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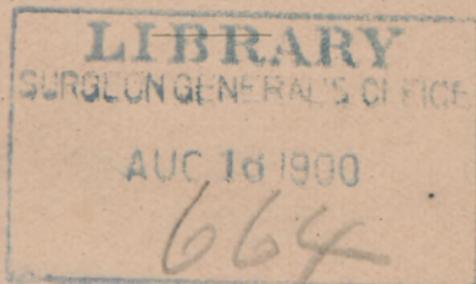
BY

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CONGENITAL DEFECT OF THE FIBULA.

BY F. J. COTTON, M.D., AND A. L. CHUTE, M.D.

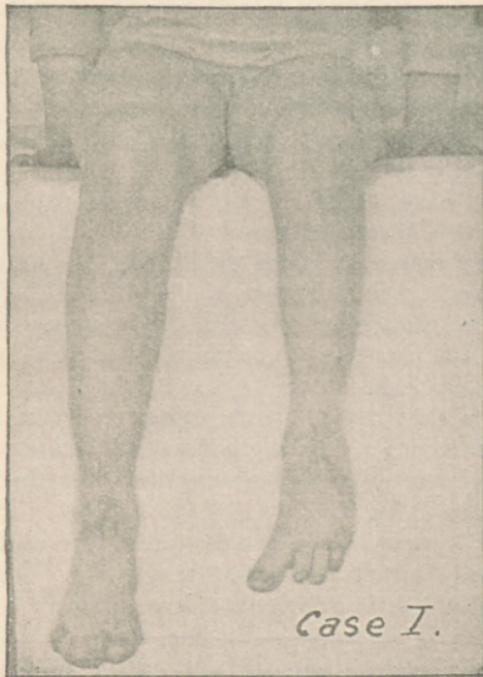
THE cases we have to report are of those classed in the literature either as congenital defect of the fibula or as intrauterine fracture of the tibia, according as the one or the other feature has chanced to impress the individual observer.

The first case, G. H., a boy of seven years, was an inmate of the Marcella Street Home. There was no history of trauma at birth or before, no deformities noted as having occurred in the family. The deformity was first noticed when the child began to walk. The limp then noticed has rather increased with his growth, though the disability has never been extreme. The boy is now robust, and, except for the left leg, normally formed. This leg shows marked shortening, the tibia shows a bend forward, and the foot, which has but four toes, is in marked valgus. The total shortening is two and one-half inches, one-half inch of which is due to shortening of the femur. The girth of the left thigh is two inches less than on the right. The patella is distinctly smaller than on the sound side. The circumference of the calf is one and one-quarter inches less on the left. No trace of the fibula is to be felt. At the middle part of the tibia there is a distinct bend forward. Over the most salient point of this bend is a faint scar-like line something over one-half inch in length.

The foot shows no equinus, and the whole sole is used in walking. The foot is, however, markedly

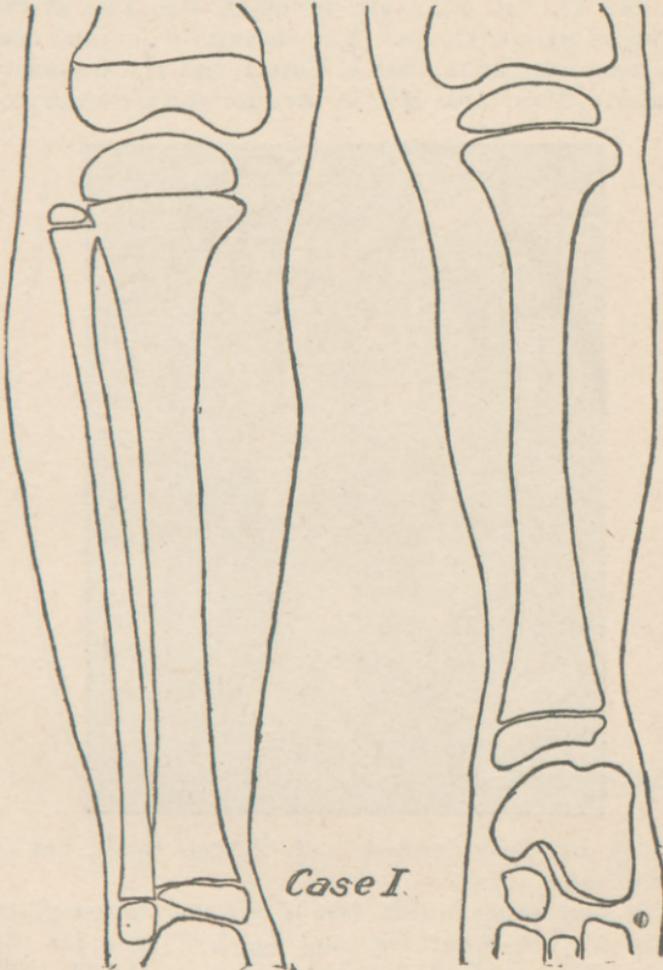
everted when weight is borne on it, the inner malleolus coming much nearer the ground than in the sound foot. There are but four toes, the great toe being considerably larger in all dimensions than that of the other foot, the other toes of normal size and shape.

