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Talipes equinovarus and vertical talus produced experimentally in newborn rabbits

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Helsinki, 1969.

Veijo Ritsilä

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I. INTRODUCTION
PURPOSE OF THE INVESTIGATION

Despite the increase in our knowledge of various malformations, the etiology of many common deformities such as club-foot, and vertical talus remains unknown. Detailed patho-anatomical observations have not led to the solution of the problems of the etiology and pathogenesis of talipes equinovarus. The reason for such divergence may be that there have been differences of etiology in different cases. But in such clinical and patho-anatomical studies it is difficult, especially at late stages, to distinguish secondary changes from primary. It is also evident that in congenital deformities, different etiological factors may lead to similar structural changes. The orthopedic treatment of deformities, however, aims at the correction of changes in the musculo-skeletal system or the prevention of their progression or recurrence. Thus information on the nature of the peripheral deforming mechanism may be of great importance in planning effective treatment.

In a study concerning the development and pathogenesis of the experimental dislocation of the hip in the rabbit, *Langenskiöld et al.* (1962) emphasized some of the basic principles of teratology in the following statement. "We know that by altering the environment of the embryo we can produce abnormalities simulating those of genetic origin. We know too that the nature of the alteration or injury is less important in determining the result than its site of action and the stage in embryonic development at which it is applied". Since the development of the skeletal tissue continues after birth, it is possible to extend this statement to postnatal developmental events as well. Thus, conditions simulating true congenital defects may result from injurious environmental factors affecting the child after the intrauterine period.

Congenital skeletal deformities in humans may have a genetic origin as a rule. But in children we see infections and injuries damaging growing bones and producing deformities similar to those of genetic origin. Observations of foot deformities caused by poliomyelitis and certain

patho-anatomical studies of congenital foot deformities have given many investigators reason to suppose that soft tissue anomalies in some way cause bone deformity. On this basis it seems reasonable to try to imitate experimentally conditions which have been shown to result from abnormalities of the germ plasm.

Therefore, the purpose of the present study was:

- to investigate whether it is possible to produce foot deformities in newborn rabbits phenotypically similar to human congenital deformities, especially talipes equinovarus deformity, by means of various surgical and non-surgical, soft tissue procedures;
- to carry out a detailed anatomic study of deformities produced by these procedures;
- to elucidate the peripheral deforming mechanism in congenital foot deformities by comparing observations in humans with experimental results achieved.

II. REVIEW OF LITERATURE

A. TALIPES EQUINOVARUS

1. Etiology

The first etiological theory was that of *Hippocrates* (*Kite* 1964). He believed that club-foot was the result of intra-uterine pressure. This idea has been elaborated with convincing arguments by *Denis Browne* (1933, 1936, 1967). Many others have returned to the notion that intra-uterine position or mechanical factors may be the cause of club-foot (*Parker* and *Schattock* 1884, *Ketch* 1892, *Nutt* 1925).

Club-foot has also been considered to be secondary to a lesion of the external popliteal nerve caused by pressure at the intra-uterine stage (*White* 1929). Another neurological theory of the cause of club-foot is that it might result from a nerve lesion, similar to deformities which result in nerve lesions after birth (*Little* 1839, *Adams* 1873). *Chapman* (1839) wrote, "Club-foot originates in the nerves supplying the flexor and extensor muscles of the foot". *Nutt* (1925) also mentioned as one etiological possibility of the cause of the club-foot a nerve anomaly. A primary defect of the spinal cord has also been considered to be the cause of club-foot (*Courtililer* 1897, *Dittrich* 1930).

The part played by heredity was stressed by *Aschner* and *Engelmann* in 1928, and by many since. *Compere* (1951) considered hereditary factors leading to neuromuscular imbalance as a cause of club-foot.

Disturbances in the development of striated muscle fibres (*Middleton* 1932), a primary muscular defect (*Smith* 1948), an imbalance of muscles resulting from their hypo- or hypertonicity (*Lombard* 1950 and 1952), a primary muscular imbalance due to dysplasia of the peroneals (*Flinchum* 1953), a relative shortening of degenerating muscle fibres during growth (*Bechtol* and *Mossman* 1950) are muscular changes on which the blame has been laid for the origin of club-foot.

That the activity of the anomalously inserted muscles can lead to foot deformity has been suggested by many authors (*Scherb* 1930, *Stewart* 1951, *Fredenhagen* 1955, *Inclán* 1958 and 1960).

Wiley (1959) thought the primary cause of club-foot was an extrinsic factor maintaining the foot in a deformed position and causing deficiencies in the growth of the leg muscles.

According to one theory, the cause of club-foot was developmental inhibition or arrested development at the embryological stage when muscles, tendons and joints develop (*Bessel-Hagen* 1889, *Ketch* 1892, *Böhm* 1929, *Schinz et al.* 1952, *Fripp* and *Shaw* 1967).

The hypothesis that club-foot is due to congenital dislocation of the head of the talus has been advanced by *Brockman* (1930, 1937). Also *Reimann* (1967) believed that congenital club-foot is a deformity of local nature, caused by primary dislocation between the tarsal navicular and the head of the talus during fetal life.

Mesenchymal dysplasia in the medial part of the foot has been considered the mechanism of club-foot (*Keith* 1940). Changes in connective tissues have also been suggested as the cause of club-foot by others (*Scheel* 1951, *Ode* 1952).

2. Pathological anatomy

Patho-anatomical observations of foot deformities have been made at autopsy since antiquity, and operative examination has become common during the last few decades. But dissection of fetal and immediately postnatal club-foot in order to determine primary changes of the intrauterine period has not been seriously undertaken until very recent times. After the first few years of life, secondary, adaptive changes lead to a confusing picture.

Formerly, the pathology of congenital skeletal deformities was studied almost entirely from the bone deformity point of view, and the skeletal changes were considered primary. Nowadays, however, the belief is widespread that pathological changes in the muscle-tendon apparatus precede and even initiate bone changes.

a) Bone changes

After examining the bones of children with club-foot some 165 years ago, *Scarpa* believed that the deformity was due to medial twisting of the navicular, cuboid and calcaneus in relation to the talus. The talus itself was considered normal in shape and position. Muscle abnormalities were considered secondary. *Adams* (1873) asserted that the essential change involved in idiopathic club-foot occured in the tarsus. He found the principal deformity in the talus, particularly in the neck and head of the

talus. *Scudder* (1887) noted "that the neck of the astragalus turned inward instead of being straight". In the 19th century there were several German accounts of club-foot dissection with particular elucidation of bone changes (*Hueter* 1863, *Kocher* 1878 etc.). Similar work was done by *Parker* and *Schattock* (1884) who noted talar deformity as did the Russian, *Shevkunenko* (1899).

Burrell (1893) found no important muscle changes in a seven-month fetus with a club-foot. He found bone changes, however, and said, "The astragalus of the deformed foot was small and its neck short. The outer part of the articular surface of the deformed astragalus, as it articulated with the scaphoid, was distinctly diminished in size."

Nichols (1897) in his dissection of a stillborn child with club-feet noted only directional changes and shortening in the tendons of tibialis anterior, posterior and gastrocnemius. He stated: "The deformity of the foot in cases of congenital equinus is due essentially to an alteration in the shape of the bones of the foot. The greatest and most important abnormalities are the obliquity of the facets and the change in the shape of the bones which enter into the information of the midtarsal joint, that is the astragalus and the os calcis posteriorly and the scaphoid and cuboid anteriorly. All the bones are altered."

Until quite recently, in fact, the main emphasis in dissection reports was placed on bony changes in congenital foot deformity. This was the case in dissections performed by *Elmslie* (1920) and *Pfrang* (1920). *Böhm* (1935) also noted that the soft tissues were normal and the equinovarus foot depended upon serious deformities of the talus and calcaneus. In their publications and monographs *Kreuz* (1927), *Mau* (1927), *Virchow* (1933), *Debrunner* (1936), *Bernbeck* (1950, 1955) and *Schlicht* (1963) have described in detail the bone changes of talipes equinovarus with emphasis on changes of shape in the talus and its joint surfaces to which other tarsal bones (cuboid, navicular and calcaneus) adapt themselves with lesser changes.

Three careful studies of the anatomy of club-foot in fetuses and stillborn children have been published very recently. *Irani* and *Sherman* (1963) examined eleven cases of club-foot and three calcaneo-valgus foot. Their conclusions were: "In idiopathic club-foot there are no primary abnormalities of vessels, nerves, muscles or tendon insertions. Only the anterior part of the talus is always abnormal. The anterior part of the calcaneus is usually abnormal, but much less so and to variable degree. These deviations probably result from a defective cartilaginous anlage which is dependent upon a primary germ-plasm defect." *Settle* (1963)

presented his anatomical findings from the dissection of sixteen infantile club-feet. Anatomical abnormalities were surprisingly uniform. He writes: ". . . congenital talipes equinovarus is a composite deformity involving all tissues of the foot. There are no simple or isolated defects such as contracted bands or peroneal muscle atrophy, but each tissue conforms to the equinus and varus position. The major bone deformity resides in the talus. Its neck and articular surface for the navicular deviate to face medially and plantarward. Congenital club-foot is a primary developmental deformity of the hind part of the foot". *Reimann* (1967) presented the dissection material of six cases comprising fetuses, stillborns and persons with club-foot who had died. Her conclusions were: "Gross and histological studies of the spinal cord and nerve roots, including ganglion cell counts, disclosed no abnormality in the club-foot cases. Histological study of muscles showed no signs of neurogenous atrophy. Histological study of the muscles showed normal muscles. Microscopically the average diameter of the muscle fibres was diminished. Histologically the vessels have proved normal. Skeletal changes have been a constant finding, consisting of dysplasia and changes in shape mostly affecting the talus. The skeletal changes are considered to be secondary."

b) Soft tissue changes

Little (1839) was the first to assert that uncoordinated muscle activity or abnormal tendon insertions might be the cause of club-foot and mentions the notion of Hippocrates that muscles maintain the clubfoot. *Mackeever* (1820) (quoted by *Little*) and *Tourtual* (1832) had made earlier observations on muscles in this connection.

Adams (1854—1855) described the dissection of immediately postnatal cases of club-foot and discovered anomalies in the gastrocnemius, the tibialis anterior and the tibialis posterior muscles which were "very tense and possibily hypertrophied". All three muscle bellies were shortened. The extensor digitorum longus muscle was "very small" and microscopically "degenerate". In another case he found several tendinous abnormalities. Nevertheless he considered soft tissue changes to be secondary and was convinced that the talus changes he had found were primary.

Von Volkmann (1863) also described changes in the muscles of club-foot and noted small peroneal muscles and a small extensor digitorum communis muscle.

Bissell (1888) studied the muscles of the equinovarus feet of an infant and noted that the tibialis anterior and posterior tendons had

shortened and moved inwards. Like many other investigators, however, he was more deeply concerned with detailed description of bone changes, especially in the talus.

Elmslie (1920) discussed the anatomy of the club-foot. He wrote that the chief factor of the deformity was a displacement of the bones, and that the importance of the tendons in maintaining a deformity had been much exaggerated. But he also said, "Doubtless, the tendons of the tibialis anticus and posticus and tendo Achillis have a considerable part in the original production of the deformity."

Dittrich (1930) published the results of a dissection of an infant with bilateral club-foot and came to the conclusion that, "the position and form of the various components of the foot are secondary to changes in the soft tissues which control them".

Middleton (1932) studied the pathological anatomy of congenital anomalies and assembled material which included congenital tibial kyphosis and arthrogryposis multiplex congenita. He suggested the imperfect development of striated muscle as a possible cause of certain congenital deformities of the extremities.

Mau (1938) made an examination of three fetuses with club-foot and other congenital abnormalities. In muscles he found macroscopically nothing of note, but histologically "there was a loss of striation". He considered this secondary to "myelodysplasia". He noted changes in each muscle of his histological specimen and could not attribute the deformity to any particular muscle.

Scherb (1930) also noted anomalies in club-foot muscles. In operations he often noticed an attachment of the peroneus brevis muscle to the cuboid instead of the usual attachment to the fifth metatarsal. This, he asserted, was the cause of a depression of the lateral edge of the foot and a relative increase of supination power due to the abductive effect of the peroneus brevis muscle on the metatarsal plate decreased. Similar changes in the lateral tendinous region have been noted by *Debrunner* (1957) and also by *Fredenhagen* (1954).

Bechtol and *Mossman* (1950) studied embryonic club-foot and observed changes in muscular development, some cases being more imperfectly developed in the histological sense. This, the authors maintained, was the cause of the muscular imbalance of club-foot.

Stewart (1951) presented his findings from the dissection of one stillborn and twenty operated patients. He found several abnormalities of tendon insertion, but most common was the unusual attachment of the Achilles tendon to the medial side of the tuber calcanei. His findings were in full

agreement with the results presented earlier by Scherb. In 1954, *Penners* published similar insertion anomalies noted in operated patients. They occurred in Achilles tendon insertions, in the tibialis anterior and extensor hallucis longus tendons, in the plantaris muscle, in the insertion of the tibialis posterior tendon and in the plantar fascia.

Flinchum in 1953 published the results of his dissection of a club-foot in a six-and-a-half month infant. He found an insertion anomaly of the Achilles tendon, a large tibialis anterior muscle and small peroneal muscles. The tibialis posterior muscle was normal compared to the standard of a healthy foot. The author considered bone changes secondary resulting from muscular imbalance.

Wiley (1959) gathered material in six cases of club-foot from post-mortem examinations performed on infants. He noted that the bellies of the long muscles were shorter and thinner and commented, "In the severe cases abnormal musculature was noted throughout the long muscles. In talipes equinovarus the principal muscles affected were the gastrocnemius and soleus, the tibialis posterior and the tibialis anterior, in that order." The cause of these muscle changes, in his opinion, was an extrinsic factor maintaining the foot in a deformed position and causing growth deficiencies in the long muscle.

B. VERTICAL TALUS

The congenital flat-foot deformity connected with the vertical position of the talus received no attention in the literature until this century. The vertical position had evidently remained unnoticed, and vertical talus cases had been discussed in connection with ordinary flat-foot cases. It was the development of the X-ray technique which made a consideration of the various forms of flat-foot possible.

The first complete radiological, anatomical and pathological study of extreme flat-foot with vertical talus dates from 1914 (*Henken*). Numerous publications followed in which the deformity was described. They were mainly from continental Europe in the period 1920—40. All authors noted the special features of the deformity. These are, clinically, convexity of the sole, deviation into valgus of the posterior part of the foot, abduction and dorsiflexion of the forepart of the foot and, radiographically, the vertical or oblique position of the talus.

There have been divergent opinions on the nature of the deformity, however, some considering it a specific congenital malformation (*Chrysospathes* 1938) and others not considering it a morbid entity (*Ombrédanne*

1928). It has been recognized by most authors recently as an individual entity (*Herndon* and *Heyman* 1963). The latter used the term congenital convex pes valgus which was first used in 1939 by *Lamy* and *Weissman*, who list nine other terms in use. The terms vertical talus or congenital talonavicular dislocation or subluxation are recent additions. All these reflect different points of view in the interpretation of this deformity.

As with the etiology and pathology of talipes equinovarus deformity, there are a great variety of corresponding theories on the etiology and pathogenesis of congenital flat-foot and congenital vertical talus deformity. They can be divided into exogenic or extrinsic and endogenic or intrinsic theories.

Many have believed congenital flat-foot is an exogenic malformation caused by intra-uterine pressure. (*Henke* 1859, *Küstner* 1880, *Joachimsthal* 1903, *Lange* 1912, *Henken* 1914, *Krukenberg* 1934—1935, *Denis Browne* 1966, *Harrold* 1967). According to *MurkJansen*, (quoted by Spiro 1934—1935) pressure arises from a deficiency of amnion. *Chrysospathes* (1938) thought that pressure of the tibia against the tarsal bones in the early embryonic stage with the foot in dorsiflexion would cause deformity.

Many factors have been suggested which support the theory of endogenic origin. The deformity is often accompanied by other congenital malformations such as congenital luxation of the hip, club-foot, arthrogryposis, spina bifida, congenital kyphosis, hypospadia and microcephaly. Some have found evidence of heredity in this condition (*Aschner* and *Engelmann* 1928). Also, congenital talus verticalis has certain features in common with certain stages of the normal embryogenic development of the foot. Various authors consider that disturbed development of the bones of the foot is the primary reason for the deformity, as in club-foot.

Franke (1901) considered disturbances of muscle balance to be the cause of congenital flat-foot. He asserted that the insertion of the tibialis anterior had moved from the plantar to the dorsal side and that its supinatory effect was thus eliminated. *Braus* (quoted by *Lamy* and *Weissman* 1939) has noted in 50 per cent of congenital flat-foot cases that the supinatory effect of the tibialis anterior is lacking because of insertion variation. According to *Lamy* and *Weissman*, *Mau*, pursuing the same line of thought, assumed that a spinal lesion could cause a predominance of peroneal muscle action at a certain stage of fetal development, leading to a valgus position of the foot.

Some have surmised that abnormal, congenital laxity of muscles and ligaments is the reason for congenital convex pes valgus (*Hagenbach-Burckhardt* 1907, *Hackenbroch* 1924).

Some connect the changes with spina bifida occulta (*Peltesohn* 1920, *Beck* 1922, *Hackenbroch* 1924, *Spiro* 1934—1935).

Fuchs (quoted by *Spiro*) speaks of "myelodysplastic" changes which may sometimes be merely microscopic.

Böhm (1932) has shown that at the end of the second fetal month and the beginning of the third the long axis of the talus is the same as that of the leg and the position of the bones of the foot corresponds most closely to that seen in congenital vertical talus. It is thus suggested that the deformity is a cessation of development at that stage (*Böhm* 1932, *Spiro* 1934—1935). *Mau* (1930) has suggested that the primary cause of this arrested development is a lesion of the spinal cord, and that the foot deformity is secondary.

Lamy and *Weissman* (1939) state that all explanations of congenital pes valgus are far from satisfactory and the cause is still to be found. It should be sought among endogenous factors, however, since external factors only occasionally intervene. The role of the nervous system appears to be the most important. This is their opinion of the pathogenesis of club-foot in general.

Hark (1950) emphasized two facts concerning the etiology; one is the multiplicity of other deformities accompaning "rocker-foot", and the other is the occurence of only one case of spina bifida in his series. He believes that spina bifida is merely one manifestation of a developmental disturbance which may include "rocker-foot" as well as other deformities.

Osmond-Clarke (1956) suggested the possibility that in the early intrauterine life the foot with vertical talus has moved its position laterally in relation to the talus. The thought appealed to him that this may be an extreme form of congenital talipes valgus and that vertical talus is a subluxation of the talonavicular joint as in club-foot, but in the opposite direction.

Herndon and *Heyman* (1963) are inclined to believe that congenital convex pes valgus is related to an embryological fault during the first three months of pregnancy and that the deformity is closely akin to club-foot in etiology.

Støren (1967) said, "Observations made at operation support those made during closed reduction: The vertical position of talus is secondary to the subluxation seen in Choparts joint, which is in a great deal believed to be the primary event."

C. EXPERIMENTAL PRODUCTION OF SKELETAL MALFORMATIONS

The etiology of congenital defects in human beings is virtually unknown and evidently both genetic and environmental influences are involved (*Kalter* 1965, *Saxén* and *Rapola* 1969). For many years congenital anomalies were generally assumed to be wholly due to defects of the genes, chromosomes and germ plasm. Genetic considerations have tended to overshadow the importance of the environmental influences (*Ingalls* 1955). However, the association of developmental anomalies with epidemics of rubella, noted first in Australia by *Gregg* (1941) and attributed to maternal infection in the early months of pregnancy by *Swan* (1949) demonstrates clearly enough that changes in the genome cannot always be invoked as the cause of congenital abnormalities. Systematic studies of the congenital anomalies demonstrates that part of them are hereditary, genotypic, but also that environmental factors have teratogenic influence. The recent appearance of newborn babies with malformed limbs due to maternal ingestion of thalidomide as a tranquilizing drug during pregnancy has revealed the sensitivity of the fetus to these teratogenic factors (*Lenz* and *Knapp* 1962).

Experimental teratology has made great progress in the last few years and many excellent articles have been written on the experimental production of malformations (*Fraser* 1964, *Salzgeber* and *Wolff* 1964, *Saxén* and *Rapola* 1969). The list of exogenous factors causing skeletal malformations in experimental animals is long and includes mechanical injuries, hypoxia, ionizing radiation, a great variety of chemical compounds, dietary deficiences, hormonal factors, virus inoculation etc. Such factors may act on a genetically normal embryo causing different skeletal anomalies, but several examples have been described in which synergistic effect with a mutant gene has been demonstrated (Fraser, *Walker* and *Trasler* 1957, *Landauer* 1965, *Dagg* 1967). Only a few pioneering experiments in this field have been chosen for discussion, and reader is referred to recent articles where the problem has been dealt with in greater detail.

Earlier mechanical factors were generally considered to be an important cause of skeletal anomalies. *Browne* (1936, 1967) has presented a vast material on deformities of mechanistic causation., *Trasler, Walker* and *Fraser* (1956) have experimentally produced congenital malformations in mice by amniotic-sac puncture.

Hypoxia is a teratogenic factor. *Romanoff* (1930) found that hypoxia caused anomalies in the chick embryos. Both *Ingalls, Curley* and *Prindle*

(1952) and later *Degenhardt* and *Knoche* (1959) have published detailed material on the skeletal defects in the offspring of mothers subjected to low oxygen pressures.

Ionizing radiation has been used for the experimental provocation of congenital deformities. The first experiments on mammals were carried out in pregnant rabbits by v. *Hippel* in 1905. In 1920—29 *Bagg* published his important studies. He investigated the effect of X-rays, radium emanation and injections of radio-active solutions on the development of mice, rats and dogs. The most constant lesions occurred in the head region of embryos. There were also abnormalities of the limbs; various forms of club-foot, syndactylism, hypodactylism, congenital amputations and polydactylism. Later *Warkany* and *Schraffenberger* (1947) in rats and *Russel* and *Russel* (1954) in mice produced primarily skeletal anomalies by X-ray irradiation.

"Chemoteratogenesis", the production of malformations by means of chemical substances, has after the thalidomide disaster attracted much interest. Many active teratogens, both naturally occurring and artificially produced, have since been described. However, as early as 1921 *Stockard* published a classic study in which he succeeded in reproducing malformations by means of chemical agents. The work of *Ancel* and *Lallemand* (1942) has contributed considerably to the development of experimental teratology. They succeeded in producing malformations of chick embryos by eserine sulphate and sulphonamide. Since then, a great variety of chemical compounds have been tested for their teratogenic action, and in most cases a suitable combination of dose and animal species have yielded positive results. As regards human development, proven chemical teratogens, are, however, few (*Cahen* 1966). The thalidomide accidents prompted a large number of investigators to test thalidomide in various animal species. In mice and rabbits it had a teratogenic effect. The first experiments were carried out by *Giroud, Tuchmann-Duplessis* and *Mercier-Parot* (1962). With regard to the limbs, the most common findings were malformations of the club-foot type. *Drachman* and *Coulombre* (1962) induced club feet and arthrogryposis curarizing chick fetuses.

Dietary deficiencis have a teratogenic effect. The first research on vitamin deficiencis was carried out by *Hale* (1933). He produced eye anomalies in pigs by vitamin A deficiency. *Warkany* and *Nelson* (1940) used the deficiency of riboflavin in the mother's diet to induce experimentally skeletal malformations like many other anomalies. Deficiency

of folic acid, panthotenic acid and vitamin E have also been used to produce skeletal anomalies: cleft palate, club-foot etc.

