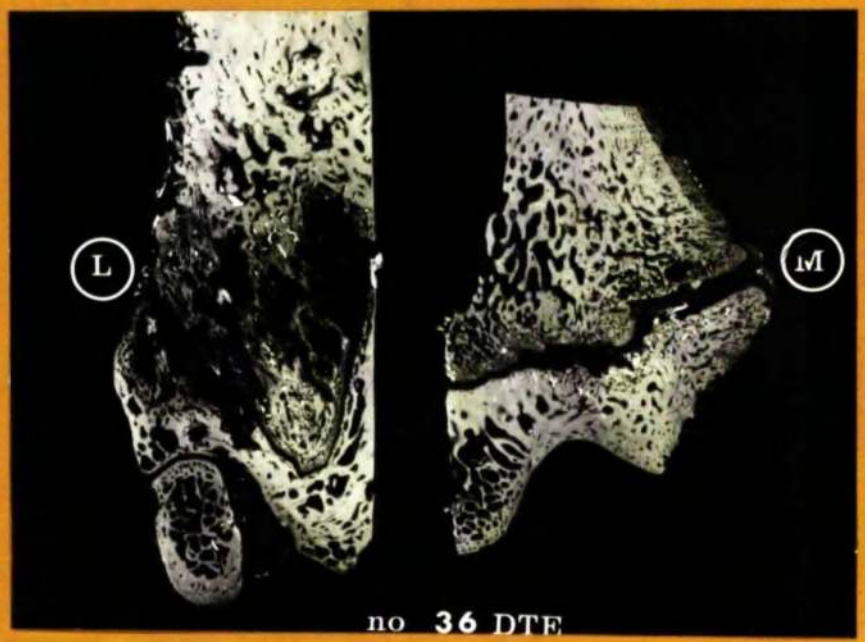


PLATE 30B Microradiograph of distal tibia, lamb no.36 (5months postoperatively). N.B. Gross metaphyseal defect laterally and fusion of lateral periphery of growth plate.

C

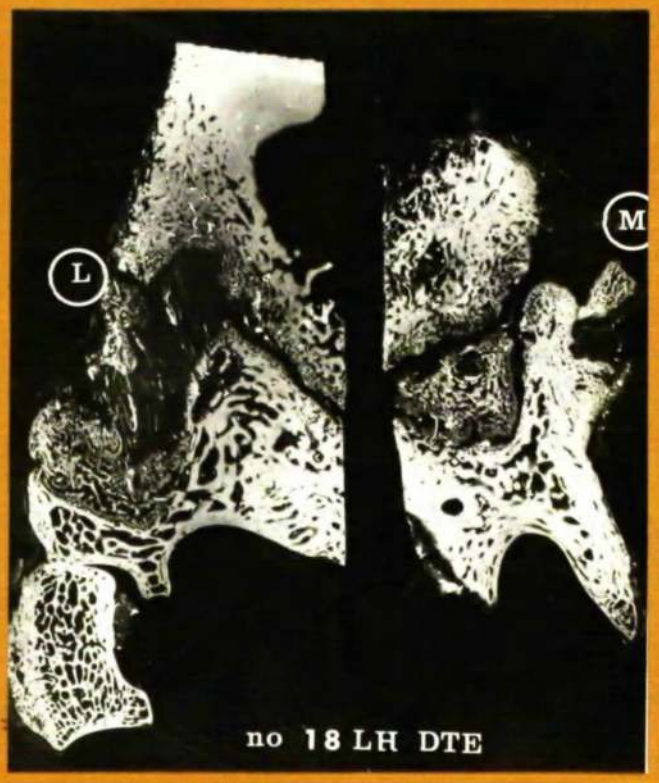


PLATE 30C Microradiograph of distal tibia, lamb no. 18 (8months postoperatively). N.B. Gross lateral metaphyseal defect and fusion of lateral growth plate periphery. Also double medial growth plate line.

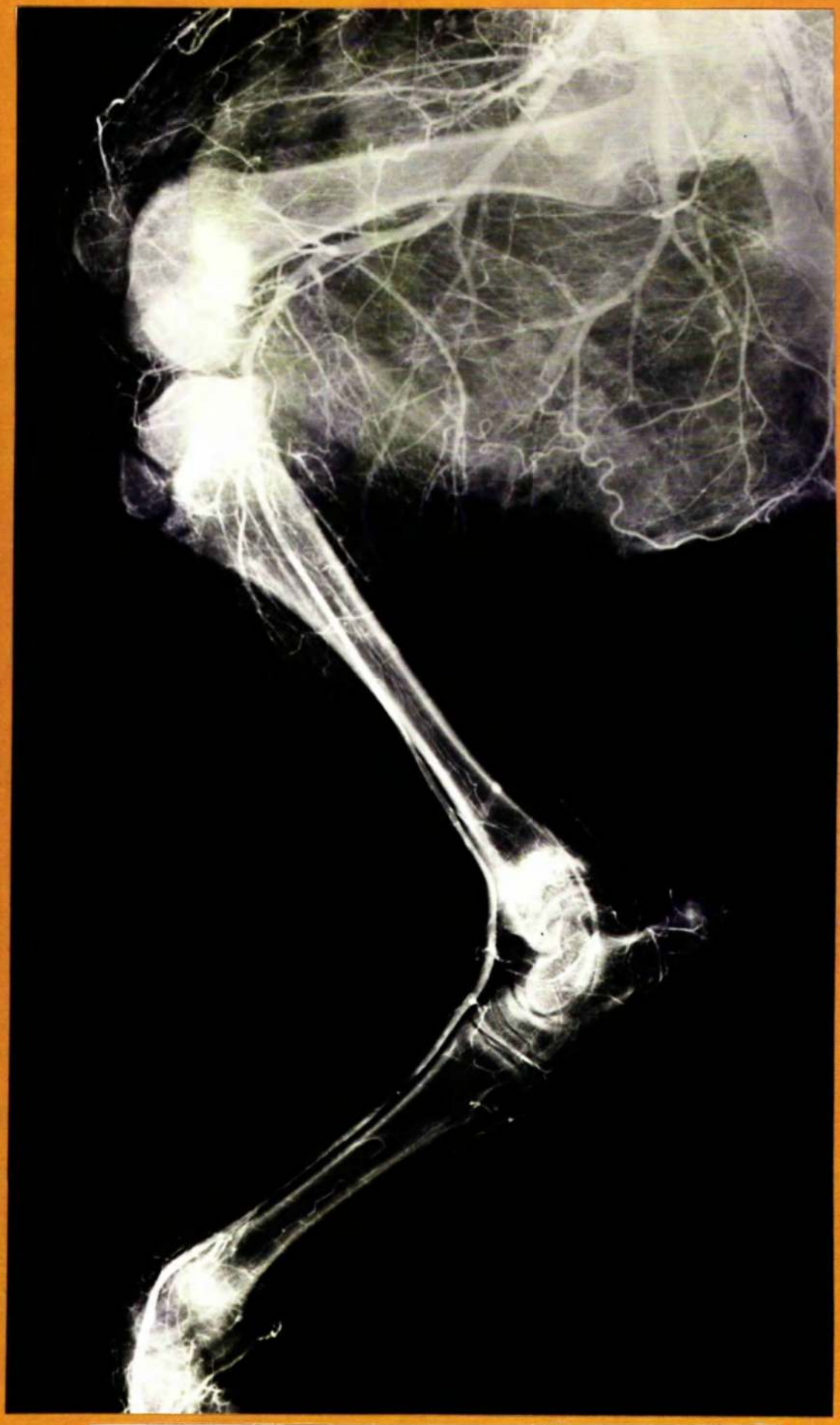
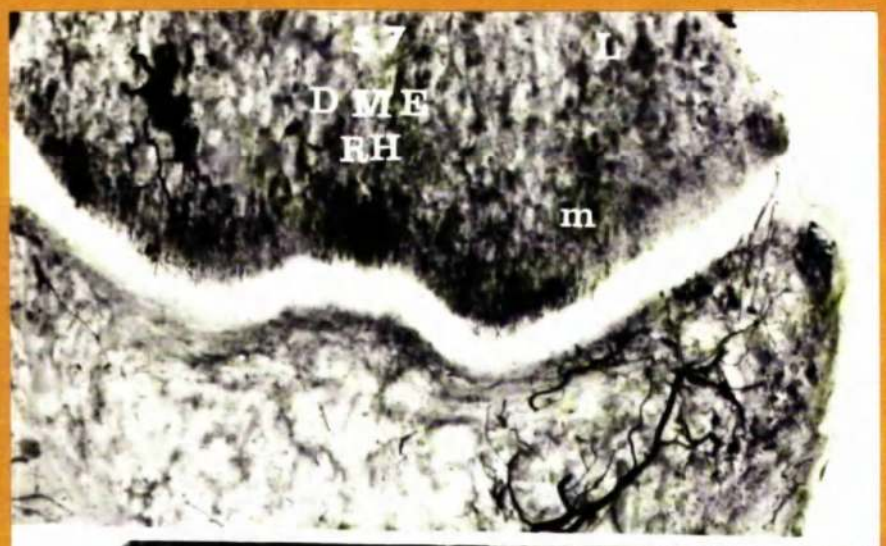


PLATE 31 Example of radiographs taken prior to limb dissection to check efficiency of perfusion.

A

PLATE 32A
Spalteholz prepared section demonstrating epiphyseal vessels and their ramifications which traverse the bone plate.

B



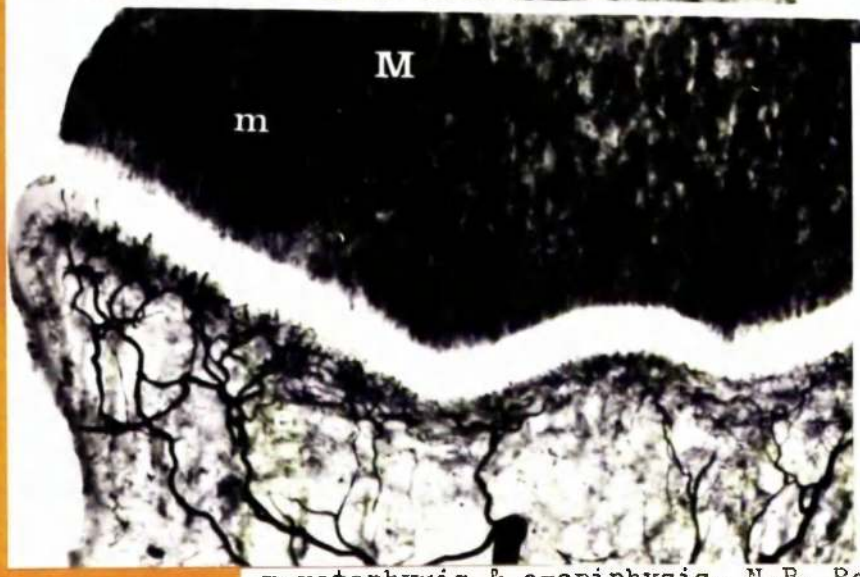
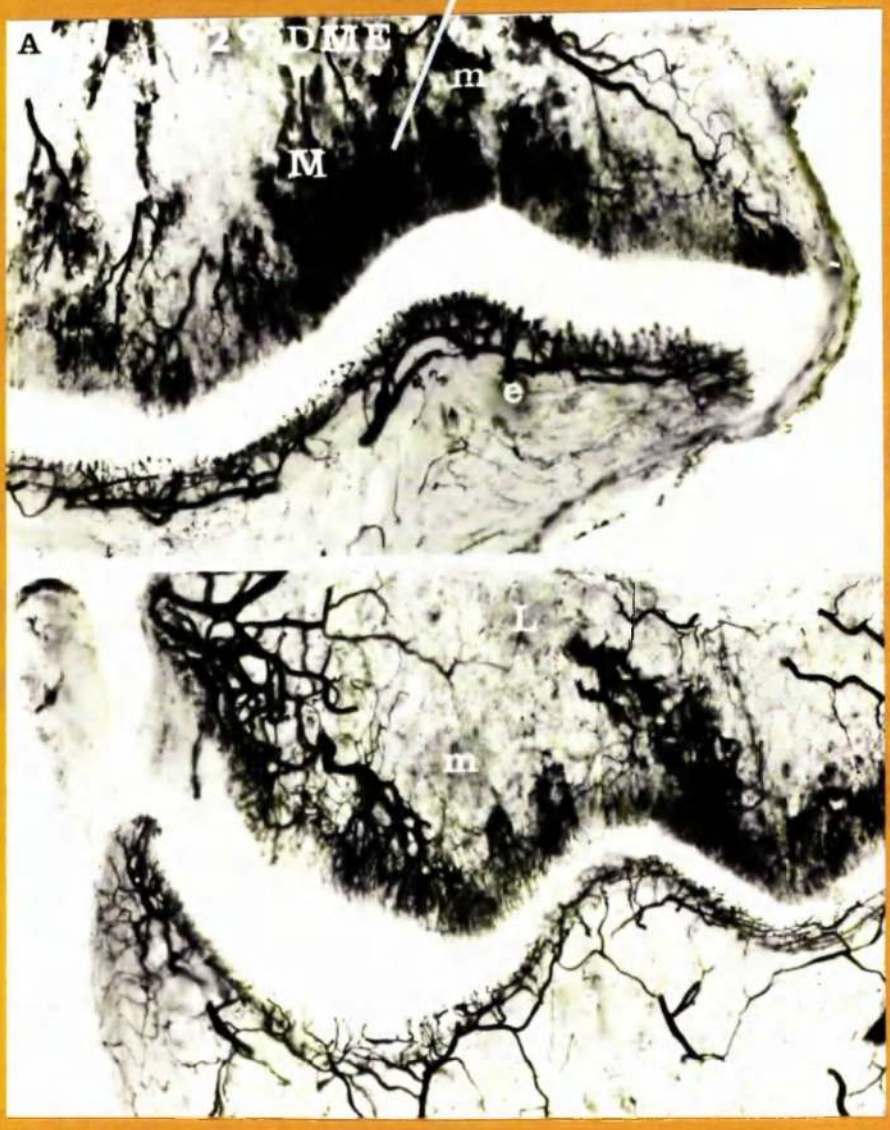


PLATE 32B
Spalteholz prepared section of distal metatarsus, lamb no.57 RH (2week control). The medial (M) and lateral (L) aspects of the same section are presented. m=metaphysis & e=epiphysis. N.B. Relatively more profuse medial epiphyseal and metaphyseal vasculature.