Examination with the fluoroscope and x-ray photo-



graphs showed no trace of the fibula. There was defect of one toe and of the corresponding metatarsus. The astragalus, calcis, and probably the cuboid, are represented by a fused mass of bone of irregular shape; the scaphoid and the three cuneiforms were normal, at least in position.

The boy had, as a result of the shortening, a marked scoliosis, with rotation, the dorsal convexity to the left;



this disappeared on lying down. He had a marked limp, but never had any pain, and was active. The

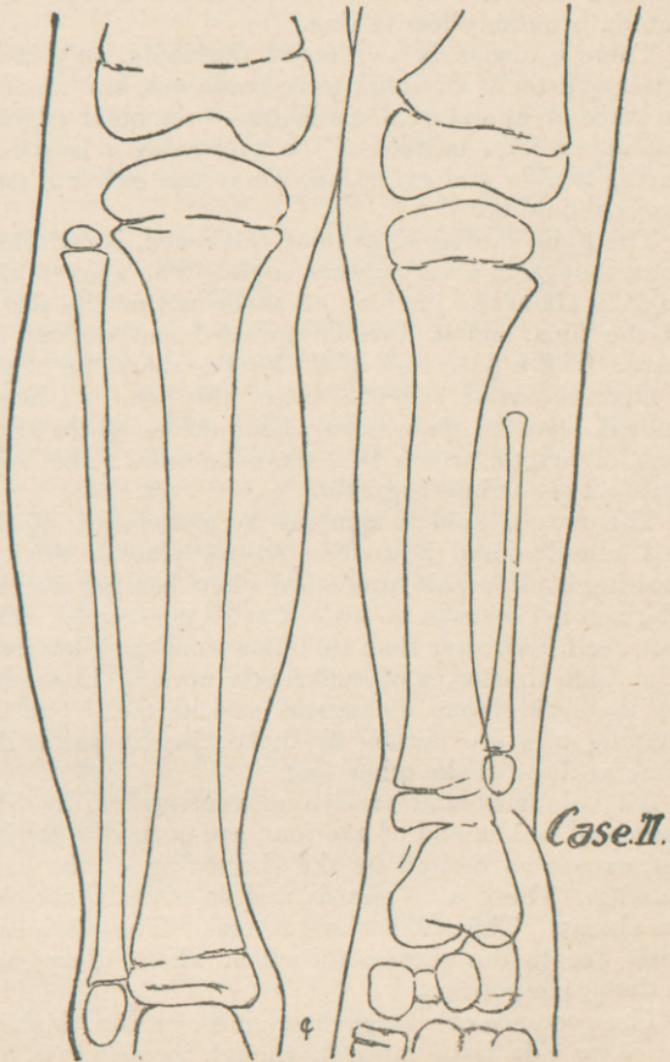
boy left the institution shortly after being seen, and has never had other treatment than a high sole.

CASE II. E. F., a girl of eight, was also at the Marcella Street Home. The deformity in this case was very similar to that in Case I, but the disability greater. This child also is, save for the deformity of



the leg, normally formed, and, though small, yet of strong frame.

The shortening in this case is two and three-eighths inches for the lower leg, and one-half inch for the thigh. The whole limb is evidently atrophic, the difference in girth of the two thighs being three-quarters of an inch; of the calves, two inches difference. The



femur at its lower end is obviously less in width. The patella is notably less in size.

There is obviously a defect of the fibula, only in its lower quarter is anything to be made out, and there is no trace of an external malleolus — the outer edge of the upper joint surface of the astragalus is palpable, during flexion and extension, where the external malleolus should be.

The tibia, shortened but not thickened, is definitely bent, though at a very obtuse angle. The apex of this angle is about the junction of lower and middle thirds of the tibia, and is directed forward and slightly inward. There is no sign of thickening about the angle.

Over the most salient point of this bend is a longitudinal scar-like depression, about three-eighths of an inch in length, which is more adherent to the bone than is the surrounding skin.

The foot is held in equinus by contraction of the calf muscles, and is used in strong plantar flexion, showing considerable pronation when bearing weight. The peronei seem to be little if at all contracted. The foot itself, narrower than its fellow, and three-quarters of an inch shorter, is of sufficiently normal shape save for the lack of one metatarsus and its toe. The remaining toes are normal in shape; in size about the same as those of the other foot.