Hormonal factors can also induce experimental malformations. Insulin has a strong teratogenic effect on the limb in the chick embryo (*Landauer* 1945). Very interesting studies to orthopedic surgery were carried out by *Duraiswami* (1952), who induced congenital skeletal defects in chick embryos also by insulin. Using certain pituitary preparations, *Jost* (1953) obtained degeneration of the extremities in the rat fetus. Cortisone has been used as a teratogenic agent by many. *Hicks* (1954) provoked, among other defects, skeletal anomalies by large cortisone doses in mice. The investigations of *Walker* and *Fraser* (1956, 1957), who induced cleft palate deformity by cortisone both in mice and rats, are classical.

In experimental teratology, many other environmental agents such as virus infection, have been used to produce congenital anomalies. By means of influenza-A-virus in chick embryos, *Hamburger* and *Habel* (1947) provoked a typical anomaly, microcephaly, and *Ferm* and *Kilham* (1964) induced congenital anomalies in hamster with H-1-virus.

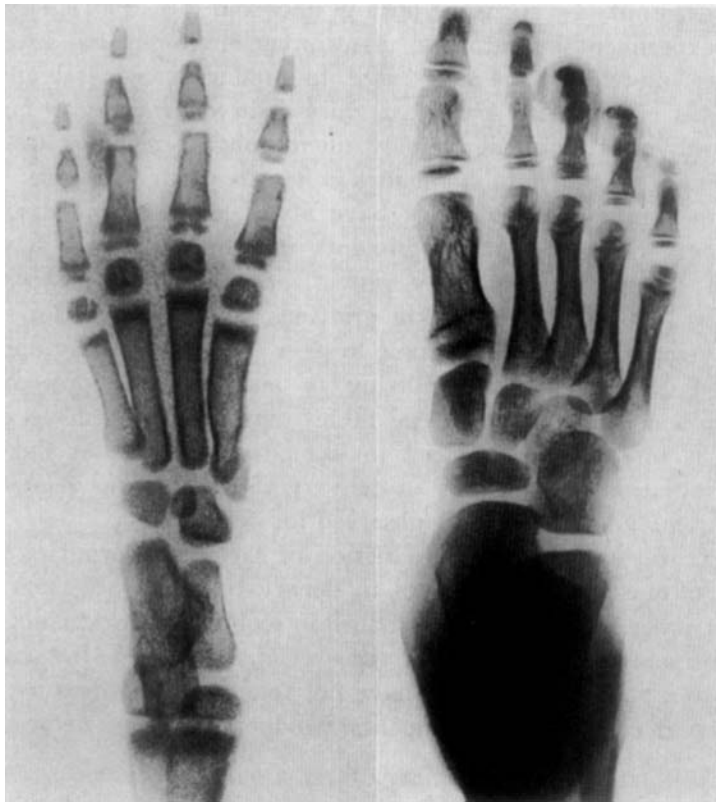
Intra-uterine manipulations have also been used in experimental teratology. Some recent experiments in which embryos hawe been treated locally *in utero* may prove very important. *Schinckel* and *Ferguson* (1963) performed skin graftings on fetal lambs for studying the development of the immune system. In 1964, *Spencer* and *Peltier* reported their attempts, utilizing *in utero* surgical procedures, to produce a deformity in the fetal rabbit extremity. By means of a silk suture a fore-foot was placed in a moderate degree of flexion and ulnar deviation. On the day of delivery the suture was removed from the extremity and the rabbit observed for deformity.

Postnatal experimental production of skeletal deformities by transection of muscles and ligaments, by denervation of muscles and by bone or joint procedures has been described in scoliosis, dislocation of hip etc. (*Schwartzmann* and *Miles* 1945, *Langenskiöld, Sarpio* and *Michelsson* 1962, *Michelsson* 1965). However, I have not found in the literature a single such report concerning experimental production of limb deformity.

III. MATERIAL AND METHODS

A. EXPERIMENTAL ANIMALS

The present study is based on the results of operations on 402 growing rabbits. Approximately 75-100 growing rabbits were used in preliminary experiments and are not included in the results. The rabbits were operated on at the age of four to twenty-eight days, most of them being six to seven days old at operation.



Rabbit Human

FIG. 1. X-rays in the dorsoplantar projection of the normal foot of a rabbit and of a man. General skeletal structure and relative positions of tarsal bones are strikingly similar. The rabbit has four metatarsals and four digits (the first row is missing).

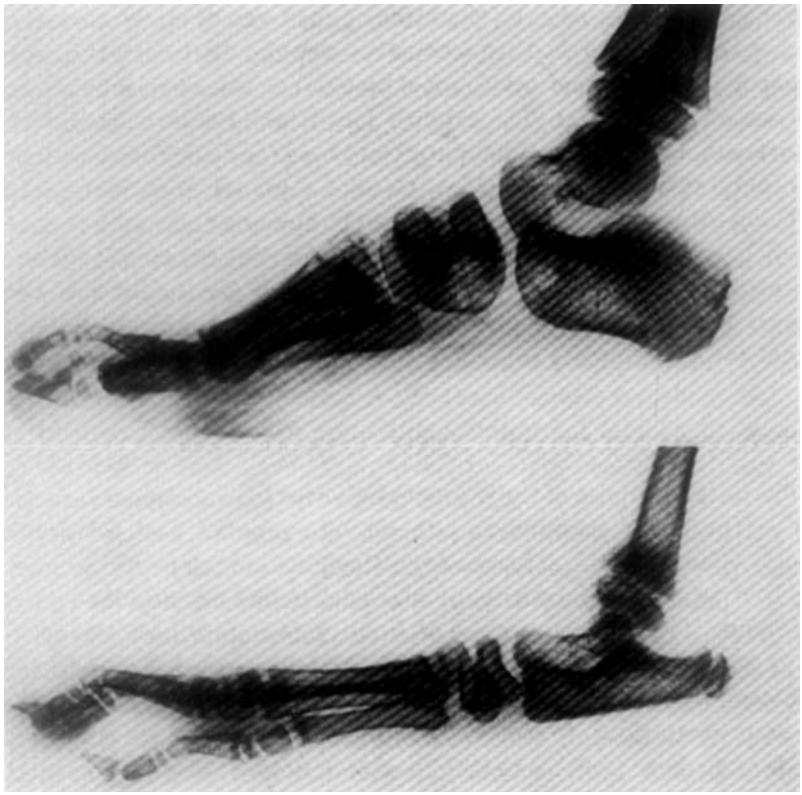


FIG. 2. X-rays in the lateral projection of the normal foot of a man (top) and of a rabbit (bottom). General skeletal structure and relative positions of bones are strikingly similar.

B. ANATOMY OF THE FOOT OF THE RABBIT COMPARED WITH THE HUMAN FOOT

The human foot is unlike any other. It is the most distinctly human part of the whole of man's anatomical make-up. It is by his feet that man is distinguished from all other members of the animal kingdom. (*Jones* 1944).

Despite this, however, there are remarkable resemblances between the hind feet of quadrupeds and the human foot. The resemblance is of course greatest in anthropoid apes, but the use of such large and expensive animals in the study of foot deformities would present difficulties if a sufficiently large material were to be obtained. In many other four-

footed plantigrades such as the rabbit, moreover, there is a basic structure of the foot which conspicuously resembles that found in humans. In particular the structure of tarsal bones and their anatomical interrelation are almost similar. The rabbit's foot is admittedly flatter, while the metatarsals and toes are comparatively long, and the fore-foot has only four metatarsals and digits (Figs. 1 and 2). But the skeletal age at birth and soon afterwards is almost the same in humans and rabbits, as *Heikel* (1960) has noted. Similarities in leg muscles and other soft tissues are also striking, as the study of the anatomy of the rabbit has verified (*Krause* 1884). Hence the young rabbit seems to be a suitable laboratory animal for experiments involving imitation of human foot deformities.

C. DEVELOPMENT OF OPERATIVE METHODS

The initial purpose of the present study was to cause muscular contracture of the leg in rabbits, and thereby to produce foot deformities if possible. This notion had arisen from the quite common clinical observation that in foot deformities in man shortening of muscles and other soft tissue structures appear. The immediate impulse for these experiments was given by shortening of the tibialis posterior muscle and its transformation into a tight string, which had often been noted in club-foot operations (*Fried* 1959, *Langenskiöld* 1961). An attempt was therefore made to produce ischemic contracture by using various methods of blocking the blood circulation. It proved very difficult, however, to produce a permanent contracture in the leg muscles of a growing rabbit in accordance with earlier studies (*Brooks* 1922). An attempt was also made to produce contracture and fibrosis of the muscles both chemically (by the infiltration of carbolic acid, for instance) and by means of electrocoagulation. No lasting contractures were obtained.

Younger and younger rabbits were taken as test animals, as it was noticed that more reliable and lasting changes were produced in young rabbits than in older animals. Lasting changes in four-week-old rabbits rarely occurred. This is in agreement with the observations of *Crawford* (personal communication to *Wiley* 1959) when he immobilised the feet of a rabbit in dorsiflexion and caused increased resistance to full plantarflexion of the foot by means of resting tension. This myostatic contracture effect was less in adult rabbits than in young ones. For practical reasons the young of rabbits were usually 4—7 days old. Among still younger animals mortality was greater, mainly because of abandonment by the mother.

Another method of producing the effect of deformity corresponding to contractures in the periphery was considered. The leg muscles including the gastrocnemius and soleus muscles were fixed with nylon thread through a hole bored through the tibia at its halfway point. It was expected that the muscles would thus be hampered in growth and normal function with resulting constriction and foot deformity. But this, too, was not wholly successful. The muscle evidently either stretched or grew, but while attached was able to adapt itself to the growth of the bones so that no true deformities occurred. A possible explanation may be found in the tests performed by *Lange* (1929). In a series of experimental rabbits, the Achilles tendon and one-third to one-half of the belly of the gastrocnemius were excised. A silk thread connected the remaining muscle to the calcaneus with approximately normal tension. Regeneration of muscle started down the thread in eight weeks and had the appearance of normal muscle and tendon.

Tendon fixation or tenodesis was the next method to be tried for provocation of foot deformity. The Achilles tendon, which is very strong in rabbits as it is in humans, could as a rule be fixed successfully to the tibia. This fixation maintained the calcaneus in a position of plantarflexion and the Achilles tendon could not adapt itself to the growth of the bones. According to *Haines* (1932), a tendon under tension is unable to increase its length. But it proved technically impossible to achieve this fully with the very thin and muscular tendons of the tibialis anterior and the extensor digitorum longus at the front of the leg of a rabbit. An attempt was therefore made to cause imbalance of muscle power on this side by muscle-tendon transections and transection of the ligamentum transversum cruris. By this means it was also thought possible to bring about changes in the direction of action of the muscles. Observations obtained from paralytic deformities such as those seen in poliomyelitis, were considered when planning the experiments. But over a year's work on more than 75 rabbits had been done before a method of operation was evolved which brought about a progressive deformity in the rabbit, resembling the talipes equinovarus deformity in man. (*Ritsilä* 1964).

D. TECHNIQUE

1. General operative technique

The operations were performed under aseptic conditions on the left leg under local anaesthesia (0.25% Xylocain exadrine®), while the right leg was left intact as a control. Longitudinal, ventral and dorsal skin incisions

were made near the ankle (ventral) and Achilles tendon (dorsal). Using normal tendon-stitching technique, a nylon thread was sewn to the Achilles tendon. The tendon was cut between the tendinous and muscular part, drawn out and fixed through a hole bored in the middle of the tibia on the medial side so tightly that the talocrural joint was in extreme plantar flexion at the time of the operation. On the ventral side, transection of the muscles was performed, followed later as a rule by complete removal of part of the muscle and tendon to eliminate possible regeneration. The same ventral incision enabled transection of the peroneal muscles and of the ligamentum transversum cruris to be performed.

2. Plaster of Paris technique

Plaster immobilisation in fixed positions was performed by means of rapidly-hardening, Cellona® plaster bandage. In order to avoid disturbance of blood circulation, this was not applied to the whole circumference of the leg. The plaster of Paris splint was tied loosely to the limb by gauze bandage. As a rule the plaster remained firmly attached to the limb but because of the rabbit's fast growth it had to be changed once a week. Like other procedures, plaster was applied to the left hind limb leaving the right as a control.

3. Fixation technique

With experimental animals on whom other methods of fixation than a plaster splint were applied in order to keep the foot in a fixed position, the following technique was used. By manipulation of the left hind limb, the foot was brought into the desired position. It was kept there by a nylon thread drawn through the foot between the metatarsals. This thread was attached through the tibia halfway along the leg, avoiding the principal nerves and blood-vessels. (Fig. 3).

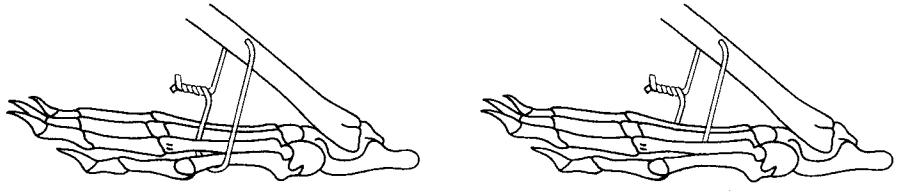


FIG. 3. Diagrams to show examples of fixation of the foot of a growing rabbit in a calcaneovalgus position (left) and in a calcaneovarus position (right).

E. METHODS OF INVESTIGATION

1. Radiological study

The deformity and position of the bones were radiologically recorded in all animals. The first examination was made one week after operation. Radiographs were subsequently taken 3—4 weeks, 2 months, 3—4 months, 6 months and at least 1 year after operation. Further radiographs were taken even later if the animal remained alive. Finally, radiographs of the dissected specimens were taken. Dorsoplantar or anteroposterior and lateral projections of both limbs were used.

2. Gross-section study

Almost all animals were dissected post mortem. Specimens were examined by inspection. Soft tissue changes, deformity and the position of bones were recorded and the degrees of deformity measured from radiographs. Photographs of specimens were taken when indicated.

3. Histological study

Specimens of eight animals were histologically examined. After fixation in neutral formalin and decalcification in ethylene-diaminetetraacetic acid, the specimens were embedded in paraffin, sectioned in the desired planes and stained with haematoxylin *van Gieson*.

F. OPERATIONS PERFORMED

The operations were confined to the leg region as fas as possible, since operations performed on the foot itself would make estimation of peripheral changes impossible (Fig. 15; p. 48). Operations on the leg muscles and tendons together with the ligamentum transversum cruris offer an infinite number of possible combinations, though not all combinations are adequate for the production of specific effects of muscle imbalance. Nineteen different measures were used besides four fixation methods (Table 1).

Table 1. Operative and plaster immobilisation measures, with abbreviations, taken in experiments with soft tissue components.

A_F = Achilles tendon fixation (tenodesis)
A_T = Achilles tendon transection or resection
E_F = extensor digitorum longus muscle or tendon fixation
A_S = Achilles tendon shortening
$A \rightarrow TA$ = Achillo-tibial tenoplasty
$A \rightarrow (E+TA)$ = Achillo-extensoro-tibial tenoplasty
E_T = extensor digitorum longus muscle or tendon transection
TA_F = tibialis anterior muscle or tendon fixation
TA_T = tibialis anterior muscle or tendon transection
P_T = transection of all peroneal muscles or tendons
PB_T = peroneus brevis, tertius and quartus muscles or tendons transection
PL_T = peroneus longus muscle or tendon transection
"TP_F" = tibialis posterior muscle (extensor digiti primi) or tendon fixation
"TP_T" = tibialis posterior muscle (extensor digiti primi) or tendon transection
F_F = flexor digitorum longus muscle or tendon fixation
F_T = flexor digitorum longus muscle or tendon transection
L_T = transection of ligamentum transversum cruris
K_E = plaster of Paris immobilisation in equinus position
K_V = plaster of Paris immobilisation in varus position

Possibilities of combinations of these measures were too great in number for a completely systematic study. It was therefore necessary to start from certain hypotheses and continue the choice of combinations on the basis of results in order to achieve specific deformities. The next aim was to make a selective search for significant factors in the cases of experimental deformity produced, by analysing the dissected specimens. The simplest possible combinations for the production of deformity were thus determined. Negative results were also important in explaining the factors leading to deformity.

A total of 76 operations or other measures, including plaster or other fixation, were performed (Table 2).

Table 2. Operations performed and equinovarus and vertical talus deformities.

Combination	Number of experiments	Number of equinovarus	Number of vertical talus
A_F	4		
L_T	3		
A_F+L_T	13	1	7
$A_F+E_T+L_T$	11		7
$A_F+TA_T+L_T$	9		6
$A_F+P_T+L_T$	14		5
$A_F+E_T+P_T$	19	9	
$A_F+TA_T+P_T$	29	1	1
$A_F+E_T+P_T+L_T$	29	11	4
$A_F+E_T+TA_T$	1		
A_F+E_T+"TP_F"	6		
$A_F+TA_F+P_T$	4		
A_F+TA_F	10		
A_F+E_T	11		3
A_F+TA_T	3		
A_F+P_T	4		
$A_F+TA_T+P_T+L_T$	11		7
$A_F+E_T+PB_T+L_T$	1		
$A_F+TA_F+F_T$	1		
$A_F+TA_T+F_T$	1		
$A_F+TA_T+F_F$	1		
$A_F+TA_F+PL_T$	4		
$A_F+E_F+TA_F+PL_T$	5		
$A_F+E_T+TA_T+PL_T$	2		
$A_F+TA_F+F_T+PL_T$	1		
$A_F+E_T+TA_T+P_T$	13		
$A_F+E_T+TA_T+L_T$	6		4
$A_F+E_T+TA_T+$"TP_T"	5	2	
$A_F+E_T+TA_T+$"TP_F"	1		
$A_F+E_T+P_T+F_T$	3	1	
$A_F+P_T+L_T+F_T$	3		
$A_F+E_T+TA_T+P_T+$ $+$"TP_T"	8		
A_T	7		
A_T+E_T	1		
A_T+TA_T	2		
A_T+TA_F	3		
A_T+P_T	3		
A_T+L_T	5		
$A_T+E_T+L_T$	1		
$A_T+TA_T+L_T$	2		
$A_T+TA_T+P_T$	2		
$A_T+TA_T+P_T$	1		
$A_T+TA_F+PL_T$	1		
$A_T+TA_F+P_T+L_T$	1		

Table 2. Continued.

Combination	Number of experiments	Number of equinovarus	Number of vertical talus
A→TA	8		
A→TA + P_T	6		
A→TA + P_T + L_T	5		
A→(E + TA) + P_T	4		
A_S + P_T	5		
A_S + E_T + P_T	5		
A_S + TA_T + P_T	4		
TA_T	1		
PB_T	3		
„TP_F"	6		
TA_F + P_T	3		
TA_T + P_T	8		
TA_T + L_T	1		
E_T + L_T	2		
E_T + PL_T + L_T	3		
E_T + PB_T + L_T	4		
E_T + P_T + L_T	4		
K_E	4		
K_E + K_V	11	4	
K_E + K_V + L_T	4		1
K_E + L_T	14		3
K_E + TA_T + L_T	2		
K_E + E_T + L_T	2		
K_E + P_T	5		
K_E + E_T + P_T	5	2	
K_E + K_V + A_T	3		
K_E + K_V + A_T + TA_T + „TP_T"	3		
Fixation in calcaneo-varus position	3		1
Fixation in calcaneo-valgus position	2		2
Fixation in equino-varus position	1	1	
Fixation in equinus position	1		
Fixation in equinus + L_T	5		
Total	402	32	51

IV. RESULTS

A. TALIPES EQUINOVARUS

1. General description of deformity produced and factors leading to deformity

When the records of the experiments were reviewed it was found that there was a clear regularity in the procedures leading to talipes equinovarus deformity. (Table 3). Achilles tenodesis was always performed producing an equinus position of the calcaneus if it had not been already produced by plaster immobilisation or other fixation. In most rabbits with equinovarus deformity transection of both the extensor digitorum longus muscle and the peroneus muscles had been performed. (Table 3).

Table 3. Structural talipes equinovarus deformity in rabbits resulting from various procedures.

Series	Rabbit	Operation	Remarks
LVII	No. 7	$A_F + L_T$	Good Achilles tenodesis, lesion of peroneal nerve: extensor and peroneal muscles atrophied.
XIII	No. 5	$A_T + TA_T + P_T$	Good Achilles tenodesis, extensor muscles also atrophied.
XXXII	No. 6	$A_F + E_T + P_T$	Good Achilles tenodesis, tibialis anterior and flexor digitorum muscles contracture
XXXII	No. 7	—»—	—»—
XXXVIII	No. 3	—»—	—»—
—»—	No. 4	—»—	—»—
—»—	No. 5	—»—	—»—
XLIII	No. 3	—»—	—»—
—»—	No. 5	—»—	—»—
XLVII	No. 3	—»—	—»—
—»—	No. 4	—»—	—»—

Table 3. Continued.

XXVI	No. 10	$A_F+E_T+P_T+L_T$	Good Achilles tenodesis, tibialis anterior and flexor digitorum muscles contracture
XXVII	No. 4	—»—	—»—
—»—	No. 7	—»—	—»—
XXVIII	No. 1	—»—	—»—
—»—	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
—»—	No. 6	—»—	—»—
—»—	No. 8	—»—	—»—
XXXII	No. 1	—»—	—»—
XLIX	No. 1	—»—	—»—
—»—	No. 5	—»—	—»—
XXXIII	No. 1	$A_F+E_T+P_T+F_T$	Good Achilles tenodesis, flexor muscles reattached
XXIV	No. 3	$A_F+E_T+TA_T+$"TP_T"	Good Achilles tenodesis, tibialis anterior and "tibialis posterior" muscles reattached
XXVI	No. 5	—»—	—»—
LVIII	No. 1	K_E+K_V	Achilles tendon, tibialis anterior and flexor digitorum muscles contracture
—»—	No. 2	—»—	—»—
—»—	No. 7	—»—	—»—
LXI	No. 1	—»—	—»—
LV	No. 1	$K_E+E_T+P_T$	—»—
—»—	No. 4	—»—	—»—
XII	No. 7	Fixation in equinovarus position	—»—

Transection of the ligamentum transversum cruris was also performed on several animals developing equinovarus deformity, but the deformity also appeared without this measure. Severing the ligament clearly accentuated the deforming effect of the tibialis anterior and augmented the adducto-varus position of the metatarsus. In some rabbits where the equinovarus position appeared in other combinations, careful dissection explained the factors which led to the changes. Here, too, soft tissue changes were in accordance with the deformity and explained its development. Thus in Series XIII no. 5, in which the combination; Achilles tenodesis and transections of the tibialis anterior and peroneal mucles, had produced equinovarus, it was noted that the extensors were atrophied,

while the tibialis anterior had become reattached. Muscle-tendon resections had not yet been performed. When the test series were renewed using resections, this combination did not lead to the equinovarus deformity. Similarly, in Series XXIV no. 3 ($A_F+E_T+TA_T+"TP"_T$) and Series XXVI no. 5 ($A_F+E_T+TA_T+"TP"_T$), in which muscle-tendon resections had not yet been performed, but discisions had been extended to include the tibialis anterior and the "tibialis posterior", it appeared at macroscopic examination that re-adherence and contracture had occurred in the region of the tibial muscles. In Series XXXIII no. 1 where, in addition to the combination; Achilles tenodesis and transections of the extensor digitorum and peroneal muscles, severing of the flexor digitorum had been performed, dissection revealed that the flexors had become attached and were tight. In Series LVII no. 7 with Achilles tenodesis and transection of the ligamentum transversum cruris, atrophy of the extensor and peroneal muscles was noted as a possible result of a lesion of the peroneal nerve arising accidentally with Achilles tenodesis.

