THE AETIOLOGY OF CONGENITAL DEFORMITIES DUE TO

DISPROPORTIONAL GROWTH DURING DEVELOPMENT

OF THE FOETUS.

Gavin C. Gordon.

First Assistant,
Orthopaedic Department,
Sheffield Royal Hospital.

GAVIN. C. GORDON.   F.R.C.S. (Ed.)

1st. ASSISTANT ORTHOPAEDIC DEPT.

SHEFFIELD ROYAL HOSPITAL.

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In 1939 I was fortunate to be present at a precipitate birth when the malposition, which I will describe, was at once apparent in the infant. This stimulated an already keen interest in congenital deformities, which, although interrupted by the war, has led me to write this thesis to further elucidate a subject which presents many puzzling features. It has as its basis the discussion of malposition of the foetus in utero. The earliest records of cases of congenital deformity, show that malposition in utero from causes unknown was the then accepted explanation. Its popularity has waxed and waned, and the literature has become more confusing due to the fact that there has been no true basis on which differing degrees and varieties can be classified.

Later this theory appeared to be deficient in explaining all cases, further study leading to the belief that malposition in utero could not explain all the varieties met with, particularly those that relapsed after apparently efficient treatment, and a period ensued when all the structures of the leg were in turn implicated in attempts to explain all varieties.

Attention has always been mostly centred on club-foot or talipes equino varus, as it is the commonest of the foot deformities (77%), and is often gross, crippling and permanent if untreated. In other words it offers the greatest field for investigation, both from an academic and operative view-point.

Dr. Little, in 1839, believed that any cause which disturbed the equilibrium between antagonistic sets of muscles, brought about club-foot.

In 1851 Eschricht described the unfolding of the mammalian embryo during development, and the rotation of the limbs, and he claimed that all lower limb deformities were due to an arrest of development, occurring before the rotation of the limbs.

Berg, in 1882, put forward a somewhat similar theory.

In 1852, Adams after thorough study of the bones of the foot in cases of club-foot, gave it as his opinion that the alteration in shape of the bones was the result rather than the cause of the deformity.

In 1882, and again in 1887, Parker wrote producing sound evidence that the deformity of club-foot was due to malposition in utero, but he failed to explain the different varieties encountered. Middleton in 1934, in describing several cases of congenital deformity, gave it as his opinion that they resulted from myodytrophia foetalis, occurring before the birth of the child and resulting in contractures. He failed to establish the histological picture necessary for such a theory.

Brockman, 1930., favoured a theory which drew a parallel between congenital dislocation of the hip and club-foot, suggesting that the latter was due to an aplasia of the calcaneo navicular socket. This theory of ideopathic dislocation does not bear scrutiny.

Denis Browne, 1934 and 1936, revives the theory of malposition in utero but fails to produce sufficient evidence to support it. However, his is the first real attempt to correlate the various deformities of the lower limbs.

The key to the situation lies in the fact that most of the deformities of the lower limbs can be caused by at least two entirely different mechanisms, and if the lower part of the spinal cord is involved, them a complete variety of deformities arises which has defied elucidation until the present time.

It has been said (Adams) that the aetiology of club-foot is of no interest nowadays as the treatment is so well developed. This is incorrect. The aetiology of club-foot is only part of the large subject of congenital deformities in general and I believe that treatment will be considerably altered by thorough knowledge of the factors operating to bring about deformities.

For the proper accommodation of the foetus in utero three factors must be considered.

    1) Growth of the foetus.
    2) Rate and volume of uterine expansion.
    3) Amount of liquor amnii.

If a woman bears five children at different times their weights normally vary considerably, usually in an upward direction for each consecutive child. This suggests that the uterus enlarges with more ease at each succeeding pregnancy, and means that there is an adjustment possible between the size of the uterus, the size of the foetus, and the amount of liquor amnii.

Again, the first born may weigh the least, but this does not affect the subsequent growth of the child, so that the size of the foetus would appear to be regulated by factors other than purely hereditary or nutritional. In other words, it is regulated, at least to some extent, by the accomodation available.

From this it would appear that there is a mechanism of adjustment operating between the three factors mentioned above, and on this mechanism depends the comfort and well-being of the foetus during growth. Conditions which would operate to modify any one factor and bring the mechanism of adjustment into play would be :—

    1) Nutrition of the mother.
    2) Heredity as exemplified by the growth potential of the foetus.
    3) Inherent growth potential of the uterus.

None of these factors are fixed. All are capable of some modification according to the conditions which exist in any pregnancy. On the other hand, none of them are easily or quickly adjustable, and it is in this connection that the liquor amnii plays an important role. It is labile but plays its part in distending the uterus as the foetus grows, acting in a similar capacity to the C.S.F.

This mechanism will be sufficient to adjust all degrees of disproportion within a certain range, but it appears to me from the view-point of genetics that there must, on many occasions, be a degree of disproportion which cannot be adjusted fully.

This may only be of a temporary nature but if it persists, then certain well defined events will occur.

If the adjustment fails completely the foetus will die, but failing this catastrophe, there will be varying degrees of maladjustment allowing the pregnancy to continue to term. The degree of adjustment will finally be reflected in the amount of liquor amnii present, as mechanically it is the most freely variable factor, and, as it were, fills in the interval between the foetus and the uterus.

If the capacity of the uterus develops more quickly than the foetus, moderate degrees of disproportion will be of no significance, as it will result in nothing more than increased freedom for the foetus with increased liquor amnii, but if the foetus develops quicker than the capacity of the uterus, then the disproportion will be followed by decrease in liquor amnii and by certain pressure effects, brought about by constant pressure of the uterine wall on the most prominent parts of the foetus. The existence of such a condition does not preclude the foetus rotating in the uterine cavity. Movement of the foetus as a whole would still be possible, so that foetal movements can still be appreciated by the mother. Balottement will also be possible in the earlier months of pregnancy.

These pressure effects, if taken in conjunction with certain other factors, will bring about fixed malposition, leading to,

1) Deformities of the limbs.
2) Malformation of the spine.

I would like to emphasise at this stage that the amount of liquor amnii is regulated by the proportional growth between the foetus and the uterus and that it is not of a predetermined fixed quantity. Accurate measurement of the amount of liquor amnii at birth would be useless as a guide to the condition of the foetus during pregnancy, as it is only of significance when taken in conjunction with the other two factors and the amount of liquor amnii which would represent the difference between freedom and constraint of the foetus must be very small. The condition I describe occurs in healthy representatives of the human species and is an expression of conditions arising from indiscriminate breeding.

Many examples of gross deficiency or abundance of liquor amnii have been described (Parker and Middleton, Denis Browne). The results of such disproportion are gross deformities which are mostly of academic interest. Between these two extremes lies the aetiology of all minor and moderate deformities of the lower limb and spine.

It is presumed that disproportion in the circumstances set forth becomes evident fairly early in intra-uterine life, but not at any fixed time, while the foetus is establishing itself. Once established, constant proportional
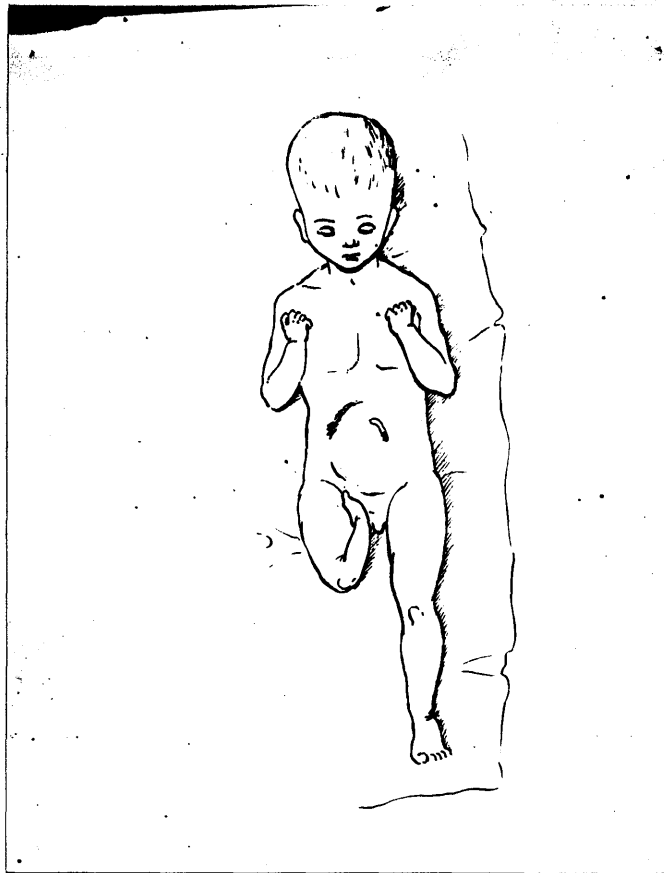
Fig. 1.

The marked asymmetry of the lower limbs is due to the difficulty of the artist in appreciating the mal-position. There was no asymmetry in the living infant. (S.G.S.).

growth will take place so that the disproportion is maintained throughout intra-uterine life.

The malpositions are dependant on skeletal, not soft tissue development, so that the total bulk of the foetus is a minor factor.

The following is a description of the malposition found in a newly born child in 1939.

The right leg was fully flexed on the thigh, lying closely opposed to it, the foot being internally rotated, adducted and inverted, so that the outer side of the foot was applied to the pubes, the toes pointing up over the abdomen and the sole lying against the upper inner aspect of the thigh of the affected leg.

The thigh was flexed on the abdomen but could be extended through an angle of just over 90°, the movement occurring at hip and ankle, and the movement was free and parallel to the sagittal plane of the body, that is, movement was in one plane only. At full flexion the foot lay parallel to the tibia. Vernix caseosa filled the interval round the point of impaction of the outer side of the foot on the pubes, showing that the malpositon had existed for some time, and the point of impaction corresponded to the dimple found over the outer side of the foot in cases of talipes equino varus. See Figs. 1 and 11.

I realised I was viewing the explanation to the aetiology of at least one type of talipes and examined the child accordingly. Unfortunately I had no camera.

The malposition was very stable and showed no sign of spontaneously reducing itself, even when roughly handled. The child could be rolled over and picked up without effecting the malposition in any way. Its plane and range of movement remained constant. When attempts to adduct or abduct the thigh were made, elastic but definite resistance showed that the malposition was maintained by reason of the impaction of the foot on the pubes. Movement of the thigh produced no reaction in the child which was in other respects, a normal, healthy, full-term female child.

I then reduced the deformity with my left hand on the knee and my right hand ready to manipulate the foot. No amount of manipulation of the knee had any effect on the position of the foot and eventually I gently applied increasing pressure to the foot in a downward and inward direction. I did not grasp it but merely pushed it so that any movement could be carefully noted.