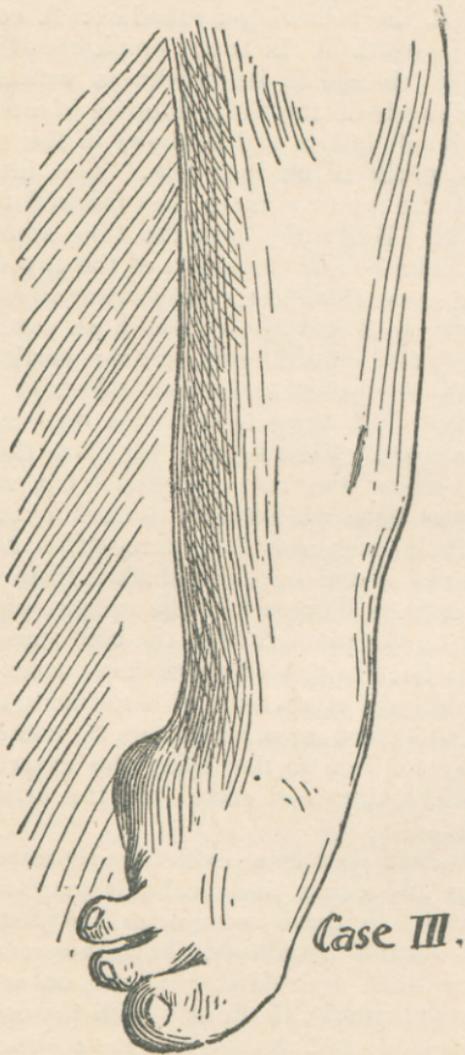
All voluntary and passive movements of the hip, knee and ankle, and of the foot, are normally carried out, except as limited by the shortening of the calf-muscles. There is no paresis, and no essential muscles are absent. The reflexes are normal. There is a scoliosis due to the shortening which wholly disappears in the prone position.

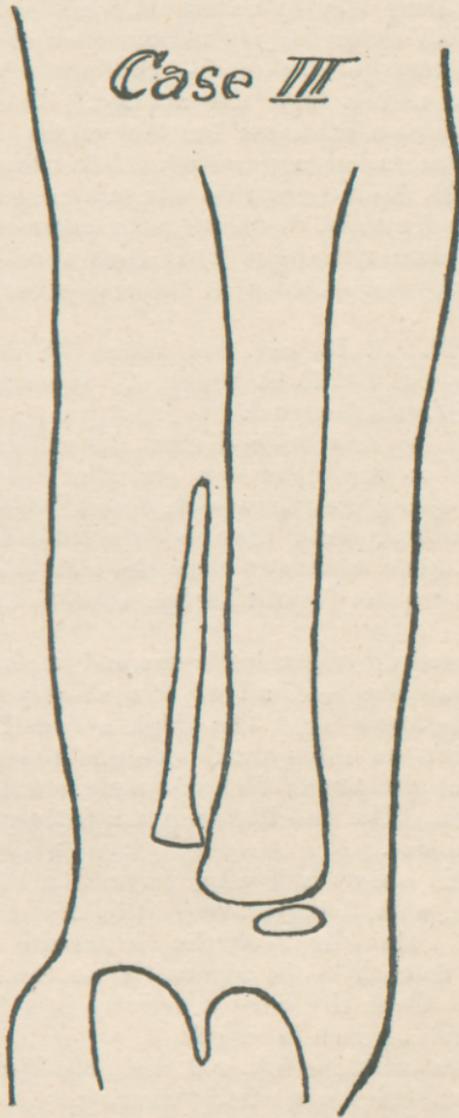
The presence of a large hairy mole on the thigh of the lame side is of interest, though probably of no significance.

The x-ray examination showed a normal right leg; on the left the following anomalies: A considerable decrease in width of the lower epiphysis of the femur, no marked change in its thickness vertically. The upper epiphysis of the tibia, likewise of normal thickness but much less in width, shows in the photograph as three-eighths of an inch less. The intercondylar groove of the femur is much less marked than on the sound side. Below the head the tibia approaches the normal thickness and the shafts of the two tibiæ are of about the same thickness. The lower tibial epiphysis is narrower (one and one-sixteenth *vs.* one and seven-sixteenths) and considerably thinner vertically. The epiphyseal line grows somewhat indistinct toward the outer side. The angle in the tibia does not show in the front view. Viewing the leg from the side with the fluoroscope one sees a definite but very obtuse angle in the bone without any thickening of the shaft.

The fibula measures but three-fifths as much in length as the sound one, the shaft is little more than half the normal diameter. The upper epiphysis does not show on the plate though the conformation of the end of the bone suggests its presence, unossified. The lower epiphysis is present, of two-thirds the normal size, but so shifted upward that its tip comes opposite the epiphyseal line of the tibia; the external malleolus, therefore, does not enter into the articulation at the ankle.

In the tarsus is a mass evidently representing fused astragalus and calcis, and probably scaphoid as well. The cuboid and three cuneiforms are distinct. The first and second metatarsals have the normal articulation, the third articulates with the outer cuneiform and with the cuboid, the fourth with the cuboid alone. The metatarsals and phalanges are, save for the defect of one toe and metatarsal, entirely normal.



Case III

Before being seen by us the child had had no treatment. With a view largely to correction of the scoliosis, a tenotomy of the tendo-Achillis was performed and a boot with a high sole applied. This reduced the scoliosis to a minimum but the valgus position of the foot was rather aggravated. Accordingly, a sole support with double uprights was so arranged by raising the heel end in the boot as to leave the foot in moderate plantar flexion. This apparatus in connection with the high-soled boot has somewhat improved the gait.