In other animals, an equinovarus deformity developed either through external fixation with nylon thread in the equinovarus position (Series XII no. 7) or through fixation in the equinovarus position by means of plaster (Series LVIII no. 1 etc.). But immobilisation must be continued sufficiently long time, three weeks at the least. Then a permanent, even progressive deformity developed.

Two interesting experiments are in Series LV nos. 1 and 4. Transections of the extensor digitorum and peroneal muscles were performed in connection with immobilisation by plaster in the equinus position producing a typical equinovarus deformity.

On the other hand, no deformity with equinovarus position developed when resections of the Achilles tendon, the tibialis anterior and tibialis posterior muscles were made in connection with plaster immobilisation in the equinovarus position. Nor was equinovarus deformity produced in series where one of the factors; Achilles tenodesis, transections of the extensor digitorum or peroneal muscles was omitted from the combination.

Talipes equinovarus deformity was produced in the following combination of three components, summarized as follows:

First, the proximal part of the Achilles tendon was drawn through a hole bored in the middle part of the tibial diaphysis and fixed to the bone. Tenodesis was thus performed.

Second, transection of the peroneal muscles and the extensor digitorum longus muscle was performed. A pronation effect was thus eliminated.

Third, the tibialis anterior, the tibialis posterior and flexor digitorum longus muscles with their supinatory effects were left intact.

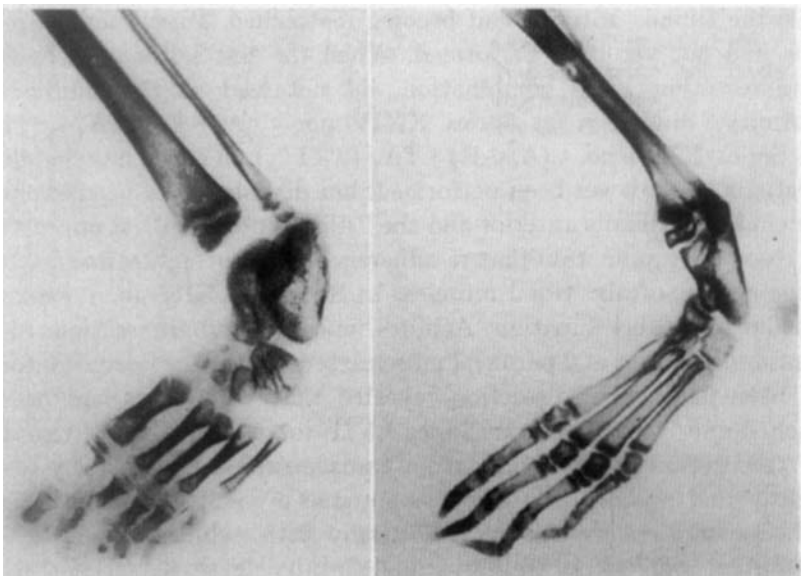


FIG. 4, X-ray pictures in the dorsoplantar projection of talipes equinovarus of a child (left) and of experimental talipes equinovarus in a rabbit; Series XXVIII no. 1 (right).

General skeletal structure and relative positions of tarsal bones are strikingly similar.

2. Comparison of experimental talipes equinovarus in the rabbit and talipes equinovarus in man

a) General comparison

The deformity known as talipes equinovarus or club-foot comprises the following components; adduction of the fore-foot, varus or inversion of the heel and equinus of the entire foot (*Kite* 1964). Club-foot also has a cavus deformity (*Ponseti* and *Smoley* 1963).

In all experimental foot deformities noted in Table 3, it was possible to show these different components (Figs. 4 and 12).

Clinical examination of the pathology of talipes equinovarus in man is an easy matter since the bones of the foot can be clearly palpated. In a club-foot the talus is in approximately the same position as in a normal foot that has been strongly adducted an plantar flexed. The fore-foot and os calcis are medially rotated and displaced around the talus. The lateral malleolus is prominent, but the medial malleolus is difficult to find by touch. Also, the head of the talus is prominent on the lateral side of the dorsum pedis. The calcaneus is in inversion and equinus deformity.

In rabbits with equinovarus deformity the same general changes could be found (Figs. 5 and 7).

X-ray examination clarifies the position of the bones in the foot and their relationship to one another. Comparison of the radiographic appearance of the experimental equinovarus deformity in rabbits with equinovarus deformity in children revealed striking similarities; in the dorsoplantar projection it can be seen that the calcaneus has turned into the inversion position under the talus. The fore-foot is adducted. The navicular, cuboid, cuneiforms and metatarsals are displaced medially and the fore-foot is thus in adduction deformity. The navicular is medial to the head of the talus and the cuboid medial to the distal end of the calcaneus (Fig. 4).

b) Patho-anatomical comparison of position of bones in relation to each other.

The patho-anatomical changes in the bones of club-feet were already noted at the end of the 18th century (*Blumenbach* 1786, *Clossius* 1798, *Wanzel* 1798, etc. according to *Kreuz* 1927). *Antonio Scarpa* in 1803 thought that the deformity was caused by the navicular, cuboid and calcaneus moving medial to the talus. He observed this while examining club-feet in children. Renewed study has led to the conclusion that the most notable divergence from normal in the relation of the bones of a club-foot apart from the plantar flexed position of the talus is the more medial position of the navicular in relation to the head of the talus (*Kreuz* 1927, *Virchow* 1933, *Wiley* 1959, *Irani* and *Sherman* 1963, *Ponseti* and *Smoley* 1963). *Debrunner* in his monograph suggests that the structure of a club-foot is understandable as a twisting of the calcaneus, the cuboid, the navicular and the whole fore-foot into inversion and adduction around the talus. *Brockman* (1930) noted that in a club-foot there is subluxation of the talocalcaneonavicular joint and alterations in the position of the navicular and calcaneus in relation to the talus. The talus is also in the equinus position in the tibio-fibular mortise, and it has even been said that a forward subluxation of the talus is present (*Marique* 1946).

When comparing the position of the bones of experimentally-produced equinovarus feet with that in a normal foot, the changes observed have been very similar to those noted in the literature concerning club-foot in man (Figs. 6 and 8).

FIG. 5. Gross anatomical structure of the bones in rabbit talipes equinovarus, Series XXVIII no. 3, front view.

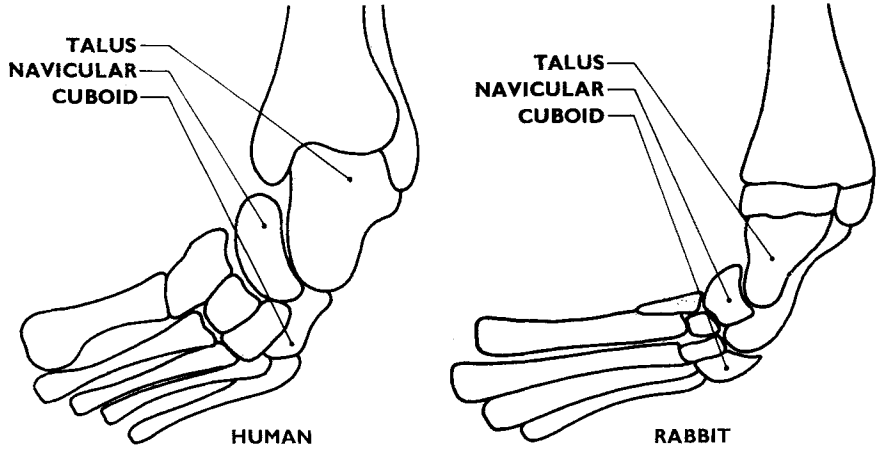


FIG. 6. Diagram of Fig. 5 compared with diagram of corresponding human talipes equinovarus according to *Kreuz* (1927). (By courtesy of Arch. orthop. Unfall-Chir.)

In both, the foot is displaced and rotated medially below the talus. The head of the talus is on the lateral side of the dorsum of the foot owing to the medial and plantarward displacement of the navicular.

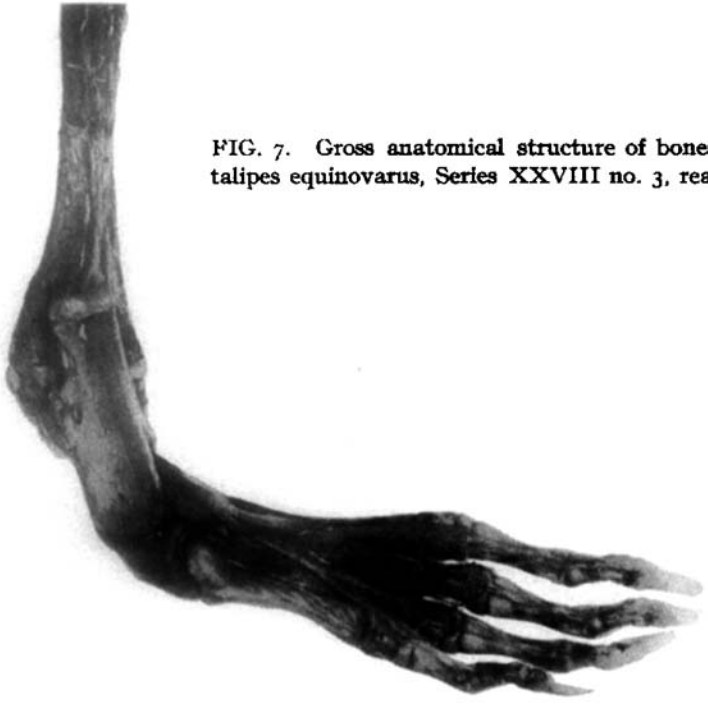


FIG. 7. **Gross anatomical structure of bones in rabbit talipes equinovarus, Series XXVIII no. 3, rear view.**

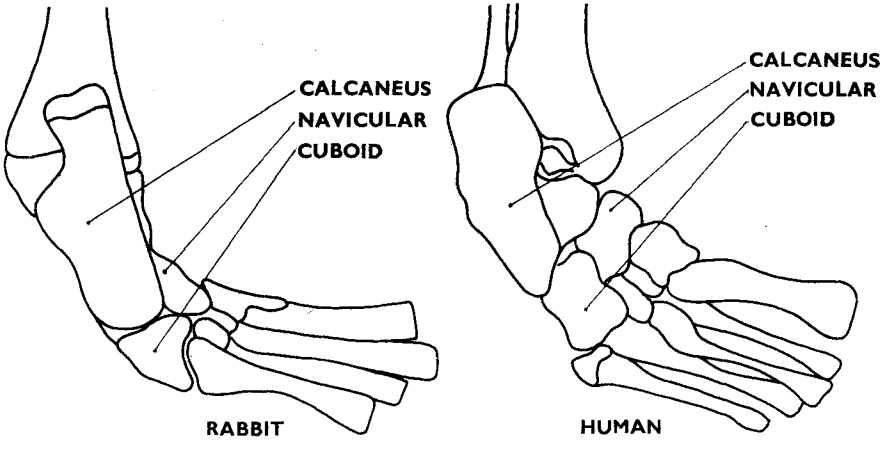


FIG. 8. Diagram of Fig. 7 compared with diagram of corresponding human talipes equinovarus according to *Kreutz* (1927). (By courtesy of Arch. orthop. Unfall-Chir.)

In both, the calcaneus is in a severe equinus position with its anterior portion lying below the head of the talus. The cuboid is also displaced inward.

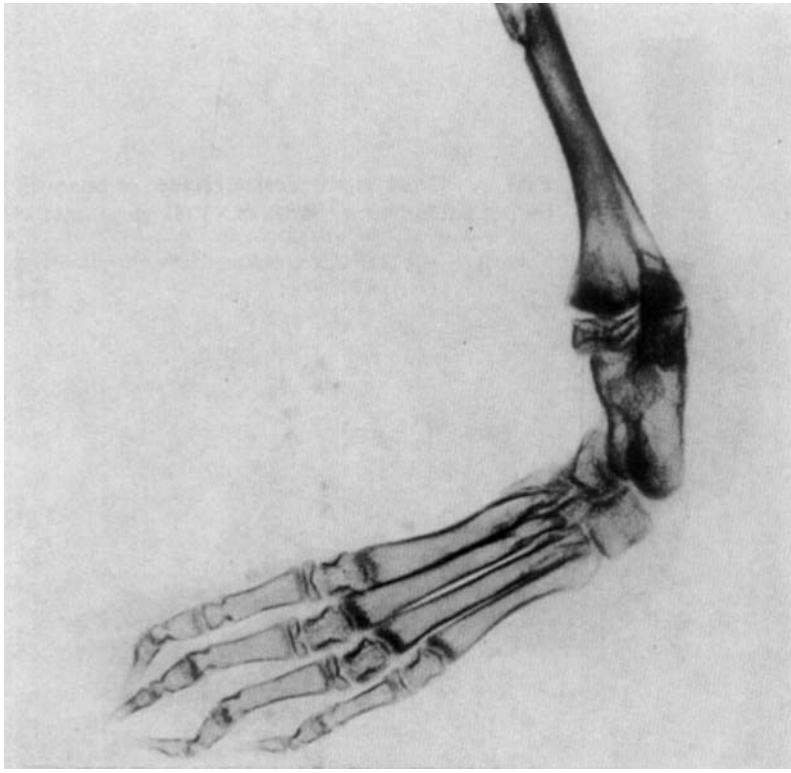


FIG. 9. Roentgenogram in the dorsoplantar projection of a rabbit talipes equinovarus; Series XXXII no. 7.

c) Radiological comparison of the talo-calcaneal angle

X-ray studies add much information about talipes equinovarus to that obtained from clinical examination.

The essential difference between a club-foot and a normal foot in man is the difference in the angle formed by the longitudinal axes of the talus and the calcaneus. According to *Debrunner* (1957), the difference in this angle is so great that a diagnosis can be made on the basis of the roentgenogram alone. Small changes in the talo-calcaneal angle can be seen on a lateral X-ray, but the most significant pathology is best seen in the dorsoplantar projection. In the normal human foot the talo-calcaneal angle in the dorsoplantar projection is a distally open angle of 30—45 degrees according to *Debrunner*, and 35—40 degrees according to *Thomasen* (1941). In a club-foot the calcaneus is under the talus, and the angle between their longitudinal axes, the talo-calcaneal angle, decreases with the degree of severity. Thus in an extreme

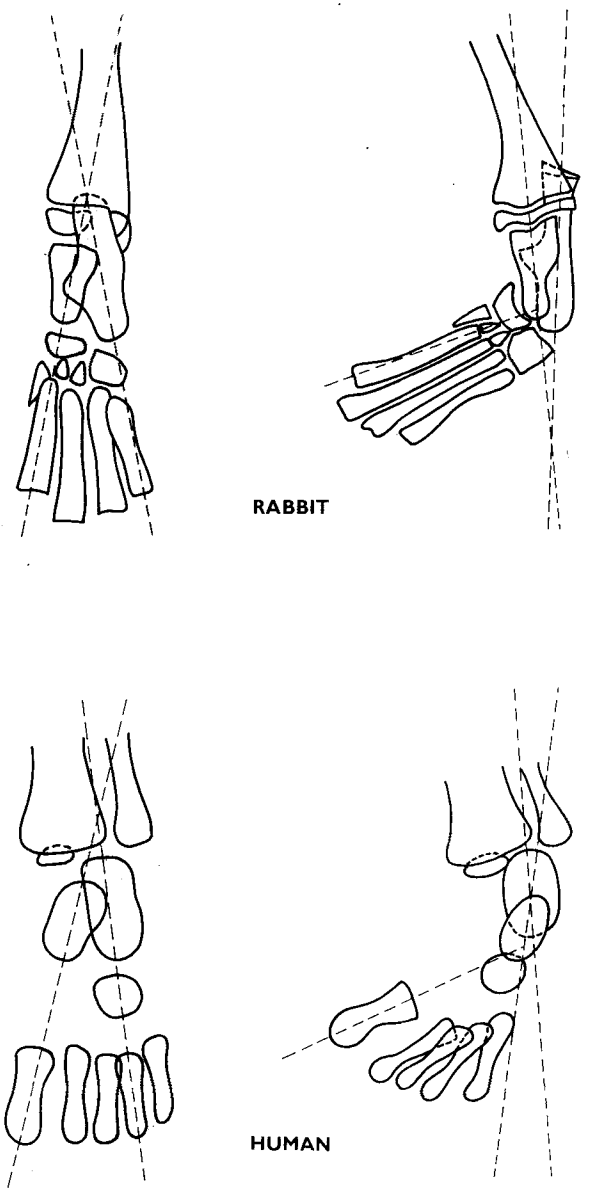


FIG. 10. Top: diagrams of the dorsoplantar roentgenograms of rabbit's normal foot and club-foot; Series XXXII no. 7. Bottom: corresponding diagrams of human feet (*Davis* and *Hatt* 1955). (By courtesy of Radiology.)

Both in rabbit club-foot and human club-foot, the same radiological changes can be found; the talo-calcaneal angle approaches 0° or is reversed, the mid-talar line lies lateral to the normal position, the mid-calcaneal line lies medial to the normal position, the mid-talar line and the line through the shaft of the first metatarsal form an angle.

case the axes are parallel or the angle between them may even be reversed (*Davis* and *Hatt* 1955).

It was possible to measure a similar change of angle between the longitudinal axes of the talus and the calcaneus in the dorsoplantar projection in all experimental talipes equinovarus feet (Figs. 9 and 10).

d) Comparison of changes in the shape of the talus and the joint surfaces

Descriptions in the literature of bony abnormalities of dissected infantile club-feet in humans are in close agreement with each other. They suggest that among the changes in the shape of separate bones the most important is the change in the shape of the talus. *Adams* (1851—1852) found the greatest changes in the neck and head of the talus. The same was noted in later dissection studies (*Kocher* 1878, *Parker* and *Shattock* 1884, *Bissell* 1888, *Bessel-Hagen* 1889, *Burrell* 1893, *Nichols* 1897, *Shevkunenko* 1899, *Böhm* 1929) and described in many other publications (*Mau* 1927, *Kreuz* 1927, *Trèves* 1931, *Virchow* 1933, *Debrunner* 1936). *Irani* and *Sherman* in 1963 dissected eleven extremities with equinovarus deformity recovered from stillbirths or from neonatal deaths and constantly found abnormality in the anterior part of the talus. In his publication of 1963, *Settle* presented the earlier literature on dissections of infantile congenital talipes equinovarus collected by him. He also described his anatomical findings in sixteen dissected infantile club-feet. In the literature dealing with 52 club-feet, he noted in 44 cases the same deformity, severe distortions of the head and neck, as in his own cases. *Reimann* (1967) dissected six fetuses, stillborns and children with club-feet who had died, and found almost constantly skeletal changes affecting the talus.

The main deformity in the bones of club-feet in fetuses was in the neck of the talus which was markedly deviated medially and plantarwards on

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FIG. 11. Top: the talus of a normal foot (R_n) and of a talipes equinovarus foot (R_t) of the same rabbit; Series XXXII no.6 seen from the calcaneal side.

Diagrams of these photographs compared with corresponding diagrams of the tali of human normal foot (H_n) and club-foot (H_t) from the late fetal period according to *Settle* (1963). (By courtesy of J. Bone Jt Surg.)

Similarities between the normal talus and the talus of club-foot in both human and rabbit are marked; the tali of club-feet have an obvious medial rotation of the neck, the articular surface towards the navicular is displaced medially and plantarward in club-feet, the anterior articular surface towards the calcaneus is missing.

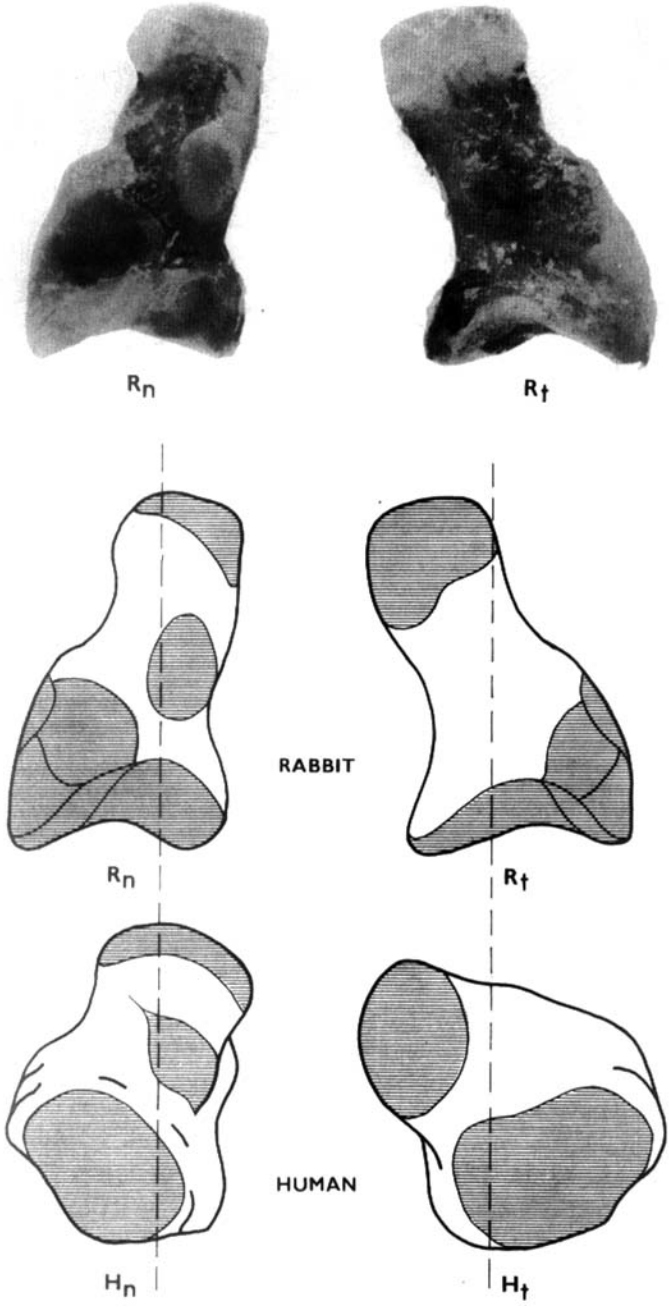


Fig. 11.