Very definite pressure had to be used to push the foot from the pubes and it slipped off reluctantly - it was no mere temporary position.

As the foot left the pubes the tibia rotated outwards or forwards on the femur with a marked sudden slipping movement, showing that the maintenance of the malposition depended on inward rotational subluxation of the tibia on the femur combined with impaction of the outer side of the foot on the pubes.

A.

B.
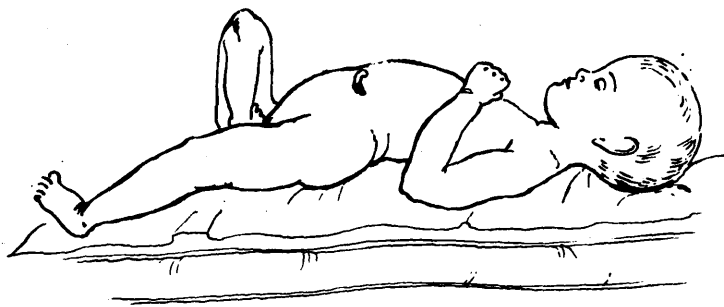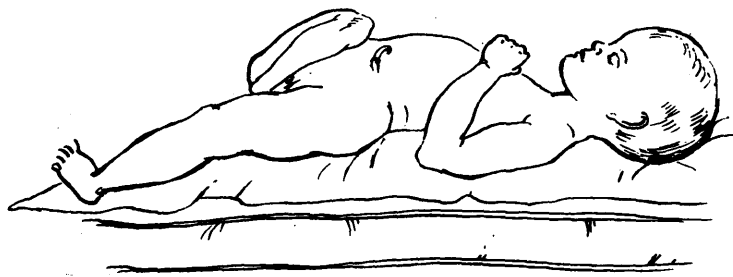
FIG. 2.

Reduced, the foot showed the typical talipes equino varus deformity. The knee and hip showed no observable abnormality and all movements were free. The malposition could not be reproduced, although I made several serious attempts, but it became obvious that considerable force would have to be used to rotate the tibia into its former position.

Examination of the pubes showed a distinct ring of vernix caseosa but no mark of pressure on the skin. On the other hand, a well marked dimple was present on the outer side of the foot.

The child was first-born, the mother had had a normal pregnancy with no adverse symptoms.

Most authorities are agreed that talipes equino varus is merely an exaggeration of what is a normal movement of the foot, and it does not require a pre-existing talipes equino varus to enable the foot to reach this malposition. A normal one could be placed in such a position by exaggerated movement, and once fixed for any length of time, then a classical deformity would result. The structures on the inner side of the foot would contract, those on the outer side would lengthen, and the deformity would persist when the malposition was relieved. Later the osseous elements would adapt themselves to the altered position and the typical changes would be seen.

The following points regarding one type of classical club-foot or talipes equino varus existing without other detectable deformity, are fully accounted for by the malposition described.

1) Adduction of the forefoot.
2) Inversion of the foot.
3) Plantaris of the foot.
4) Internal rotation of the foot.
5) Inward rotation of the lower end of the tibia.
6) Pressure area over outer side of the foot.   I believe that the dimples often seen in association with congenital deformities are merely due to deficient laying down of subcutaneous fat consequent on pressure between foetus and uterine wall. Subcutaneous/fat is not laid down until the 8th month of pregnancy and any pressure occurring between foetus and uterine wall will prevent it being laid down, leading to the dimpling effect. Gross pressure would lead to atrophy of the skin or in a lesser degree to a type of bursa formation which is sometimes described.

7) Tilting of the calcaneus leading to prominence of lateral malleolus.
8) Equinus. This is an inconsistant part of the deformity and it is by no means always present. If Figs. 1 and 11 are studied, it will be seen that when the thigh is in full flexion the foot is in a calcaneus position. How does this disappear when the malposition is relieved ? Part of the deformity is due to the pitch and roll of the calcaneus whereby the anterior part of the calcaneus comes to lie below the head of the talus. This produces the furthest upward shift of the heel possible. This, plus the

acutely flexed and fixed knee leads to the development of a short gastrocnemius so that when the knee extends the foot is pulled down into a more normal position. The ankle has been able to move through a restricted range and shows no contracture.

As the knee is fixed why does it show no contracture ? The rotational subluxation of the tibia on the femur causes sufficient stretching of the capsule to allow it to extend, but there probably exists a minor degree of contracture for the first few days. As there are no muscles, such as would hold the knee flexed, the capsule is easily stretched. (The gastrocneumius plantar flexes the ankle and the ham-strings will not be short).

If the origin and insertion of the hamstrings is envisaged it will be appreciated that when the thigh is flexed these muscles are lengthened and vice versa, so allowing extension of the knee when the thigh is extended.

If a newly born infant flexes its thigh its knee bends, and if it straightens its knee its thigh extends.

This rotational subluxation also explains why nutritional changes are minimal, as it will ease the pressure which would otherwise be exerted on the popliteal vessels.

The hip joint is allowed a certain range of movement and so shows no gross contracture.

9) Laxity of the ligaments of the knee joint with a tendency to genu valgum.

As can be seen, inward rotation of the tibia round an axis approximately in the region of the external collateral ligament would lead to lengthening of the capsule and ligaments on the inner side of the knee, or if the subluxation is a rotation of the tibia round its own axis, the laxity will be even more marked.

10) Luecke in 1871 stated that children born with talipes equino varus had a deformity of the whole limb, and Berg in 1882 stated that the deformity follows failure of the thigh to rotate inwards. If the malposition is studied it can be seen that the thigh is held everted and persistence of such eversion is seen in the early days of classical talipes equino varus. This also explains the use of the Denis Browne splint with external rotation of the leg.

11) The absence of a dimple over the pubes is due to the modified nature of the skin in this area.

Changes in bone structure have frequently been described but only after the child is some years old at least, and the moulding of the bones is to be expected. It would be more a matter for comment if it did not occur.

The Chinese bind, or used to bind their children's feet, so permanently moulding them and retarding growth, as can be seen from specimens in many anatomical museums.

Some tribes bind their children's heads and produced permanent deformity,
so that the final adjustment of ligaments and bones in an uncorrected deformity
is not strange, but must be put in its proper place, that is, not among
the problems of aetiology, but as an after-effect or sequel due to lack of correction.

One of the apparent weaknesses of this explanation is that if the malposition
occurs at a relatively early stage of development, any change in the proportional
growth of the different parts of the leg would invalidate the explanation given.

However, proportionate changes in the limbs after the embryonic period
may be given as follows (after Retzius).

"The arm reaches its greatest length relative to the body during the fourth
month, the leg its greatest relative length during the fifth month. There
are no changes in the proportionate length relations between upper arm, forearm
and hand after the third month, and the same may be said of the proportionate
relations between thigh and leg. In the case of the foot, however, a definite
elongation when compared with the leg is found between the sixth and eighth months."

There is nothing then, to negative the view that a leg may adopt the above
malposition accidentally when minimal disproportion occurs between foetus and
uterus towards foetal preponderance. This may occur at any time after the third
month, although due to the fragile nature of the preosseous elements, it will
be most likely to occur from the sixth month onwards when the logitudinal pressure
necessary to maintain the malposition can be borne by the leg and thigh without
distortion.

The elongation of the foot between the sixth and eighth months will not
affect the position. If this is accepted, then the next problem is to explain
how such a malposition can occur.

Normally, although the foetus lies in a curled up position, a certain amount
of involuntary or reflex movement occurs in the limbs and this movement serves
to prevent any contractures occurring. If this small but necessary range of
movement is abolished for any reason, certain adjustments in the soft tissues
would be expected, and contractures would occur, as they do in adult limbs
under similar circumstances.

Intermittent pressure does not, as a rule, give rise to permanent effects,
so that to bring about such a well developed deformity a continuous degree of
pressure or maintained malposition must be described. The degree of constraint
is very important. If the foot is merely restricted in its variety of
movement, and, while being inverted, is still able to carry out a certain
range of movement in this position, then the resulting deformity will be capable
of easy correction as there will be no gross contracture, or gross change in
the cartilaginous elements, so that the deformity will tend to disappear
as the child grows up. This no doubt explains many of the cases described in
the older text-books which spontaneously cleared up. Nervous, muscular and
skeletal causes, and arrest of development have been postulated but no tangible
proof can be advanced to uphold any of these theories. Nervous causes have
some basis in fact but only as a late result.

Berg in 1882, working on data from Billroth, St. Hillaire, and Wagner, and by the study of various museum specimens, stated that in early foetal life – 6 – 7 weeks – the sole of the foot is turned in in the varus position, and that arrest of development at this early stage would perpetuate the early foetal position of the foot. "Arrest of development" is an unfortunate phrase as it suggests a growth defect, but if the foot was prevented from rotating outwards, then the persistence of an inverted position of the foot, would lead to a varus deformity, differing in degree and type according to certain factors.

Bessel Hagen 1899, and Scudder 1887, on the other hand, state that the position of the limbs bears no necessary relationship to the age of the foetus.

The difficulty in studying such a subject lies in the fact that most of the specimens and data available will be of a pathological nature but the truth probably is that the feet in a normal pregnancy with adequate adjustment can assume a variety of positions, but that with the onset of disproportion, which will occur at an early stage when the yolk sac is disappearing and the foetus filling the uterine cavity, pressure by the uterine wall will be exerted on the lower limbs, limiting movement.

Parker and Shattock 1884, offer a somewhat similar explanation.

The probable course of events in classical talipes equino varus is as follows.

A minimal or moderate degree of disproportion arises which, while not restricting the movement of the lower limbs, tends to make them slip and slide over one another in the crossed position. The foot of the leg nearest or lying closest to the abdomen becomes trapped on the inner side of the opposite thigh while it (the foot) is in an inverted position, after which any sudden strain such as lifting a weight or straining at stool, causing a rise in pressure on the knee of the trapped leg will drive it down between the thigh and abdomen. Once trapped, the leg will be at a mechanical disadvantage and be unable to free itself. Inevitably pressure on the knee will drive the foot down on to the pubes and terminal rotational subluxation at the knee will fix it there. Release will only be possible at birth, and will occur as the child is delivered to reveal the deformity but not the malposition.

If this explanation is correct then classical talipes equino varus becomes understandable as the result of an accidental malposition in utero.

This explanation is only applicable to cases of the accidental variety, and is always unilateral. Bilateral cases and unilateral cases of a second type cannot be explained in this way, and fall into a group which will be described later.

This explanation fits all the facts and cannot reasonably be repudiated.

This explanation does not fit all the other forms of talipes which occur, and other factors must be sought.

