THE MANAGEMENT OF DEFORMITY AND PARALYSIS OF THE FOOT IN MYELOMENINGOCELE

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The combination of deformity, motor paralysis and sensory loss in the feet of children born with myelomeningocele produces some difficult problems in management. Before the development of means to control hydrocephalus, and in the absence of closure of the spinal defect in the newborn, most of those who survived were likely to be confined to a wheelchair because of mental defect and loss of sense of balance arising from gross hydrocephalus, or because of poorly healed skin or cystic swelling in the lumbar region that limited the use of supportive apparatus. The almost complete elimination of these two complications means that nearly all children with myelomeningocele are potentially capable of walking. Their upper limbs are normal and very often there is normal trunk musculature to enable them to elevate the pelvis. The most serious liability that many have is anaesthesia of the sole of the foot. Prompt operation on the spinal lesion after birth has caused many more children to have active innervation of muscles controlling the ankle and foot. Useful though this muscle activity may be, muscle imbalance may lead to progressive deformity in growing children or may have already given rise to deformity during intrauterine development. The object of this article is to describe the results of an experience of ten years in the management of various combinations of deformity and paralysis in the foot and ankle in children with myelomeningocele and to try to formulate principles of treatment.

TYPES OF DEFORMITY AND THEIR CAUSATION

A study of paralysis and deformity in the lower limb in spina bifida cystica (Sharrard 1966) has shown that several factors may be responsible for the production of deformity. In a consecutive series of seventy-one infants seen during the first twelve hours of life, sixty-five limbs had no deformity at birth and seventy-seven limbs showed various combinations of equinus, calcaneus, varus and valgus deformities of the ankle and foot. When the examination was made, before the spinal lesion had been operated upon, there was often no correlation between spontaneous motor activity and the type of deformity, but a strong correlation was found between deformity and the response of the lower limb muscles to faradic stimulation. Severe calcaneus deformity, for instance, was associated with a strong response to stimulation of all the dorsiflexor muscles and a weak or absent response in the triceps surae and other ankle plantar-flexors. Clawing of the toes was associated with a good response in the long flexor and extensor muscles of the toes and a poor or absent response in the intrinsic muscles of the sole of the foot. These results suggest that intrauterine paralysis is the predominant cause of birth deformity in myelomeningocele, probably supplemented by the effects of intrauterine pressure and posture. Unfortunately, this relatively simple situation is often upset by subsequent events. Loss of function in the neural tissues associated with the spinal defect may result in an alteration in the neurological picture in survivors. When there has been no deformity at birth, secondary deformity may develop from muscle imbalance in partial flaccid or spastic paralysis of some of the muscles controlling the ankle and foot. Deformed feet in which there has been muscle activity before birth may later become completely paralysed. The postural effects of gravity may produce equinus and, when the child starts to bear weight, the foot may deviate into valgus.

The resulting variety of deformities and distributions of paralysis makes analysis extremely difficult. Calcaneus, equinus, varus or valgus of the hindfoot can be combined with calcaneus,
equinus, varus, valgus, adduction or abduction of the forefoot and with clawing or valgus deformity of the toes. In practice, six varieties of deformity predominate in myelomeningocele: equinus, equinovarus, equinovalgus, calcaneus, calcaneovarus and cavus deformity with clawing of the toes (Table 1).

**TABLE 1**

**SHAPE OF FOOT IN CHILDREN WITH MYELOMENINGOCELE: 296 FEET**

<table>
<thead>
<tr>
<th>Deformity</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Equinus</td>
<td>64</td>
</tr>
<tr>
<td>Equinovarus or varus</td>
<td>78</td>
</tr>
<tr>
<td>Equinovalgus or valgus</td>
<td>24</td>
</tr>
<tr>
<td>Calcaneus</td>
<td>20</td>
</tr>
<tr>
<td>Calcaneovarus</td>
<td>42</td>
</tr>
<tr>
<td>Cavus and claw toe deformities</td>
<td>13</td>
</tr>
<tr>
<td>No deformity (flail)</td>
<td>28</td>
</tr>
<tr>
<td>No deformity (normal)</td>
<td>27</td>
</tr>
</tbody>
</table>