PLATE 32C Spalteholz prepared section demonstrating periosteal/ perichondrial complex of vessels supplying the growth plate/metaphysis periphery. N.B. Vessels traversing the extreme periphery of the growth cartilage.

PLATE 33A Spalteholz prepared section of distal metatarsus, lamb no.29 LH (5months postoperatively). Medial & lateral aspects of same section. N.B. More profuse epiphyseal vasculature medially & epiphyseal vessels entering growth cartilage in areas of thickening.



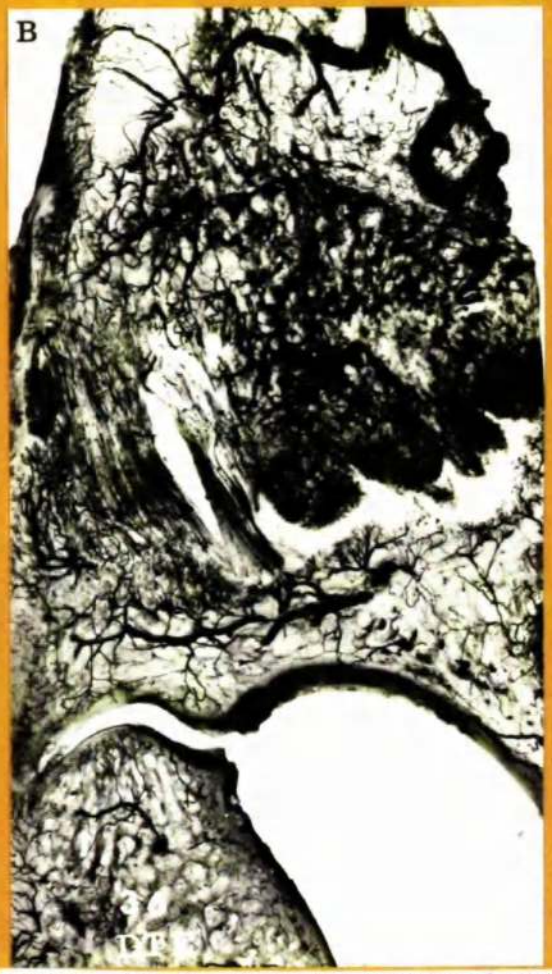


PLATE 33B Spalteholz prepared section of distal lateral LH tibia, lamb no.36 (5months post-operatively). N.B. Tongue of epiphyseal blood vessels entering metaphyseal defect.

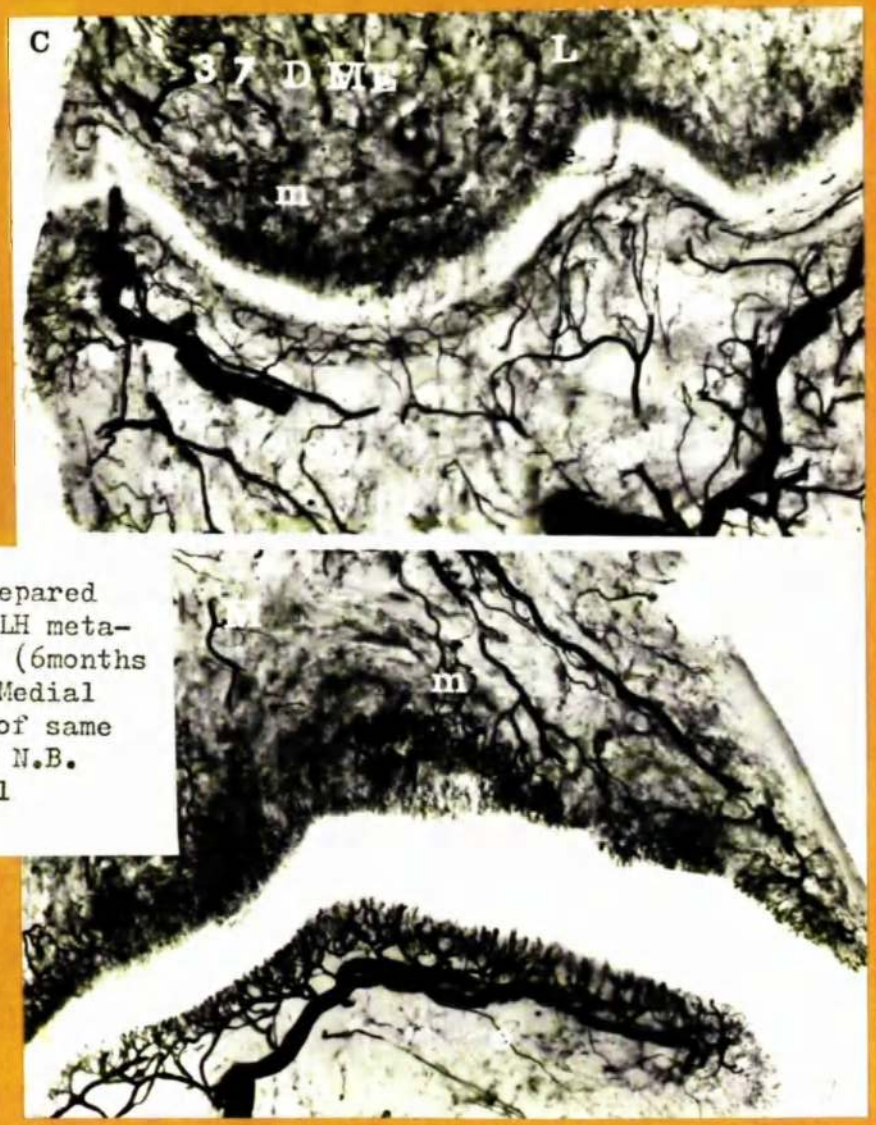


PLATE 33C Spalteholz prepared section of distal LH meta-tarsus, lamb no.37 (6months postoperatively). Medial & lateral aspects of same section presented. N.B. More profuse medial vasculature.

PLATE 33D,E&F Spalteholz prepared sections of lateral distal LH tibiae, lambs 55 (6months postoperatively) and 18 (8months postoperatively). Plate 33E is an anteriorly derived section of no.18 whereas 33F is posteriorly derived. N.B. Epiphyseal blood vessels penetrating growth plate and profuse vasculature associated with gross metaphyseal defects.

G

PLATE 33G Spalteholz prepared section of lateral distal LH tibia, lamb no.14 (10months postoperatively). N.B. Profuse vasculature associated with metaphyseal defect.

H

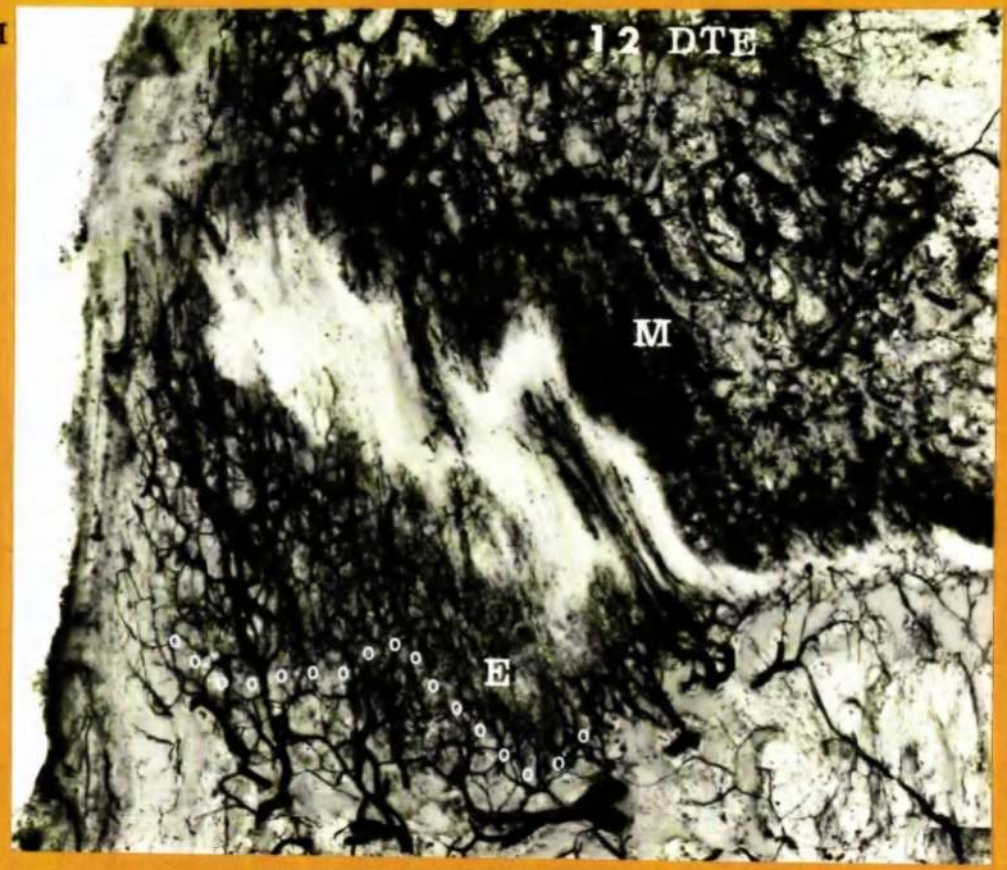


PLATE 33H Spalteholz prepared section of lateral distal LH tibia, lamb no.12 (12 months postoperatively). N.B. Tongues of epiphyseal vessels penetrating metaphyseal defect. Site of original growth plate is demonstrated (dotted line).

A

PLATE 34A Spalteholz prepared section of medial distal metatarsal growth plate, lamb no.36 (5months post-operatively). N.B. Metaphyseal protrusion of growth cartilage and associated penetration of growth plate by epiphyseal vessels.

B

C

PLATE 34B&C Spalteholz prepared section of distal medial LH tibiae, lambs no.36 (5months postoperatively) and 39 (6months postoperatively). N.B. Profuse medial vasculature and bulging medial growth plate periphery.