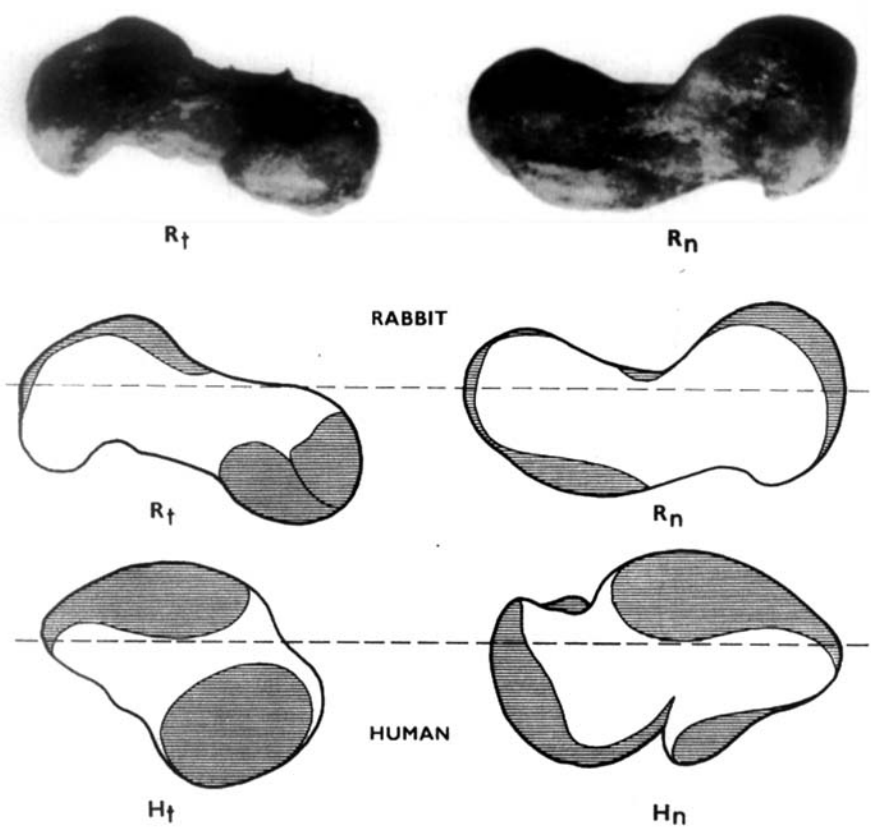


FIG. 12. Top: the medial side of the tali of a rabbit; left from a club-footed specimen (R_t). right: from a normal one (R_n); Series XXXII no. 6.

The centre pictures are diagrams of these tali.

The bottom illustrations are corresponding diagrams of tali in human congenital equinovarus foot (H_t) and in human normal foot (H_n) (*Settle* 1963). (By courtesy of J. Bone Jt Surg.)

In this direction the similarities between the normal talus and the talus of club-foot in both human and rabbit are also obvious; both tali of human and rabbit club-foot have the same kind of plantar deviation of the neck and medial displacement of the articular surface towards the navicular.

the body of the talus. The angle between the long axis of the neck and the long axis of the body of the normal talus is 150—155 degrees, while in a club-foot it is 115—135 degrees (*Irani* and *Sherman* 1963). Upon the deformed and deviated neck of the talus, the talo-navicular articular surface displaces still further medially and plantarwards increasing the deformity even more. The subtalar articular surfaces fuse together in a

single distorted articular surface which faces medially in internal rotation and equinus (*Settle* 1963).

The same pathological changes as in the talus of an infantile club-foot have also been noted in these macro-anatomical studies of experimental club-foot in rabbits. The degree of deformity in the talus was directly proportional to the degree of equinovarus deformity. The more extreme the equinovarus position, the greater the medial deviation of the neck of the talus and the medial displacement of the talonavicular articular surface (Figs. 11 and 12).

The navicular in experimental equinovarus feet is regularly smaller than in a normal foot in the same rabbit. This has also been noted in fetal club-feet (*Irani* and *Sherman* 1963, *Settle* 1963).

Changes in the shape of other bones and articular surfaces of club-feet in both humans and rabbits are smaller. The calcaneus and cuboid remain almost normal as the literature has suggested (*Irani* and *Sherman* 1963, *Settle* 1963). In extreme experimental equinovarus feet, however, these bones show greater changes corresponding to the deformity.

e) Comparison of changes in soft tissues

There are very few good anatomical studies on soft tissue changes in club-foot deformity. Clinical observations and those made during operations on club-feet are numerous, but they are partial and separate observations and do not cover all the factors having an effect in these cases. Earlier authors mainly clarified the bone changes and, not until recent decades, have soft tissue changes received the attention they deserve. However, *Little* (1839) had already shown that uncordinated muscle function or abnormal tendon insertions could cause club-foot deformity. Most investigators have studied tendon insertions and found anomalies in them (*Scherb* 1930, *Penners* 1954). *Debrunner* (1957) wrote that these anomalies have been found in almost all the tendons of club-feet.

A general observation in talipes equinovarus specimens in rabbits produced both by means of Achilles tenodesis and by plaster and fixation immobilisation was the displacement of the Achilles tendon insertion to the medial side of the tuber calcanei. On the other hand, the calcaneus, in relation to the talus, rotates into inversion and thus it is difficult to say whether it is a displacement of the tendon insertion or a displacement of the bone in relation to the tendon. A similar Achilles tendon displacement has, however, been observed in many examinations

of club-foot in man (*Flinchum* 1953, *Penners* 1954, *Inclán* 1958). In his studies *Stewart* (1951) frequently found this tendon insertion anomaly which strengthens the supinating effect of the Achilles tendon.

When analyzing the gross talipes equinovarus specimens in rabbits, it was always noticeable that supinating muscle-tendon structures; the Achilles tendon, the tibialis anterior and "tibialis posterior" muscles and the flexor digitorum muscles, were shortened and taut compared with the normal foot of the same rabbit. These changes occurred in experimental deformities produced by Achilles tenodesis and by transection of the peroneal muscles and the flexor digitorum longus muscles or in club-foot deformities produced by immobilisation into the equinovarus position. In club-foot in man, the Achilles tendon contracture is a constant phenomenon. From his investigations *Stewart* draws the conclusion that the triceps surae muscle, the tibialis anterior muscle and the short plantar muscles are positively deforming forces, while the peroneal muscles are insufficient and do not prevent the foot from turning towards equinovarus deformity. Other authors have also placed emphasis on either the contracture or fibrosis and shortening of the tibialis posterior muscle (*Mau* 1930, *Fried* 1959) or the abnormal resistance to eversion of the tibialis anterior muscle (*Flinchum* 1953). Recently *Attenborough* (1966) has confirmed the finding of small muscle bellies and shortness of the gastrocnemius, soleus, tibialis posterior and flexor digitorum longus muscles in stillborn infants with club-feet.

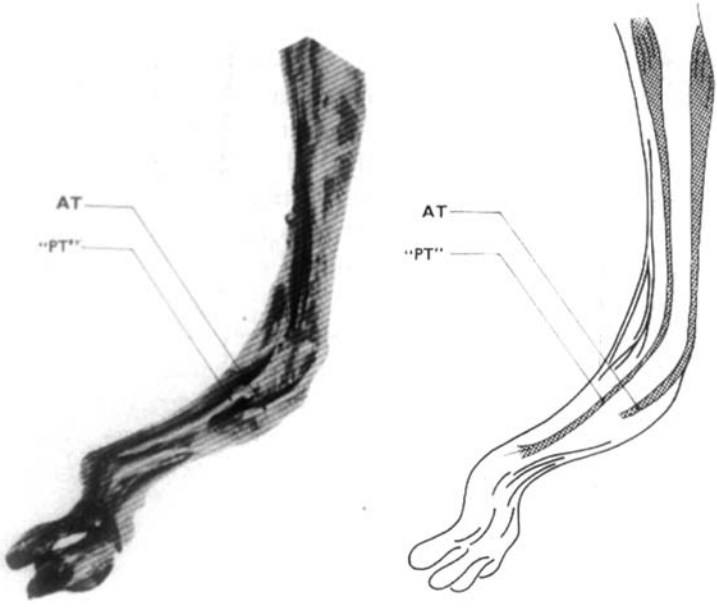
In the experimental talipes equinovarus specimens, it has been possible to observe similar shortening and contractural tightening of the Achilles tendon, the tibialis anterior and posterior muscles, and the flexor digitorum longus muscles as described in the literature (Fig. 13).

FIG. 13. Top left: a picture of a rabbit club-foot; Series LVIII no. 7; top right: a diagram of the same picture.

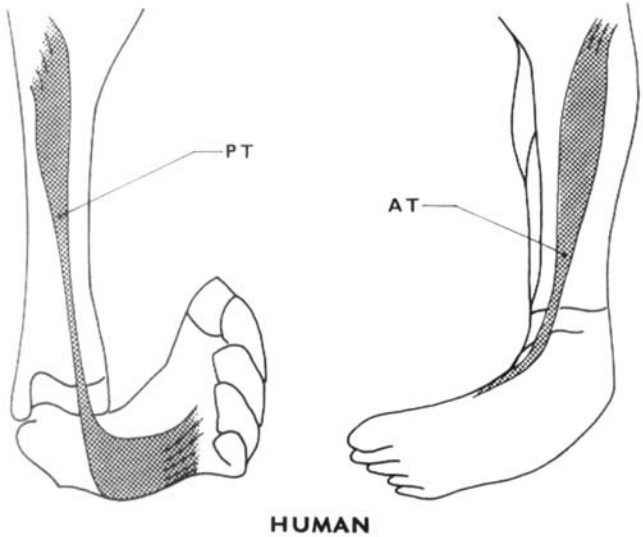
Bottom: illustrations taken from the literature showing human fetal club-foot specimens. Bottom left: club-foot of an eight-month-old fetus showing the tightened and shortened tendon of the tibialis posterior (PT) muscle and its longer tendinous part (*Mau* 1930). (By courtesy of Arch. orthop. Unfall-Chir.)

Bottom right: a club-foot of a six-and-a-half month old fetus showing the same change in the tibialis anterior muscle (AT) (*Flinchum* 1953). (By courtesy of J. Bone Jt Surg.)

Experimentally the same changes in the supinator and adductor muscles; the tibialis anterior (AT), the extensor digiti primi ("PT") (in rabbit homologous perhaps to the tibialis posterior in man) and the flexor digitorum longus, could also be found in all club-foot specimens. These muscles offered abnormal resistance of eversion.



RABBIT
Medial view



HUMAN

f) Micro-anatomical comparison

Bernbeck (1954) has presented micro-anatomical studies on talipes equinovarus specimens of fetuses in which a marked bone deformity was clearly seen. The greatest change was in the talonavicular joint, where the navicular had shifted medially and plantarward around the talus. The fore-foot and cuboid also shifted with the navicular.

In all three micro-anatomical studies of vertical sections of experimental talipes equinovarus deformities in rabbits, a similar striking bone change could be found (Fig. 14).

In these experiments on rabbits as well as in *Bernbeck's* studies, molding of the head of the talus and the navicular can also be seen. The neck of the talus has deviated medially, the navicular articulation with the talus has an inclination towards the medial surface of the foot and the talonavicular and the calcaneocuboid joints have turned medially.

FIG. 14. Top left: a micro-anatomical section in the frontal plane of a club-foot in fetus (*Bernbeck* 1954—1955). (By courtesy of Ferdinand Enke Verlag, Stuttgart).

Top right: the same section of a club-foot of a rabbit; Series XXVIII no.1.

Bottom: corresponding diagrams.

See text above.

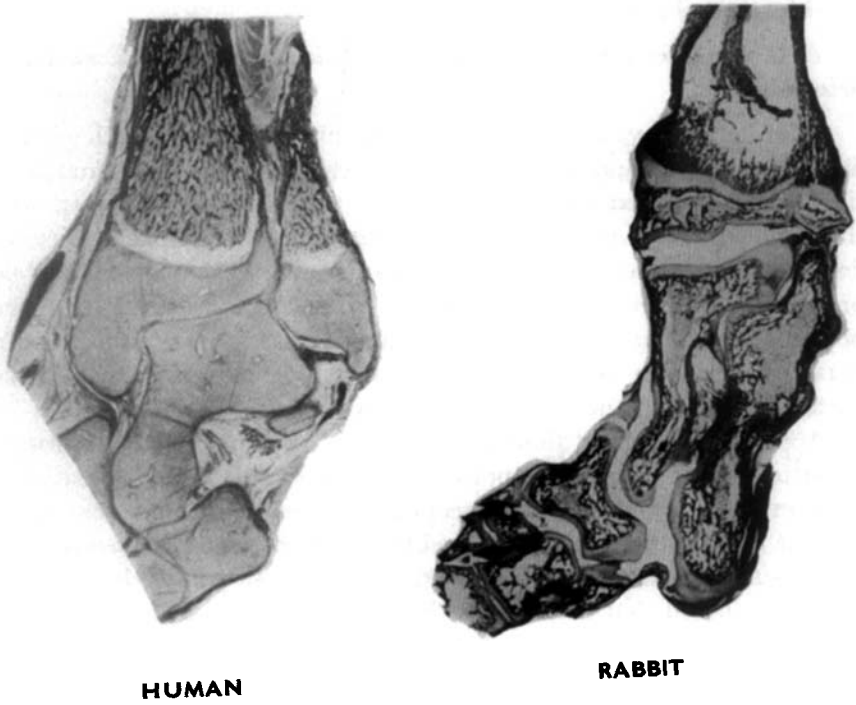

HUMAN
RABBIT
TIBIA
TALUS
CALCANEUS
NAVICULAR
CUBOID
TIBIA
TALUS
CALCANEUS
NAVICULAR
CUBOID

Fig. 14.

B. VERTICAL TALUS

1. General description of deformity produced and factors leading to deformity

In order to provoke vertical talus deformity, fixation of both extensor digitorum longus and tibialis anterior muscles or tendons through the tibia was performed on the ventral side as previously explained while the Achilles tendon was fixed on the dorsal side of the tibia. Progressive deformity with the talus turning towards a vertical position was not produced, however, although the sole of the foot assumed a "rocker bottom" form. In some series, the plantar fascia and short plantar muscles were also severed, but this merely emphasised the "rocker bottom" form without producing a vertical talus.

Accidentally while aiming specifically at equinovarus deformity, a vertical talus deformity appeared as a sequel of the combination of fixation of the Achilles tendon, section of the extensor digitorum longus and the tibialis anterior muscles and the ligamentum transversum cruris. This result was surprising. However, when analysing animals in this series it was noted that discision of the extensor digitorum longus and the tibialis anterior muscles had led to refixation and contracture on the front side of the talocrural joint at the point of discision. At a later stage, resections were performed instead of tendon-muscle discisions. On the basis of a selective search for deforming factors, the following simple combination was arrived at: *fixation of the Achilles tendon in equinus to the tibia dorsally, and resection of the ligamentum transversum cruris.* This produced progressive vertical talus deformity in the growing rabbit. (Table 4).

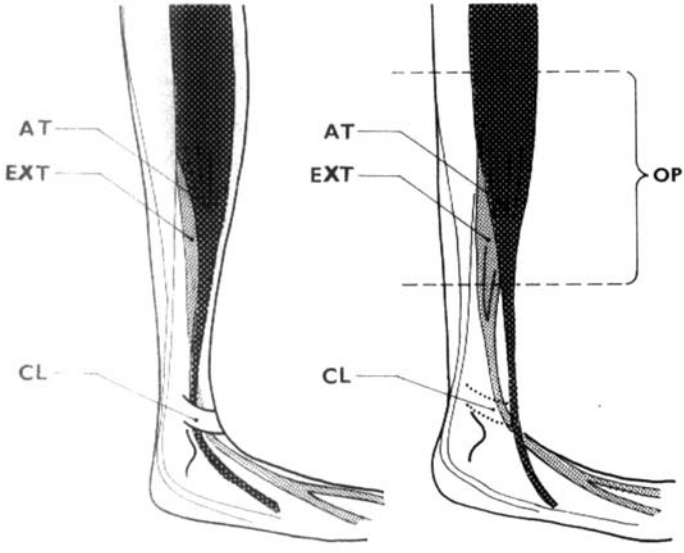


FIG. 15. Left: the normal position of the tibialis anterior muscle (AT), and the extensor digitorum communis longus muscle (EXT) in a rabbit; right: the position after division of the ligamentum transversum cruris (CL). OP: the operation level of the transections of the muscles and tendons.

Table 4. Structural vertical talus deformity produced in rabbits by various procedures.

Series	Rabbit	Operation	Remarks
XL	No. 1	$A_F + L_T$	Good Achilles tenodesis, tibialis anterior and extensor digitorum muscles contracture
—»—	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
—»—	No. 4	—»—	—»—
XLVIII	No. 1	—»—	—»—
—»—	No. 2	—»—	—»—
LVII	No. 8	—»—	—»—
XXXI	No. 1	$A_F + E_T + L_T$	Good Achilles tenodesis, and tibialis anterior muscle contracture
LII	No. 1	—»—	—»—
—»—	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
—»—	No. 4	—»—	—»—
—»—	No. 5	—»—	—»—
—»—	No. 6	—»—	—»—
XLI	No. 2	$A_F + TA_T + L_T$	Good Achilles tenodesis, and extensor digitorum muscle contracture
—»—	No. 3	—»—	—»—
L	No. 1	—»—	—»—
—»—	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
—»—	No. 4	—»—	—»—
XXIX	No. 5	$A_F + P_T + L_T$	Good Achilles tenodesis, tibialis anterior and extensor digitorum muscles contracture
XL	No. 5	—»—	—»—
LIII	No. 4	—»—	—»—
LVII	No. 1	—»—	—»—
—»—	No. 4	—»—	—»—
XXVI	No. 7	$A_F + E_T + P_T + L_T$	Good Achilles tenodesis, tibialis anterior muscle contracture
XXVII	No. 1	—»—	—»—
XXVIII	No. 5	—»—	—»—

Table 4. Continued.

Series	Rabbit	Operation	Remarks
XLIX	No. 4	$A_F+E_T+P_T+L_T$	Good Achilles tenodesis, tibialis anterior mucle contracture
XLIV	No. 1	$A_F+TA_T+P_T$	Good Achilles tenodesis, extensor muscle attached
LIV	No. 4	A_F+E_T	Good Achilles tenodesis, tibialis anterior muscle attached
—»—	No. 5	—»—	—»—
—»—	No. 6	—»—	—»—
XXIX	No. 1	$A_F+TA_T+P_T+L_T$	Good Achilles tenodesis, extensor digitorum muscle contracture
XXIX	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
XLI	No. 6	—»—	—»—
—»—	No. 7	—»—	—»—
LI	No. 2	—»—	—»—
—»—	No. 2	—»—	—»—
XXIII	No. 1	$A_F+E_T+TA_T+L_T$	Good Achilles tenodesis, not resections, but discisions, dorsiflexor muscles reattached
—»—	No. 2	—»—	—»—
—»—	No. 3	—»—	—»—
—»—	No. 6	—»—	—»—
LVIII	No. 6	$K_E+K_V+L_T$	Achilles tendon and dorsiflexor muscles contracture
LXI	No. 2	K_E+L_T	—»—
—»—	No. 3	—»—	—»—
LXIV	No. 2	—»—	—»—
XII	No. 3	Fixation in calcaneovarus position	—»—
—»—	No. 4	Fixation in calcaneovalgus position	—»—
—»—	No. 5	—»—	—»—

Macroscopic analysis of the vertical talus specimens thus obtained led to the conclusion that sufficiently tight fixation of the Achilles tendon to the tibia causes the calcaneus to turn downwards in plantar flexion. At the same time it is clear that the talus also turns more vertically.

Severing of the ligamentum transversum cruris causes the tibialis anterior and extensor digitorum longus muscles to become insufficient at first, but later they shorten and begin to tighten (Fig. 15; p. 48). In addition, *the tendons of the extensor digitorum longus adhere to the dorsum pedis and the muscle effect is transferred from the toes to the tarsus.* Similarly the pull of the tibialis anterior muscle is exerted more directly towards the insertion point at the base of the first metatarsal. In operations on several children with vertical talus deformity at the Orthopaedic Hospital of the Invalid Foundation, it was noted that the tendons of the extensor digitorum longus in these cases adhered to the dorsum pedis in the same way (*Langenskiöld* 1964), thus causing a transference of muscular pulling effect from the toes to the forepart of the tarsus, as in the rabbits with the experimentally produced deformity.

Vertical talus deformity also developed *when either the extensor digitorum longus or the tibialis anterior muscles were resected in addition to sectioning of the ligamentum transversum cruris and fixation of the Achilles tendon.* The two forms of vertical talus thus obtained differ from each other. If the tibialis anterior is resected, a deformity arises with dorsiflexion, pronation and abduction in the distal part of the foot (Fig. 20; p. 55), the changes occurring mainly at the mid-tarsal joint. If in turn the extensor digitorum longus muscles are resected, a slight supination towards dorsiflexion in the distal part of the foot and a tendency to varus position are provoked (Fig. 18; p. 54). Vertical talus deformity produced merely by discision of the ligamentum transversum cruris and fixation of the Achilles tendon are predominantly of the first type, the extensor digitorum longus and the peroneal muscles strength overcoming the effect of the tibialis anterior. When the children with vertical talus deformity seen at the Orthopaedic Hospital of the Invalid Foundation were examined, it was possible to distinguish both these vertical talus deformity types (*Langenskiöld* 1964).

In the so-called "tibialis anterior" type (Figs. 17 and 18; p. 54) the tibialis anterior tendon is a tightened and shortened element on the anterior side of the ankle, while in the so-called "extensor digitorum" type (Figs. 19 and 20; p. 55) the extensor tendons and the peroneal tendons are contracturally tightened and shortened.

It is a fact of great interest that when tension of the Achilles tendon, which is produced by evident myostatic contracture of the gastrocnemius and soleus muscles secondary to plaster immobilisation alone, was combined with a myostatic dorsiflexion contracture produced by transection of the ligementum transversum cruris, a vertical talus deformity also developed (Fig. 16).

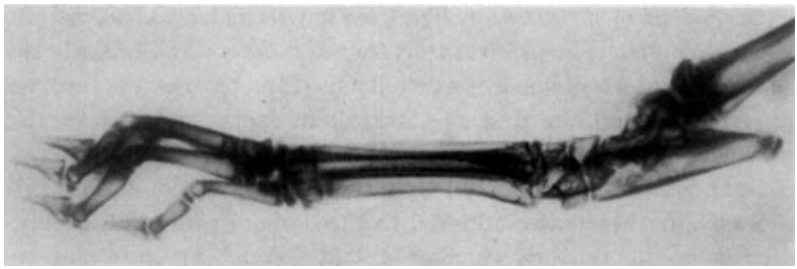


FIG. 16. Vertical talus deformity in a rabbit, Series LXI no. 3, produced by plaster immobilisation and transection of the ligamentum transversum cruris. The sole is convex, the calcaneus is in the equinus position, the fore-foot is in dorsiflexion and the talus is in a clearly vertical position.

2. Comparison of experimental vertical talus in the rabbit with vertical talus in man

a) General comparison

Though the clinical picture should cause suspicion of the vertical talus deformity, it is the radiograph which is characteristic. Therefore the general comparison of both the "tibialis anterior" and the "extensor digitorum" types in children and rabbits is presented by means of X-ray pictures.

In the lateral projection in both types in rabbits and in man, the sole is convex, the forepart of the foot is in dorsiflexion and the talus points downwards into the sole of the foot. The calcaneus is carried into the equinus position with the talus. The navicular does not articulate with the head of the talus but faces the dorsal surface of its neck (Figs. 17 and 19).

In the anteroposterior projection the "tibialis anterior" type has a slight supination of the forepart of the foot while the "extensor digitorum" type has clear pronation (Figs. 18 and 20).

There are remarkable similarities in the general shape of the bones and the relative positions of separate bones in children and in rabbits.