CASE III. J. R., age two years. Seen through the kindness of Dr. E. G. Brackett. Is an out-patient at the Children's Hospital.

At birth it was noticed that the child had but three toes on the right foot and that the foot was held in a queer position which, from the description of the parents, must have been a calcaneo-valgus. Under no other treatment than the mother's manipulation this "queer" position has almost entirely disappeared.

At present the right leg is one and one-half inches shorter than the left, all of this shortening apparently in the lower leg. The thigh and calf are both smaller than on the normal side and the shaft and condyles of the femur feel distinctly slenderer than on the left. The patella is not much over one-third the normal size.

The tibia shows a bowing forward a little below the middle, with, however, very little lateral deviation. Over and about the bend the tissues are somewhat thickened but there is apparently no thickening of the bone. Over the bend anteriorly is a faint scar-like line half an inch in length.

No fibula could be felt and the prominence of the external malleolus was wholly wanting.

The radiographs, however, show a rudimentary fibula about two inches in length, the lower end of which is nearly one-half inch above the lower epiphyseal line of the tibia. Nothing is to be seen of either epiphysis, but from the shape of the lower end of the fibula it seems probable that the lower epiphysis exists, though it probably, as in the last case, does not reach the ankle-joint. The upper end of the fibula, unlike that in Case II, tapers off gradually and there is no reason to suppose the presence of an epiphysis.

The foot in this case has but three toes, sufficiently normal in size and shape, and is one and one-half inches shorter and much narrower than the left. It is held in very slight equinus, while the valgus deformity is extreme. The foot can be brought up nearly to a right angle with the leg, but correction of the valgus is only partly possible. This is evidently due to contraction of the peronei, which are drawn tense long before the normal position can be attained.

Radiographs show an apparent fusion of the talus and calcis. The cuboid is much smaller than normal, — the scaphoid and the three cuneiforms somewhat small. Two metatarsals with the corresponding phalanges are wanting.

The boy, who has walked for eight or ten months, has an extreme limp and is not active, but has no pain. He is now wearing a valgus shoe with a T-strap.

In neither of the two cases (I and III) in which a complete history was obtainable could we get any account of hereditary deformity, prenatal trauma or abnormal labor.

Many cases of this sort have been reported and there has been much discussion as to their etiology, with a number of theories to account for the rela-

tively constant association of these apparently non-related deformities.

Most of the earlier observers (Gurlt, Brodhurst, Swan and others) and some of the later (as Brinton, Ithen, Braun and Busachi) have believed the bend in the tibia a result of intrauterine fracture, while the scar was supposed to indicate compound fracture. The accepted etiology was either external trauma or excessive muscular contraction on the part of the fetus. These observers had no clear theory to advance as to the cause of the defect of toes, metatarsus and fibula.

Little accounted for the fracture which he accepted as proximate cause, by supposing an overaction of muscles due to some vice of innervation — which, of course, explains nothing.

Broca supposed a defect of development with fracture from muscular action.

Adams believed the condition not an absence of the fibula but a fusion of tibia and fibula — a view not borne out by dissections.

Pressure from twin pregnancy and compression by the cord have been made to serve as causes, though with no support of facts.

Blasius noting the lack of growth of the limb in later life found this hard to reconcile with the theory of trauma as a cause, but he advanced no other theory.

Chance supposed compression in abnormal position (against other leg) by the cord to be the cause; the scar a result of decubitus.

Fricke noting that the fetus in the fifth and sixth week shows a bend of the tibia and assuming the factor in its straightening to be the growth of the fibula — was inclined to assume, in cases where the fibula was wanting, a mere persistence or exaggeration of this fetal condition.

Brinton, writing in 1884, considered the trauma primary and tried to explain the associated defects as in some vague way secondary.

Ithen, in 1885 and Braun in 1886, still held to trauma; Braun holding the fibular defect to be a predisposing cause of the fracture, in that it weakened the leg. The defect he did not explain. Recognizing the uncertainty whether the dimple over the angle was or was not a true scar, he suggested the possibility of decubitus as a cause.

Temesvary, six years later, reiterated these views, and the theory now usually accepted, that of amniotic pressure and adhesions, though suggested by Dareste in 1882, seems to have been overlooked by most authors until 1892, when Sperling, in a masterly paper, reviewed the whole subject and challenged all evidence supposed to prove that these so-called intra-uterine fractures were fractures at all, and pointing out the possible explanation of their causation by abnormalities of the amnion. His arguments in brief were:

A fracture *in utero* by indirect or direct violence would be hard to produce, the fetus being suspended in amniotic fluid and protected by the uterine and abdominal walls.

Violence sufficient to produce such a fracture would usually at least produce miscarriage.

If uterine pressure were the cause of fracture we should have fractures occurring with some frequency *intra partum*, when the intrauterine pressure is greatest; in fact, however, such fractures occur only at or near full term, and as a result, not of intrauterine pressure alone, but of the counterpressure of the bony pelvis as well.