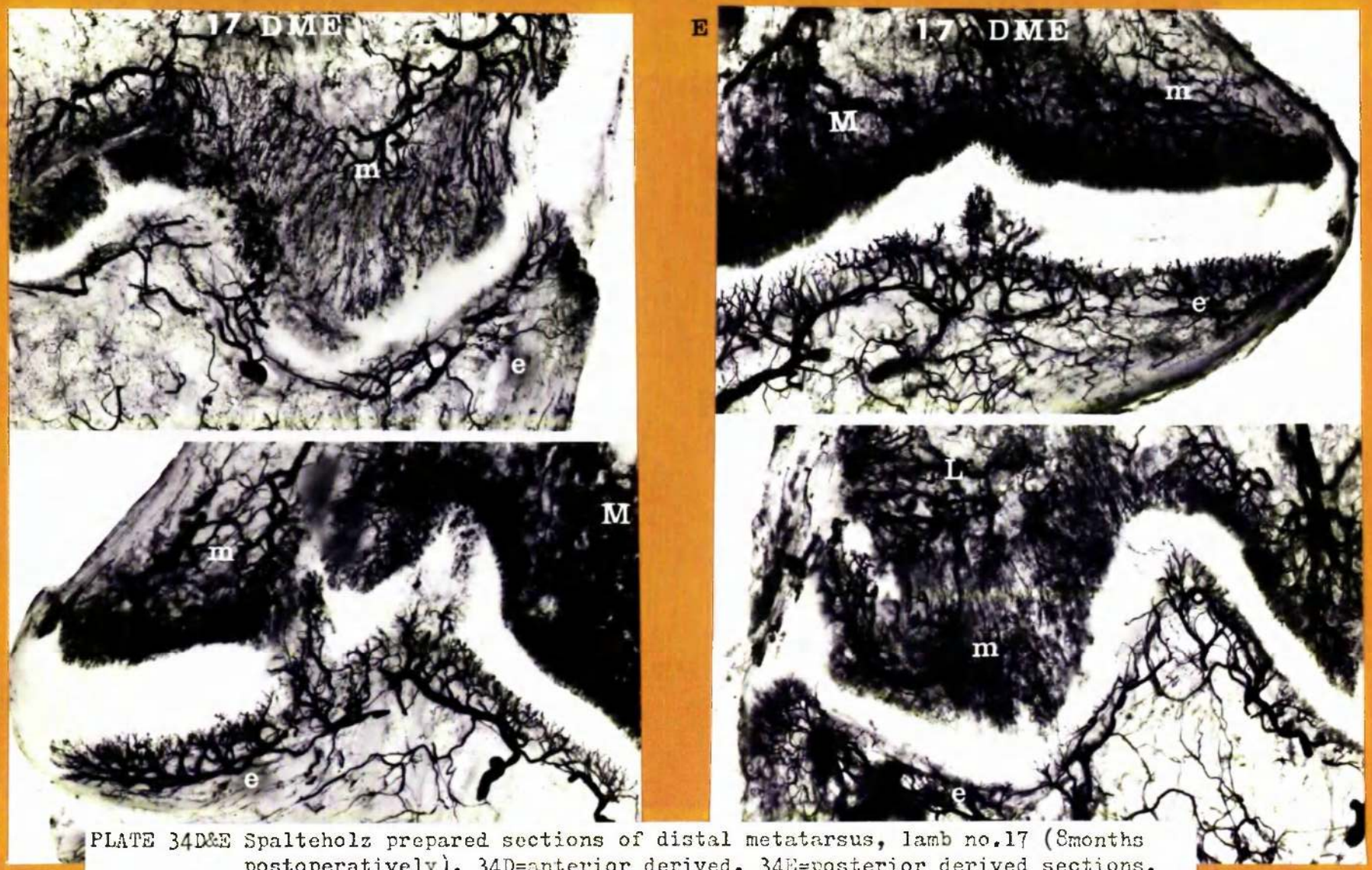


PLATE 34D&E Spalteholz prepared sections of distal metatarsus, lamb no.17 (8months postoperatively). 34D=anterior derived, 34E=posterior derived sections. Medial & lateral aspects of same sections presented. N.B. Metaphyseal protrusions and tongues of epiphyseal vasculature. Also fusion of metaphyseal & epiphyseal vasculature with epiphyseal union.

F

G

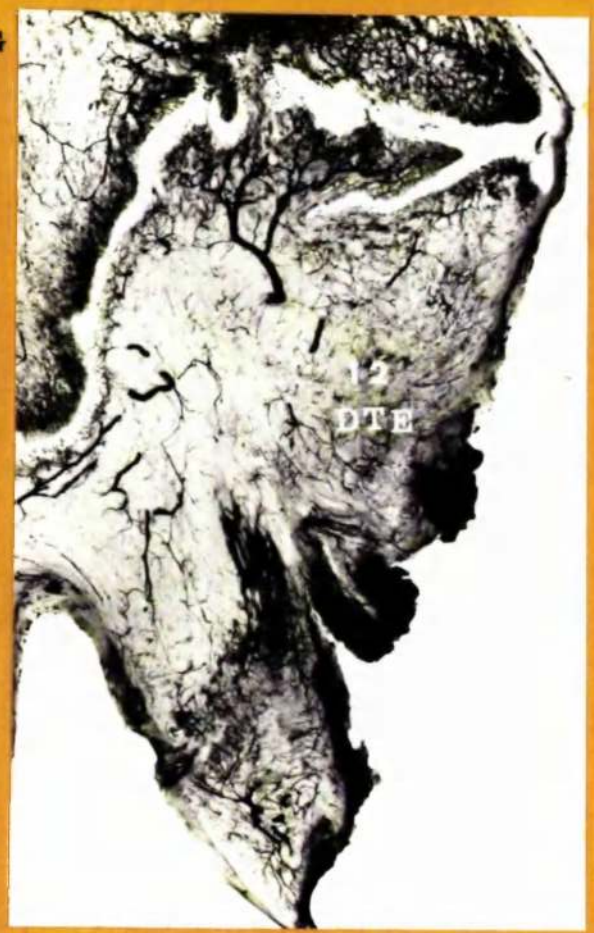


PLATE 34F&G Spalteholz prepared sections of distal medial LH tibiae, lambs 14 (10months postoperatively) and 12 (12months postoperatively). N.B. Fusion of metaphyseal and epiphyseal vasculature with epiphyseal union (34F) and double growth plate appearance due to large bone plate protrusion (34G).

A

PLATE 35A Spalteholz prepared section of distal lateral metatarsus, lamb no.55 (6months postoperatively). N.B. Scant metaphyseal vasculature and periosteal/ perichondrial vessels.

B

PLATE 35B Spalteholz prepared section of distal lateral metatarsus, lamb no.14 (10months postoperatively). N.B. Scant metaphyseal vasculature.

A

PLATE 36A Microradiograph distal tibial metaphyseal cortex, lamb no.19 LH (8months control). In plates 36A-E the surface cut is on the medial aspect and the anterior aspect is at the top of the photomicrograph.

B

PLATE 36B Microradiograph distal tibial metaphyseal cortex, lamb no.51 LH (3months postoperatively). In both of these sections much of the cortex consists of primary osteonal or plexiform bone. In addition compacted coarse cancellae can be identified endosteally in 36B. Resorption cavities demonstrate sites of remodelling to secondary osteonal bone.

C

PLATE 36C Microradiograph distal tibial metaphyseal cortex, lamb no.37 LH (6months postoperatively). Most of the cortex consists of plexiform bone and an eccentric location of resorption cavities and thus internal remodelling is present.

D

PLATE 36D Microradiograph distal tibial metaphyseal cortex, lamb no.18 LH (8months postoperatively). Massive endosteal and periosteal deposition of plexiform bone demonstrated. Internal remodelling confined to limited area medially.

E

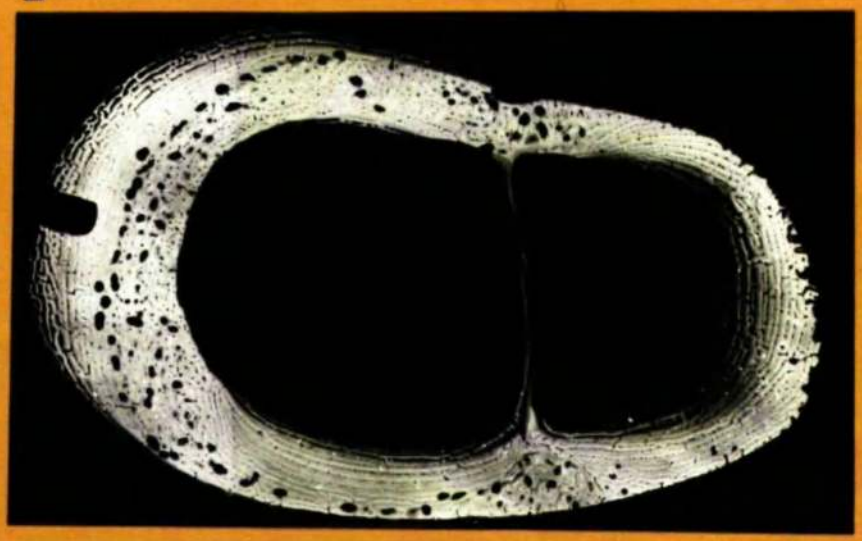


PLATE 36E Microradiograph of distal metatarsal metaphyseal cortex, lamb no.37 LH (6months postoperatively). Endosteal and periosteal deposition of plexiform bone demonstrated. Most of internal remodelling medially.

F

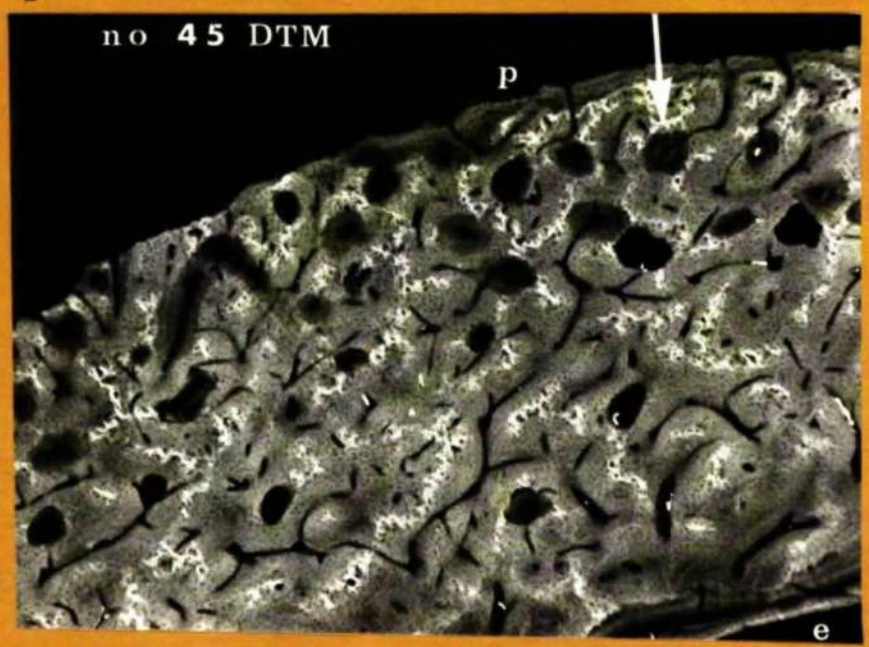


PLATE 36F Microradiograph of section of distal tibial metaphyseal cortex, lamb no.45 LH (1month postoperatively). p=periosteal surface, e=endosteal surface. N.B. Dark lines within osteones (arrowed).

A

PLATE 37A Photomicrograph of distal tibial growth plate, lamb no.57 LH (2week control) demonstrating normal growth cartilage morphology (H/E).

B

PLATE 37B Photomicrograph of distal tibial growth plate, lamb no.59 LH (1week control) demonstrating epiphyseally derived cleft entering growth cartilage. Bone plate at foot of photomicrograph (V.G.)

C

PLATE 37C Photomicrograph of medial distal metatarsal growth plate, lamb no.53 LH (3 months control) demonstrating rounded chondrocyte nuclear shape. Primary spongiosa at top of photomicrograph (V.G.)

D

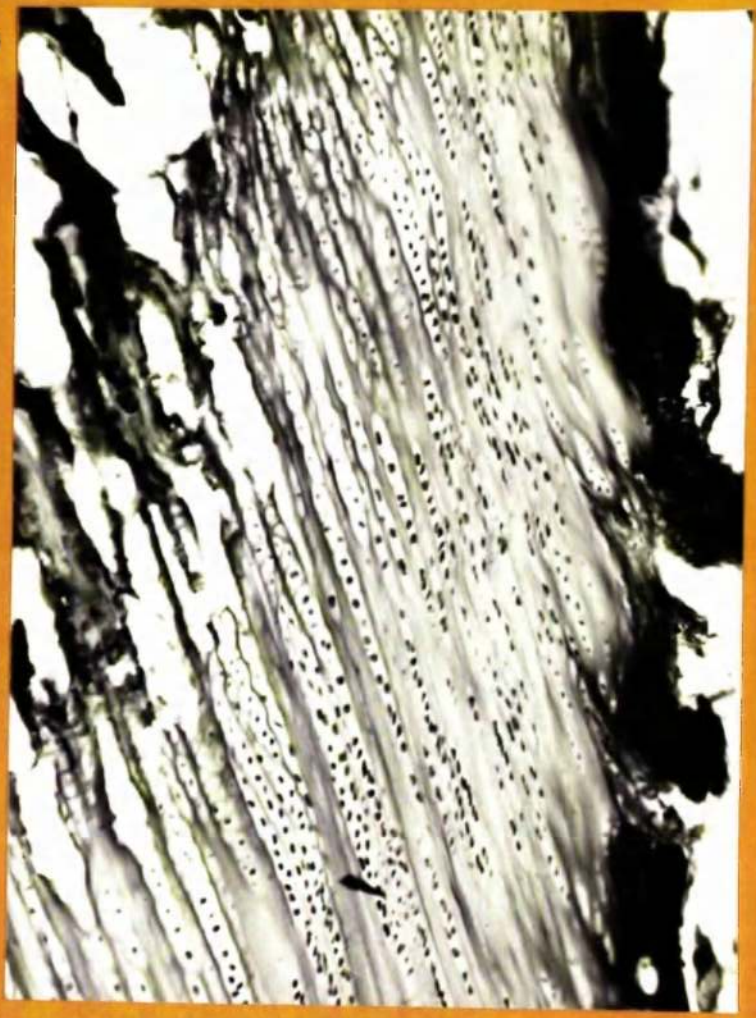


PLATE 37D Photomicrograph of lateral distal metatarsal growth plate, lamb no.53 LH demonstrating more elongated chondrocyte nuclear shape (V.G.)