FIG. 3. (Proc. of N. Soc. Med)

FIG. 4. (Mercer's Orthopaedic Surgery)

If there occurs a failure of the mechanism of adjustment which brings about disproportion between foetus and uterus to such a degree that constant pressure is exerted on the foetus by the uterine wall, such pressure will bear on certain points.   On the head, on the length of the spine, on the upper limbs at the shoulder, elbow and hands, and on the lower limbs on the sacrum, knees, and feet.

The upper limbs will be protected from moderate degrees of pressure by further flexion of the head and the effects of pressure. due to disproportion will first be seen in the lower limbs, and vary according to the position occupied by the feet when the pressure develops.

Only the commoner forms of malposition will be discussed here.   There must be many atypical and probably unique forms but they do not concern us at the moment.   For the sake of clarity I will presume that the normal position of the foot in the foetus is plantar surface to the uterine wall and crossed.

If Fig. 3 .is studied it will be seen that if the feet are trapped in inversion with legs crossed, when pressure becomes operative, the pressure will be exerted on the outer toes, giving rise to the condition known as minimi digiti quinti varus.   This may also effect the 4th toe.   This is a common condition.

If the disproportion is greater, then the foot will be further inverted and pressure will be brought to bear on the dorsum, resulting in matatarsus varus, a much commoner deformity than is usually recognised.

Further pressure will result in grosser degrees of inversion, when the fore-foot is fully inverted the hind foot will follow, giving rise to deformities approximating to, but never quite so severe as classical talipes equino varus. In this type an equinus deformity will be the rule and the area of pressure, which will only occur with the more severe degrees, will be more diffuse.

These deformities will be unilateral or bilateral according to whether one or both feet are trapped in inversion, and if bilateral never quite equal due to the crossing of the legs resulting in a different angle of contact between the feet and the uterine wall.   This inequality of practically all bilateral deformities of the feet is a point very strongly in favour of this explanation.

This malposition is the source of cases of so called bilateral classical talipes equino varus, unequal on each side as explained above, and with a more pronounced equinus deformity.   Tenotomy of the tendon Achilles, Stromeyer 1831, probably had its greatest successes in these cases.   The cases of club-foot which after apparent adequate treatment, relapse, are fully dealt with later on in this paper (p. 21.).

The first two degrees of this deformity may also result if one or both feet are trapped in an inverted position side by side, and if disproportion is more severe, the tibia may bend and the lower limbs assume a position as in Fig. 4.

The degrees of pressure resulting in the above deformities in an inverted foot will show a corresponding degree of calcaneus: in the normally placed or everted foot which will, to a great extent, correct itself when relieved. Bilateral calcaneo valgus will tend to become a simple valgus when the knees are extended.

Denis Browne describes these three degrees of deformity but places accidental classical talipes equino varus at the top which is incorrect.

Parker describes a case of double talipes equino varus in which, after appropriate staining, no lesion of the spinal cord or muscles of the legs was found.

After removing all muscles, the deformity was still unreduced and remained so until the anterior part of the internal lateral ligament of the ankle blended with the astragalo-scaphoid ligament and the calcaneo scaphoid ligament was divided. He describes the extension of the astragalus trochlear surface backwards and lessening of it in front, indicating extension of the ankle joint.

This case is typical of those which would occur when the legs are crossed with the feet in inversion, pressure occurring after full spinal closure.(↑. 20).

The trochlear surface of the talus would be unaltered in the accidental variety of talipes equino varus. It would also be unilateral, otherwise the changes would be the same.

He also describes a case of calcaneus in which the ankle did not admit of extension to 90° until the anterior ligament of the ankle was divided. The sole of the foot was convex towards the plantar surface, i.e. outwardly. This case is typical of the unilateral deformity when the feet are trapped side by side, dorsiflexed and one foot obtains leverage over the other so that it adopts a natural position with the other foot constrained in extreme calcaneo-valgus. (vide infra).

Finally he describes a case showing a sloughing spina bifida and extreme equino varus of both feet. This is a case of crossed inversion of extreme degree with disproportion arising before full closure of the spinal canal (see Spinal Malformations. p.20.).

Now, if the legs are not crossed but are trapped side by side, then a totally different set of deformities arise. In feet with plantar surface to uterine wall, the feet will be forced into a calcaneus position, and if one limb gains an advantage over the other, which is most likely, and so pushes it laterally, then one foot will develop normally, as in a crossed leg position, while the other is constrained in a severe calcaneo-valgus position which will persist after birth. A severe degree of this condition can only be unilateral. The plantar surface will show a convex surface from heel to toes as it conforms to the uterine wall, with the apex of the arch of the foot the most prominent part. Extension of the limb will not correct this deformity much, as it has been held firmly in a calcaneus position with no movement allowed so that the capsule of the ankle joint will be grossly contracted anteriorly, resisting correction. The same will apply to the muscles. Contracture of the anterior group will prevent any convincing degree of correction.

The capsule, ligaments and final formation of the bones in the vicinity of joints are the greatest factor in maintaining most of the deformities described, the muscles play a secondary role. Neglect of a deformity, or on the other hand forcible manipulation, leads to continuance of the deformity owing to changes which occur in the bones and ligaments, not the muscles. This point is discussed further in the treatment of these cases.

When one foot is trapped in a plantar flexed position side by side, the normally placed foot with plantar surface to uterine wall will be capable of greater pressure and will push the other foot to the side. In this malposition, the limb will conform to the uterine wall, which will force it into a gross equinus position, when, with the ankle in full plantar flexion the strain will be transmitted to the lower third of the tibia, the leg being acutely flexed on the thigh and in the same plane. This last is an important point when considering nutritional changes in the limb. This will result in bending of the tibia in the lower third leading to the condition known as congenital angulation of the tibia.

Angulation can be bilateral if the feet are both plantar flexed side by side.

In cases of unilateral angulation of the tibia with legs side by side and one plantar flexed a type of pseudarthrosis may occur if the malposition occurs early in intra-uterine life. Then the angulation may go on to the formation of a Greenstick fracture in the lower third of the tibia. Muscular contraction which begins about the 11th to 12th week of intra-uterine life will prevent immobilisation of the fracture, which will tend to progress. When the bone ends become sufficiently soft the foot will be released from its position of acute equinus and as this part of the deformity corrects itself to some extent, or becomes distorted, so the evidence of the malposition will be missing at birth. At this stage the pseudarthrosis will be established. The dimple over the fracture is due to the pressure of the uterine wall at the apex. Nutritional changes will occur in the muscles causing fibrosis, or the fibula may fracture and be partially absorbed.

Middleton's three cases can be explained in this way, the associated deformities of the digits being merely coincidental in case 1. Congenital dislocation of the hip may be present and will depend on the period of intra-uterine life when angulation occurs. Before the angulation becomes established, pressure of the uterine wall on the dorsum of the foot will counteract pressure on the knee, but once angulation or pseudarthrosis has occurred this counterbalancing pressure will be lost and pressure effects will occur as is discussed under congenital dislocation of the hip. If angulation or pseudarthrosis is late in appearing, then congenital dislocation is unlikely as the acetabulum will be more developed and insufficient time is left to produce atrophic changes in the acetabular rim. (see p. 19).

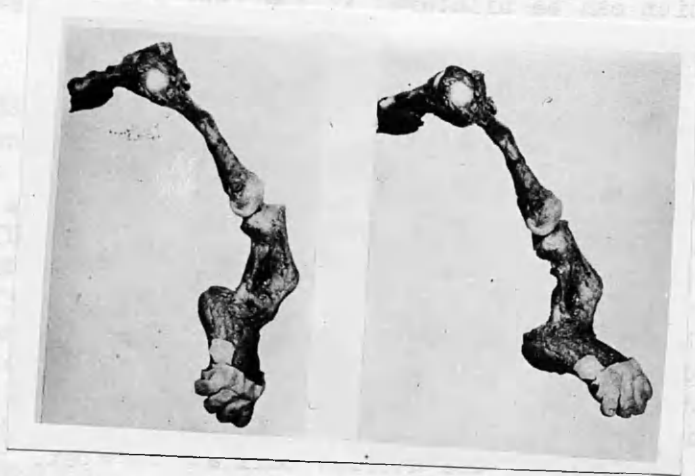FIG. 7.—Case III.

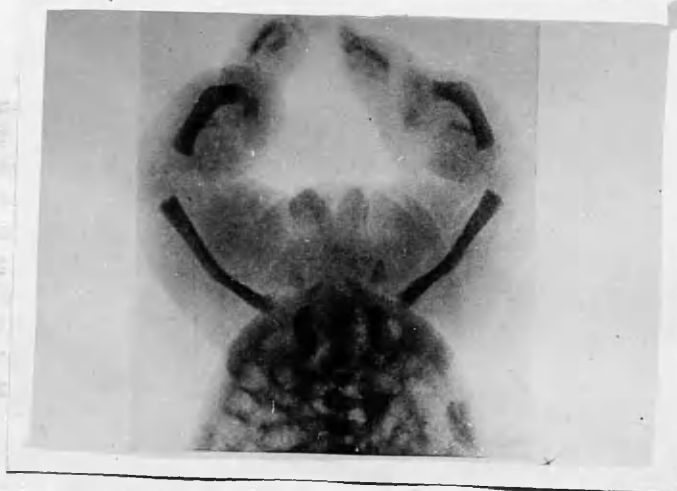FIG. 5. (Edinburgh medical Journal)

FIG. 6. (Edinburgh medical Journal)

Fig. 7. (Edinburgh medical Journal)

It can be argued that in the malposition of classical talipes equino varus, angulation does not occur. It does. There is a rotational angulation, but the other lines of strain are in the natural weight-bearing axis of the bone and so no distortion occurs.

If Figs. 5, 6, and 7 . are studied, it will be appreciated that if the feet are side by side in plantar flexion under pressure, once the angulation occurs in the tibiae, the heels, lying in the same plane as the tibiae and femora, are brought, by further pressure, into contact with the posterior aspect of the femora, giving rise to the angulation seen in the specimens which need only be bent to demonstrate this point.

In conformity with these views, pseudarthrosis of the tibia is always unilateral, congenital angulation may be unilateral or bilateral.

Further Evidence Regarding Pseudarthrosis of the Tibia.

This explanation of pseudarthrosis of the tibia did not entirely satisfy me and in searching for a further explanation I came across a specimen in the pathology laboratory of the Sheffield Royal Hospital, which Dr. J.M. Barrie kindly presented to me. It is a foetus which, according to the usual standards of estimation is 10 to 11 weeks old, and it shows a typical pseudarthrosis of the right tibia.

There is no history available for this specimen. It has lain about in the pathological laboratory for some ten years. The scalp has been lacerated and tends to fall over the face, but it is a complete foetus in all respects. The irregular appearance of the skin above the deformity of the left leg is due to cuffing of the skin, inflicted accidentally due to repeated handling.