Callus is practically never found in the cases seeming to belong to the class under discussion.

The presence of the so-called scar is not valuable evidence of compound fracture — it is not even proved that it is a scar.

The defects of fibula and toes, so frequently associated with the tibial bend, point to an origin as early as the second month of fetal life, for at this time the parts are laid down, and defects in the true sense cannot arise later. These defects cannot be, so far as we can see, either cause or result of the so-called fracture. Hence the association points to a common cause for both. It seems likely that both are due to pressure and pull of the abnormal amnion and its adhesions — the more likely inasmuch as this seems to be the cause of other similar deformities.

Haudek, in an extensive article in 1896, accepting Sperling's conclusions, adds to the evidence the report of the microscopic examination of one of these so-called scars which he had excised. It showed no interruption of the skin layers, and only such fibrous and atrophic changes as could best be explained as the result of pressure, in short, could not be called true scar. He also called attention to the fact that defects of the radius, which in early fetal life lies exposed in supination, are, with the associated defect of thumb and fingers, the not infrequent counterpart of defect of the similarly exposed fibula.

Kirmisson notes in this connection the scar-like mark over the bowed ulna which occurs in some of these cases of radial defect.

Nelaton, Kirmisson, Hoffa, Walsbam and Hughes, and very recently Nasse, in the new *Deutsche Chirurgie*, accept these views in general, and of late other theories have been largely given up. Delanglade, in an article which has just appeared, accepts the amniotic theory in general, but is inclined to lay more stress on narrowness of the amnion than on the action

of adhesions — a reservation also tacitly made by several others of the above list.

In considering this list of theories we find that several need not detain us. The theory of changes in bone texture lacks all evidence. That the pressure of twin pregnancies is not the cause appears from the fact that we have found no note of a case where the child was one of twins. The defect is not a fusion of tibia and fibula for, as Gould has pointed out, the interosseous membrane is present. Moreover, where the fibula is defective in part only, its rudiment occupies its normal position.

Fricke's theory of persistence of the fetal bend does not account for the usual direction of the bend forward and inward, instead of forward and outward, and presupposes, without explaining, the absence of the fibula.

The theory of nerve changes as a cause lacks evidence and is not in accordance with recent work which tends to show that growth *in utero* is independent of the nervous system (Schaper).

There remain only the theories of damage to, or absence of, original cell-elements, of fracture, and of amniotic changes. The arguments of Sperling and Haudek are, we think, enough to dispose of the fracture theory. It is conceivable and cannot be disproved that the defect may be one of development *per se*, but the theory of defect of development of "rays" lacks proof that such rays exist, and cannot explain the undoubted cases of defect of the fibula or radius without defects of the foot or hand. On any other theory of defect due to absence or faulty arrangement of original cell-groups, it is hard to see how to explain the relative constancy of type and the limited extent of the deformity. Nor can such a theory explain the tibial bend or the so-called scar.

The amniotic theory, however, seems to explain all the lesions pretty clearly. We cannot but accept it as at least the most plausible theory yet advanced. The origin of the typical deformity would be, then, as follows: Between the fifth and the eighth week, pressure of a too tightly-fitting amnion interferes with the development of the exposed fibula and the outer toe or toes of the exposed foot. Lack of space determines the bend of the growing tibia; the adhesion which produces the so-called scar is a result of contact of the most salient point of the tibia with the enveloping wall. The persistent lack of growth of the whole limb is probably a result of deficient vessels and nerves due to the early pressure, as well as of disuse.

As to the frequency of this deformity it is not so rare as would be supposed from some very short lists recently published. Haudek collected, with his own case, 103 in all, and we have found others to bring the list up to 127, including the cases here reported:

Haudek's list: Ztschr. f. Orth. Chir., 1896, iv, 326	103
Kirmisson (besides 2 cases included by Haudek): <i>Traité des mal. ch. d'origine congen., Paris, 1898; Revue de Chirurgie, 1897, p. 524</i>	6
Walsham and Hughes: <i>Deformity of the Human Foot</i>	1
Steele: <i>Transactions American Orthopedic Association, 1896, ix, 175</i>	3
Frees: <i>Transactions British Orthopedic Society, Birmingham, 1896, i, 10</i>	1
Humphrey: <i>On the Skeleton (cited by Gould, Transactions Pathological Society, xxxii, 153)</i>	1
Proudfoot: <i>New York Journal of Medicine, 1846 (cited by Brinton, Transactions American Surgical Association, 1884, ii, 425)</i>	1
Langton: <i>St. Bartholomew's Hospital Report, 1879, xv, 272</i>	1
Kaufmann: (cited by Braun, <i>Arch. f. klin. Chir., xxxiv, 1886</i>)	1
Delanglade: <i>Rev. des Malad. de l'Enfance, April, 1898</i>	1
Tyrie: <i>Journal Anatomy and Physiology, xxviii, 4, p. 411, 1894</i>	1
McKenzie: <i>New York Medical Journal, 1897, lxx, 241</i>	1
Morestin: <i>Revue des Malad. de l'Enfance, April, 1898</i>	1
Rose: <i>Cbl. f. Chirurgie, 1897, No. 12, p. 357</i>	1
Whitman: <i>Rev. d'Orthopedie, 1898, ix, 86</i>	1
Tubby: <i>Deformities, 1896, p. 359</i>	1
Cases here reported by us	3
Total	128

Nasse¹ speaks of having seen this defect repeatedly in v. Bergmann's clinic but gives no detailed account of cases, and we find reference to a report by Burmiester,² which we have not verified.