E

PLATE 37E Photomicrograph of distal metatarsal growth plate, lamb no.57 LH (2week control) demonstrating bending of cell columns in association with normal undulations of epiphysis/metaphysis (V.G.)

A

PLATE 38A Photomicrograph of lateral distal tibial growth plate, lamb no.58 LH (1week post-operatively) demonstrating core of growth cartilage in metaphysis. N.B. De-ranged spongiosa formation associated with cartilage protrusion. (V.G.)

B

PLATE 38B Photomicrograph of lateral distal tibial growth plate, lamb no.43 LH (3weeks postoperatively) demonstrating fracture line traversing growth cartilage. N.B. Rounded chondrocyte nests adjac-ent to fracture line. Bone plate at foot of photomicrograph. (V.G.)

C

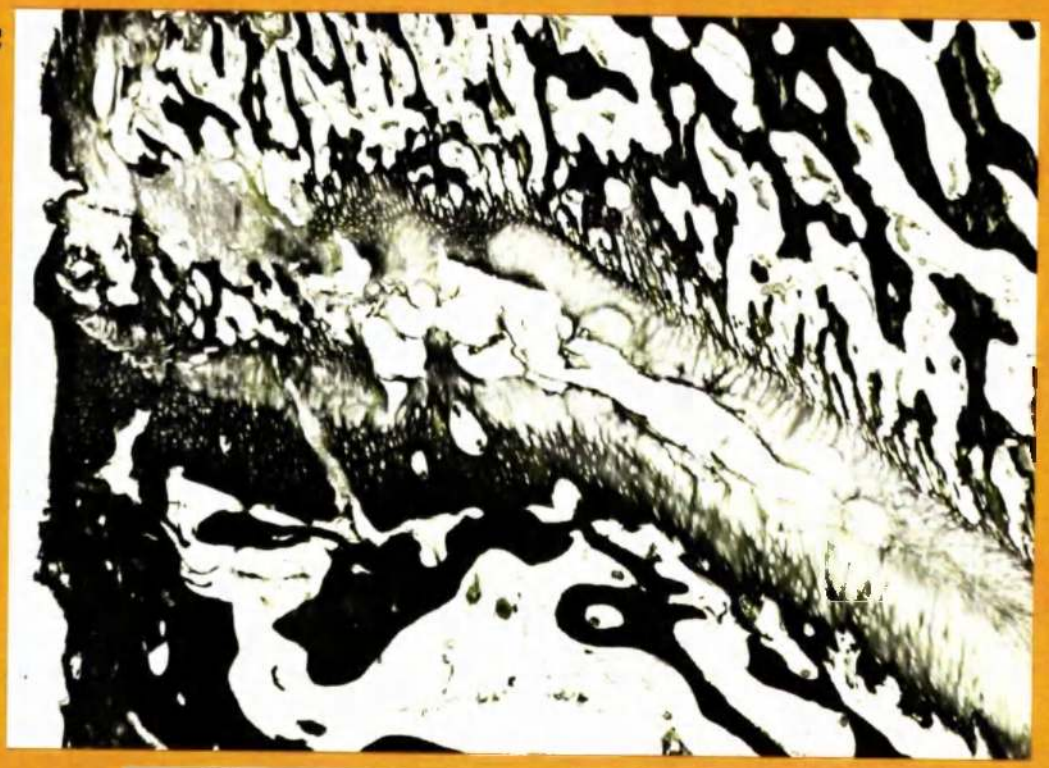


PLATE 38C Photomicrograph of lateral distal tibial growth plate, lamb no.52 LH (3months postoperatively), section 2, (anteriorly derived) (V.G.)

D

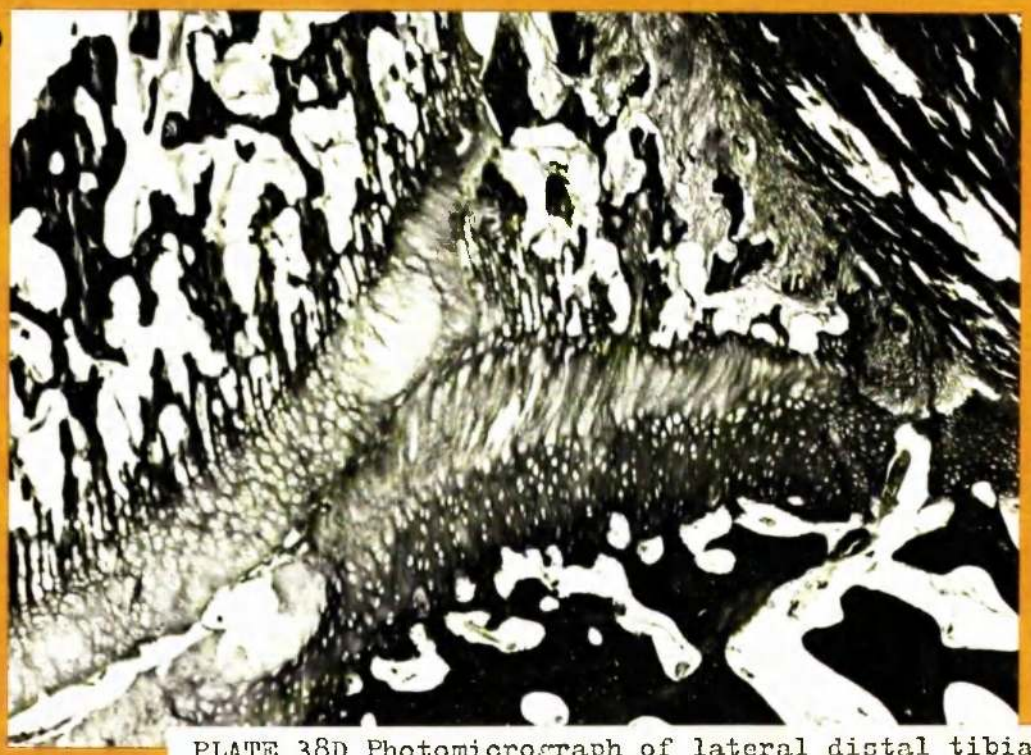


PLATE 38D Photomicrograph of lateral distal tibial growth plate, lamb no.52 LH, section 3, (more posteriorly derived). N.B. Larger dimensions of bone island in posterior section. Also bone island is seen to be derived from continued maturation of growth cartilage. (V.G.)

E

PLATE 38E Photomicrograph of lateral distal tibial growth cartilage, lamb no.50 LH (2months postoperatively) demonstrating bending of chondrocyte columns. Bone plate at foot of photomicrograph. (V.G.)

F

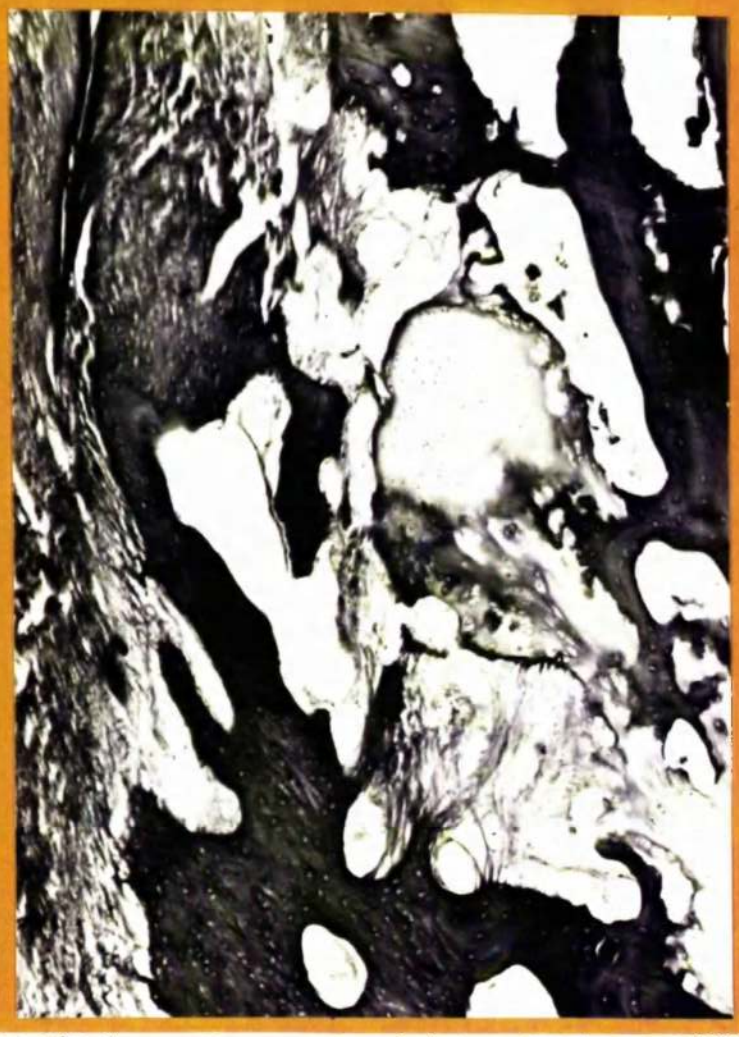


PLATE 38F Photomicrograph of lateral distal tibial metaphysis, lamb no.36 LH (5months post-operatively) demonstrating chondrocyte nest located amongst trabeculae and failing to degenerate. (V.G.)

i

ii

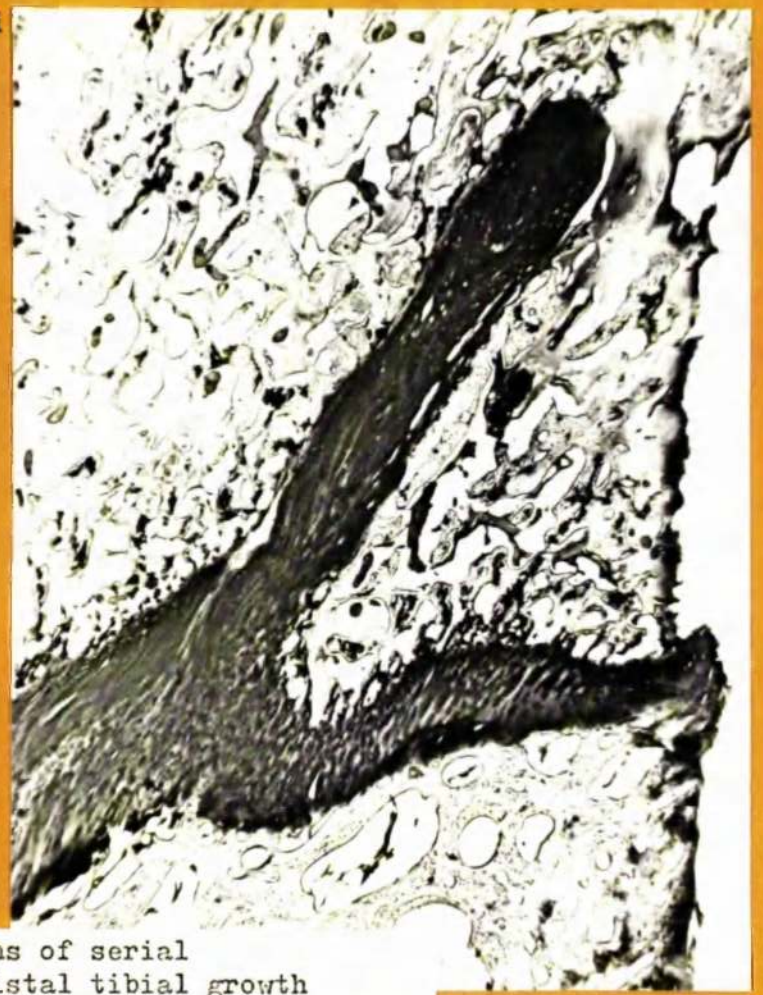


PLATE 38G i & ii Photomicrographs of serial sections of lateral distal tibial growth plate, lamb no.55 LH (6months postoperatively). Gi=V.G., Gii=T.B. N.B. Tongue of growth cartilage which demonstrates normal matrix staining density.