It is a well formed foetus except for the left lower leg and foot. The legs lie naturally crossed and at the point of crossing the left leg is bent over the right acutely and a pressure area on the posterior surface of the left leg fits over the right leg in its lower third. In addition to this angulation at the point of crossing, the foot of the left leg is in very acute equinus, the toes pointing down and to the right. The forepart of the foot shows adduction and the toes being given a "spraying" effect to the right.

Movement is possible at the point of angulation of the left tibia, indicating interruption of the cartilagenous continuity of the tibia.

The right leg shows slight anterior bowing of the tibia, otherwise it is well formed. The foot is normal and is at right angles to the leg.

Close examination revealed no other detectable deformity, particular attention being paid to the spine.

Fig. 8.

Scale
is
in
cm.

Fig. 9.

Scale
in
cm.

Fig. 10.

Figs. *8,9,* and *10.* show that the legs fall naturally into a crossed position with one foot plantar flexed, dorsum to uterine wall, and the other dorsiflexed sole to uterine wall. Although this foetus shows a very convincing picture of pseudarthrosis of the tibia, the possibility of post-mortem changes occurring during its ten years sojourn in the laboratory, could not be completely discounted. How much of the deformity was ante-mortem and was any of it post-mortem ?

To elucidate such queries the foetus had to be sacrificed and after adequate photography serial sections were made of both lower limbs and stained.

Before proceeding further it would be as well to recapitulate the main facts regarding the development of the limbs and the earlier stages of ossification of the skeleton.

The limbs first begin to appear during the fourth week, as mesodermal elevations ventral to the line of somites, and they arise opposite the somites associated with the limbs. Fourth cervical to second dorsal for the upper limbs and twelfth dorsal to fourth sacral for the lower limbs.

They are derived partly from the lateral meso-dermal sheet and partly from the mesoderm of the somites which migrates into the limb buds.

The limbs develop rapidly and at the end of the second month the various parts are clearly seen. The arms are longer than the legs and this persists until the second year after birth.

The embryonic cartilage formed by the central condensation of mesoderm is formed in the sixth week and by the end of the eighth week, conforms accurately to the final shape of the skeleton. The muscles form early and may be recognised in the sixth week. Nerve elements are recognisable as soon as muscle tissue appears. The muscles and cartilagenous elements of the hand and foot are slower than the rest of the limbs in becoming defined. The limb buds are usually described as having dorsal and ventral surfaces and preaxial and post-axial borders. The ventral surface being applied to the body wall and the pre-axial border includes the thumb for the upper limb and the great toe for the lower limb.

Further development brings about an inward rotation of the lower limb through 90°, by which the extensor surfaces are brought forwards and the flexor surfaces backwards. Outward rotation through 90° occurs in the upper limb, so that the flexor surface comes to lie anterior.

The inward rotation of the lower limb occurs at a very early stage and is followed by crossing of the legs, a position which is seen about the seventh to eighth week. Muscular contractions first become possible about the eleventh week.
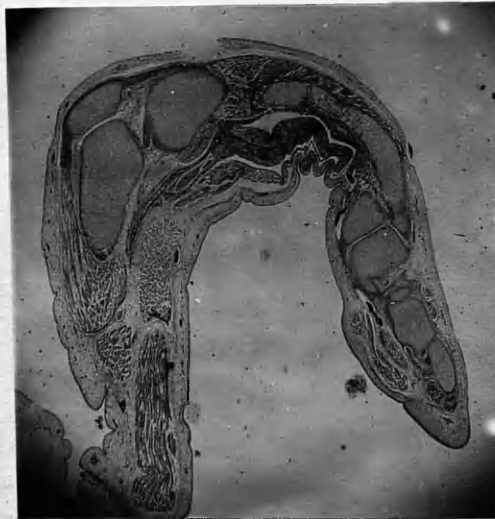
FIG. 11.

X 11.5.

LEFT LEG.

With regard to the ossification of the skeleton, each long bone is represented in early foetal life by a rod of hyaline cartilage which arises primarily from a central condensation of mesenchyme.

The primary centre of ossification for the shaft appears in the centre of the rod of cartilage and the process of ossification proceeds towards the ends. Secondary centres appear at one or both ends later and form epiphyses. The centres for the long bones of the limbs appear about the eighth week of intra-uterine life.

The cartilagenous model of the bone is surrounded by a very vascular and thick membrane known at the perichondrium, the outer layer of which is the fibrous periosteum and the inner layer the osteoblastic layer.

When ossification begins the cartilage lacunae in the middle of the shaft become greatly enlarged and the cartilage cells undergo degenerative changes and lie in spaces which are termed primary lacunae. The spaces are invaded by osteoblasts from the deep layer of the perichondrium and primary bone is deposited. Osteoclasts now appear and absorb part of the primary bone to form the medullary spaces, which are filled by marrow consisting of osteoblasts and vessels derived from the osteogenic layer of the perichondrium.

Towards the ends the cartilage cells undergo division and give rise to parallel rows of cells. These cells continue dividing during the period of growth of the bone and are responsible for the growth in length.

Osteoblasts and osteoclasts from the primary centre extend the process of ossification in the longitudinal direction by invading these columns of cells, removing the trabeculae and laying down new bone. This series of changes proceeds gradually from the true bone at the centre towards the ends of the diaphysis.

The epiphyseal cartilage immediately in front of the bony tissue continues to grow until the adult length of bone is reached.

One or more secondary centres may appear in the epiphysis but throughout the process of growth the epiphysis is separated from the growing shaft by the epiphyseal cartilage.

Description of Sections of Lower Limbs of Foetus.

Left Leg: There is no evidence of post-mortem distortion of the specimen and the sections stained well. (Fig. 11)

The lower end of the tibia shows a uniform curvature of the cartilage from the vacuolated area downwards, this curve being prolonged by the cartilagenous elements of the foot which are in complete equinus, in the same axis as the tibia. The foot could not be further plantar flexed.

FIG. 12.

The vacuolated area in the centre of the tibia indicates that the process of ossification had begun. It tapers off into normal cartilagenous areas at either end.

If the upper part of the tibia is studied there can be seen, first the vacuolated area, an area of degenerating cartilage and cutting across this, an area where ossification is proceeding. Osteoblasts are numerous and primary bone is obvious. This area runs completely across the tibia. There is an interval in the cortex at all points as shown by serial section. The perichondrium is thickened in this area. This is a fracture (or at least a line of stress which has resulted in premature ossification). (Fig. 14).

If a line is taken through this fracture and continued out through the soft tissues of the leg, it can be seen that,

1) The perichondrium is thickened and buckled or folded on itself in this region.

2) The muscular tissues shows normal histology and are folded on themselves, indicating approximation of origin and insertion of these muscles due to bending of the tibia. (Fig. 12).

3) The site of maximum infolding of the skin is directly opposite this fracture as with all the other soft tissues.

4) Above the fracture the cartilage is normal in appearance.

It may be argued that this fracture area through the upper part of the tibia merely represents premature ossification due to the presence of a blood vessel in this area, developing from the perichondrium and leaving an intervening area of cartilage.

This is unlikely, as blood vessels develop from the deep surface of the perichondrium and it is unlikely that one abarrant vessel would cause ossification completely across the shaft of the tibia, or that a circular ingrowth of vessels occurred at this area.

This interruption in the continuity of the tibia is complete, as shown by all the sections. Also see the description of the other tibia. Bilateral abberant vessels at this level is not a reasonable assumption.

There is a definite angulation of the tibia at this level, as compared with the curvature lower down, so that in effect, there is shortening of the posterior length of the tibia, and it is as if there is a wedge removed, the deepest part posteriorly.

The rest of the bones of the leg are normal. Further, the kinking of the soft tissue indicates a pathological process.

This bend of the tibia which is reflected in the buckling of the perichondrium, muscles, skin etc., i.e. the soft tissues, is unassociated with a redundancy of preosseous tissue at the site of fracture, which is further proof that it is a fracture.

FIG. 13.   × 11.5.
RIGHT LEG.

There has been stress in this area which has brought about
premature ossification and absorption of the cartilage under pressure.
The soft tissues are not subject to the same changes and merely fold.

The curvature of the lower tibia and equinus of the foot would
result in shortening the muscles, but the bend in the tibia can be seen
to be the most potent factor in causing in-folding of the soft tissues.

Right Leg: In contrasting these appearances with the left leg,
I was dismayed to find an incomplete fracture of the upper end in the
right tibia. This, at first sight was a very puzzling feature, but
if the malposition which brought about this deformity is studied, the
reason for the kinking in the normally placed leg can be seen, and
it becomes an interesting contrast study of the stresses and strains
brought about by the malpositions. The right leg shows a kink
posteriorly on the tibia. (Fig.13).

Any kinking of the right leg would, at first sight, necessarily
appear on the anterior surface of the tibia.

However, on closer study of the innermost leg, and the position it
assumes, it can be seen that the pressure of the outermost will acutely
flex the knee of the other leg, which will be forced back on to the thigh.
The foot will be acutely dorsiflexed. Increase of pressure will cause
strain on the upper half of the tibia causing anterior bowing or kinking
of the posterior surface, as it conforms to the curve of the thigh. This
can be demonstrated if one's ankle is grasped, and the leg flexed on the
thigh.

It is, in fact, further proof of the malposition.

All these changes are ante-mortem and cannot possibly be put down
to post-mortem trauma to the foetus. Efforts of the perichondrium to
effect a repair are illustrated by the thickening of this membrane in the
vicinity of the fracture.

There is no evidence of a fibula in the sections examined, and
it would appear that if the deformity is established as early as the 10th
week, absence of the fibula is possible. Certainly the occurrence of a
pseudarthrosis at this stage, would mean pseudarthrosis of the fibula also.

The amount of movement of the foetus would be a factor, also the exact
time of the fracture, allowing freedom of movement of the foot and lower
tibia, giving rise to varying degrees of formation of the middle third of
the tibia.

Once the process of ossification is well advanced in the diaphysis,
this process of bending, followed by fracture higher up in the tibia, is
unlikely, unless the degree of pressure is severe. Angulation will be
more likely to occur in the lower third, but if it does, evidence of
pressure on the other leg will be expected.