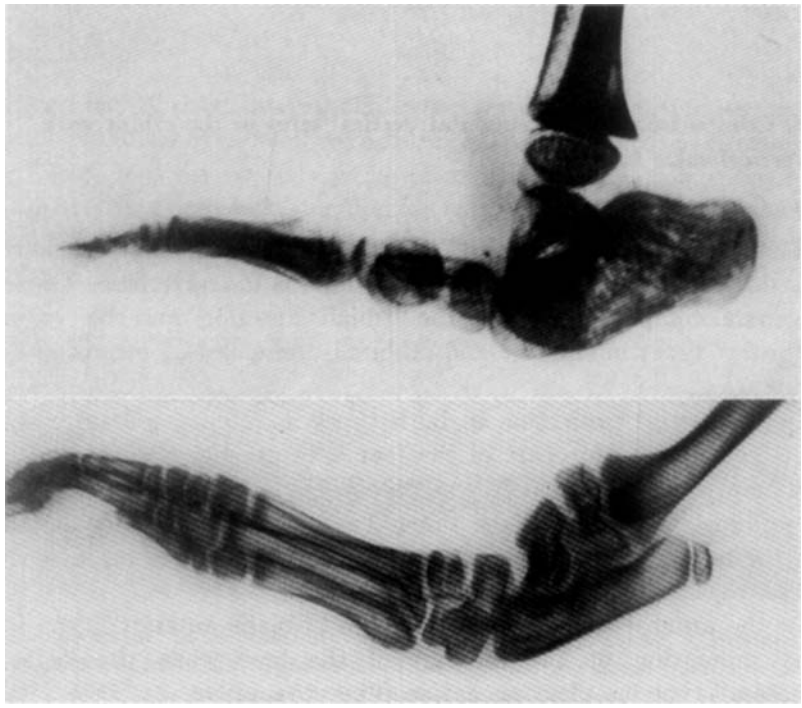


FIG. 17. Lateral roentgenogram of a vertical talus, the "tibialis anterior" type. Top: deformity in a child; bottom: in a rabbit; Series LII no. 1.

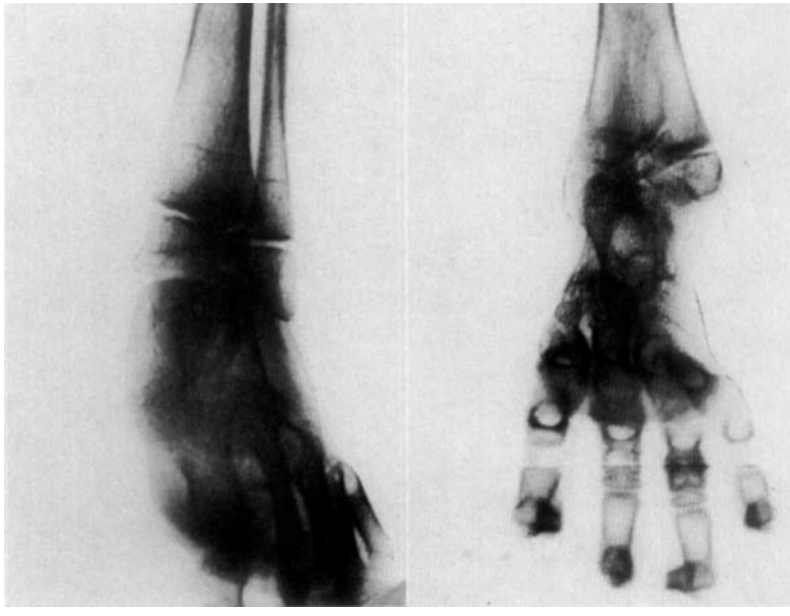


FIG. 18. Anteroposterior roentgenogram of the same vertical talus deformity in a child and in a rabbit as in Fig. 17.

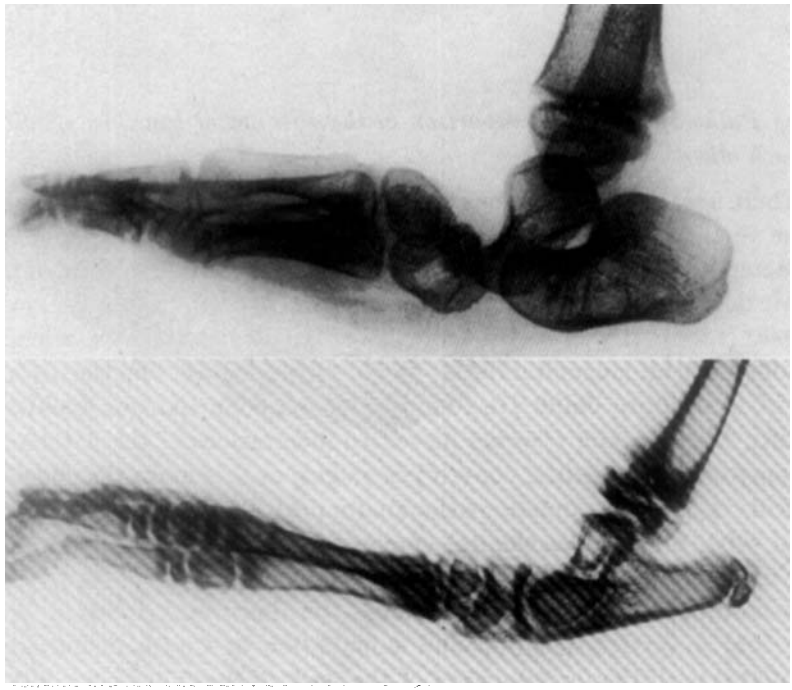


FIG. 19. Lateral roentgenogram of a vertical talus, the "extensor digitorum" type. Top: deformity in a child; bottom: in a rabbit; Series I, no. 1.

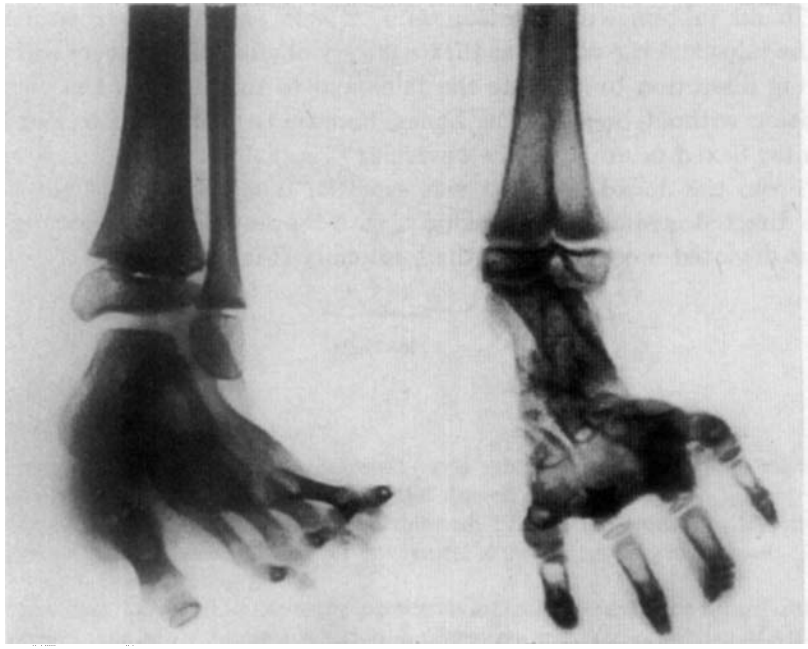


FIG. 20. Anteroposterior roentgenogram of the same vertical talus deformity in a child and in a rabbit as in Fig. 19.

b) Patho-anatomical comparison of the position of bones in relation to each other

There are few investigations dealing with the gross-anatomical changes in the vertical talus deformity. Usually only the clinical and radiological characteristics of the deformity have been described. The only detailed description from earlier times is, according to *Güntz* (1939), that of *Küstner* (1880). It was not until 1939 that *Güntz* carefully examined the pathology of the congenital flat-foot deformity by dissecting the deformed feet of a stillborn child. He found the same bone changes as *Küstner*. In 1963, *Herndon* and *Heyman* presented illustrations of the pathological anatomy of the congenital convex pes valgus, as they term the congenital vertical talus. A complete patho-anatomical study of a six-week-old infant with bilateral congenital convex pes valgus has been published very recently (*Patterson, Fitz* and *Smith* 1968).

According to earlier examinations, the downward rotation of the talus into a vertical position and subluxation of the navicular, which articulates with the dorsal aspect of the neck of the talus locking it in a plantar flexed position, are essentials in the bone relationships of congenital flat-foot or congenital convex pes valgus deformity.

In all rabbits with vertical talus, it was exactly this relationship of the talus and the navicular that was very obvious. It was very difficult during dissection to separate the talus and to turn it from the vertical position without breaking the bones, because the talus was locked in a plantar flexed position by the navicular (Fig. 21).

From the dorsal aspect it was essential that the head of the talus was directed medially and inferiorly, with the result that the axis of the talus deviated more medially than normally (Figs. 22 and 23).

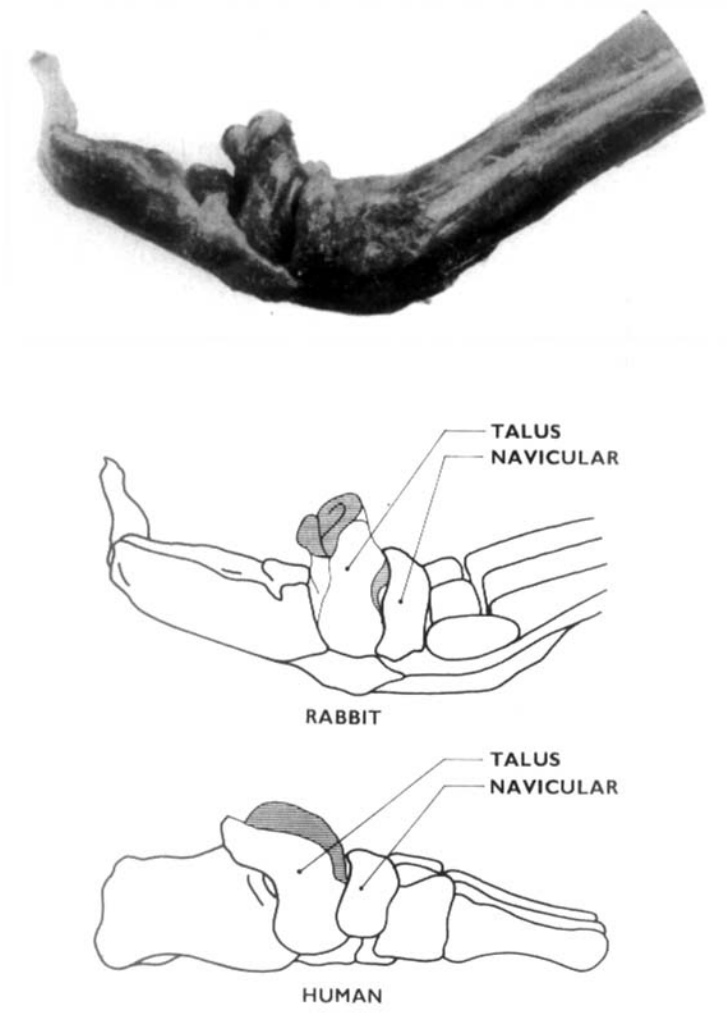


FIG. 21. Top: the vertical talus deformity in a rabbit, Series XL no. 2, showing the bone relationships seen from the medial aspect.

Centre: a corresponding diagram; bottom: an illustration from a publication by *Herndon* and *Heyman* (1963) of congenital convex pes valgus in man. (By courtesy of J. Bone Jt Surg.)

In both man and rabbit the same phenomena can be found; downward rotation of the talus into a vertical position and subluxation of the navicular which articulates with the dorsal aspect of the neck of the talus, locking it in a plantar flexed position.

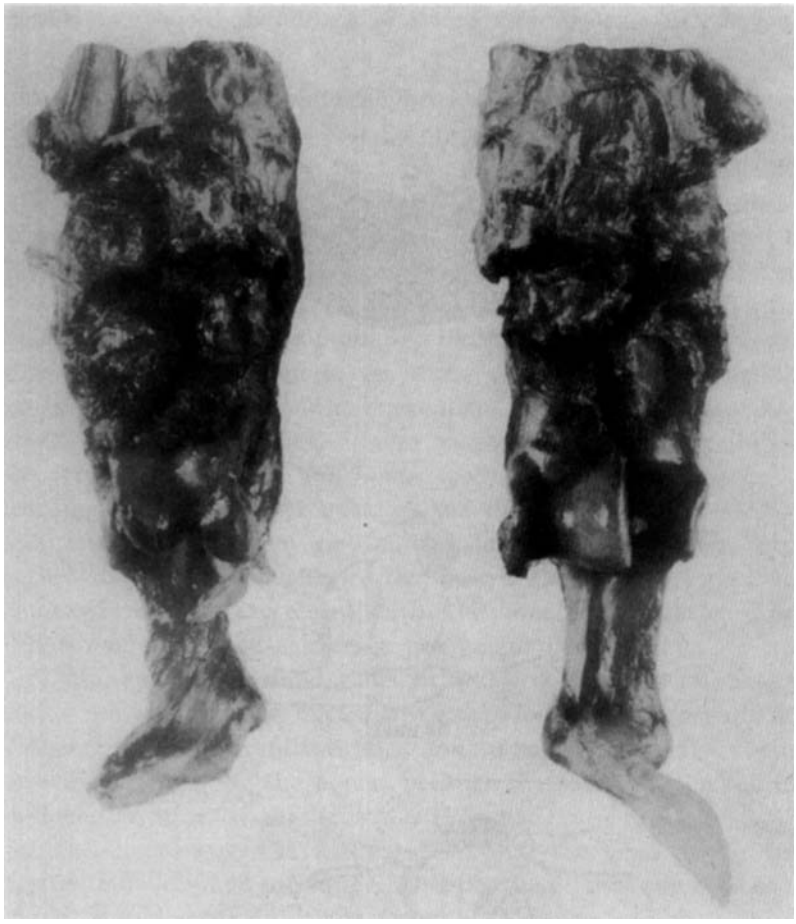


FIG. 22. Patho-anatomical pictures of the dorsal aspect of vertical talus deformity (left) and (right) normal foot in a rabbit; Series XL, no. 2. Tibia has been removed. In vertical talus deformity the long axis of the talus deviates medially and the neck has been turned medially and plantarward.

FIG. 23. Top: diagrams of Fig. 22. Bottom: corresponding illustrations of human "Knickplattfuss" according to Niederecker (1950). (By courtesy of Z. Orthop.)

In both human and rabbit there is a similar medial deviation of the long axis of the talus in the deformed foot.

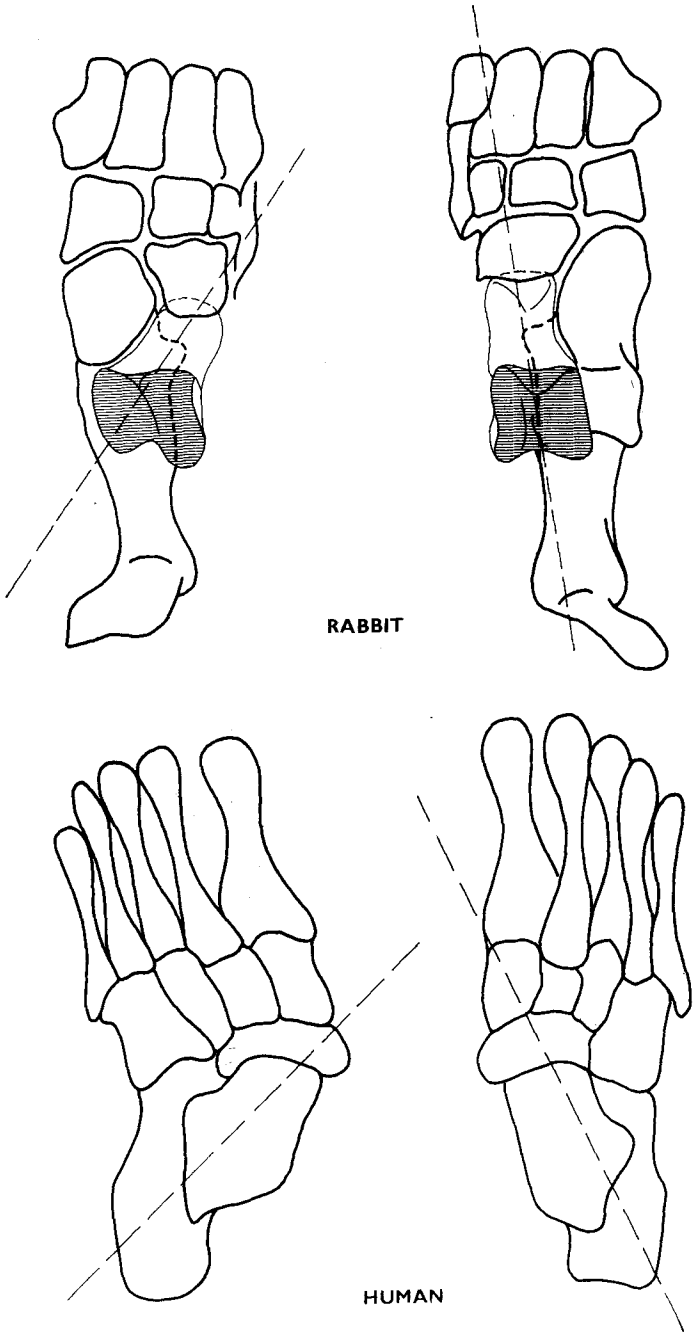


Fig. 23.

c) Radiological comparison of the talo-calcaneal angle

In order to estimate and compare the X-ray examinations of the foot objectively, it is necessary to draw axes of the bones and to measure the angles formed by them. Changes in the angle formed by the longitudinal axes of the talus and the calcaneus (the talo-calcaneal angle) are characteristic of the congenital vertical talus and also of the congenital club-foot. *Güntz* (1939) has made a thorough radiological comparison of various foot deformities. He has found that the talo-calcaneal angle both in lateral and dorsoplantar projections in all flat-foot deformities is wider than normal. *Hüner* (according to *Niederecker* 1959) has measured the talo-calcaneal angle in the lateral projection of the normal foot at a minimum of 20 degrees while it can be 90 degrees in congenital flat-foot. *Siegmund* reported (according to *Hohmann* 1951) the talo-calcaneal angle in the lateral projection to be 50—90 degrees in congenital flat-foot and 28 degrees in normals.

Radiographs taken in the dorsoplantar projection reveal medial displacement of the distal part of the talus. This radiographic finding is similarly described by *Osmond-Clarke* (1956), *Lloyd-Roberts* and *Spence* (1958), *Haveson* (1959), *Outland* and *Sherk* (1960), *Herndon* and *Heyman* (1963). Apart from *Güntz*, other German authors have also observed the increase in the talo-calcaneal angle in the dorsoplantar projection. According to *Hohmann*, the talus in congenital flat-foot can be directed transversally to the calcaneus which is in the direction of the longitudinal axis of the foot. In clinical material *Hüner* has found the talo-calcaneal angle in the dorsoplantar projection to be about 20 degrees in normals, but much wider in congenital flat-foot up to 45 degrees. On the other hand, *Stören* (1967) claims that the talo-calcaneal angle is reduced in the dorsoplantar projection in congenital convex pes valgus with vertical talus, but not in all cases.

In rabbits with vertical talus, an increase in the talo-calcaneal angle both in the lateral and the dorsoplantar projection in comparison with the normal foot could be found (Figs. 24, 25 and 26).

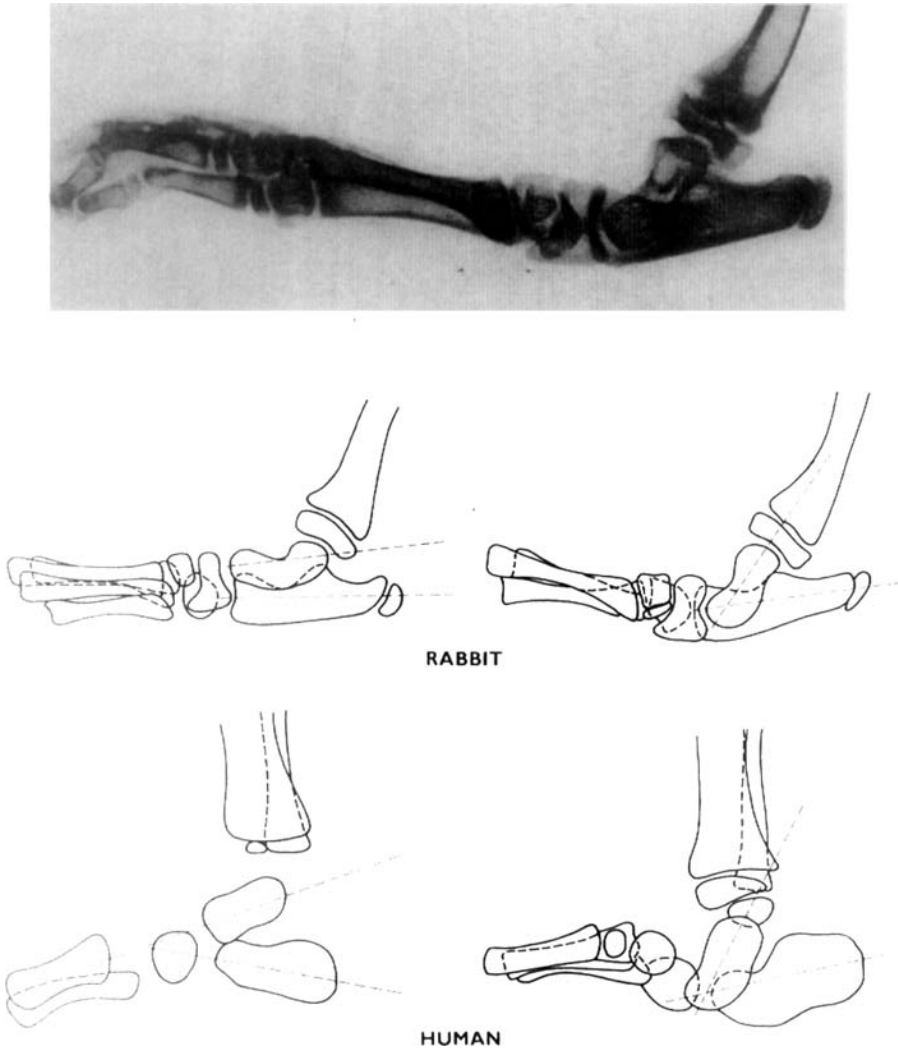


FIG. 24. Top: lateral roentgenogram of a vertical talus foot in a rabbit; Series L, no. 1.

Centre right: diagram of the same foot; centre left: diagram of the rabbit normal foot.

Bottom right: diagram of human congenital flat-foot; bottom left: diagram of human normal foot from a publication by *Davis* and *Hatt* (1955). (By courtesy of Radiology)

The diagrams show an increased talo-calcaneal angle in the deformed feet on the lateral projection both in man and rabbit.

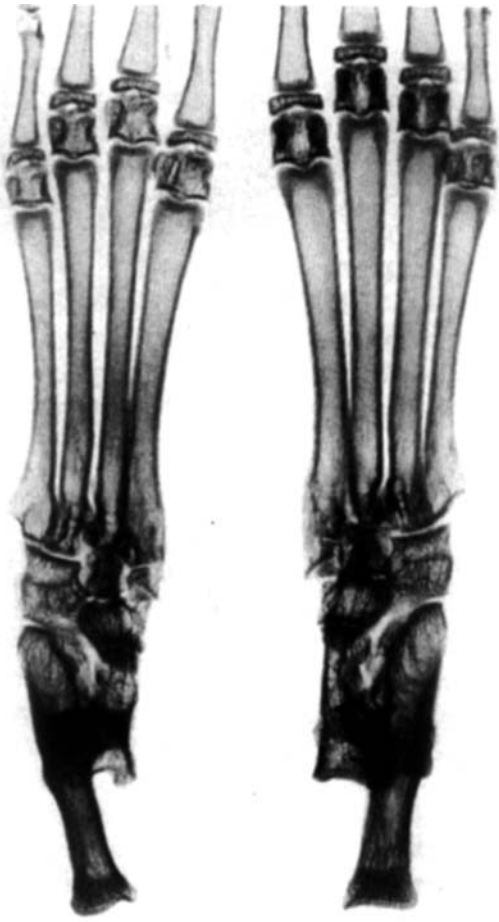


FIG. 25. Left: dorsoplantar roentgenogram of a vertical talus foot in a rabbit of the "tibialis anterior" type: Series LII no. 2. Right: the normal foot of the same rabbit.