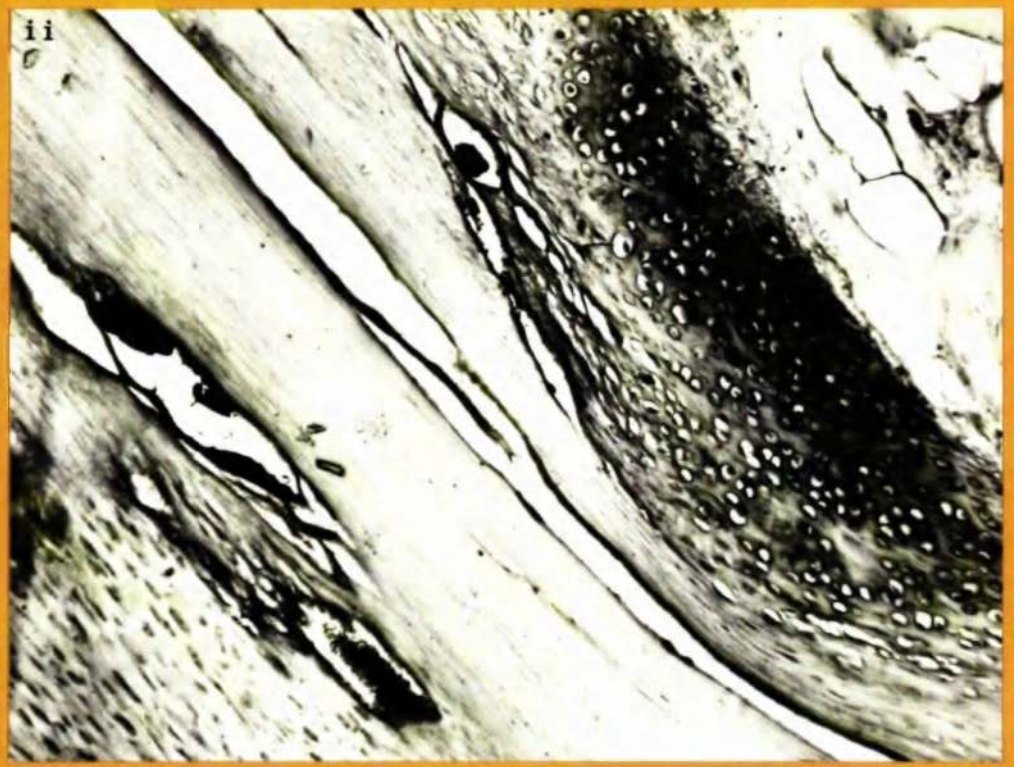


PLATE 38H i & ii Photomicrographs of comparable sections of lateral metaphyseal defect, lamb no.18 LH (8months postoperatively). Hi=G.T., Hii=T.B. N.B. Grossly attenuated nuclear shape with poorly staining matrix in toluidine blue section.

I

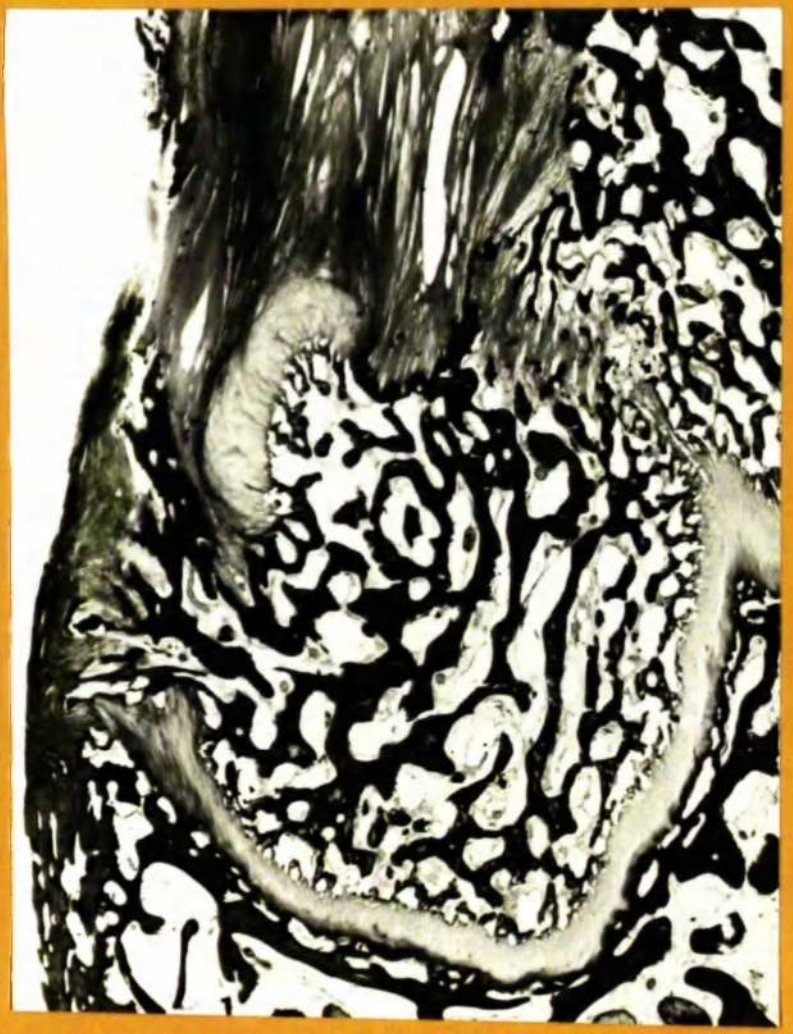


PLATE 38I Photomicrograph of lateral distal metatarsal growth plate/metaphysis, lamb no.37 (6months postoperatively). N.B. Reorientation of a portion of growth cartilage which is producing primary spongiosa on the bone island. Bone plate at foot of photomicrograph, periosteal surface at left. (V.G.)

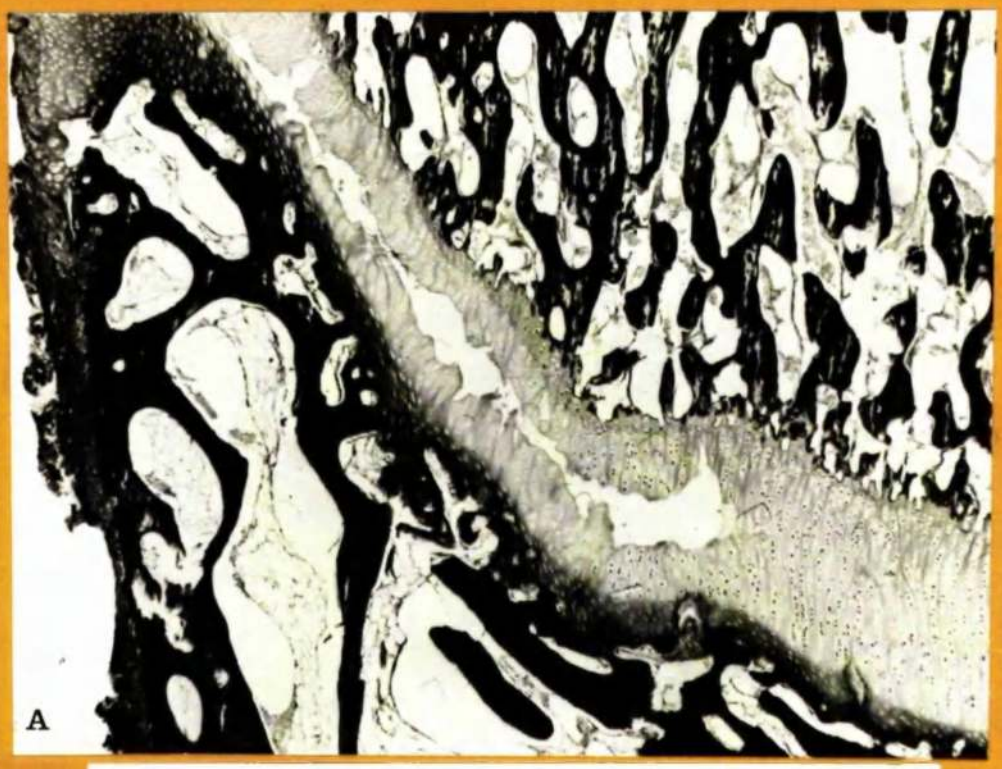


PLATE 39A Photomicrograph of medial distal metatarsal growth plate, lamb no.33 LH (2weeks postoperatively) demonstrating fracture line traversing cartilage columns. (V.G.)

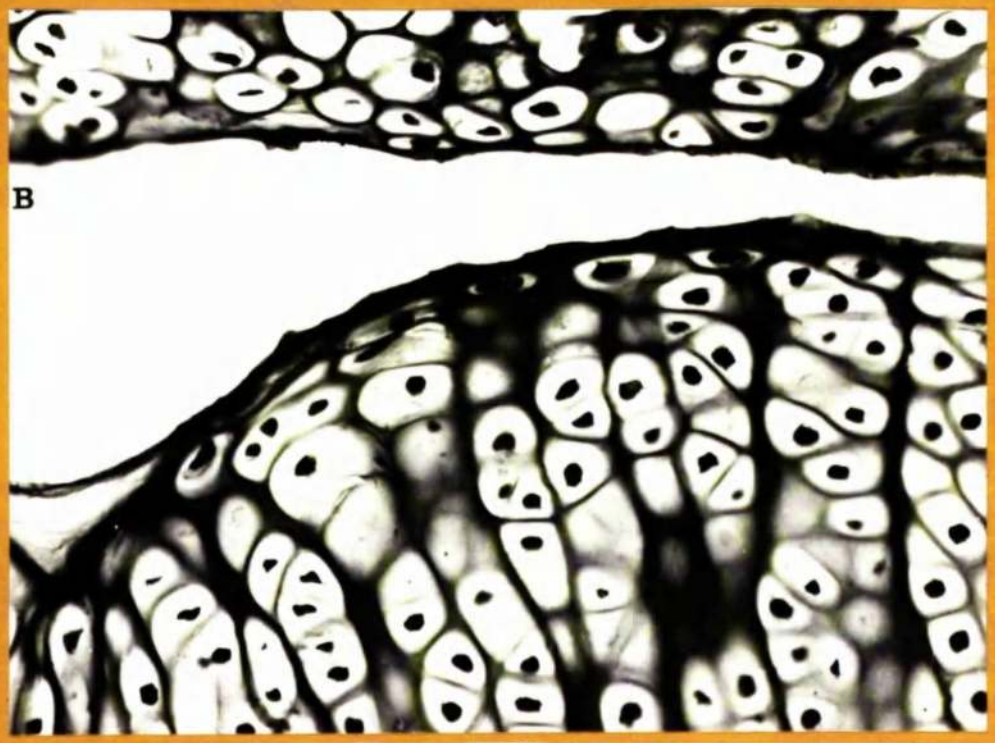


PLATE 39B Photomicrograph of medial growth plate fracture line, lamb no.54 LH distal tibia (4months postoperatively) demonstrating flattened chondrocytes at cartilage surfaces. (H/E).

C

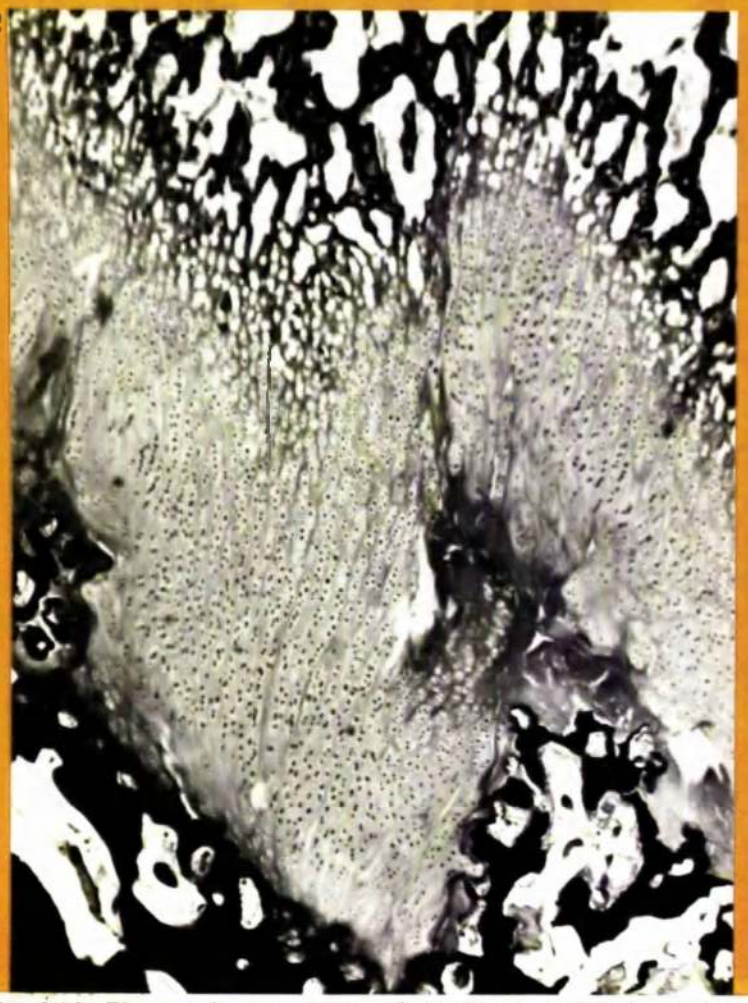


PLATE 39C Photomicrograph of medial distal metatarsal growth plate, lamb no.55 LH (6months postoperatively) demonstrating eosinophilic septae and areas of relatively amorphous matrix. N.B. Bone plate protrusions into growth cartilage. (V.G.)

D

PLATE 39D Photomicrograph of medial distal metatarsal growth plate, lamb no.36 LH (5months postoperatively) demonstrating eosinophilic septum with canal connecting epiphysis and metaphysis. N.B. Disorganised primary spongiosa. (V.G.)