Or, pseudarthrosis will occur but with a different sequence of
events. Once ossification has developed well towards the extremities
of the tibia, pressure developing at this stage will cause angulation
in the lower third, followed by fracture in the lower third leading to
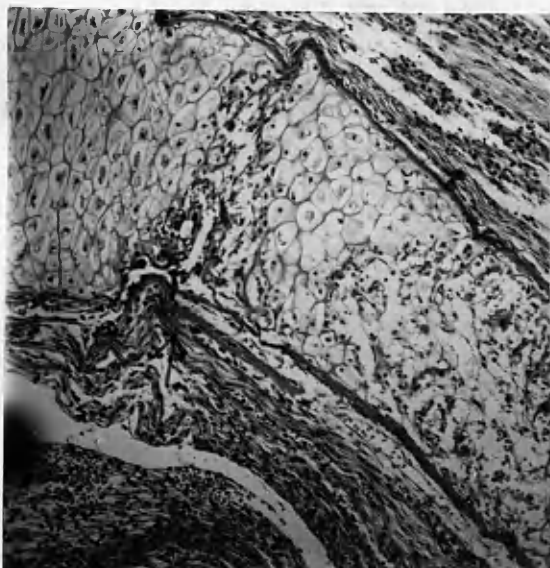
Contrasting fractures of tibiae

FIG. 14.   ×105.
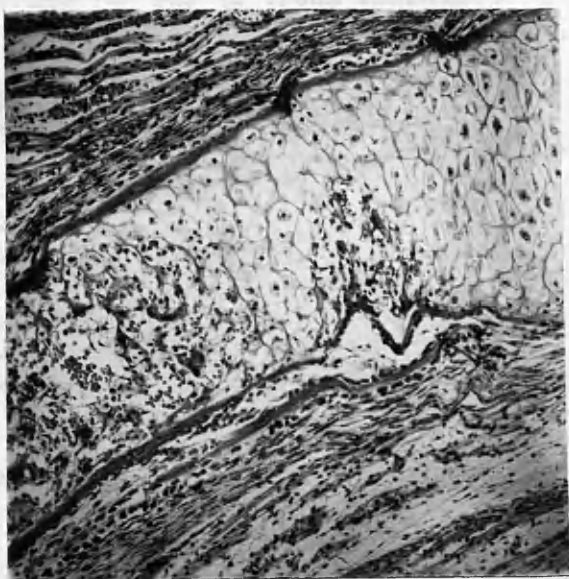LEFT LEG

To show complete fracture.

FIG. 15.   ×105.
RIGHT LEG.

To show incomplete fracture.

pseudarthrosis.   In these cases the fibula will be present and may only show bending.

With regard to the case under discussion, the course of events is probably as follows.   A minimal degree of disproportion arises between foetus and uterus about the 7th to 8th week of intra-uterine life. At this time there is no muscular contraction occurring, so that it would appear the feet adopt the position of plantar flexion of one and dorsiflexion of the other, when the legs cross during development. The disporportion fixes the malposition.   To result in a pseudarthrosis it must be the foot of the outermost leg that is trapped in plantar flexion.

Following this, the leg in correct position, sole to uterine wall, forms a bar across which the outermost leg is bent.   At first this results in curvature of the lower part of the tibia following pressure which is not fully balanced by acute equinus of the foot.

This process occurs during a period of 3 - 4 weeks and once the foot is in full equinus, and the lower tibia in sufficient curvature to correspond to the curve of the uterine wall, a pressure point will develop on the posterior aspect of the outermost tibia where it crosses the inner tibia, and at this point the cartilagenous tibia will kink, forming a fracture, the perichondrium remaining intact.   Once fractured, the perichondrium in this region will attempt to heal the lesion, but although the enchondrial bone is produced, the gap in the cortex of the bone cannot be bridged over, due to lack of such reparative powers or to slight movement occurring at the site of fracture.

When contraction of muscular tissue begins at the 11th and 12th weeks, movement at the site of fracture prevents any adequate repair and a pseudarthrosis occurs.

Again due to the muscular contractions, the limb will later escape from its confined position, but by this time the pseudarthrosis is established and healing cannot take place.

The other leg also suffers in the first stages, but the fracture is incomplete and never becomes mobile, so that when the legs free themselves, it is able to heal and develop normally. (FIG. 14 + 15).

Once the pseudarthrosis is developed, the muscles, which had been developing normally, are left with no fixed points on which to act, and are subject to no physiological demands, such as the stretch reflex.   Under these circumstances, the muscles will degenerate and be replaced by fibrous tissue which will further accentuate the deformity when it contracts. In congenital angulation of the tibia, the posterior tibial group of muscles will still have fixed points to act on and no degenerative changes will take place, although due to the fixed malposition, contractures will occur and some fibrosis of the muscles will be present.

So that pseudarthrosis of the tibia can be brought by two different mechanisms, for I believe that a severe unilateral congenital angulation of the tibia, occurring with the feet side by side and one plantar flexed, can result in a pseudarthrosis, just as a severe unilateral calcaneo-valgus results if the feet are side by side and both dorsiflexed.

Gosne in 1907 divided cases of pseudoarthrosis of the tibia into four groups.

1) Those in which there is a well marked anterior projection, but no fracture at birth.

2) Same as Group 1, but pseudoarthrosis occurring after slight trauma.

3) Those in which pseudoarthrosis is present at birth.

4) Those in which the patient was normal at birth but in which a fracture, occurring after slight trauma, develops into a pseudarthrosis.

I suggest that 1) is a case of severe unilateral angulation which stops short of pseudarthrosis.

2) is a stress fracture occurring in an angulated tibia.

3) is one or other of the types of pseudarthrosis described by me.

4) is either a condition quite unrelated to congenital pseudarthrosis, with a nutritional basis or due to inefficient immobilisation in Plaster of Paris, a mode of treatment difficult if not impossible to carry out in an infant and probably resulting in fixation of the foot, but not of the knee, and allowing movement at the site of fracture.

Obviously, now that the mode of causation of pseudarthrosis of the tibia is known, no classification of this condition, or of congenital angulation of the tibia will be adequate.

Each case will require to be studied closely and an estimate made as to the time when disproportion became operative. A complete review of all cases would be necessary before a new classification would be possible.

If both feet are trapped in plantar flexion, the probability is that a type of angulation of the tibia or a simple bilateral equinus or high arch would result, but there will be no fulcrum across which one leg can bend to cause fracture as the dorsal surface of the innermost foot will slide on the uterine wall.

If the foot of the innermost leg is in plantar flexion, an equinus deformity would result.

In conformity with these views pseudarthrosis is always unilateral.

It might be argued that a lower spinal lesion should accompany this condition according to my reasoning with regard to spinal malformations, but this type of pseudarthrosis is due to one leg being trapped in a particular position where it is at a grave mechanical disadvantage and the degree of maladjustment is not such as to bring about pressure on the foetus resulting in maldevelopment of the spine. The condition is akin to accidental talipes equino varus where the malposition occurs with a minimal amount of maladjustment between foetus and uterus. Serial sections of the spinal cord did not reveal any lesion.

Pressure on Joints

We have already described how pressure may effect the ankle and knee joints in the lower limb.   There remains the hip joint.

This joint comes between the sacrum and the knee joint and it has no adequate support posteriorly apart from the rim of acetabulum, to counteract any pressure on sacrum and knee.

Any pressure on the knee will normally be compensated for by pressure of the sole of the foot on the uterine wall acting along the axis of the tibia and so exerting an opposite pressure effect on the lower end of the femur at the knee.   This balancing of pressure on the hip is well illustrated by the malposition of accidental talipes equino varus in which congenital dislocation of the hip does not occur.

If this fails, as it will do if the foot is prevented from rotating sole to wall, then pressure on knee and sacrum will be concentrated on the postero-superior rim of the acetabulum due to the curve of the lower spine and pelvis, and depending on the duration and intensity of the pressure, the following degrees of instability of the hip joint will occur.

1) A strong tendency to dislocate when the child walks, due to under-development of the acetabular rim.

2) Various degress of subluxation present at birth.

3) Complete dislocation of the hip present at birth.

The intensity of the pressure will vary in inverse proportion to the degree of pressure exerted by the uterine wall and will be most apparent when the disproportion is minimal but constant.   The hip will be weakly supported and all pressure on knee and sacrum will act directly on the acetabular rim.   All further degrees of pressure will result in closer apposition of the limb to the body, greater support for the hip and indirect pressure on the foot acting obliquely upwards alongthe tibia, tending to cancel any pressure on the hip.

A gross example of the effects of unopposed pressure exerted along the axis of the femur on the hip joint, is seen in cases of arthrogryphosis multiplex congenita, where the legs have developed in full extension with flexed hips, and pressure is exerted on the feet and on the sacrum.   The hip joints are inadequately protected and show a strong tendency to dislocate.   As the child is very late in walking, if ever it walks, this tendency is not always apparent.

The effects of such pressure will tend to dislocate the femoral head oftener in the female foetus due to the more perpendicular walls of the pelvis.

A slight tendency to inversion in the early days of life will be the only observable abnormality of the feet and it will soon disappear.

FIG. 134.—A human embryo about 9 mm. long. (Drawn from a stereoscopic photograph.)

Yolk sac
IV Ventricle
Auditory vesicle
Hyoid arch
Mid-brain
Mandibular arch
Maxillary process
Heart
Eye
Upper limb
Olfactory pit
Somite
Vitello-intestinal duct
Umbilical cord
Liver
Tail
Lower limb
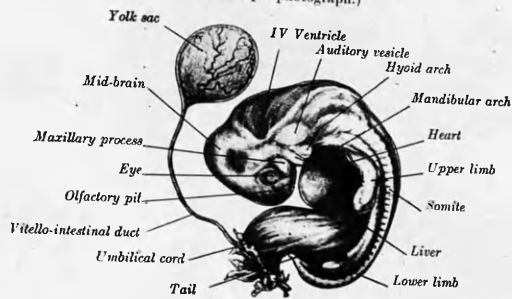
F 1 G. 16. (Gray's Anatomy)

FIG. 135.—A human embryo about 15 mm. long. (Drawn from a stereoscopic photograph.)

Roof of IV Ventricle

Developing external auditory meatus

Rudiments of fingers
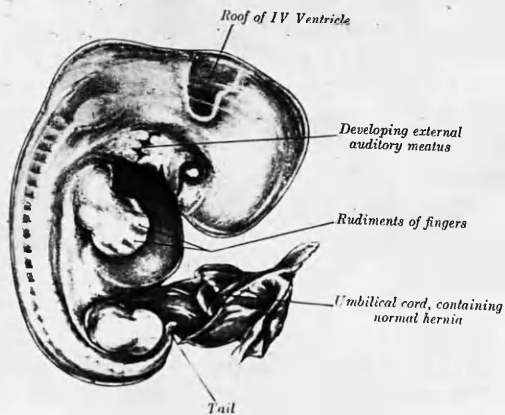
Umbilical cord, containing normal hernia

Tail

F 1 G. 17. (Gray's Anatomy)

I must emphasise that these pressure effects will occur only in the crossed leg position apart from legs in full flexion, congenital angulation of tibia and pseudarthrosis of tibia.