As to the details of cases reported, and the variations occurring, the following notes are presented:

The fibula defect is noted as total in 72 cases.

A fibula of not over half the normal length with both epiphyses present, one or both small, is thrice noted. In Volkmann's dissections, the upper epiphysis of the fibula was prolonged upward by a fibrous band. This band may, in part, replace a fibula which has no upper epiphysis (Braun) or may replace the whole fibula (Gould, Wagstaffe). Where an epiphysis is present it may show lack of ossification; this is probably the condition in Case II of our series.

Where the lower epiphysis is present it may lie higher up than normal and not serve as a part of the ankle-joint. This is shown by Bidder's dissections and by the radiographs of our Case II. Probably this condition may often obtain, for in the absence of dissections or radiographs, where no external malleolus is to be felt, it is very hard to say whether a rudimentary fibula may not be present.

A bend or angle of the tibia, directed forward and usually slightly inward, usually accompanied by the so-called scar, is noted in 67 of the published cases. The acuteness of the angle varies widely. There may be thickening of the tibia, or its head and shaft may be slenderer than normal. The shortening of the shaft is present to a greater extent than the bend can account for and may be marked. As to the lower end of the bone, it may show no obvious change or may

¹ Deutsche Chirurgie Lief, 66, i, Hälfte sq. 165.

² Cerberten ans des Geb. des Guburkh. ü. Gynack, zum Feier von Carl Ruger, Berlin, 1896, sq. 215 (1 plate).

show a knobby internal malleolus and an oblique surface for the ankle articulation.

This latter condition is the classical one in the cases of Volkmann's "Congenital Dislocation of the Ankle." It is a question, however, whether these cases do not belong in a separate class. The original cases, described by Volkmann, Kraske and Bidder, though they had defective fibulæ, showed no defect of toes, were all bilateral, and all occurred in members of one family, seven out of seventeen of whom had the deformity. This hereditary character does not accord with either the facts or the theory of cases of fibula defect in general. For these few cases, however, the dissections reported prove a definite obliquity of the ankle-joint, primary, and the cause of the extreme deviation of the foot. Bidder found in one case that the lower epiphysis of the tibia, 1.2 cm. thick at the inner side, fell away to 2 mm. at the outer.

We have no evidence of the existence of such obliquity in the majority of cases, but it is very probable that it exists to some extent. The radiograph of our Case I shows some obliquity and that of Case III suggests it.

Defect of one or more toes associated with the fibular defect is noted expressly in 67 cases. In one case, however (Kirmisson), there were seven toes. Apart from one case (Otto) where the great toe is said to have failed, the defect seems always to have affected the outer toes. Several cases are reported where all save the hallux were lacking. There is often some disproportion in size of the toes. Webbing, especially of the second and third, is frequent. In some cases there is associated defect of toes on the other foot without fibular defect. The metatarsals corresponding to the missing toes are uniformly absent. Some of the tarsal bones may be lacking or more often there is

fusion of several of them: the calcis, talus, scaphoid and cuboid, or some of these, being especially likely to fuse. There seems to be no regular type of deformity here. The foot is in equinus, usually in equino-valgus of varying degree. In five cases, however, varus or equino-varus was reported. In one of our cases the position in the beginning seems to have been a cal-



canéo-valgus. The contracture of the tendo-Achillis accompanying the equinus is not entirely owing to the disuse of dorsal flexion in a short leg, for it may, as in a case of Bidder's, occur where the deformity is bilateral and equal.

In 21 cases the patella is noted as of less than normal size. It may be wanting or, at least, not distin-

guishable clinically. A lack of development of the whole limb, femur as well as tibia, is usual, varying widely in degree. Whether this may be in part, as Nasse seems to think, due to the atrophy of disuse, or from congenital deficiency of vessels and nerves, is hard to say. The shortening is progressive and its increase as the child grows is a very important factor in determining the disability.

Braun reports two cases, of sixteen and seventeen years, with a shortening of 7 and of 8 cm. respectively — the femur being responsible in each case for 2 cm.

Blasius found in one case at ten years, 2 inches shortening; in another, at nine years, $2\frac{1}{2}$ inches.

Kirmisson had a case with 12 cm. shortening at two years, with atrophy of the whole extremity.

Schnelle reported a case with 3 inches shortening at three years, 5 inches at ten years, 9 inches at twenty-nine years.

Kaufmann found the increase of shortening between two and one-half and six and one-half years of age to be 2 cm. in one case.