PLATE 39E Photomicrograph of medial metaphyseal/ growth plate junction distal tibia, lamb no.36 LH (5months postoperatively). N.B. Chondrocyte nest located amongst trabeculae & disorganised spongiosa formation. (V.G.)

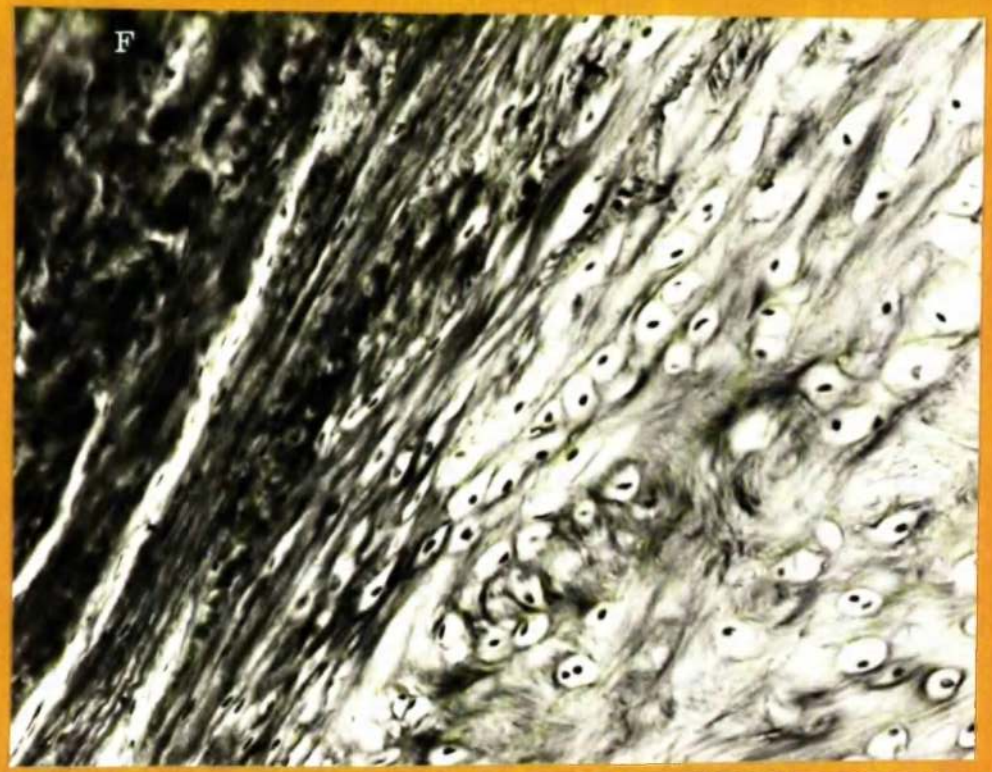


PLATE 39F Photomicrograph of medial growth plate periphery distal tibia, lamb no.18 (8months postoperatively). N.B. Transition of cell shape from chondrocyte to fibrocyte and occasional mitotic figures. (V.G.)

In plates 40 A-F & 41 A-B the times of fluorochrome administration were as follows:

Lamb No. 53	- 3 months survival =	84 days
	AC administered	day 24
	OTC "	day 62
	XO "	day 72
	FC "	day 82
Lambs No 17 & 18	- 8 months survival =	224 days
	AC administered	day 112
	OTC "	day 180
	XO "	day 201
	FC "	day 222
Lamb No. 14	- 10 months survival =	280 days
	AC administered	day 112
	OTC "	day 236
	XO "	day 257
	FC "	day 278
Lamb No 12	- 12 months survival =	336 days
	AC administered	day 112
	OTC "	day 292
	XO "	day 313
	FC "	day 334

A

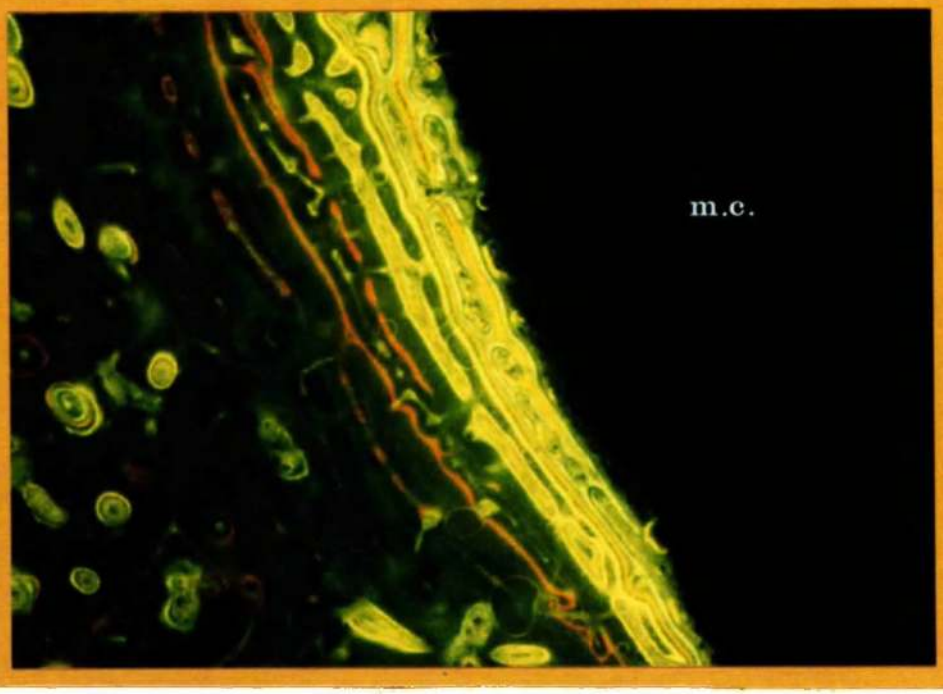


PLATE 40 A Lamb no. 53 LH DTM. m.c.=medullary cavity. N.B. Osteonal label on left and bands of endosteal label. Part of endosteal surface consists of compacted coarse cancellae.

B

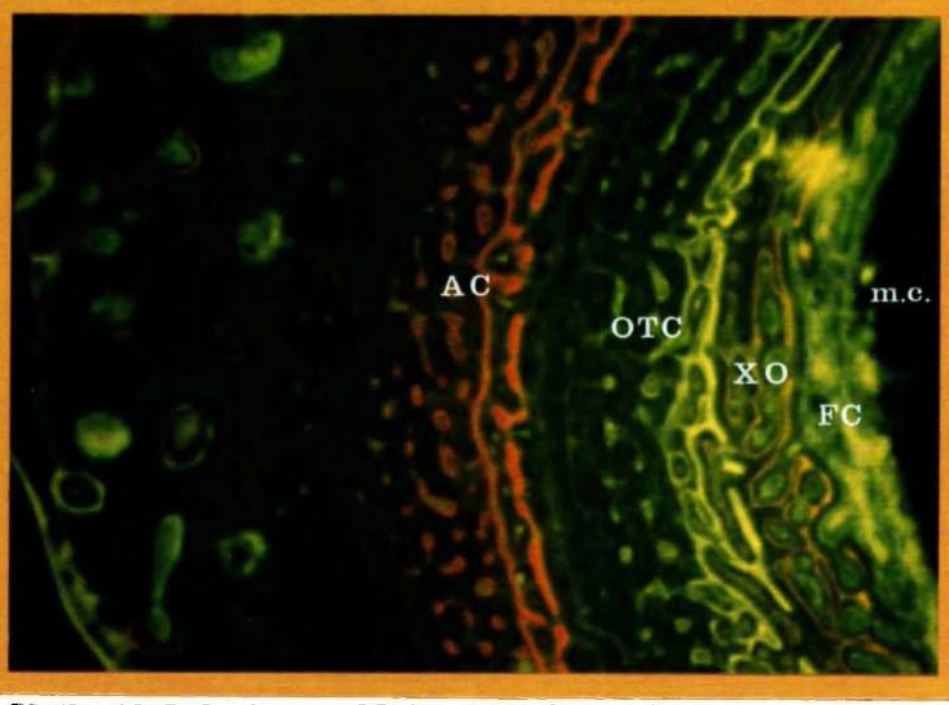


PLATE 40 B Lamb no. 17 LH DMM. Lateral cortex. m.c.= medullary cavity. N.B. More than half of cortical width consists of endosteally derived bone.

C

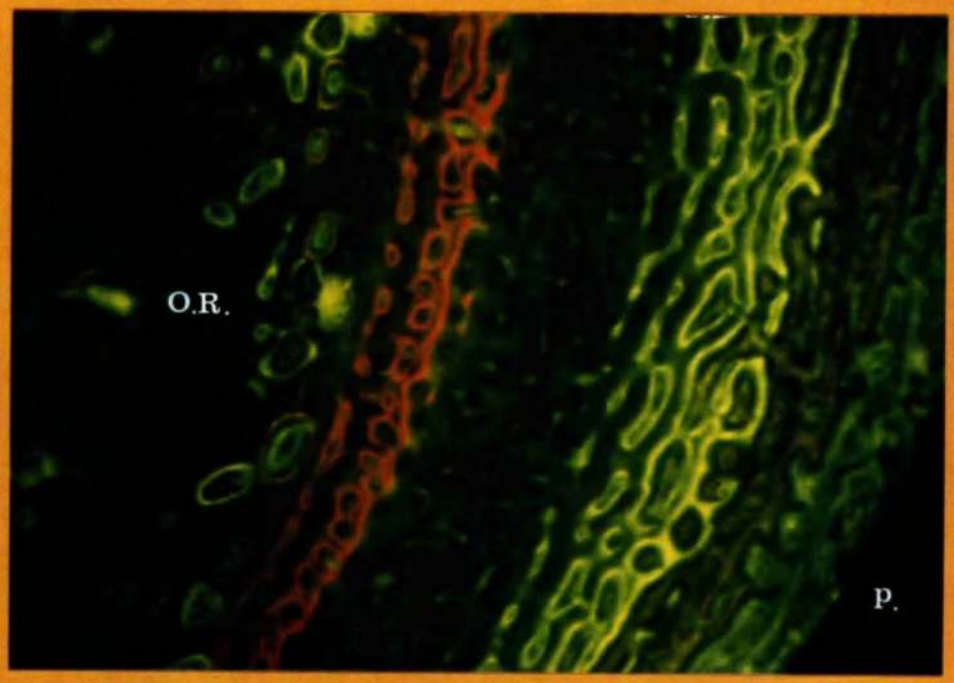


PLATE 40 C Lamb no. 17 LH DMM. Medial cortex. p.= periosteal surface. N.B. More than half of cortical width consists of periosteally derived bone. O.R.= Osteonal remodelling.

D

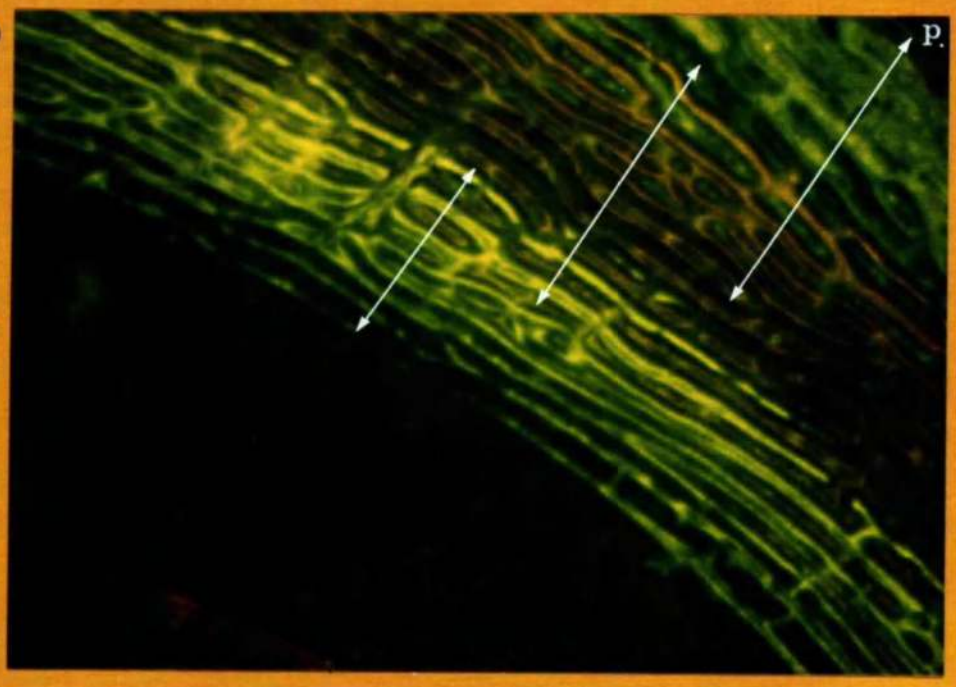


PLATE 40 D Lamb no. 14 LH DTM. Medial cortex. p.= periosteal surface. N.B. Gradual increase in label width of successive fluorochromes OTC,XO & FC.