With the legs side by side in plantar flexion, compensating pressure will be exerted through the dorsum of the foot in an oblique direction upwards, and the closer apposition of the thigh to the abdominal wall would also tend to neutralise pressure on the knee, until angulation or pseudarthrosis occurs, when pressures on the knee will be transmitted directly to the acetabular rim without counterbalancing pressure. In addition, the internal rotation of the thigh in these cases will render the hip joint less stable.

This is well exemplified in Dott's case as described by Middleton, where, in a case of Arthrgryphosis multiplex congenita affecting upper and lower limbs in a male infant, a congenital dislocation was present in one hip and an exaggerated coxa vara in the other.

The condition can be unilateral or bilateral according to whether one or two feet are inverted, but never quite equal on both sides due to crossing of the legs and hence variation in angle of pressure. The outer leg will suffer most.

On the rare occasions when congenital dislocation of the shoulders occurs, it will be due to one arm lying wholly in front of the other, causing prominence of one elbow, hence a backward dislocation by pressure. Disproportion is minimal in these cases.

Spinal Malformations

Figs. 16 and 17 show how acutely curved the foetus is in the early stages of development and comparison with later foetal forms demonstrates the fact that the spine straightens as it develops. The head, the most acutely flexed part (it is in contact with the umbilical cord at the 6th week, and may almost touch the toes), is extended by the development of the face in the 3rd month and the spine follows as the legs develop. The most acute flexion of the spine occurs towards the lower end as can be seen. The use of the word "unfolding" to describe the extension of the spine as the foetus develops would not be inappropriate.

Eschricht in 1851 described this spinal curvature along with early curvature of the limbs and explained all congenital deformities as being due to an empirical arrest of development leading to defective formation of the pre-osseous elements. He, however, repudiated any mechanical factor and brought evidence to show that this acute curvature is a normal feature of the early foetus.

Denis Browne in 1936, on the other hand, states that meningocele and other spinal malformations are due to excessive bending of the developing spine by mechanical pressure exerted by the uterine wall on the foetus, so bringing about a kinking which is followed by non-union of the two sides.

I believe the true explanation lies between these two theories.

There is no arrest of development if by that is meant an inherent growth defect, nor is the spine excessively bent, being already sufficiently curved.

If it is prevented from straightening, then that would be sufficient to explain non-fusion of certain laminae. Although chondrification of the vertebrae begins in the 7th week, the laminae do not fuse until the 4th month.

If disproportion between the growth of the foetus and uterus towards foetal preponderance occurs early in pregnancy, the results will first be seen in the 3 - 4 months as the foetus begins to fill the whole uterine cavity.

Cephalic and caudal extremities will take the brunt of any disproportion, being in the long axis, and as the head is already extended by the 3rd month, the results of retraint will be seen mostly in the lower spine. This, I maintain, will lead to non-fusion of laminae and varying degrees of malformation governed by the extent to which pressure is exerted by the uterine wall as the disproportion develops.

The part of the spine where this resistance to extension is most felt will be the lower lumbar region.

This persistance of flexion in the lower spine will result in the legs tending to develop upwards in front of the body.

In other words, the deformity of the lower limbs usually present with certain spinal deformities is not the result of a nervous factor, but is due to distorted development resulting from non-straightening of the spine.

If cases of arthrogryphosis multiplex congenita with or without spinal malformation are studied, it will be seen that there is a continuity between thigh, leg and foot, showing that although the joints are present they have never been flexed properly. The legs may develop upwards in front of the body or laterally round the body, but the knees never flex and the feet merely assume the position dictated by the constrained and abnormal position. The term club-foot should be discarded in describing these cases. This position of the lower limbs results in loss of the bracing effect to the lower spine, further prejudicing any chance the lower spine has of straightening and so completing the fusion of the laminae. Wuth the legs developing in extension they invade the regions reserved for the upper limbs and produce deformity which may assume a variety of forms. Muscle degeneration will occur in the more severe cases of disproportion and will be nutritional in origin. Middleton states "the muscular degeneration arises fairly late in intra-uterine life, after the muscle fibres are fully differentiated, and it progresses rapidly and ceases at birth", when the pressure is relieved and nutrition improves.

In this lies the explanation of cases of talipes which apparently adequately treated, relapse after a period. These cases have been beyond explanation up till now and although the usual explanation is that treatment was abandoned too soon, this has been felt to be quite inadequate.

They relapse only after some years when it appears that cure is complete, the reason being that the disproportion causing the talipes occurred early enough to cause a minor spinal lesion which only comes to light as the child grows and traction is exerted on the spinal cord. This traction results in changes in the plantar ligaments fully described from p.23 onwards.

As weakness of the leg muscles develops the foot tends to adapt the inverted position, the foot "heels over" just as people with weak ankles complain of "going over", weight is borne on the outer side of the foot more and more and the club foot relapses. It is a gradual, uncontrollable, unconscious and insidious process. Weakness of the leg muscles and contracture of ligaments is overshadowed by the awful progress back to club-foot. The paralysis is never absolute, the feet are distorted and the true explanation is missed.

So that a nervous factor may well exist in cases of talipes, but it is one which only operates in the late stage and is inconsistant.

This explanation fits the facts, for, although the vertebrae are deformed, and the spinal cord in many cases, the elements of the spinal cord are always present.

It goes far in throwing light on spinal malformation, such as spina bifida occulta and membrana reuniens, centreing round non fusion of the laminae, but it does not explain other malformations such as hemi vertebra, or anterior rachischisis, or complete rachischisis cleido cranial dysostosis, Sprengel's shoulder, which are more in the nature of products of dysphasia.

So that it may be postulated that the existance of spinal malformation concerned with the partial or non fusion of laminae indicates that disproportion between foetus and uterus towards foetal preponderance occurred early in pregnancy and is always associated with other stigmata, although they may be of a very minor character.

And, deformity of the lower limbs which does not increase in severity as the spinal cord relatively shortens in the spinal canal, indicates that the disproportion only exerted its full influence after the spinal canal was fully closed, and the components of the spinal cord fully developed.

The development of the lower limbs in full extension with flexed hips without obvious spinal malformation, is also capable of explanation with the above principles, and if they develop over instead of under the arms, then hyperextension of the knees would be a natural sequel, a deformity further accentuated by extension of the thighs as the quadriceps would have developed in the shortest possible length.

The deformities seen at birth in cases of pes cavus, and the associated spinal malformation are products of the same cause. It is likely that this lesion arises due to the pressure effects of disproportion becoming operative in the late stages of closure of the spinal canal, bringing about a foot deformity associated with a minor spinal deformity, the effects of which are apparent later in life when the spinal cord shortens in the bony canal. The spinal deformity will vary from an obvious spina bifida to a condition where there exists a membrana reuniens or maldeveloped filum terminale without a detectable bony defect.

Beykirch believes there exists a myelo dysphasia in 50 % of these cases, but such a stipulation is unnecessary, and if it was present would be obvious much earlier in life.

The effects of the spinal cord lesion are only apparent after 7 - 8 years of life and there has been no evidence brought forward to repudiate the belief that there is in all cases a membrana reuniens or maldeveloped filum terminale.

The occurrence of spina bifida of the lumbar region will require only a minimal degree of disproportion occurring before closure of the laminae and need not be followed by deformity of the lower limbs.   However, it is probable that in the great majority of cases, a certain degree of calcaneo valgus leading to a minor degree of pes valgus after birth is present, or minimi digiti quinti varus. Close examination will no doubt reveal other stigmata.   Parker described some cases of spina bifida, seven of which showed no deformity of the feet, but he was obviously looking for gross evidence of deformity, which will be unusual, as degrees of disproportion which give rise to well-marked club-foot, if it operates before closure of the laminae, will bring about much grosser spinal malformations than spina bifida. Any variations are possible, but minor degrees of disproportion will tend to give minor deformities of spine and lower limbs and major degrees, severe deformities of spine and lower limbs.

PES   CAVUS

The final shape of the bones of the foot decide its form, the ligaments maintain this form and the muscles render it functional.   The function being naturally of an intermittent nature, the elastic tissue present in the ligaments is able to reconstitute the form of the foot after active distortion, unless such distortion is of a prolonged nature, when the elasticity or recoiling power of the ligaments is exhausted.

The strong ligaments of the foot predominate on the plantar aspect and further, the ligaments on the inner part of the plantar aspect of the foot are stronger than those on the outer.   This follows because the axis of weight-bearing falls to the inner side of the calcaneus, as is shown in Fig. 569 (Gray's).

The foot should be regarded as a forked appendage, the body of the talus being at the apex of the fork, and the heel and metatarsal heads forming the points. Viewed laterally, the anterior prong is longer and more oblique than the posterior. Viewed antero-posteriorly the posterior prong is directed slightly laterally and ends on a blunt point, i.e. the under-surface of the calcaneus, while the anterior prong is directed slightly medially and on a base represented by the head of the first metatarsal, the other metatarsals acting as an outrigger to steady this position. This is necessary if the spreading effect of depression of the apex is to be minimised and the elasticity of the longitudinal arch centred at the apex.

So that when the body is erect on one or both feet, the axis of weight-bearing lies to the inner side of the calcaneus and the prongs of the fork are the body of the talus and the calcaleus posteriorly and,anteriorly, the neck and head of the talus, the navicular, first cuneiform and first metatarsal.

In motion, as the body goes forward and the heel rises from the ground, the axis of weight-bearing travels along the neck of the talus until it reaches the head of the talus. In other words, the apex of the fork moves forward along the head of the talus.

Also, in order that the function of walking may take place without
a sideways lurch, the actual area of weightbearing, must describe part of
a circle or curve round this axis.　　　As the C.G. is thrown forward and to
the side on one foot, so must counter pressure be exerted to maintain its
persistence in a straight line, so that the weight bearing point on the foot
proceeds from the heel, along the outer side of the foot and then back across
the heads of the metatarsals to a point of neutral balance as opposed to
active balance, being again in line with the axis of weightbearing.

The foot is beautifully fashioned to carry out this function, but as
the action is of a very complex nature, any discrepancy in the various parts
of the foot will be shown by disordered action.

One of the tissues of the foot which has been given scant attention
up-to-date is the elastic tissue contained in the ligaments.

A great deal of literature has been written to elucidate the nature
of elastic tissue contained in the respiratory tract, the vascular system,
and the skin etc., but as far as it is possible to ascertain, no information
is available regarding the elastic tissue contained in the ligaments of the foot.

Hass, 1939 wrote exhaustively on the nature and function of elastic
tissue and several conclusions were drawn which are of considerable interest
when applied to the function of the foot.　It is believed that physical
factors of stress and strain were of great importance in the development of
connective tissue structures.