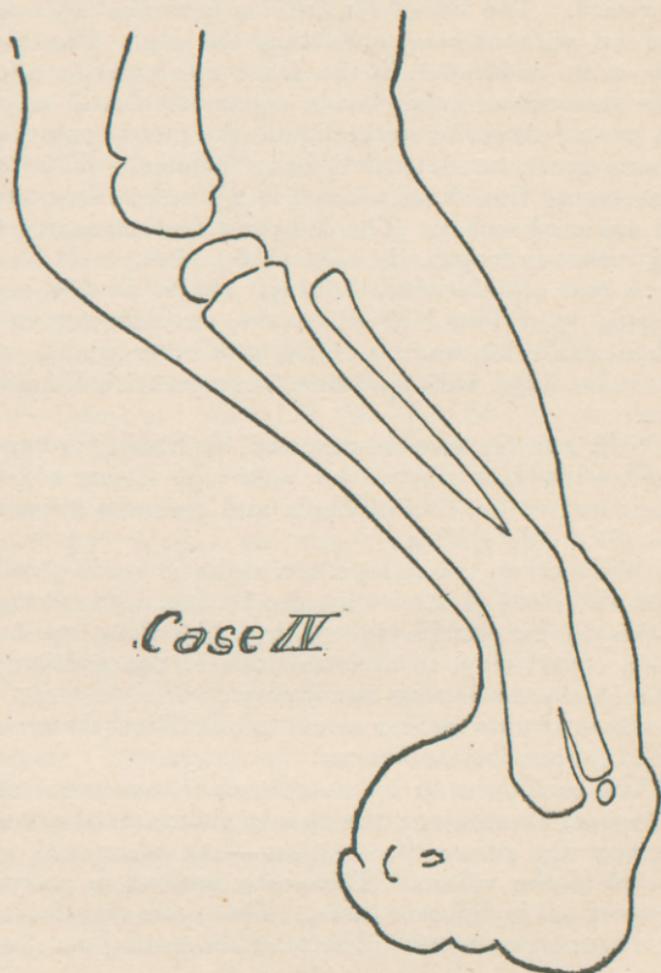
Kemke in a case seen at birth found the tibia $1\frac{1}{2}$ cm. short; at one and one-half years the shortening was $2\frac{1}{2}$ cm.

The deformities associated with fibular defect have been most various, including hare-lips, skull defects, visceral anomalies, etc.; but, as would be expected from the accepted theory, defects of the foot, as a whole, of the femur, of the whole upper extremity, or especially of the ulna, or radius and fingers, are most common.

Before leaving this part of the subject it may be well to call attention to certain deformities of the leg which seem to be of similar origin with the fibular defect — those where some constriction is apparently the cause.

CASE IV. Age four. An unreported case; seen

by kindness of Dr. J. E. Goldthwait; has a shortened and deformed leg and foot, as shown in the accompany-



ing figures. The x-ray shows no defect either of tibia or fibula, but at the point of constriction the bones lie

in contact and seem to be fused. At this point there is also a bend in both bones forming an angle open forward. The site of constriction is marked by a deep sulcus, without scar, encircling the leg. The x-ray shows no ossification in the foot; manipulation shows the presence of some bones, apparently fused, in part at least. Anterior to the tarsus, the foot is pulpy, and there must be extensive bony defects. The total shortening from knee to heel is $2\frac{1}{4}$ inches, from knee to ankle $1\frac{1}{2}$ inches. The deformed foot measures but $2\frac{3}{8}$ inches in length, the normal, 5 inches.

A case of deformity, strikingly similar to this, is reported by Ridlon,³ in which the circular depression about the ankle was associated with other similar constriction rings, with spontaneous amputation of fingers, etc.

Still another case is reported by Steele,⁴ where a depression just above the ankle, as from a cord, coexisted with defect of fibula and toes and bend and so-called scar of tibia.

These cases, taken together, make it seem possible that this class of cases may also be due, not to strangulation by the umbilical cord, but, like those we have been considering, to abnormalities of the amnion, although the mechanism is not clear.

Chance⁵ cites similar cases, and explains them as a result of amniotic adhesions.

The conditions to be considered in treatment are: The total shortening; the abnormal axis of the lower part of the tibia; the equinus—the shortened calf muscles; the valgus. These are present in varying proportions in different cases. There can, therefore, be no routine treatment. The total shortening may be so

³ Ridlon: Transactions American Orthopedic Association, vol. ix, 179, 1896.

⁴ Steele: *Ibid.*, p. 175.

⁵ Chance: *Bodily Deformities*, London, 1862, p. 135.

great, especially if the other deformities of the limb are also extreme, that amputation will be the only resort. This has been repeatedly performed. So far as the shortening is due to the bend in the tibia, it is amenable to treatment by osteotomy or osteoclasis. Brinton, in one case, reduced the shortening in this way from $2\frac{1}{8}$ to $1\frac{1}{2}$ inches, and Rose reports the successful correction by osteotomy of a right angled bend in the tibia. Tenotomy of the tendo-Achillis has usually been necessary as an accessory to this as to other operations on these cases; it has also been performed as the only means of relief, but without much result. In some cases a wedge osteotomy has been performed, in one case (Langton) followed by non-union. There have been other unsatisfactory results after osteotomy, but there seems no reason, if the operation is carefully done, why the tibia cannot be accurately straightened in this way, as has been done by Rose, Brinton, Kirmisson and others, nor why there should be a return of the deformity, as has occurred (Küster).