E

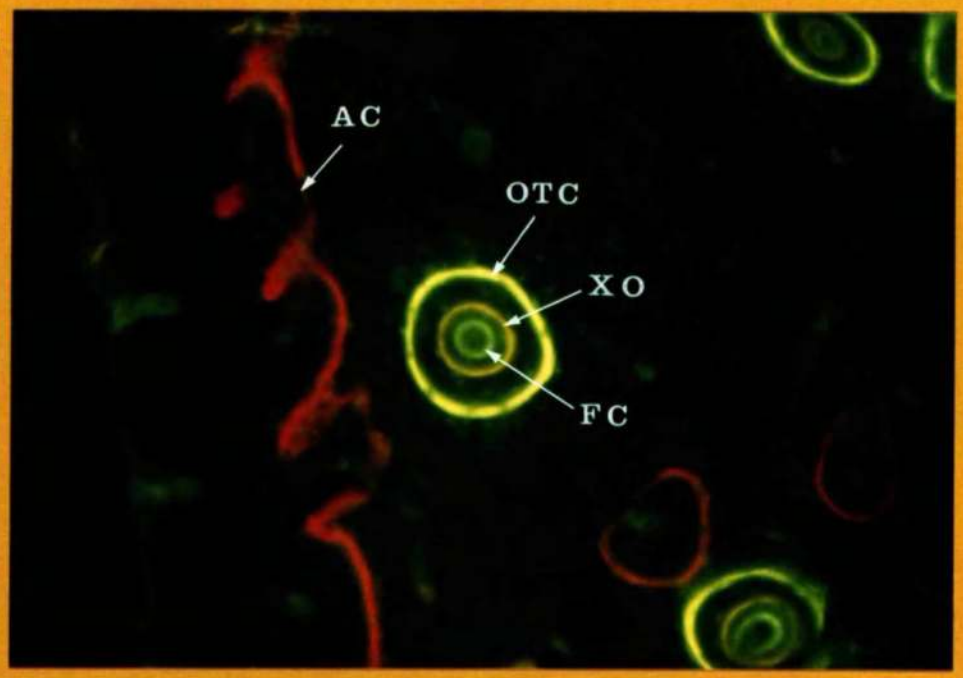


PLATE 40E Lamb no.12 LH DTM section 6. N.B. Osteonal labels.

F

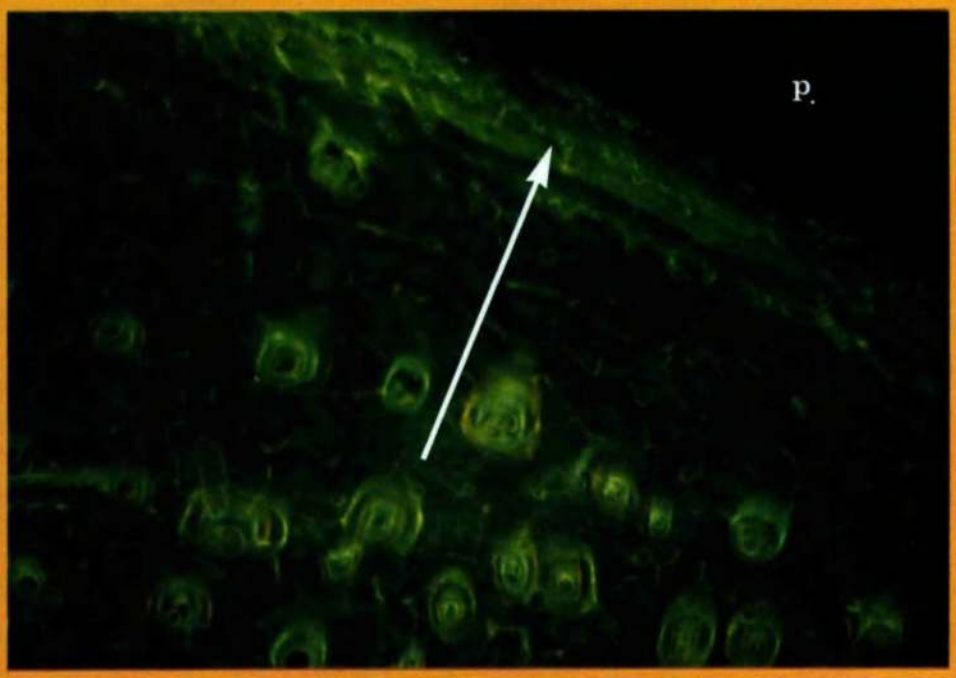


PLATE 40F Lamb no.12 LH DTM, medial cortex section 1. p=periosteal surface. N.B. Scew osteones with thickest label towards direction of drift (arrow).

A

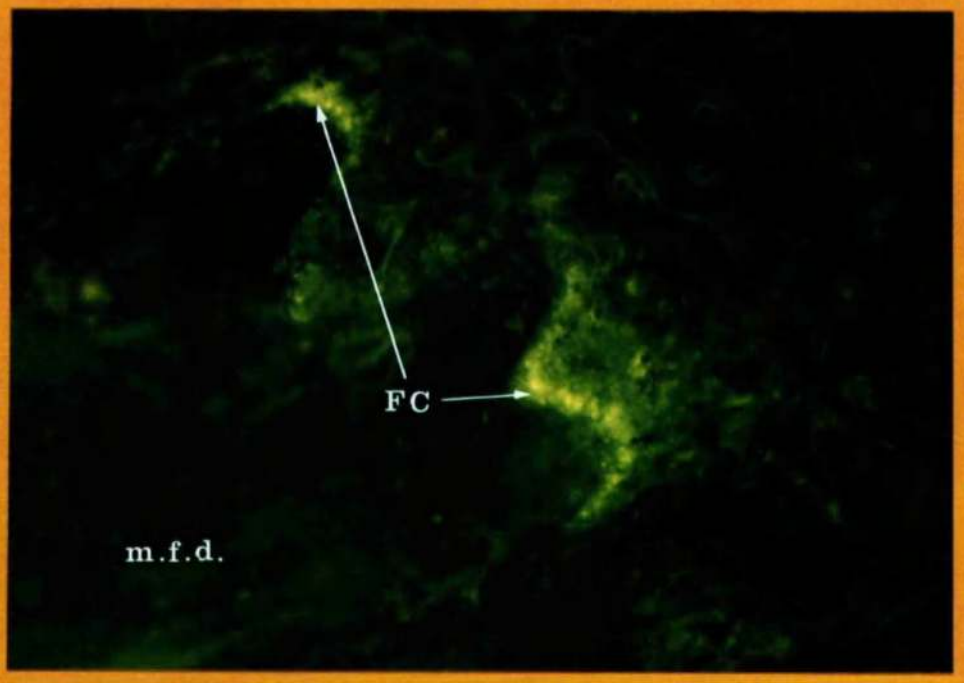


PLATE 41A Lamb no.18 LH DTE, lateral aspect.
m.f.d.= lateral metaphyseal fibrous defect.
FC= labelled spongiosa on its metaphyseal aspect. N.B. Areas exist where no label deposited.

B

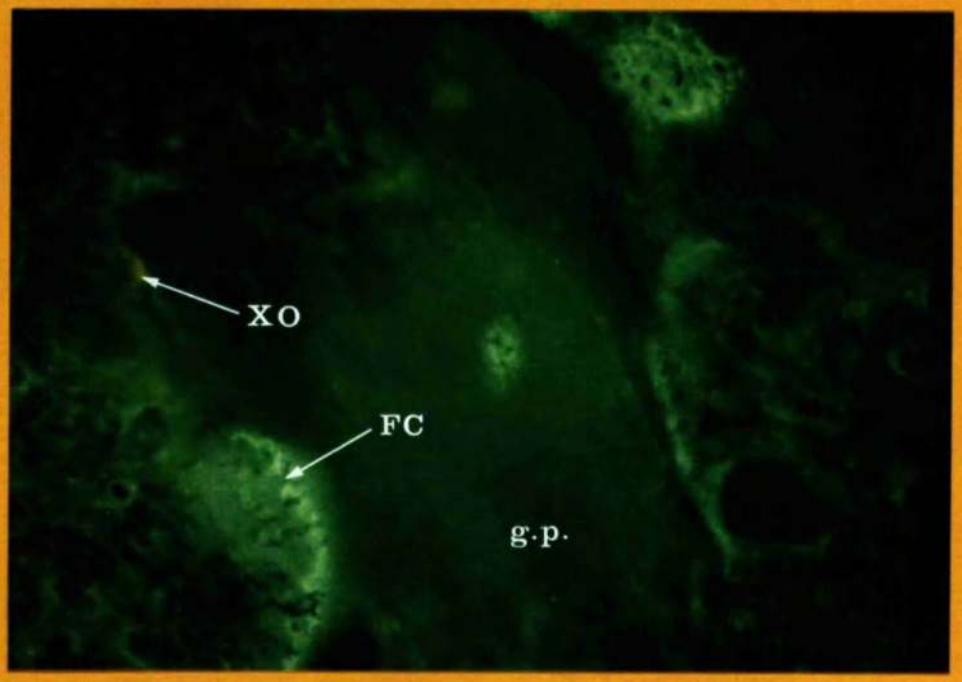


PLATE 41B Lamb no.14 DTE, medial aspect.
g.p= growth cartilage protrusion into metaphysis. N.B. Absent label over metaphyseal aspect (top left). Patchy label deposited in disorientated fashion.

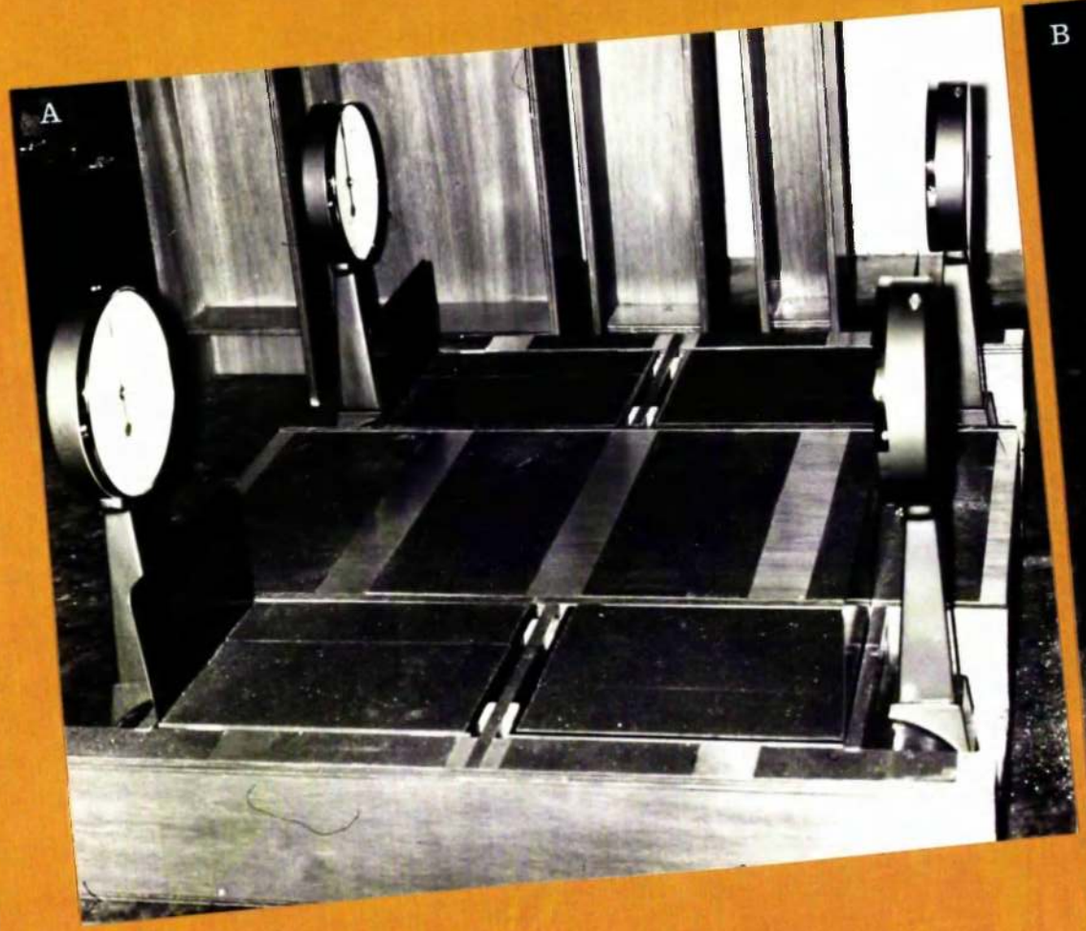


PLATE 42A&B Apparatus used for estimates of load bearing by each limb.