FIG. 26. Top: diagrams of Fig. 25.

Bottom: corresponding illustration of a congenital flat-foot in man (left) and a normal foot (right) from a publication by *Davis* and *Hatt* (1955).

(By courtesy of Radiology)

The diagrams show an increased talo-calcaneal angle in the deformed foot in the dorsoplantar projection both in man and rabbit.

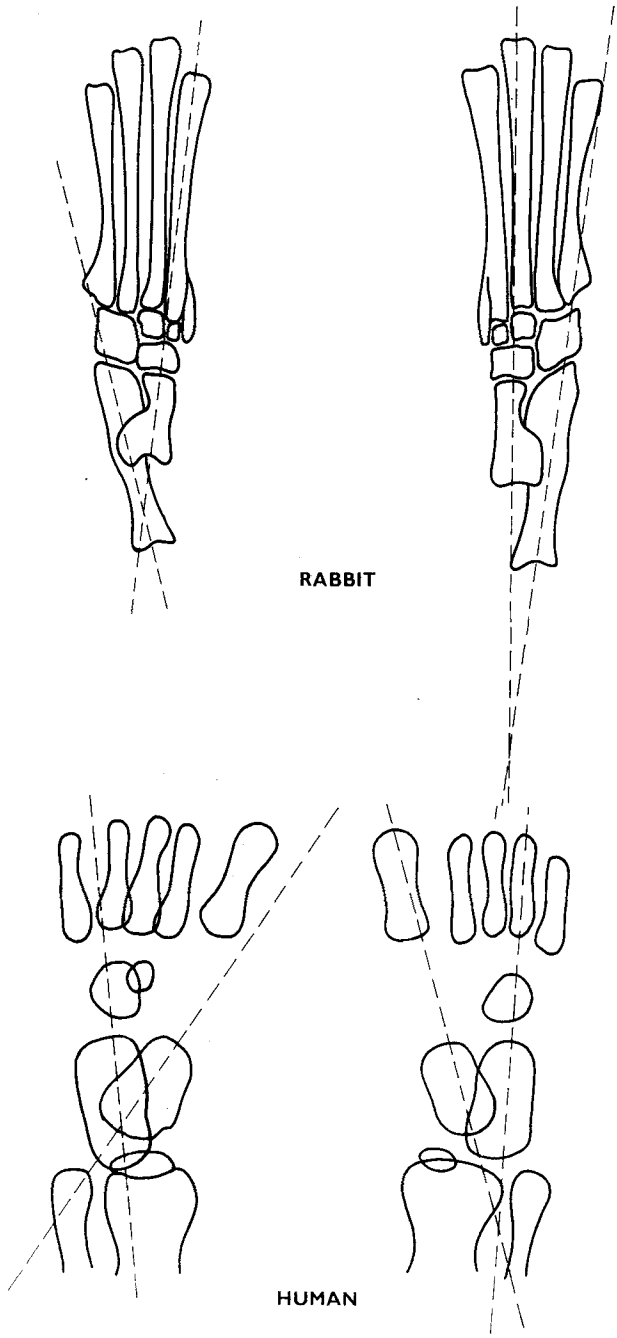


Fig. 26.

d) Comparison of changes in the shape of the talus and the joint surfaces

In vertical talus experimentally produced in rabbits, separate tarsal bones were also macroscopically examined and compared with the tarsal bones of the healthy foot of the same rabbit. Then it was found that the most obvious changes were in the talus. The smaller changes found in the calcaneus and the navicular were in conformity with the changes in the talus.

The studies on rabbits showed that the talus of the deformed foot was generally slightly smaller than that of the normal foot. The joint surface of the talus which articulates with the distal end of the tibia looked as if it had been moved backwards as a result of the vertical position of the talus which allows only the posterior part of the superior surface of the talus to articulate with the tibio-fibular mortise. This articulation has also been found in man and has been described by *Osmond-Clarke* (1956) and *Haveson* (1959). In vertical talus deformity in rabbits, the articular surface of the talonavicular joint is displaced over the caput tali on to the neck of the talus, as a result of the dislocation of the talonavicular joint (Fig. 27). Navicular articulation with the dorsal aspect of the neck of the talus has been found by many investigators in their clinical observations (*Hughes* 1957, *Lloyd-Roberts* and *Spence* 1958, *Haveson* 1959, *Herndon* and *Heyman* 1963). *Güntz* (1939) has also stated that the joint surface of the talus, which articulates with the navicular, projects dorsally and laterally. The distal head of the talus in vertical talus deformity in rabbits was flattened superiorly and pointed seen from the medial side (Fig. 28). *Güntz* has also found this in his examination of the pathology of flat-foot in a stillborn child. The same phenomen has been confirmed by *Patterson, Fitz* and *Smith* (1968) in their dissection of an infant with congenital convex pes valgus (Fig. 28).

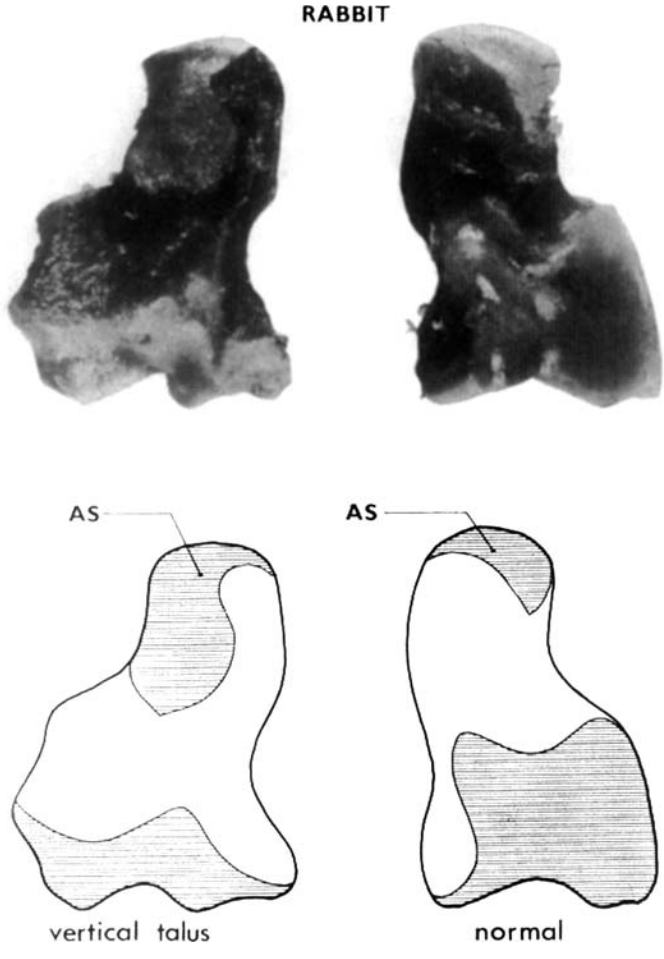


FIG. 27. Top: dorsal view of the tali of the vertical talus foot and normal foot in the same rabbit; Series XL no. 4.

Bottom: corresponding diagrams. The shaded areas are articular surfaces. AS = articular surface of the talonavicular joint. This surface is displaced over the caput tali on the neck, which deviates medially in the talus of the deformed foot. Many authors have observed this phenomenon, but no comparable pictures were to be found in the literature.

Haveson (1959) says for example: "A dislocation of the talo-navicular joint occurs, resulting in an abnormal articulation of the navicular with the superior surface of the neck of the talus".

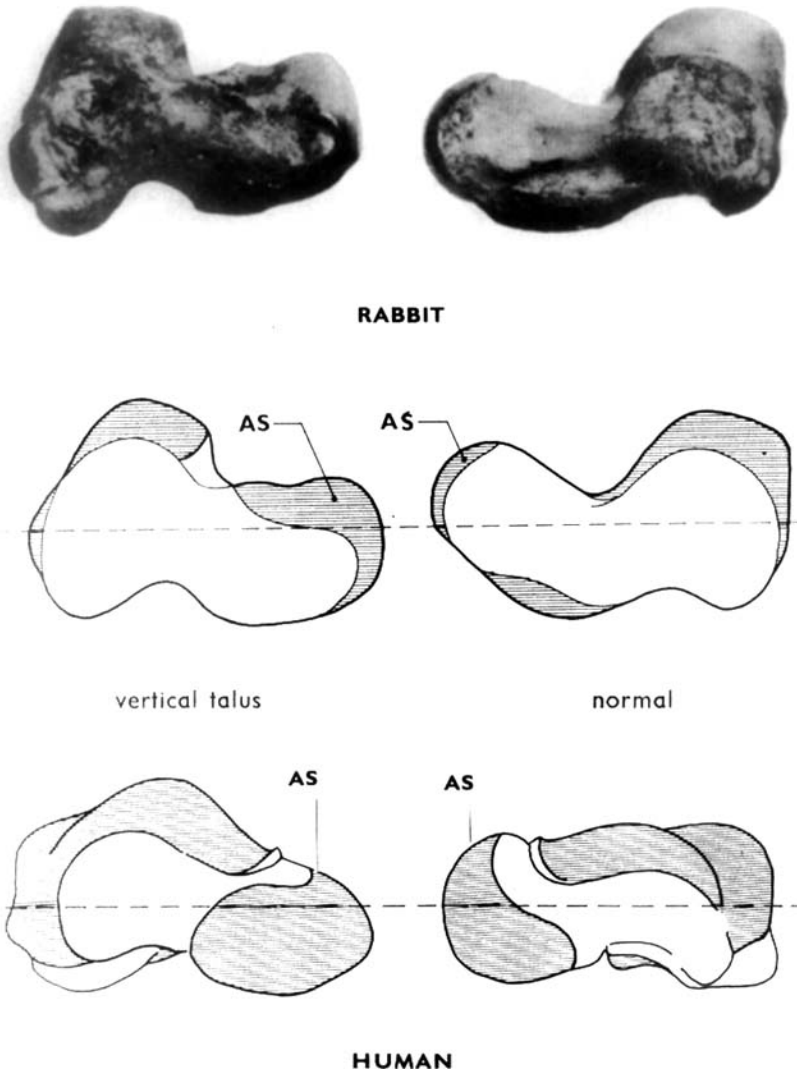


FIG. 28. Top: medial view of the tali of the vertical talus foot and normal foot in the same rabbit; Series XL no. 4.

Diagrams of these photographs compared with corresponding diagrams of the tali of human vertical talus and normal foot from a publication by *Patterson, Fitz* and *Smith* (1968). (By courtesy of J. Bone Jt Surg.)

The shaded areas are articular surfaces. AS = articular surface of the talonavicular joint. In this projection the similarities between the human and rabbit deformed tali are obvious; the talonavicular joint surface is medially displaced and the caput tali is deformed.

e) Comparison of changes in soft tissues

In order to clarify possible deforming factors in experimentally produced vertical talus deformity, muscle and tendon structures were examined macroscopically. It was found that the equinus position of the hindpart of the foot and the Achilles tendon contracture or shortening could be developed either by Achilles tenodesis or by plaster or external fixation of the os calcis in the equinus position.

Many earlier investigators have observed the Achilles tendon shortening or contracture in vertical talus deformity in children (*Haveson* 1959, *Herndon* and *Heyman* 1963, *Patterson, Fitz* and *Smith* 1968). Many authors have also suggested an elongation of the tightened Achilles tendon in operative reduction of the congenital vertical talus deformity (*Rocher* and *Pouyanne* 1934 *Hark* 1950, *Hohmann* 1951, *Outland* and *Sherk* 1960, *Mead* and *Anast* 1961).

According to *Güntz's* patho-anatomical examination of the vertical talus deformity, the Achilles tension did not seem to be noteable in the deformed position, but when the lateral dislocation of the calcaneus was reducted to the normal position, the Achilles tendon had to be tenotomized because the lateral fibres tightened and thus prevented reduction.

In one group of rabbits with experimental vertical talus deformity, tension of extensor and peroneal tendons caused a dorsiflexion position of the fore-foot. This was called the "extensor digitorum" type. In most cases described in the literature, the dorsiflexion contracture has been observed in precisely the extensor digitorum longus and peroneal tendons, (*Mead* and *Anast* 1961, *Herndon* and *Heyman* 1963), and many authors found that a transection of the tibialis anterior tendon was not indicated, or proved useful if performed (*Osmond-Clarke* 1956, *Lloyd-Roberts* and *Spence* 1958, *Herndon* and *Heyman* 1963).

In another group of rabbits with vertical talus deformity, tibialis anterior and extensor digitorum longus tendon tension could be found. The literature also contains reports of similar contractures in the vertical talus deformity in man. (*Hark* 1950, *Outland* and *Sherk* 1960, *Patterson, Fitz* and *Smith* 1968).

In the third group of rabbits with vertical talus deformity, the essential dorsiflexion contracture was found in the tibialis anterior tendon. This group was called the "tibialis anterior" type. *Hughes* (1957) has stated that in the congenital vertical talus in man there are also contractures of the anterior soft parts, particularly the tibialis anterior muscle. However, German writers in particular have put emphasis on

the tibialis anterior contracture as a deforming factor in "der angeborene Knickplattfuss", as they term the congenital vertical talus. *Güntz* (1939) made an exact patho-anatomical study of the congenital flat-foot and found the tibialis anterior tendon a factor that hindered the plantarflexion of the fore-foot. It tightened like a string from the leg to the dorsum of the foot at the same time as the ligamentum cruciatum cruris had moved more proximally fusing with the ligamentum transversum cruris (Fig. 29). According to *Güntz*, the extensor digitorum longus tendons also tighten strongly in the plantarflexion position of the fore-foot. On the basis of extensive material consisting of operated flat-feet, *Niederecker* (1950) emphasized that functional factors or muscle anomalies are more important than bone factors. He considered that the hypertrophy and contracture of the tibialis anterior muscle strengthened the development of the flat-foot deformity. He also noted that the insertion of the tibialis anterior tendon, very significant in flat-foot deformity, had moved forwards to the first metatarsal bone. *Patterson, Fitz* and *Smith* (1968) described also the "bowstringing of the anterior tibial tendon" (Fig. 29).

In rabbits in which the peroneal muscles were preserved, contractural shortening could be observed, especially in the "extensor digitorum" type. *Güntz* (1939) has referred to the contracture of the peroneus brevis muscle and its deforming effect in man. *Herndon* and *Heyman* (1963) have also noticed the contracture of both peroneal muscles. During the operative reduction of the congenital vertical talus feet of children at the Orthopaedic Hospital of the Invalid Foundation, the peroneus tertius muscle and its contracture and deforming effect have often been found (*Langenskiöld* 1964). *Niederecker* (1950) has also observed the occurrence and the importance of this muscle in flat-foot deformity.

But even if the muscle and tendon contractures in vertical talus experiments were released, it was not possible to correct the deformity and the position of the talus until the ligamentous contractures were released. In rabbits, it was found that the most important factors that hindered the correction of the vertical talus deformity were the talonavicular ligament, the talocalcaneal interosseum ligament and especially the tibionavicular ligament. Many authors have paid attention to the significance of these ligamentous factors in the congenital vertical talus deformity during the operative reduction of this deformity (*Hark* 1950, *Osmond-Clarke* 1956, *Mead* and *Anast* 1961, *Herndon* and *Heyman* 1963, *Stören* 1967).

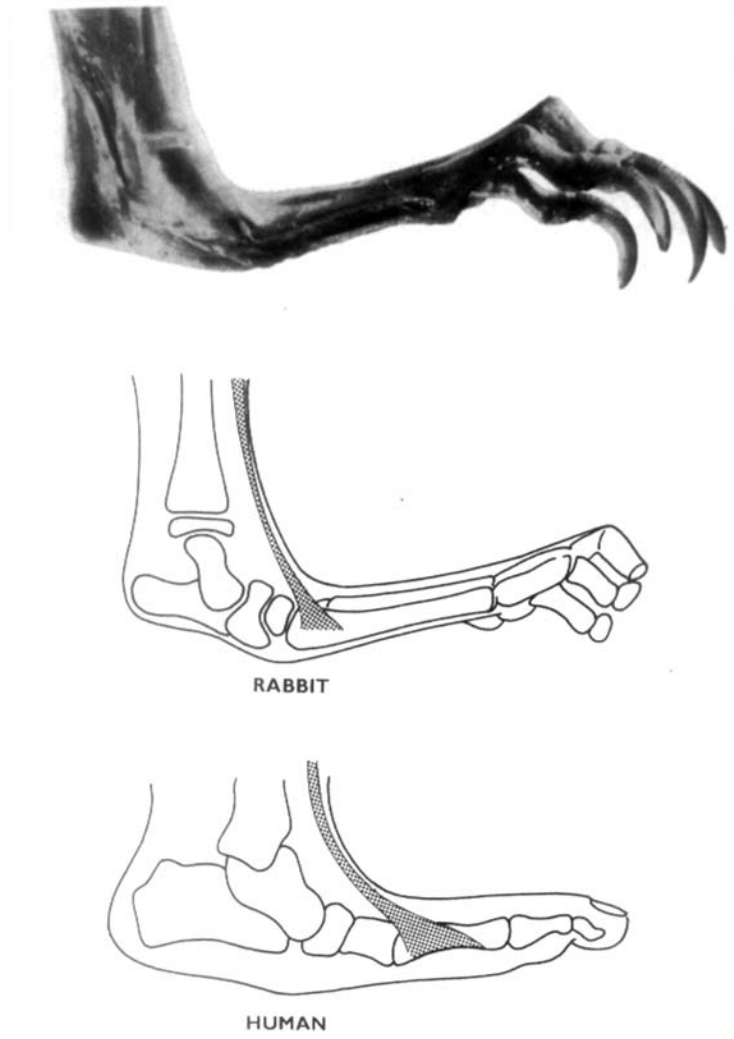


FIG. 29. Top: rabbit vertical talus foot specimen; Series LVII no. 8. Centre: diagram of the same picture.

Bottom: corresponding diagram of human "Knickplattfuss" from a publication by *Niederecker* (1950). (By courtesy of Z. Orthop.)

Both in rabbit and man a similar "bowstringing" and displacement of the effect of the tibialis anterior tendon more anteriorly can be found.

f) Micro-anatomical comparison

Five vertical talus specimens of rabbits were sectioned and examined histologically in the sagittal plane through the talo-navicular joint.

The following similar phenomena in the sagittal plane of both the vertical talus experiments in rabbits and the vertical talus deformity in an infant can be seen in the illustrations (Fig. 30). The navicular has an abnormal articulation with the superior surface of the neck of the talus, the talus rotates toward the plantar surface into a vertical position and the deformed appearance of the head of the talus is demonstrated here as in Fig. 28.

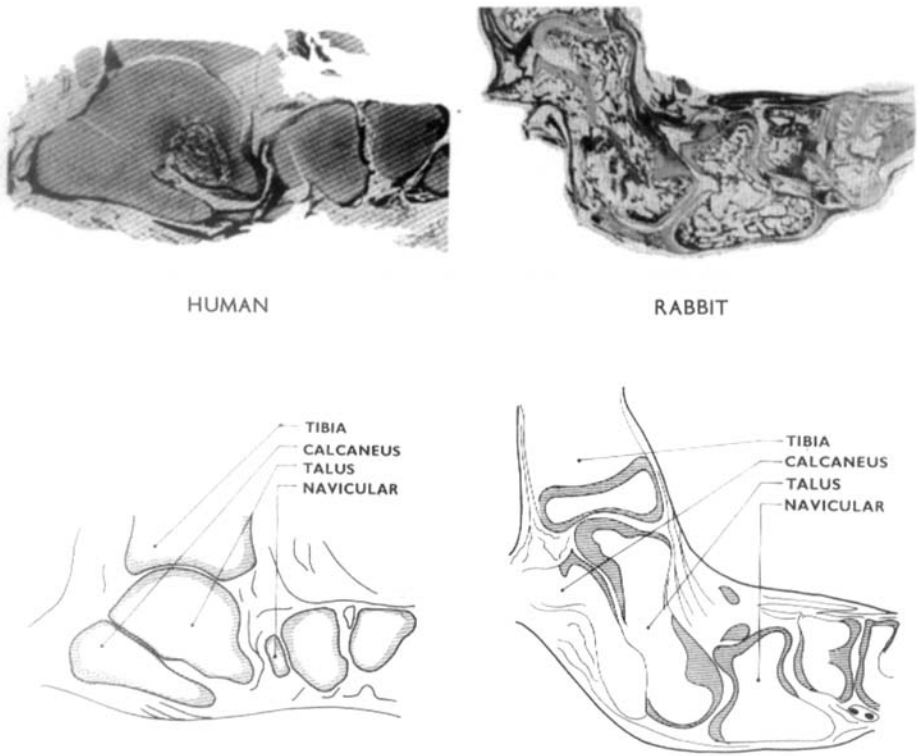


FIG. 30. Top left: a micro-anatomical section in the sagittal plane of a vertical talus in an infant (*Patterson, Fitz* and *Smith* 1968) (By courtesy of J. Bone Jt Surg.)

Top right: the same section of a vertical talus in a rabbit; Series. XL no 1.

Bottom: corresponding diagrams.

See text above.

V. DISCUSSION AND CONCLUSIONS

The purpose of the studies described above was not to determine the unexplained, primary etiology of congenital deformities of the foot, but to analyse the part played by muscle imbalance and by soft tissue factors generally in the pathogenesis of structural bone deformities of the foot. The aim was also to produce in the growing rabbit, by measures performed on soft tissues and through muscle imbalance and contracture, experimental changes which resemble foot deformities seen in humans.

Therefore only the muscle and other soft tissue contractures related to human foot deformities and to animal experiments are discussed. In addition, only the points in common which could be noted when comparing the morphological, radiological, patho-anatomical and especially soft tissue changes of talipes equinovarus and vertical talus produced in rabbits with these deformities in man are discussed. Conclusions have been made on the same bases.

A. MUSCLE IMBALANCE AND SOFT TISSUE CONTRACTURES RELATED TO CONGENITAL FOOT DEFORMITIES IN MAN AND TO ANIMAL EXPERIMENTS.

In connection with paralytic deformities, which include both traumatic deformities and those following poliomyelitis, many observations were made of bone changes arising from changes in the soft tissues. In poliomyelitis, for instance, where imbalance of muscle power is the cause of deformity, restoration of muscle imbalance when effected sufficiently early by appropriate transposition of tendons has often prevented the development of a seriously progressive deformity such as talipes calcaneus or varus, and especially its transformation into a structural bone deformity. In addition, much information on patho-anatomical soft tissue changes in congenital deformities, especially club-foot, has been obtained. The old static conception of bone deformity as a primary condition is yielding to more dynamic thinking, although studies still appear which place bone deformity in the foreground (*Thomasen* 1941, *Doyle*

1961, *Irani* and *Sherman* 1963). An increasing number of recent investigators have directed their attention to soft tissue anomalies in congenital and idiopathic deformities (*Scherb* 1930, *Stewart* 1951, *Flinchum* 1953, *Langenskiöld* and *Michelsson* 1961). *Debrunner* (1957), for instance, has suggested that an essential feature of club-foot is active contracture of the soft tissues caused by disturbed muscle balance.