There is considerable controversy as to the exact origin of the elastic
fibre.　Does it first appear as a fibril, or is it due to the fusion of
granules in the intercellular substance.　Does the fibroblast give rise to
undifferentiated fibrils which are transformed to a collagenous or elastic
element.　Is the elastic fibril formed by a specific cell called elastoblast
or is it due to an alteration in the nature of collagenous fibrils.　All
these theories have considerable support but no conclusive evidence has been
produced.

Hass describes five functions of elastic tissue in any viscera.

1) It is so placed as to aid in the dissemination of stresses directed
at isolated points.

2) It is designed for coordination of rhythmic movements of certain units.

3) It aids in the conservation of energy through partial maintenance
of tone during relaxation of muscular elements,

4) It serves as a bulwark against the possible injury of excessive forces.

5) It aids materially in the return of a tissue or organ to the natural
form after a deforming force has been removed.

The effect of fatigue on the elasticity of connective tissue was studied
by Schade who demonstrated that a sleepless night diminished the elasticity
of the skin by 15 per cent.　Katzenstein found that prolonged active and
passive movement of joints, substantially increased the capacity of the joints,
presumably due to relaxation of the joint capsule.

Further, what investigators have learned of the embryology, anatomic distribution and in vitro cultivation of elastic tissue, tends to support the hypothesis that mechanical forces may exert an influence on the genesis and development of the elastica, especially if these forces are rhythmic and fluctuating. Also it appears that future investigation will be concerned with a substance or substances which is elaborated and deposited in elective sites. It may be predicted (Hass) that such substances will be of such a labile nature as to be resorbed, redeposited and augmented in response to certain physiological stimuli or as a result of certain pathological processes.

Finally, if degeneration of elastic tissue progresses to a measurable degree from the morphological viewpoint, there is a limited capacity of the tissue to regain its structural integrity. Regeneration of the tissue does occur occasionally but the replacement is meagre and imperfect.

All the above facts have a direct bearing on the aetiology of ideopathic pes cavus, for what is this condition but a lesser degree of paralytic talipes equino varus. It merges into and cannot be distinguished from this condition in its most severe forms.

Pes cavus occurring in late childhood, or superimposed as a relapse in the treatment of a foot deformity is due to the late effects of a lower spinal lesion. The exact nature of this lesion remains in doubt although the presence of a membrana reuniens has for long been the accepted explanation. This lesion, a fibrous band extending from the skin round the cord, was thought to explain the absence of sensory signs, the fibrous sling causing pressure on the anterior horn cells alone.

Lately Beykirch has put forward the belief that there is a myelo dysplasia present but if this were so, its effects would be evident much earlier, *although the myelo dysplasia he describes is in very general terms*.

A typical case of ideopathic pes cavus shows a minimal paralysis of the leg and foot, plus certain nutritional changes which indicate involvement of the sensory nerve supply. Difficulty of micturition may occur, hyperidrosis of the effected extremity, associated with coldness and blueness. Callosities are common and intractable, and hyperkeratosis almost invariable. Actual sensory changes have been described but are uncommon.

These changes are not seen in the early stages of the condition, but occur and are marked later.

I believe that the first changes which occur in a case of pes cavus, take place in the structure of the ligaments of the foot. Degrees of motor and sensory disturbance brought about by traction on the spinal cord, not enough to cause definite signs in themselves, are yet sufficient to affect the elastic element of the powerful plantar ligaments, causing a loss of elasticity and a replacement fibrosis leading to contracture. The ligaments of the dorsum are not designed to withstand any degree of strain, as are the plantar ligaments, and any nutritional changes causing imbalance between these two sets of structures would result in considerable increase in the longitudinal arches of the foot.

This has been recognised as far as the long and short plantar ligaments are concerned but insufficient attention has been paid to the lateral and plantar calcaneo navicular ligaments and the widespread insertion of the posterior tibeal muscle.

The intrinsic changes in the ligaments have attracted no attention.

The slight motor paralysis of the first metatarsal is caused by contracture of the calcaneo navicular ligaments, one of which is the powerful spring ligament. This results in an approximation of the calcaneus and navicular, the head of the talus acting as a wedge. The calcaneus being fixed posteriorly, the navicular will be pulled round the head of the talus, and the head of the first metatarsal will drop. This is merely an accentuation of a normal resting position and is not due to muscular action. The head of a normal first metatarsal can be pushed up when at rest.

If the resting foot is studied it will be seen that all the metatarso phalangeal joints are hyperextended and remain so until the foot is placed on the ground, when this hyperextension is lost by raising of the metatarsal heads, and the toes then come in contact with the ground.

As contracture of the plantar ligaments occurs, the resulting distortion of the longitudinal arch effects the anterior component entirely, so leading to apparent shortening of the Tendo-Achilles as suggested first by Percival Mills, 1924. The navicular is pulled round the head of the talus, later the cuboid round the anterior articulating surface of the calcaneus so that the line of the tarso-metatarsal joints is altered and the metatarsal heads appear to drop. At first this deformation is reducible as the fibrosis does not extend to the ligaments and capsules of the tarso-metatarsal joints. In other words the mid-tarsal joint is the first affected. Later increasing and spreading fibrosis fixes the deformity. The apparent dropping of the metatarsal heads, causes the first phalanges to adopt a position of increased hyperextension at rest, and when weight is borne by the foot, the times comes when such weightbearing is insufficient to raise the metatarsal heads and allow the toes to reach the ground. The claw toe deformity is then established.

The flexor and extensor tendons are unable to adjust themselves to the changed position. Tension of the extensor tendons causes hyperextension at the metatarso phalangeal joints and tension on the flexors causes flexion of the interphalangeal joints.

In the early stages, tenotomy of the extensor tendons will be sufficient to correct the hyperextension at the metatarso phalangeal joints and allow the toes to reach the ground when the foot is weightbearing.

As the replacement fibrosis extends the preponderance of the medial plantar ligaments causes inversion of the foot and approximation of the head of the talus to the medial malleolus, the final stages being almost indistinguishable from paralytic talipes equino varus.

Complete contracture and fibrosis of the plantar ligaments has often been described, and the role they played in maintenance of the deformity of club-foot has been frequently commented upon, first by McKeever in 1819 and again by Parker in 1884, who recommended syndesmotomy of all medial plantar ligaments as part of the treatment of club-foot.

The reason for this contracture and its exact role in the aetiology of various foot deformities, has not, to my knowledge, been previously commented on.

Considerable further study is necessary but in the disposition, variation and reaction to various pathological and physiological stimuli, of the elastic tissue of the foot, lies the answer to many problems at present insoluble.

The exact lesion of the lower spinal cord is still in considerable doubt, but I venture to state, from a study of the development of the lower spine, that the lesion is probably a failure of full development of the filum terminale whereby it drags on the spinal cord when the upward shift takes place. This brings about varying but minor degrees of paralysis of the muscles supplied by the sacral nerves and minor degrees of sensory disturbance in the area of distribution of the sacral nerves most noticeable in its effects on the highly specialised nature and function of the elastic tissue of the limb. This explanation also accounts for disturbances of micturition if present.

This would only apply to the bilateral cases. Unilateral cases are probably explained by the presence of a previous deformity such as a well marked degree of metatarsus varus which precludes proper development of elastic tissue and eventually results in pes cavus.

Todd describes a case in which a transverse cut across the lower third of the leg damaging the extensor tendons, eventually produces a pes cavus. The photograph of this patient shows a generalised atrophy of the leg from the knee down and the condition would appear to have arisen due to disuse atrophy plus distorted weightbearing, the most marked feature of which was contracture of the plantar ligaments causing pes cavus.

Deformities of the Upper Limb which result from disproportional growth, will in most cases follow and result from malposition of the lower limbs, but an isolated club-hand deformity is possible if a palmar flexed hand, dorsal surface to chest wall, is propelled by pressure on the elbow into the axilla, becoming trapped between anterior and posterior walls. Further pressure on the upper arm will cause further flexion of the hand, resulting in the typical deformity which will only be released at birth when abduction of the arm becomes possible. Only a minimal degree of pressure would be necessary in this case.

Treatment of Congenital Deformities due to
Disproportional Growth between Foetus and Uterus:

The ultimate cure of congenital deformities as described above, lies in the study and practice of eugenics, a science which is assuming more importance as civilisation advances to

"the ultimate aimiability of all things"   Swinburne.

If war, disease, starvation and overcrowding are to be abolished
throughout the world, then the corollaries of such planning must be
faced and studied. Controlled eugenics is one of the most important ,
and may not be too far away. Magistrates can already order medical
examination, in certain cases, prior to marriage. The seed is
bearing fruit !

That "The child is father of the man", is well exemplified in
congenital deformities, but so much is the canker of specialisation
invading the art of medicine that the correlation of cause and effect
is becoming much more difficult.

Denied prophylaxis, on such a wide basis at present, the deformities
must be approached individually and treated according to the factors
causing them, and the time which has elapsed since birth.

The whole approach to congenital deformities requires revision.
Diagnosis and treatment must occur as early as possible, delay can
only result in confusion and bad results. More careful examination at
birth and in the early days of infancy is necessary to detect minor
degrees of deformity, which are easily corrected at an early stage, but
if neglected for several years lead to life-long crippling effects.

Accurate diagnosis of the various deformities and a thorough
understanding of the causes which lead to them is essential if locomotion
among the masses is to improve.

My own personal experience of these various deformities is as yet too
small to justify any detailed statements with regard to treatment, but
certain principles can be enunciated without prejudicing any particular
form of treatment which might be adopted.

It will be appreciated from the above discussion that apart from
the cases of obvious deformity, there must be many minor degrees of
distortion of the bony elements of the foot occurring in utero, which will
give rise to foot trouble later in life.   Minor degrees of valgus will
result in flat foot deformity. Any constraint placed on the feet in
utero, will result in some modification in the form of the bones, which, later,
by giving rise to minor disorders of locomotion, will so alter the
mechanics of the feet as to prevent or destroy the correct function of
elastic tissue and so lead to late foot deformity.

Missed cases of metatarsus varus, which appears sometimes to be
mistaken for pes cavus, by the persistence of distorted weightbearing,
will prevent the correct functioning of the plantar ligaments, leading eventually
to degeneration of the elastic tissue and increased deformity.   This calls
for a much more careful examination of infant's feet and more frequent
recognition and diagnosis of minor degrees of deformity, which can be
treated and corrected before the child walks, or at least, before the
deformity is fixed.

Forcible manipulation is contrary to all the facts available in relation to the function of ligaments in the vicinity of joints. This is becoming recognised with regard to the treatment of congenital dislocation of the hip, but requires emphasis and explanation of the principles involved.