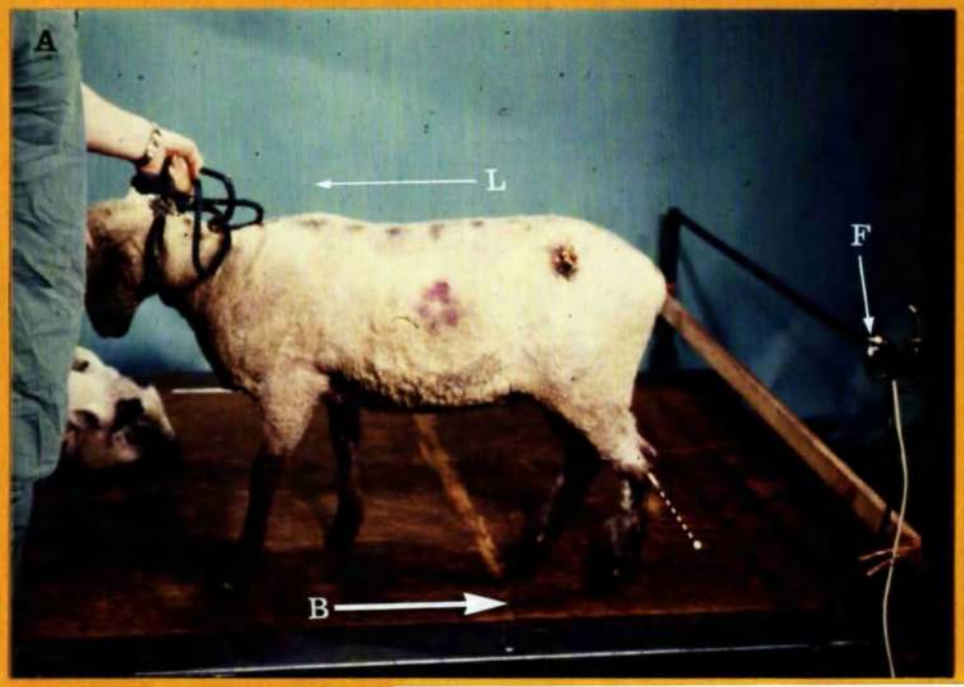


PLATE 43A Moving-road apparatus. L=direction of locomotion of lamb. B=direction of belt motion. F=flashlight to allow synchronisation of both cameras.

B

PLATE 43B Perspex extension bars and their mode of attachment by a sleeve to the orthopaedic bone screw.

PLATE 44A&B Premortem posture of lambs 41 (2weeks postoperatively) and 45 (1month postoperatively). N.B. Metatarsal varus in conjunction with internal limb torsion in 44B.

PLATE 44C, D&E Premortem posture of lambs 50 (2months postoperatively), 54 (4months post-operatively) & 36 (5months postop-eratively). N.B. Metatarsal varus in 44C & tibial varus in 44D&E.

PLATE 44F Premortem posture of lamb no.37 (6months postoperatively). N.B. Extreme metatarsal varus and marked internal limb torsion.

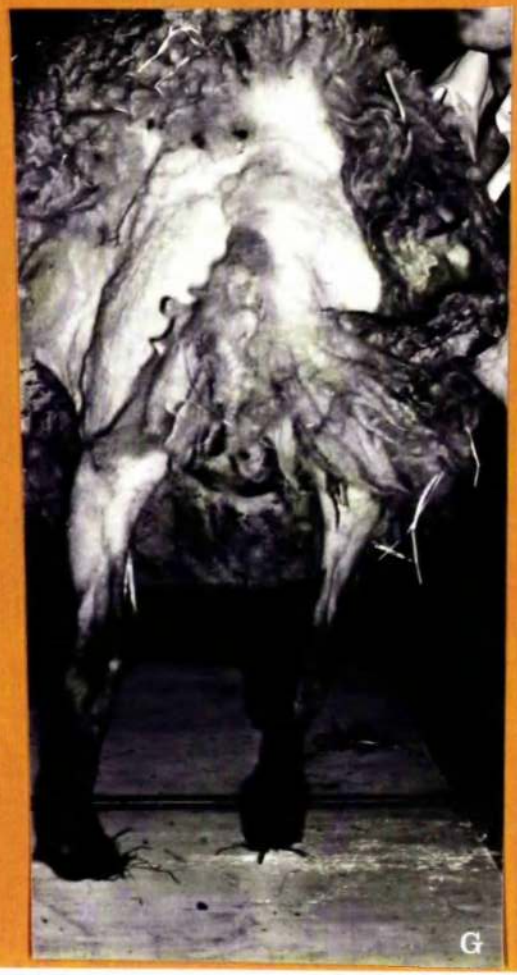


PLATE 44G Premortem posture of lamb no.13 (12months postoperatively). N.B. Metatarsal varus but greater contribution of operated (RH) limb to load bearing.

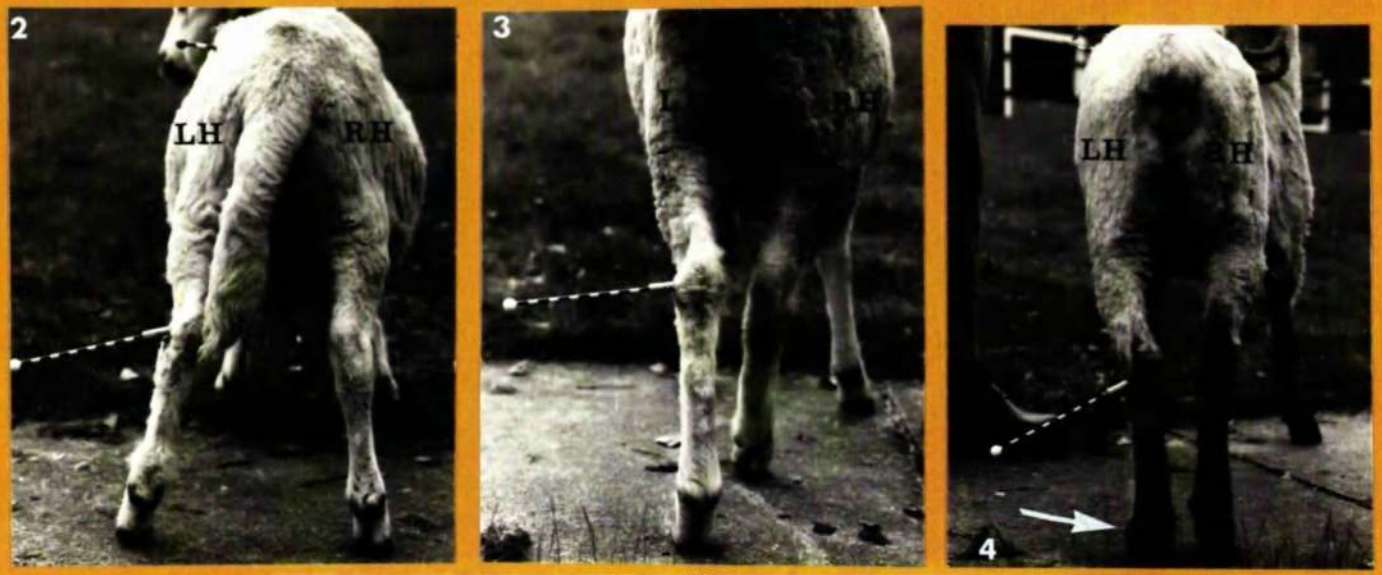


PLATE 45A Gait analysis study lambs. Posture at conclusion of experiments. Lamb no.2 (control) bone screw insertion (LH) 3weeks previously. Lambs 3 & 4 (experimental) bone screw insertion (LH) and unilateral (RH) hip excision arthroplasty 3weeks previously. N.B. Increased fetlock sinking LH lamb no.4.

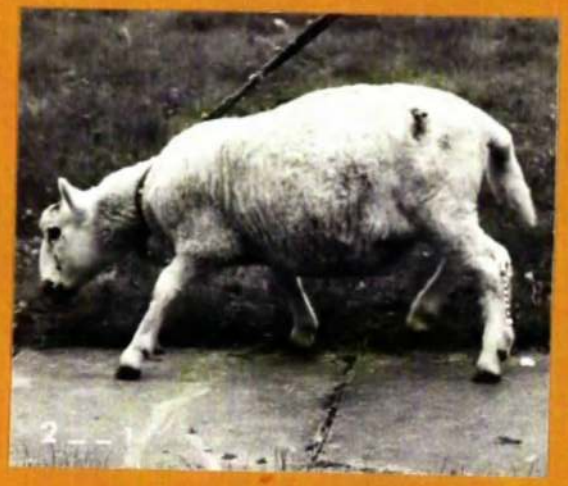


PLATE 45B Isolated phases of walking cycle in control lamb no.2.

PLATE 45C Isolated phases of walking cycle in experimental lamb no. 3. Although of relatively normal appearance in still photographs gait was abnormal when visualised.

PLATE 45D Isolated phases of walking cycle in experimental lamb no.4. Pathological gait was more severe than noted in lamb no.3. In still photographs extreme and abnormal fetlock hyper-extension can be visualised (arrowed).

PLATE 46 Case no. 73515. Border Leicester lamb, 4 months of age at presentation.

Sustained LH tibial fracture approximately 2 months previously. Malunion occurred with external rotation & a degree of valgus angulation.

The contralateral RH limb possessed marked internal rotation & moderate varus angulation was present at the distal tibial extremity.

Plate 46 demonstrates the RH limb deformity encountered.

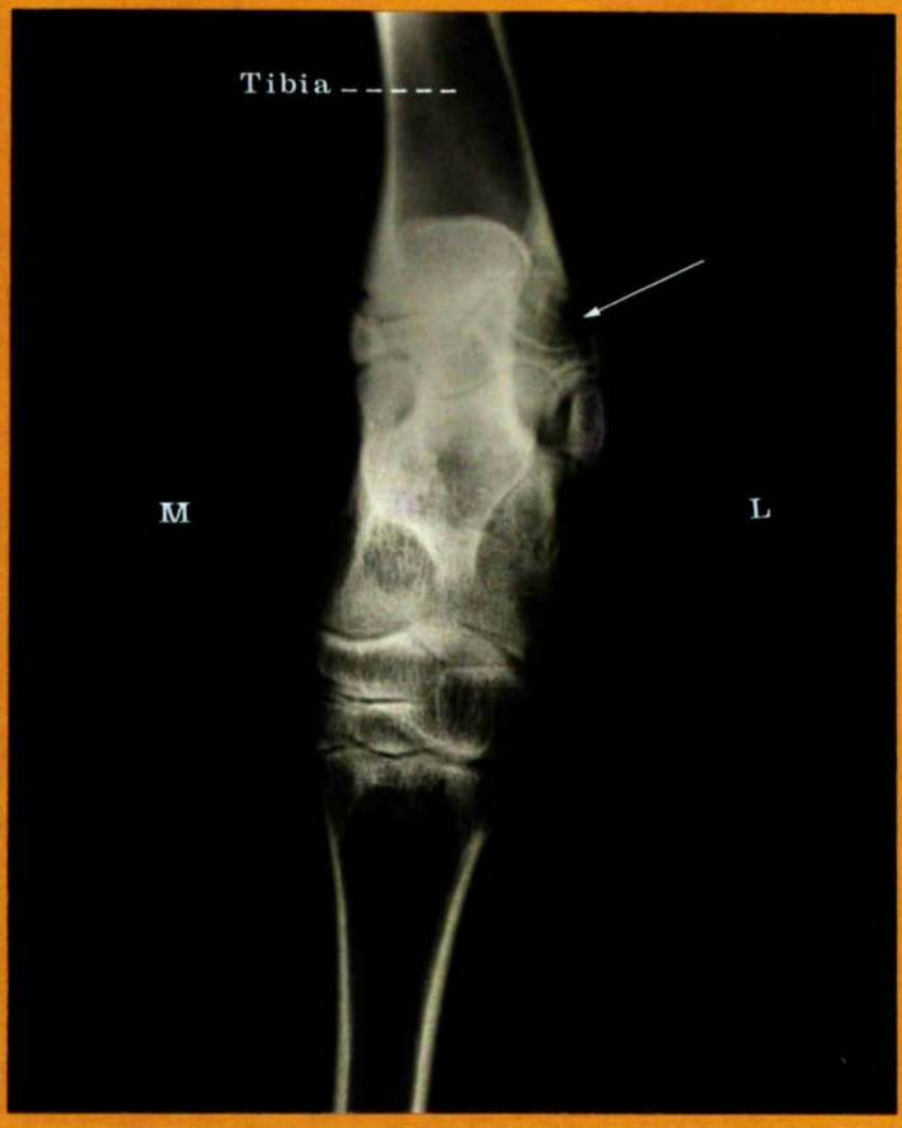


PLATE 47 Case no. 73515. Anteroposterior radiograph of distal tibia & hock, RH limb, of ewe lamb shown in plate 46. N.B. Lucent defect in distal tibial metaphysis adjacent to the lateral growth plate sector. In addition the presence of varus angulation can be appreciated. The radiographic appearance is identical to that produced in experimental lambs of this study.

PLATE 48 Case no. 72834. 18 month old Irish Setter bitch. Presented with 3 days duration spiral fracture of LH tibia. The posture adopted by the contralateral RH limb is demonstrated. N.B. The adducted position of the limb in conjunction with internal rotation. This posture is typical of that adopted by experimental lambs in the early postoperative period.