As to avoidance, so far as may be, of the increase of shortening with the growth of the child, Nasse lays stress on early use of the limb, with proper support, to avoid atrophy from disuse.

The equinus may always be remedied by tenotomy of the tendo-Achillis, which has so often been performed. The important fact to bear in mind in this connection is that the equinus is in large measure an attempt at necessary compensation of the total shortening, and, if the ankle can be supported in the equinus position, it is hard to see what is to be gained by the tenotomy, unless, indeed, the shortening is so little that a moderately high sole will fully compensate it.

The most difficult problem to treat has usually been the valgus deformity. This is not, at least in the majority of cases, a valgus of pronation and flattening

alone but rather a tipping outward of the whole foot, including the astragalus, which has no external malleolus to hold it into place. Moore⁶ has recently reported cases of valgus, due to the removal of the fibula for disease, which show this mode of production of the deformity. In some cases, at least, an obliquity of the lower surface of the tibia determines the valgus; in others there is apparently no such obliquity. The tipping outward of the foot must also be favored by the irregularity of the articular surface of the astragalus; also by the fact that the bones of the tarsus often are partly fused, and hence must have little mobility. These facts, probably, are the explanation of many failures in the use of the high sole after tenotomy of the tendo-Achillis, and must be carefully considered in outlining any treatment.

In a proportion of the cases (as in Case III of our series) there is contracture of the peronei, as well as of the calf muscles, which necessarily prevents correction of the valgus until tenotomy is performed. The section of the peroneal tendons has been performed (Brinton, Nelaton, Walsham, Kirrison and others) with good success.

In cases where there was obliquity of the ankle-joint, a wedge osteotomy above the ankle has been performed by Riedinger and by Walsham with good result. Volkmann, in one such case, took a wedge out of the articular surfaces, securing correction of the valgus and ankylosis of the ankle. The result was fair.

Simple resection of the ankle-joint has been done in a number of cases. One of the most successful is a recent case by Rose, where the lost ankle motion was compensated by extra mobility in Lesfranc's joint. If this ankylosis be so effected that the foot remains in

⁶ Moore: *Annals of Surgery*, 1896, xxiv, 634.

plantar flexion (Braun), this will lessen the necessary height of the sole. Where the deformity is bilateral this has, of course, no point, but in other cases seems desirable. The Wladimiroff-Miculicz resection, suggested by Kemke, but not carried out, would have no advantage. In two cases (Rincheval, Nasse) Bardenhauer's operation, the splitting of the tibia, and the insertion of the talus in the split has been carried out. Nasse secured ankylosis in equinus. In both cases the result seems to have been satisfactory.

In cases where the rudiment of the fibula, as shown by the x-ray, is of some size, but situated higher up than normal, it seems possible that an operation aiming to slide this down and fix it to the tibia as an external malleolus, might be worth considering. It has apparently never been done.

Attempts to remedy the defect by orthopedic apparatus have, on the whole, been about as satisfactory as the operative cases. The worth of operative procedures must vary with the case; but, in most cases, and especially where the limb as a whole is markedly atrophic, not much is to be expected. In a recent discussion by Kirmisson, Nelaton and others, it came out clearly enough that the results attained immediately after operation are not conclusive as to the end result. Bidder gives plates of two cases: one where Volkmann did ankle resection; the other one of Küster's, where there had been an osteotomy; they are not encouraging. Probably in any case, even if operated on, an apparatus of some sort must be worn to prevent recurrence or the rise of some new deformity. The limb is primarily defective as well as deformed, and correction of the deformity, even if complete, does not solve the whole problem. Probably, unless osteotomy promises some considerable gain in length, or in the position of the foot, or

unless peroneal contracture interferes with the reduction of the valgus, no operation, not even a tenotomy, will usually be worth while. If a valgus shoe with a high sole, sloped to fit the equinus, and with side irons, can be satisfactorily fitted, there seems no reason for tenotomy of the Achillis.

To sum up (in so far as one may generalize where cases so vary): Osteotomy to be performed not merely because deformity exists, but only for definite indications; tenotomy only to reduce the valgus or as an adjunct to osteotomy; resection of the ankle or Bardenhauer's operation, where apparatus will not control the tipping of the ankle; amputation only in cases utterly unfit for other treatment; treatment of all ordinary cases by apparatus to support the ankle, and prevent valgus — high sole to supplement the equinus in equalizing the shortening — these would seem to be the general lines of treatment. The treatment seems to be at best unsatisfactory, and, as always in such cases, operation is to be avoided unless we have some clear gain in view.

Appended is a partial bibliography; other references, especially to older and less accessible publications, with detailed citations of a number of cases, will be found in Haudek's article:

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