It is a well recognised fact that ligaments can be intermittently stretched with no loss of their intrinsic elasticity, but if the stretching occurs suddenly and forcefully, tearing of the ligament occurs and such a tear is repaired by fibrous tissue which lacks elasticity and prejudices the chances of the ligament resuming normal function. In the same way, constant stretching of any ligament, eventually leads to lengthening combined with loss of elasticity. For any ligament to function properly the strain placed on it must be intermittent. Under constant strain it becomes overstretched and in such a condition acts at a disadvantage. It cannot contract, its functional use disappears and the ligament fibroses.

So that any splint used in the treatment of deformity should hold the limb in the position attained by gradual manipulation, but no constant stretch should be put on the ligaments. Infants Splints used on infants are so difficult to apply that this overstretching is probably only maintained for a short time and no real damage is done. However, the principle remains.

Forcible manipulation in the early treatment of foot deformities in children is to be particularly condemned for the above reasons. The ligaments must be given time to stretch without tearing, and once this is accomplished the foot must be held in the corrected position until such time as the bones have become remoulded. They, the ligaments, must be regarded as already prejudiced with regard to the retention of their full complement of elastic tissue, and no steps must be taken which would prevent regeneration or full development of elastic tissue.

To carry out this form of treatment masseurs would require to be trained in the correct manipulation of these deformities, as it requires accurate knowledge of the anatomy and function of the foot, but would not require the constant attention of a specialist after accurate assessment of the lesion.

With regard to the case of accidental club-foot described above, treatment consisted of daily manipulation by me for a period of three months, the mother being instructed in the manipulation and encouraged to carry it out. Most mothers will not do it but this one did. This consists of rotating the foot outwards, abducting and dorsiflexing it at the talo-navicular joint. The talus is not displaced from its mortise in this deformity so that the fore-foot adducted, is rotated and inverted round the head of the talus.

The principle points where opposite pressure is exerted in the manipulation are the prominent head of the talus on the outer side of the foot, and the outer border of the foot in front of this.

This was followed by the use of the Denis Browne splint until I had to give the case up. I heard later that at four years of age the child walked normally.

This is the treatment I recommend for all cases of classical talipes equino varus, and also for the cases of third degree crossed inversion deformity described. The foot must be made malleable and loose, the deformity capable of easy correction. The Denis Browne splint should be used to maintain the reduction, not to reduce the deformity as it is in many cases. A trained masseuse should be used for the preliminary stage, the emphasis being placed on manipulation of the foot into the correct position, the splint being a very much secondary consideration. No splint, Plaster of Paris, or strapping will maintain the foot in sufficient accurate reduction until it (the foot) has been rendered sufficiently malleable by oft repeated manipulation. Distortion will occur in more severe cases and bad results are inevitable.

Theseprinciples apply to the correction of all the congenital deformities of the limbs under discussion, if the condition is correctly diagnosed and treated from birth.

In the atypical cases of club-foot, such as those associated with arthrogryphosis multiplex congenita and with spinal malformations the problem is more complex. With no spinal involvement, the treatment must be gradual correction by manipulation. If the legs have developed in extension with hips flexed, intelligent manipulation to stretch all the contracted tissues must be carried out over a period.

One has on occasions heard a nurse say, "Every time we undress the child, its legs shoot up over its shoulders". This is an admission that the implications of the deformity are not appreciated. The structures anterior to the hip joint are contracted and require time and manipulations to stretch them. Only harm can come of rough handling.

In these cases the whole limb requires re-moulding and this should commence from the first day of birth, be frequent, and not include the use of splints for some time.

If the spinal cord is involved normal voluntary control of muscles does not occur as the child develops, and this seriously prejudices any attempt at correction.

Once the deformity becomes established in later childhood, various operations have been devised for correction. They are admissions of failure to treat correctly.

The treatment of congenital dislocation of the hip has been exhaustively gone into and will not be dealt with here except to say that early diagnosis is absolutely essential. Too many cases are still missed in the early stages, which could have been prevented and too many still go on until symptoms of arthritis finally drive them to an orthopaedic clinic.

X-ray of newly born babies and in early infancy should be much more
frequent when any sign whatsoever of lower limb constraint in utero
is displayed. The practice of holding newly born babies up by the heels
should be abolished. The structures anterior to the hip joint are
contracted to some extent at birth and require a little time to stretch
and rough interference may precipitate a tendency to subluxation which
would otherwise remain latent, the period between birth and walking being
enough to allow the acetabular rim to reform.

The treatment of congenital dislocation of the hip will depend
to a great extent on the degree of the condition. If complete luxation
occurred early in foetal life, open operation will be necessary, as the
structures surrounding the joint will have adapted themselves to the changed
conditions and reduction by manipulation will be inadequate.

If the condition is a subluxation, or a luxation occurring
after birth and diagnosed early, then reposition of the femoral head in
the acetabulum, combined with recumbency until the acetabular rim forms
properly is likely to be successful.

The two movements which must be prevented are adduction and flexion
of the hip. An apparatus allowing abduction and extension would be
sufficient to keep the femoral head in position, and at the same time
allow the limb to develop normally. Fixation for prolonged periods can
only lead to degeneration in the surrounding structures at such an early
stage of life.

Open operation for congenital dislocation of the hip should not
be a matter of opinion but should be the result of full investigation
and the knowledge that manipulation will inevitably fail if the luxation
occurred early in intra-uterine life.

Pes Cavus :

A much closer study requires to be made of the treatment of
congenital pes cavus. There is always a foot deformity present in these
cases, usually bilateral and unequal, followed later at seven to eight years
of age by the development of the typical lesions, six degrees of which are
usually described. Fifty per cent show a spina bifida and Beykirch claims
a myelodysphasia causes the condition in the other fifty per cent.

A more forthright approach should be made to the spinal lesion
in these cases !

As regards operation on the foot. Transplantation of the
extensor tendons to the metatarsal heads as suggested by Mark Jansen and
described by Todd, will correct the hyperextension at the metacarpo-
phalangeal joints, but will have no effect on the deformity of the mid-tarsal
joints.

The progress of pes cavus may halt at any stage from high arch to the final equino varus deformity, so that radical forms of treatment may never become necessary, but if the lesion does progress, syndesmotomy is a form of treatment which offers considerable scope, but it must be radical.

Standler's operation is inadequate. Parker, and more recently Todd, advise syndesmotomy but both studiously avoid the calcaneo navicular ligaments. Both describe division of the astragalo scaphoid capsule and the long and short plantar ligaments : for full reduction of the deformity of the longitudinal arch, the calcaneo navicular and calcaneo-cuboid ligaments, would require to be divided. This would probably result in various degrees of pes planus but a useful foot would be obtained and lengthening of the Tendo-Achilles would be unnecessary.

Pseudarthrosis of the Tibia:

Elucidation of the aetiology of pseudarthrosis and angulation of the tibia would appear to offer little assistance in the treatment of these conditions. With regard to angulation of the tibia gradual manipulation from birth onwards to correct the equinus deformity, followed by operation to correct the angulation and so prevent the onset of a stress fracture, would appear to offer the best line of treatment.

Once a stress fracture has occurred the contracture of the muscles of the leg, by reason of diminished muscle range, associated with angulation of the fragments and lessened blood supply will lead to delayed union. If treated in plaster, delayed union will lead to non-union in most cases. Internal splinting to give firm immobilisation of the fracture would be the correct proceedure, either by massive dual onlay grafts (Moore 1941), or by vitalliam plates.

Pseudarthrosis of the tibia occurring early in intra-uterine life offers little scope for satisfactory surgery but knowledge of the pathology will lead to proper classification and concise reasons for the particular form of treatment carried out.

With regard to the further investigation of the problem of congenital deformities, the study of intra-uterine position can be further advanced by the development of X-ray technique.

This would require an enormous amount of spade work, but in my opinion, would be the logical mode of investigation, and is in fact, the only mode of investigation at present possible.

Routine X-ray, preferably stereoscopic could be made at seven months in all pregnancies at certain clinics where the rate of congenital deformity is known. Over a period the correlation of these with any deformity present at birth, suggests a line of enquiry which would probably give results if sufficiently well adhered to. The diagnosis of fixed malposition would become possible.

I suggest that the condition now known as classical talipes equino varus should be called accidental club-foot. The hereditary slur would thereby be banished, and it would conform more to the aetiology of the

condition.

Talipes equino varus should be reserved for the cases of third degree crossed inversion deformity where disproportional growth is marked.

Congenital deformities arising from disproportion between foetus and uterus towards foetal preponderance can now be classified thus : -

A. Disproportion effects accurring after full formation of the spine.

Minimal degrees may lead to :-
   a) Congenital dislocation of the hip unassociated with other deformity.
or b) Accidental club-foot, always unilateral.
or c) Congenital dislocation of the shoulder.] [Moderate to fairly severe degrees may lead to : -

With trapped feet in crossed inversion.
      a) Minimi digiti quinti varus.   ) Unilateral or
   or b) Metatarsus varus.   ) Bilateral.
   or c) Talipes equinus varus.   )

With feet trapped in crossed eversion (sole to uterine wall).

      a) Moderate degrees of calcaneo valgus.
         Unilateral or bilateral.

With legs crossed, the outer foot in plantar flexion.

      a) Pseudarthrosis of the tibia.

With feet trapped sole to uterine wall and side by side.

      a) Bilateral calcaneus.

   or b) Unilateral severe calcaneo valgus.

With feet trapped side by side and inverted.

      a) Minimi digiti quinti varus.

   or b) Metatarsus varus.

   or c) Various unique deformities.

With feet trapped side by side, one or both in plantar flexion.

   One in plantar flexion:

      a) Equinus.
   or b) Unilateral angulation of the tibia.
   or c) Pseudarthrosis, always unilateral, with severe degrees of pressure.

   Both in plantar flexion:

      a) Bilateral equinus.
      b) Bilateral angulation of the tibia.

B.   Disproportion effects occurring shortly before complete closure
     of the spinal canal.

          a) Spina bifida.
     or   b) Spina bifida occulta, with or without membrana
     or   reuniens, and associated foot deformities, leading to
             pes cavus.
     or   c) Development of legs in extension with hips flexed.

C.   Disproportion effects occurring before laminae have fused.

          a) Meningocele to myelocele according to degree of
             pressure, all associated with deformities of the lower
             limbs of varying degree.

          Conditions such as hare lip and cleft palate suggest a
disproportion occurring earlier than in group C., with probably some
temporary generalised pressure effects.

          Severe degrees of disproportion may lead to death of the
foetus, and the post-mortem changes in the muscle tissue will further
complicate the picture presented.

          The hereditary factor in congenital deformities can be
satisfactorily explained by the above account of the origin of
congenital deformities.

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