Close investigation of bone changes in various congenital foot deformities has led to greater uniformity, but observations on soft tissue changes vary. Although contractural changes in them are often observed, the microscopic findings themselves are variable. This is in agreement with the statement of *Ferguson*, *Vaughan* and *Ward* (1957) that histological changes in muscle are notoriously difficult to evalute, and disagreement as to the presence or absence of histological changes runs throughout the literature on disuse atrophy and myostatic contracture. The only difference, microscopically, between the muscles in club-foot deformity and those of a normal foot is that in the former atrophied muscle fibres are found between normal ones (*Mau* 1930) or the average diameter of the muscle fibres is diminished (*Reimann* 1967). This is most noticeable in supinatory muscles. But a similar discovery has been made in poliomyelitis, that is in muscles which have changed through disturbance of the central nervous system, and also in ischemic contractural muscles (*Debrunner* 1957). Thus *Debrunner* has said that congenital club-foot is the brother of paralytic club-foot.

As soft tissue contracture is a conspicuous feature of congenital foot deformity, at least in its later stages, the attempt to produce muscle contracture in the leg of a growing rabbit and thus possibly to imitate congenital foot deformities was of interest. Ischemic contracture seemed suitable for this purpose, but permanent contracture was not obtained by this means. Why was ischemic contracture difficult to achieve? As *Brooks* (1922) has shown in his experimental studies, blockage of the arterial blood circulation of muscles causes either complete necrosis and destruction of the muscle if collateral circulation is insufficient, or preservation of the muscle with normal appearance and functioning if collateral circulation is sufficient. Arterial production of ischemic contracture does not succeed, in fact. Fibrosis and shortening of the muscle do not occur. *Brooks* states, however, that obstruction of the purely venous blood circulation does cause fibrosis and muscle shortening and that extravasation of the red corpuscles and disturbed nutrition of tissues cause changes. In the present tests the production of disturbance in the purely venous blood circulation of rabbit muscles was hardly

successful. *Clark* (1946) has shown, moreover, that endomysium remaining after partial experimental ischemic necrosis has astonishing ability to regenerate the endomysial tubes. It is thus understandable that attempts to produce permanent ischemic muscle contracture were unsuccessful.

Soft tissue contracture which develops during plaster-immobilisation is a well-known clinical phenomenon. Also, experimental studies described in the last century, produced contractures in striated muscles when they were kept shortened for a certain time by methods including moving the muscle's points of attachment nearer to the origin (*Moll* 1886) or severing the muscle tendon to make the muscle contract. A fixation of the muscle then occurs at the length imposed upon it. This contracture is called myostatic, though its pathogenesis has not been fully explained. If immobilisation is interrupted early enough the contracture can be overcome by active or passive movements, but if the shortening of a muscle is maintained for some weeks it cannot be immediately extended without structural damage. It has also been shown that this fixation only occurs when muscle innervation is intact (*Fröhlich* and *Meyer* 1920). The change is more noticeable in growing rabbits than in those of full age when tested (*Crawford* 1954). In growing rabbits muscle growth is so inhibited that shortening remains permanent and full development is never recovered, even if immobilisation ceases (*Alder, Crawford* and *Edwards* 1959). Some writers maintain that a contractured muscle of this kind is histologically normal (*Ranson* and *Sams* 1928, *Alder, Crawford* and *Edwards* 1959), but, as *Ferguson, Vaughan* and *Ward* (1957) remark, the literature contains highly divergent conceptions of the histological changes involved in myostatic contracture. The old physiological law which states that stretched muscles (including fascia and tendons) remain insufficient and that shortened soft tissues contract is applicable to myostatic contracture.

According to the present study, plaster-immobilisation or other fixation in a certain position such as equinovarus causes a permanent postural deformity in the growing rabbit. After immobilisation of shorter duration, the posture and mobility of joints returned to normal and no permanent deformity developed. This makes an interesting comparison with the results obtained by *Ferguson, Vaughan* and *Ward* (1957) in their study of disuse atrophy of muscle in rabbits. There appeared to be no essential histological change after two weeks of immobilisation, but after four weeks a lesion could be recognized. In the present tests the main issue may be the myostatic contracture of soft tissues already described.

Three to four weeks of immobilisation are scarcely long enough for the development of bone changes which would keep the foot in a deformed position after immobilisation is ceased. The explanation of this may be found in muscle-tendon-ligament and/or cartilage changes. In the present tests, at least, radiological observation has so far revealed no morphological bone changes. These appear later if the position of deformity is maintained.

The permanent deformities achieved by the fixation method in the present study are presumably based on a similar influence: they developed when a hind foot was placed in equinus and varus position by means of nylon suture. Tests were performed on 5 day-old rabbits and immobilisation was maintained for several weeks. With several test animals in which the thread came loose in the course of a few weeks, the deformity did not remain permanent but corrected itself within one week.

It is interesting to compare the results of *Spencer* and *Peltier* (1964) when they produced plantar flexion and ulnar deviation of the fore-foot by means of silk thread in a fetal rabbit 6—7 days before birth. The thread was removed on the day of delivery. Eight rabbits born with deformities lived for more than 3 days, and 3 of these lived at least 6 weeks. In all except one, the deformity corrected itself within one week after birth. One rabbit had persistent ulnar deviation, and X-rays of the limb revealed a synostosis of the radius and ulna at the point where the suture had passed through the forearm. In these experiments, in fact, no permanent deformity provoked by a fixed position remained. Apart from the fact that their rabbits were operated on a few days before birth and in present study a few days after, the most noticeable difference is that immobilisation with silk suture lasted only one week in their experiments and over 3 weeks in this investigation. On the basis of my results it was not expected that any permanent deformity would remain, and our results are thus parallel.

B. TALIPES EQUINOVARUS IN MAN AND IN THE RABBIT

1. Morphological changes in bones

Many common points can be noted when the morphological and pathoanatomical bone changes of talipes equinovarus deformities produced in rabbits are compared with analyses of these deformities in man.

If the experimental club-foot deformities produced are examined 2—4 months after operation, changes can be noted in the morphology of individual bones as well as in the positions of bones in relation to

each other. These correspond significantly to the bone changes encountered in recently published reports of careful dissections of talipes equinovarus (*Bechtol* and *Mossman* 1950, *Irani* and *Sherman* 1963, *Settle* 1963, *Reimann* 1967). The principal change is in the neck of the talus, which is deviated medially and plantarward on the body. In addition, the navicular articular surface of the talus faces medially and plantarward. The calcaneus is almost normal in contour, but somewhat smaller than normal in size. The talonavicular and calcaneocuboidal joints are not in the frontal plane as is normal, but turned medially and plantarward. The talo-calcaneal angle is diminished. The navicular is slightly smaller than normal and has moved medially near the medial malleolus in extreme cases. Cuneiforms, cuboides and metatarsals are almost normal in shape and size. The posterior part of the calcaneus is often inclined towards the region of the lateral malleolus, where an accessory joint is formed.

2. Soft tissue changes

When the club-foot deformities produced in rabbits are examined from the functional point of view, changes can be observed similar to those encountered in man. If the Achilles tendon in the rabbit is strongly tightened, it can be clearly seen to exert a supinatory action and to cause a slight varus in the sole of the foot. Mere attachment of the Achilles tendon in a week-old rabbit causes a tendency towards the same phenomenon, which disappears, however, during growth and with use of the foot. No progressive deformity ensues, and further factors are evidently needed for its development in test animals. Mere extensor insufficiency apart from Achilles tension was unable to cause equinovarus deformity in the experiments. Peroneal insufficiency was also required. If severing of the peroneus was combined with fixation of the Achilles tendon, the result was varus and slight adduction of the fore-foot combined with dorsiflexion rather than equinus. In accordance with this are the club-foot dissections of the newborn made by *Adams* 1855. He found changes in the gastrocnemius, tibialis anterior and tibialis posterior muscles. All three muscle bellies were shortened. In 1959 *Wiley* noted the same phenomenon in his anatomical studies where in cases of club-foot among small children muscle bellies were shorter and narrower. The principal muscles affected were the gastrocnemius and soleus, tibialis posterior and tibialis anterior.

In examining experimental equinovarus deformities I observed the same phenomenon as *Stewart* (1951) in his anatomical study of club-foot, that the Achilles tendon seems to attach itself to the medial part of the

calcaneus which was inclined to varus. Muscle imbalance combination leading to equinovarus in rabbit also agrees with *Stewart's* proposition: a positive deforming force + a negative deforming force. He states that the triceps surae, the tibialis anterior and minor plantar muscles exert a positive deforming force, while the peroneus muscles, deprived of their postural activity, cannot function to inhibit the distortion. Except that the combination of fixation of the Achilles tendon, tenodesis or external, transection of peroneus and extensor digitorum longus led to equinovarus deformity it could be noted on the other hand that transection of the tibialis anterior prevented its development. It appeared that the pull of the tibialis anterior was important for the medial dislocation of the navicular and also the beginning of metatarsus varus for after the discision of the tibialis anterior this transference did not occur. The deforming effect of the tibialis anterior in present tests are in accordance with the observations of *Garceau* and *Manning* (1947) on the imbalance between the invertor and evertor muscles. They recommend the transfer of tibialis anterior more laterally in the tarsus in cases of recurring club-foot like many others (*Durbin* 1950, *Critchley* and *Taylor* 1952).

Without going further into the many soft tissue operations which were performed later, it may be mentioned that in 1888 *Vincent-Jackson* announced a successful cure for congenital club-foot immediately after severing the Achilles tendon and the tendons of the tibialis anterior and the tibialis posterior. It was probably *Lorenz* in 1784, however, who first severed the Achilles tendon in order to correct club-foot. Many others have later recommended the use of a soft tissue operations directed at the release of the contractions of the Achilles tendons and the tendons of the tibialis anterior, the tibialis posterior and the flexor digitorum longus and the capsules and ligaments on the medial side of the foot (*Codivilla* 1906, *Bankart* 1922, *Brockman* 1930, *McCauley* 1959, *Pasila* and *Sulamaa* 1961). My animal experiments. are in accordance with these operations.

Observations made by *Bechtol* and *Mossmann* in 1950 on muscle changes after dissection of two club-foot fetuses are also consistent with present experimental results. They support the theory that faulty muscle development is an important link in the pathogenetic chain of congenital club-foot. The gastrocnemius, soleus, tibialis posterior, flexor hallucis longus and flexor digitorum longus muscles showed the presence of many abnormal embryonic muscle fibres. Failure of these muscles to lengthen at the same rate as the bones of the skeleton may be the cause of the inversion of the foot. Failure in development of these muscles may

affect their physiology and ability to stretch and contract normally, and may be a cause of the recurrence of club-foot deformity.

In accordance with the present investigation are the interesting observations by *Flinchum* (1953) on the dissection of a six-and-a-half month fetal club-foot. The contour and relative size of individual bones in the club-foot and the normal foot appeared identical in the fetus at this stage of fetal growth. But the author noted that the Achilles tendon and the tendinous part of the tibialis anterior muscle were longer in the club-foot than in the healthy foot. The tibialis anterior muscle was the only structure which offered abnormal resistance to eversion. The mass of the peroneals on the affected side was only half that on the normal side. He interpreted this as muscular imbalance due to dysplasia of the peroneals.

Hirsch (1960) has noted under operative exposure in his series that in all patients the deltoid ligament was tight, the talus anteriorly displaced and the Achilles tendon medially displaced. In rabbits with equinovarus deformities very tense medial ligamentous contracture could also be found.

C. VERTICAL TALUS IN MAN AND IN THE RABBIT

1. Morphological and radiological changes

When experimental talus verticalis deformities were analysed and compared with clinical observations of *Langenskiöld* (1964), remarkable general morphological similarities were found. In clinical examination of a talus verticalis foot in a child and in a rabbit the shape of the foot is conspicuous. There is a prominence in the sole from which the heel and fore-part of the foot rise in a gentle curve. The general appearance is like a rocker, hence the term "rocker-foot". The head of the talus is prominent on the plantar aspect of the foot. This is both visible and palpable medially. Later a callus develops and sometimes also an ulceration over the prominent head of the talus. When the fore-part of the foot moves into dorsiflexion the distinct convexity is accentuated.

Remarkable similarities have been observed in radiological studies of the skeleton of the foot in children and rabbits with vertical talus. The most important was that the distal portions of the calcaneus and talus rotate towards the plantar surface, the talus rotating into a vertical position. The subtalar joint appear intact with the exceptions that the plane of the joint is more vertical and the talus is further forward on the calcaneus than in the normal foot. There is an abnormal articu-

lation of the navicular with the upper surface of the neck of the talus. In their studies, *Hark* (1950) and *Haveson* (1959) have precisely described this phenomenon and its importance in the development of deformity. There is also a widening of the calcaneocuboid joint and the cuboid is somewhat displaced upward at the calcaneocuboid articulation. The calcaneus is in the equinus position and the posterior part of the heel fails to touch the ground on standing. The anterior portion of the calcaneus deviates in a lateral direction. The calcaneus is generally underdeveloped and is convex on its plantar surface. The long axis of the talus, the keystone of the deformity, is almost in line with the axis of the tibia. This bone becomes narrow at the waist and resembles an hourglass. A moulding of the navicular occurs later.

2. Soft tissue changes

In vertical talus in rabbits the Achilles tendon was tight. In the opinion of some authors this is not always so in vertical talus in man. It has been a relative phenomenon in some cases in our clinical examinations (*Langenskiöld* and *Ritsilä* 1964) at least, and can remain unnoticed in routine examination. In the position of deformity, with the fore-foot in dorsal flexion and abduction, the Achilles tendon is not stretched, but when the position is corrected to normal, the Achilles tendon is seen to tighten so that in these cases, too, Achilles' tautness is present, whether it is a primary or secondary factor.

The fore-foot of rabbits with vertical talus was in dorsiflexion. This was maintained by the tautness of the soft tissue structures ventral to the ankle. Many authors dealing with vertical talus in man have directed their attention to this dorsiflexion contracture (*Hark* 1950, *Lloyd-Roberts* and *Spence* 1958). *Lamy* and *Weissman* (1939) said that the contracted extensor tendons can be felt as prominent and resisting all correction. *Herndon* and *Heyman* (1963) said that the peroneal and extensor tendons may be taut and may resist passive inversion.

Examinations of both rabbits and children with vertical talus reveal, as shown earlier, that dorsiflexion contractures may exist either in the extensor-peroneus group or in the tibialis anterior muscle. Thus it has been possible to distinguish these so-called extensor and tibialis anterior types. Both types have also been encountered later in children at operation (*Langenskiöld* 1964). In tests where neither the extensor digitorum longus muscle nor the tibialis anterior muscle was severed, but change in their action and myostatic contracture were produced by severing

merely the ligamentum transversum cruris, tightening of the extensor group was more severe than the tibialis anterior so that the final result was of the extensor type. This could also be noted immediately after severing the ligament following which the sole was drawn into abduction and valgus. *Herndon* and *Heyman* (1963) said that the foot as a whole is fixed in valgus and cannot be inverted. But *Mead* and *Anast* (1961) wrote that the fore-foot need not be in valgus, as is implied sometimes in the literature. The latter view agrees with present experimental results, in which, to be sure, the extensor type was in valgus in the fore-foot, but the so-called tibialis anterior type showed a tendency towards varus in the fore-foot; this is due to the elevation of the first metatarsal secondary to tightening of the tibialis anterior and transference of the point at which the pull is exerted. *Franke* (1901) has also noted that in congenital flat-foot in man the tendon of tibialis anterior was not inserted on the plantar aspect of the first metatarsal and the first cuneiform but on the dorsal surface.

Closer analysis of experimental specimens and operative findings in children showed further similarities in dorsiflexion contracture. In both of the above categories, adhesions developed in the tarsus and the peroneal and extensor tendons became so fixed that their normally more distal pull had been transferred to the dorsum pedis in the tarsal region.

D. CONCLUSIONS

In this investigation it was possible to produce foot deformities in newborn rabbits phenotypically similar to human congenital foot deformities, especially talipes equinovarus and vertical talus deformities, by means of surgical and non-surgigical, soft tissue procedures. Apart from equinovarus and vertical talus deformities produced in rabbits, other imitations of the foot deformities encountered in children, such as talipes equinus, calcaneus and varus, cavus, calcaneovalgus and metatarsovarus, have also been similarly produced in rabbits by means of operations on soft tissues; a report will be published later.

An attempt was made to elucidate the peripheral mechanisms of foot deformities in experiments and to compare these with observations in humans. It seemed clear that the talipes equinovarus and vertical talus changes produced in experiments were closely related to mechanical factors. The same procedures could lead to the occurrence of either of the deformities. The talus was in the key position in both, and the manner

in which the fore-foot changed position, especially in the talonavicular joint, was decisive. In talus verticalis the fore-foot moved into dorsiflexion, abduction and eversion. In talipes equinovarus it moved into plantar flexion or equinus, adduction and inversion. *Osmond-Clarke* (1956) suggested that club-foot is essentially a talonavicular and subtalar subluxation and vertical talus is a subluxation at the same joints but with displacement in the opposite direction. It is interesting that *Herndon* and *Heyman* (1963), too, were inclined to believe that congenital convex pes valgus or vertical talus „is closely akin to a club-foot in etiology".

The present author has assumed that a peripheral primary fault in different congenital foot deformities, which may differ in their etiology, is of a similar nature. This peripheral fault may lie in the muscles or other soft tissues. The resultant bony deformity was thus assumed to be extrinsic not intrinsic, secondary not primary. The results confirm these assumptions.

Soft tissue contractures and combinations of them resembling those encountered in man have been produced in rabbits. It has also been verified that they cause corresponding structural deformities of the bones. Thus soft tissue changes have exerted a controlling influence on bone growth, evidently following of bone transformation suggested earlier (*Hueter* 1862, *v. Volkmann* 1869, *Wolff* 1892).

VI. SUMMARY

The aim of this study was to produce in young rabbits imitations of the congenital foot deformities encountered in man, by provoking changes in the soft tissues.

A total of 76 separate procedures were performed. Approximately 500 rabbits were used, 402 for actual tabulated procedures and the rest for preliminary tests.

By means of transections and resections of tendons and muscles, tenodeses, transections of ligaments, and fixation in plaster in extreme positions, it was found possible to produce a number of imitations of foot deformities found in humans; those corresponding to talipes equinovarus and vertical talus are described. The age of the rabbits at operation varied from 4 to 28 days, most of them being from 6 to 7 days old.

Marked talipes equinovarus deformity was produced in 32 rabbits, and vertical talus in 51. The course of development of the deformity was followed during growth by repeated radiography. The deformities produced were analyzed by dissection and photography of the feet of 75 animals killed at different ages. Sections for histological examination were taken from 8 animals.

In both a macro-and micro-anatomical and a radiological sense, great similarities were observed between the deformities in rabbits and those encountered in humans.

Talipes equinovarus was produced by operation on the soft tissues by using only a combination of Achilles tenodesis, transection of the extensor digitorum longus muscle and transection of both peroneal muscles. Sufficiently long immobilisation in the equinovarus position, 3—4 weeks, also led to a permanent, even progressive equinovarus deformity. A resemblance in general structure between such deformities in rabbits and humans was noted (Fig. 4; p. 34) including the interrelation of bones of the feet (Figs. 5, 6, 7, 8; pp. 36, 37), the radiological examination (Figs. 9, 10; pp. 38, 39), the changes in the shape of individual bones (Figs. 11, 12; pp. 41, 42), the changes in soft tissues (Fig. 13; p. 45) and the microscopic examination (Fig. 14; p. 47).

Vertical talus deformity was produced most easily by causing simultaneous contracture of the triceps surae muscle group and the dorsiflexor muscles of the foot. Myostatic contracture was caused by plaster or other immobilisation and transection of the ligamentum transversum cruris, or Achilles tenodesis was used instead of the triceps surae contracture provoked by immobilisation in the equinus position. By combining these measures with transections of either the tibialis anterior or the extensor digitorum longus on the dorsiflexor side of the foot, two separate types of vertical talus deformity were produced. These two types have also been discerned in a series of vertical talus feet in children (*Langenskiöld* 1964). Similarities have also been noted in the general structure of experimental vertical talus deformities in rabbits and human vertical talus deformities (Figs. 17, 18, 19, 20; pp. 54, 55) and in the interrelation of bones (Figs. 21, 22, 23; pp. 57, 58, 59). In the same way radiological comparison (Figs 24, 25, 26; pp. 61, 62, 63) and comparison of the shape of the talus (Figs. 27, 28; pp. 65, 66) in relation to descriptions given in the literature show distinct similarities. Muscular and ligamentous contracture of the vertical talus in rabbits and in humans was found to be very similar (Fig. 29; p. 69). A resemblance was also noted in the microscopic comparison (Fig. 30; p. 70).

The results of the present study confirm the importance of considering primary soft tissues changes as a factor provoking secondary skeletal deformities.

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APPENDIX

Seven series of pictures describing development and progression of the talipes equinovarus and vertical talus deformities produced by various procedures in newborn rabbits.

In each picture from left to right: normal foot in lateral projection, deformed foot in lateral projection, normal foot in dorsoplantar projection and deformed foot in dorsoplantar (or antero-posterior) projection.

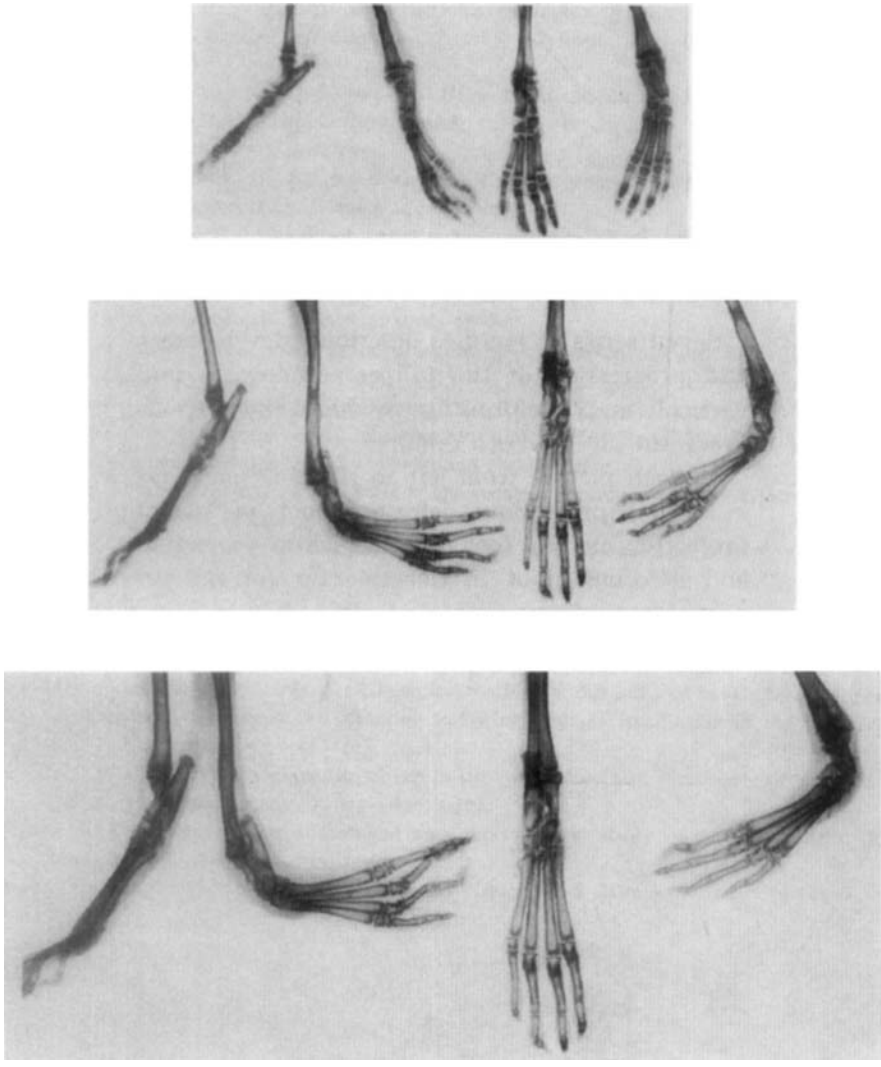

FIG. 31. Development and progression of the talipes equinovarus deformity in a rabbit; Series XXVI no. 10.

Top: lateral and dorsoplantar roentgenograms one week after Achilles tenodesis, transections of the extensor digitorum longus and peroneal muscles, and transection of the ligamentum transversum cruris.

Centre: six weeks; bottom: six months after operation.

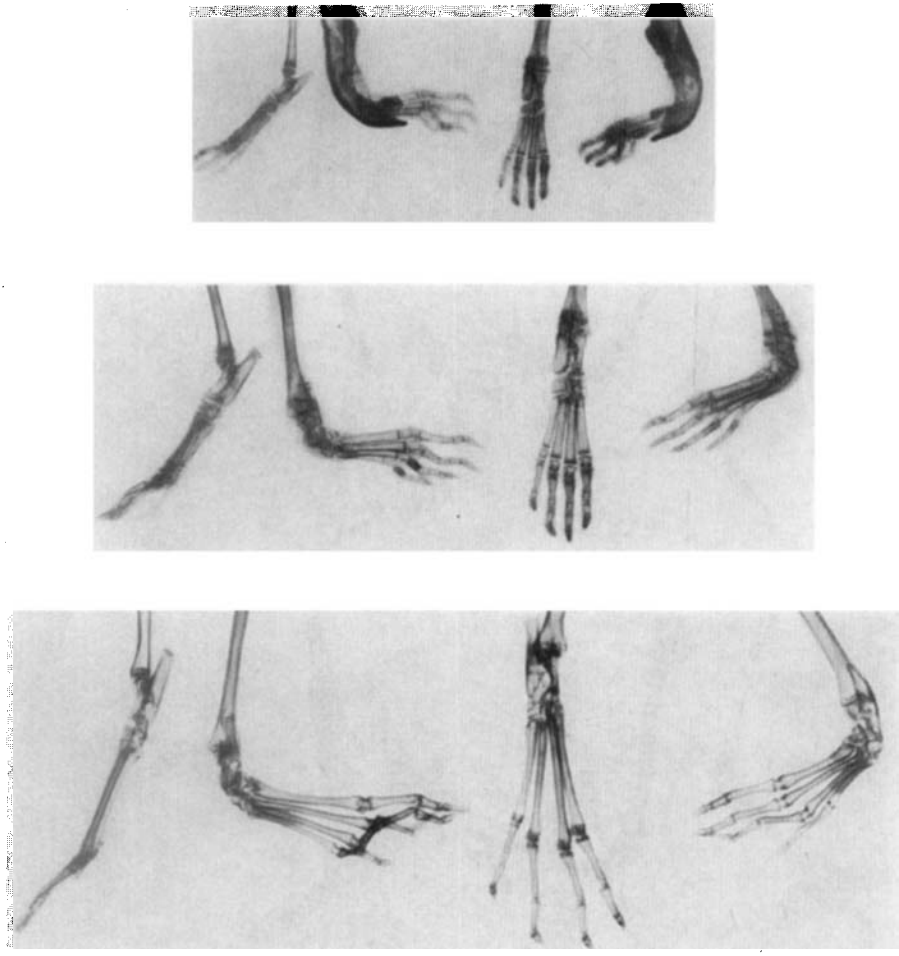

FIG. 32. Development and progression of the talipes equinovarus deformity in a rabbit produced by plaster immobilisation in equinovarus position; Series LIX no. 1.

Top: lateral and dorsoplantar roentgenograms one week after immobilisation.

Centre: six week; bottom: six months after immobilisation, immobilisation removed.

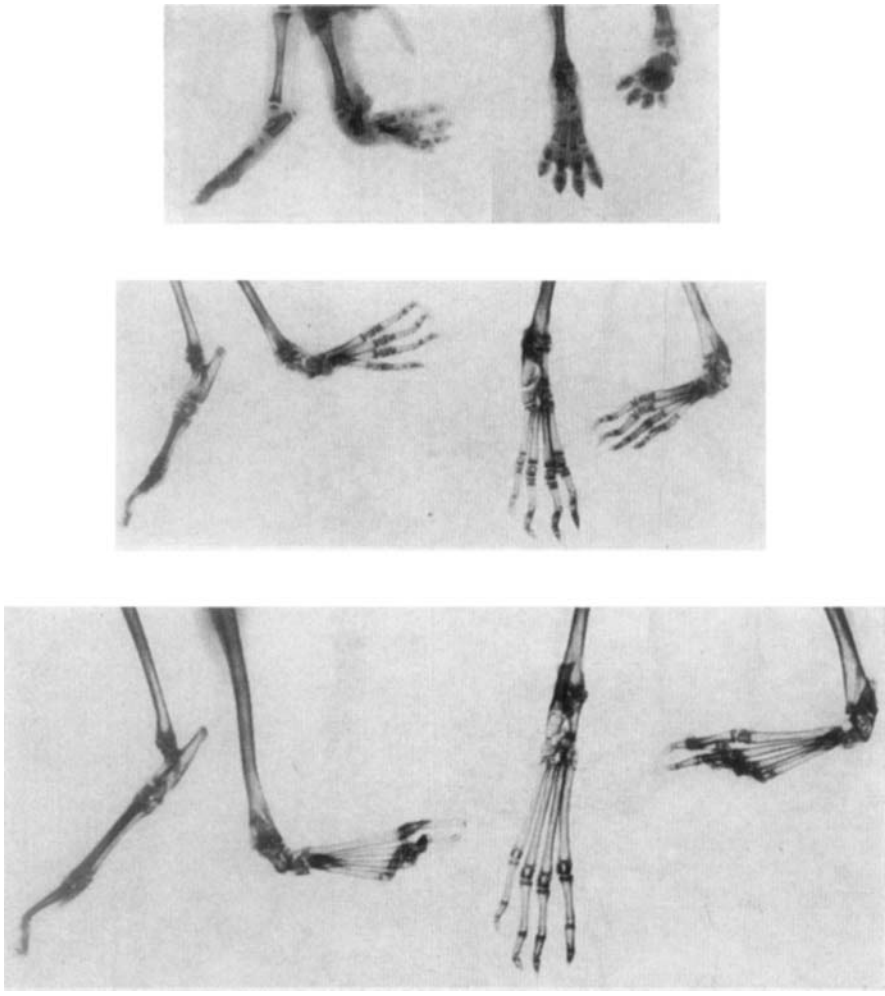

FIG. 33. Development and progression of the talipes equinovarus deformity in a rabbit produced by fixation with nylon thread in equinovarus position; Series XII no. 7.

Top: lateral and dorsoplantar roentgenograms one week after fixation.

Centre: six weeks; bottom: six months after fixation, fixation removed.

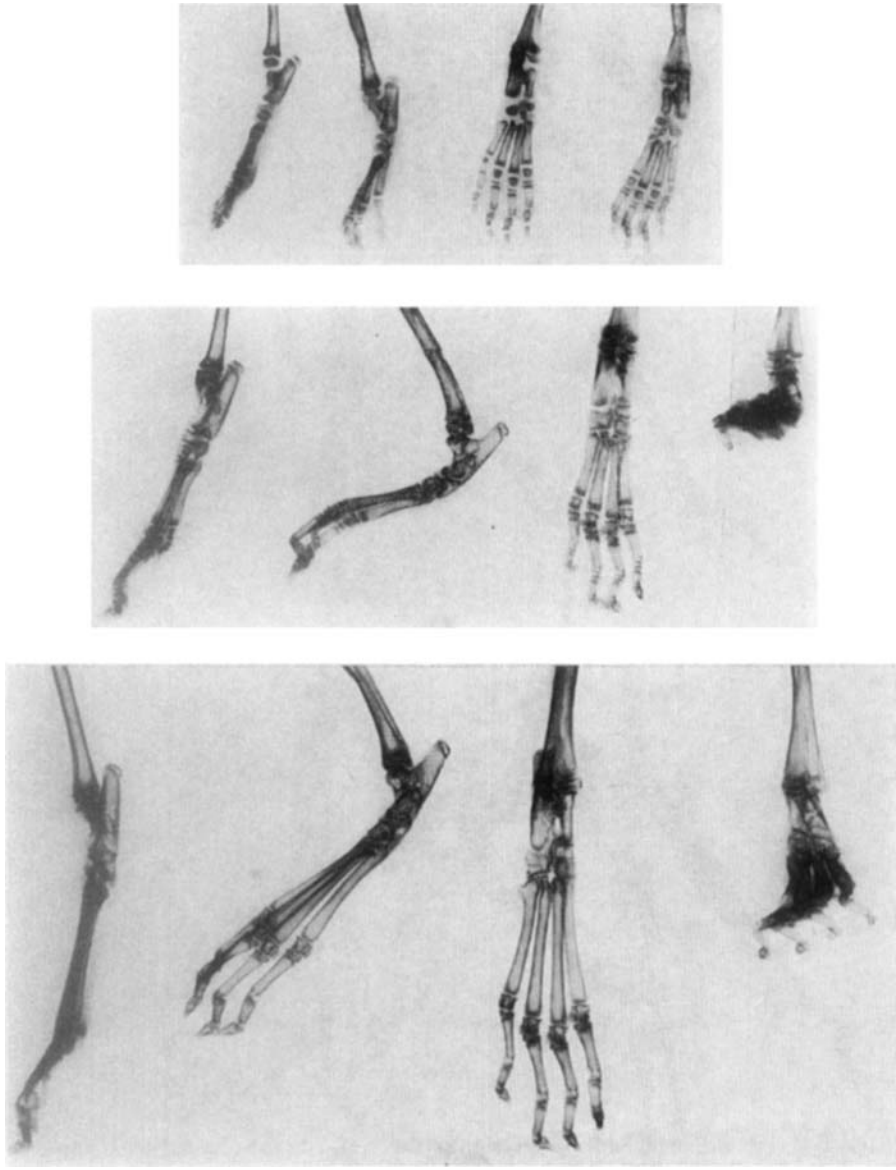

FIG. 34. Development and progression of the vertical talus deformity in a rabbit. The "extensor digitorum" type; Series XLI no. 2.

Top: lateral and dorsoplantar (or anteroposterior) roentgenograms one week after Achilles tenodesis, transections of the tibialis anterior muscle and the ligamentum transversum cruris.

Centre: six weeks; bottom: six months after operation.

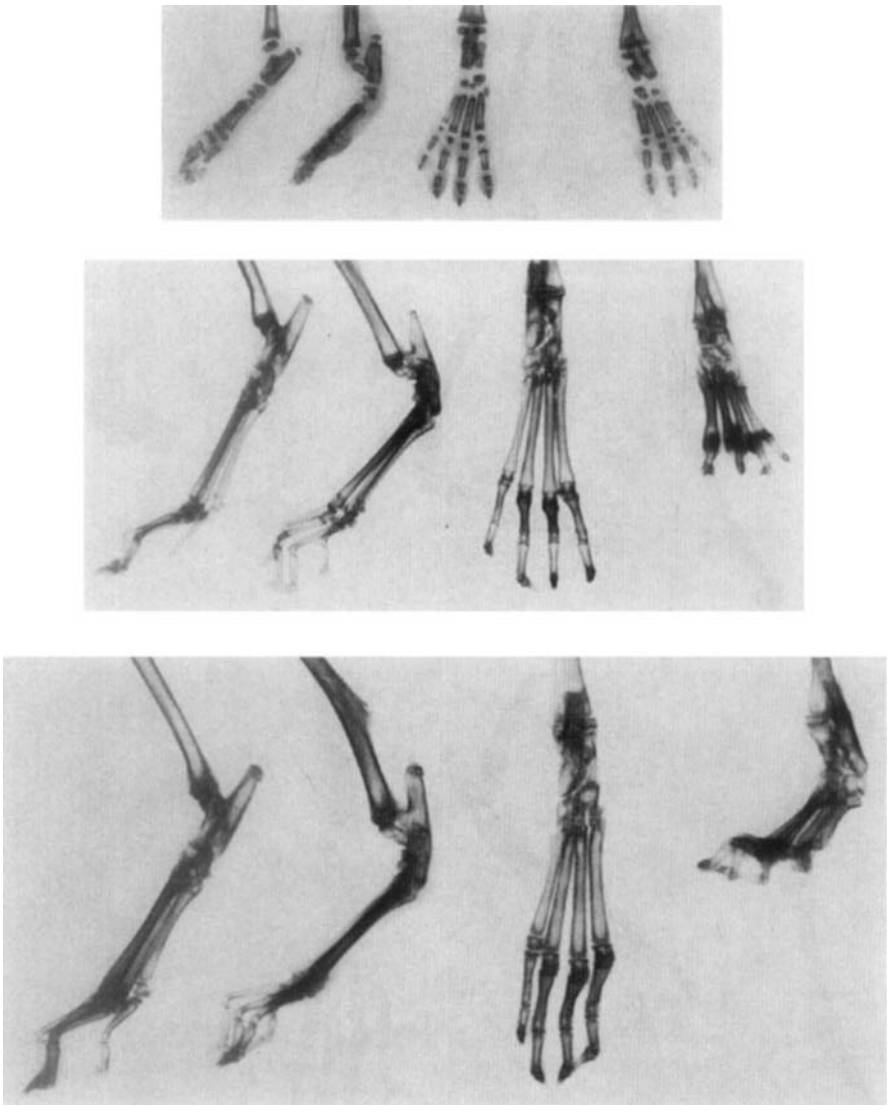

FIG. 35. Development and progression of the vertical talus deformity in a rabbit. The "tibialis anterior" type; Series LI no. 2.

Top: lateral and dorsoplantar (or anteroposterior) roentgenograms one week after Achilles tenodesis, transections of the extensor digitorum longus and peroneal muscles, and transection of the ligamentum transversum cruris.

Centre: six weeks; bottom: six months after operation.

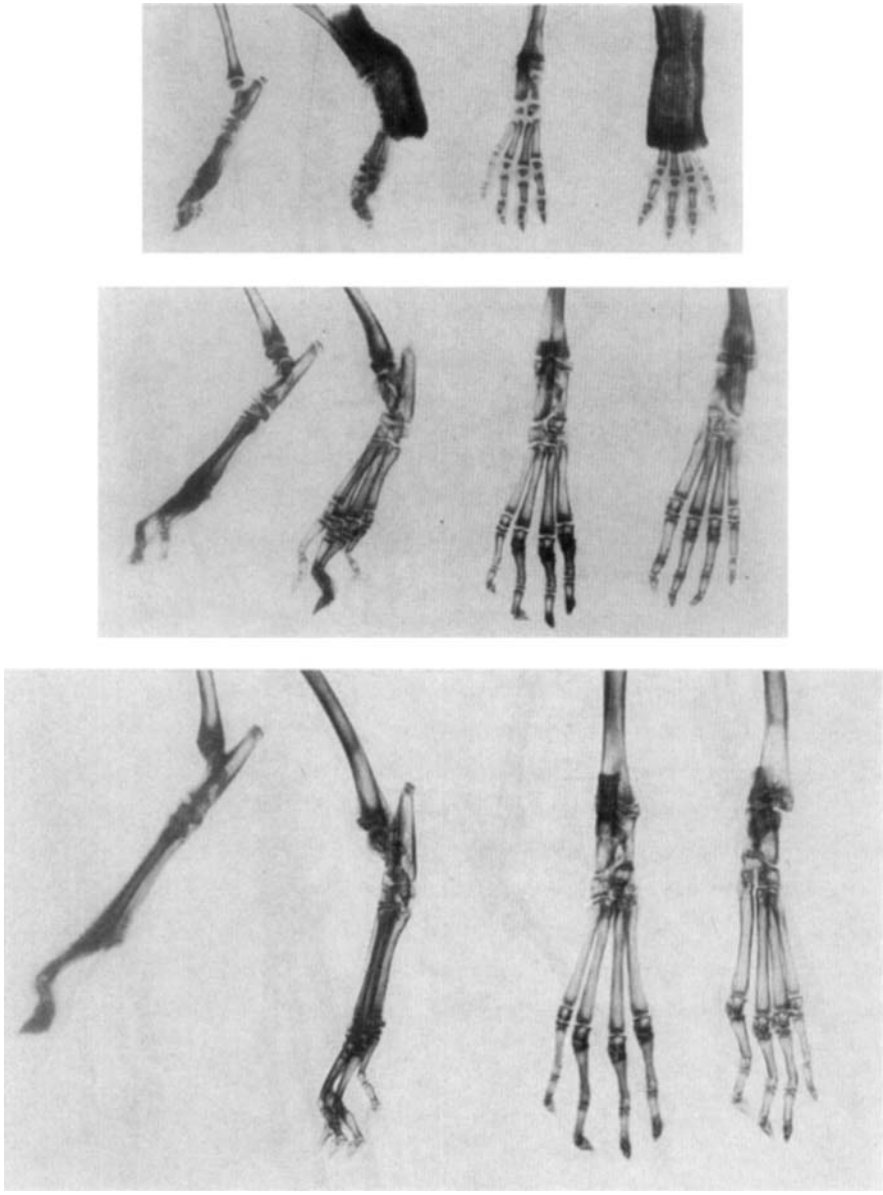

FIG. 36. Development and progression of the vertical talus deformity in a rabbit produced by plaster immobilisation in equinus position and transection of the ligamentum transversum cruris; Series LXI no. 3.

Top: lateral and dorsoplantar roentgenograms one week after operation.

Centre: six weeks; bottom: six months after operation.

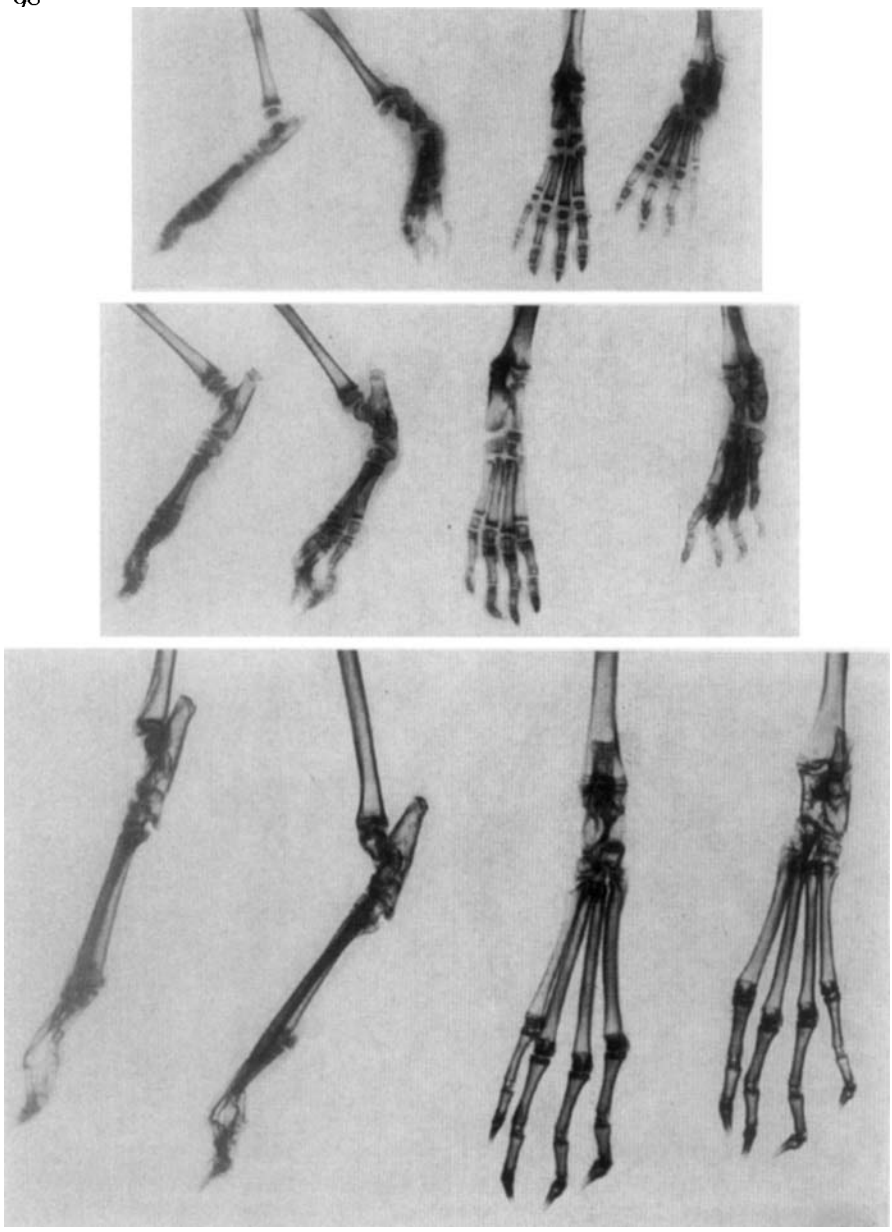

FIG. 37. Development and progression of the vertical talus deformity in a rabbit produced by fixation with nylon thread in equinus position; Series XII no. 6.
Top: lateral and dorsoplantar roentgenograms one week after fixation.
Centre: six weeks; bottom: six months after fixation, fixation removed.

ERRATA

Page 29: Table 2, right side, line No. 4 from below: **for** $A_T+TA_T+P_T$ **read** $A_T+TA_F+P_T$

Page 30: Table 2, right side: Fixation in calcaneovarus position, **strike out** the number 1 in column Number of vertical talus. Fixation in equinus position, **insert** the number 1 in column Number of vertical talus.

Page 31: Table 3, line No. 8 from above: **for** $A_T+TA_T+P_T$ **read** $A_F+TA_T+P_T$

Page 33: Line No. 6 from above: **for** discisions **read** discissions.

Page 33: Lines No. 2 and No. 14 from below: **for** tibialis posterior **read** "tibialis posterior".

Page 44: Line No. 26 from above: **for** posterior **read** "posterior".

Page 50: Table 4, line No. 4 from above: **for** mucle **read** muscle.

Page 50: Table 4, line No. 21 from above: **for** No. 2 **read** No. 4.

Page 50: Table 4, line No. 5 from below: **for** in calcaneovarus **read** in equinus.

Page 64: Line No. 3 from below: **for** phenomen **read** phenomenon.

Page 67: Lines No. 5 and No. 17 from below: **for** dorsiflexion **read** dorsiflexor.

Page 79: Line No. 12 from below: **for** non-surgigical **read** non-surgical.

Page 82: Line No. 2 from below: **for** tissues **read** tissue.

Page 96: Line No. 2 from above: **for** Series LI no. 2 **read** Series XXVII no. 1.

Page 98: Line No. 2 from above: **for** Series XII no. 6 **read** Series XII no. 3.