**MATERIAL**

The object of the study was to investigate a cross-section of the patients attending the spina bifida clinic at Sheffield Children’s Hospital. To have attempted to analyse the deformities and results of treatment in more than 900 survivors now attending the clinic would have been impossible within the time available. One hundred and forty-eight patients, seventy-six boys and seventy-two girls, were studied in 1967. Their ages ranged from two to sixteen years and all had had at least one operation on the foot between 1947 and 1965. Records had been made of deformity, paralysis and operative procedures. The object of treatment was to produce a plantigrade foot that could bear weight without causing excessive pressure in any one part of the sole. A foot should be of such a shape that shoes and appliances were not difficult to fit. The distribution of muscle activity should be such as to require the least amount of external support and guard as much as possible against recurrent deformity. The management of a given deformity was regarded as successful if it fulfilled these criteria. Failure was recorded if a foot at the time of review was unchanged from the initial deformity or still required further surgery because the residual deformity was unsatisfactory for walking.

Of the 296 feet reviewed fifty-five were without deformity. Twenty-seven were completely normal. Twenty-eight had no deformity at birth, were completely flail and did not develop deformity when weight was borne on the limb; their management required nothing except the provision of appropriate footwear with lacing extending to the toes and appliances to control the position of the foot and ankle in walking.

**METHODS OF TREATMENT USED**

In 1958 a review was made of the results of attempted conservative treatment of equinus and equinovarus deformity in children with myelomeningocele. The methods that had been used were those applicable to congenital talipes equinovarus, such as adhesive strapping, splintage, serial plasters and passive stretching by physiotherapists. The results in the twenty feet so treated were disastrous. In spite of extreme care in the application of splints or plaster casts, five feet developed pressure sores and treatment had to be abandoned; in one patient secondary sepsis in the foot eventually led to amputation. In only two of the remaining fifteen feet was correction achieved and maintained, and these were both feet in which the deformity was not severe and the foot was completely paralysed. In four feet in which adequate
correction was achieved at first, the deformity recurred within six months of discarding corrective splints because of uncorrected muscle imbalance. Splintage during the early days or weeks of life often proved difficult to maintain and interfered with the nursing and surgical management of the spinal defect and hydrocephalus. The use of night splints, however carefully moulded or padded, was found to be equally unsatisfactory even when deformity had been corrected and almost always eventually led to skin ulceration.

Since 1958 operation has been used to correct deformity. An assessment has been made of the deformity at each part of the ankle and foot, of the short soft tissues and of bony abnormalities contributing to the deformity. Voluntary or reflex activity in each muscle group has been estimated and the effects of posture and, in a child able to walk, the effects of bearing weight noted. It was not always possible to determine sensory loss in a small child, but in most instances it was assumed that the foot was partly or totally anaesthetic.

Correction of deformity was first attempted by division or elongation of short tendons, tendon sheaths, ligaments, joint capsules and fasciae. When muscles were known to be paralysed and showed no response to faradic stimulation their tendons were divided. The tendons of active muscles were either elongated or transferred to a new position in the foot. The aim in tendon transfer was to obtain, as far as possible, a balance of dorsiflexor against plantar-flexor and of invertor against evertor. To achieve this in a small child in whom the precise strength of individual muscles is sometimes extremely difficult to assess was often a matter of intelligent guesswork combined with surgical expediency. It was considered unwise to rely on a tendon transplant to complete the correction of deformity that had not been fully eliminated by soft-tissue division. Some deformities proved to be impossible to correct by soft-tissue division alone. Additional bony operations such as calcaneal osteotomy, tallectomy, resection of the tarsus or metatarsal osteotomy were used even in young children if they were needed to obtain a foot safe to bear weight. These operations were sometimes performed at the time of soft-tissue division and in other instances as a second procedure.

**TABLE II**

**OPERATIONS IN 241 FEET**

<table>
<thead>
<tr>
<th>Type of deformity</th>
<th>Equinus</th>
<th>Equinovarus</th>
<th>Equinovalgus</th>
<th>Calcaneus</th>
<th>Calcaneo-varus</th>
<th>Cavus or clawing</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of feet</td>
<td>64</td>
<td>78</td>
<td>24</td>
<td>20</td>
<td>42</td>
<td>13</td>
<td>241</td>
</tr>
<tr>
<td>Soft-tissue operations</td>
<td>77</td>
<td>122</td>
<td>24</td>
<td>10</td>
<td>14</td>
<td>15</td>
<td>262</td>
</tr>
<tr>
<td>Tendon transfers</td>
<td>1</td>
<td>30</td>
<td>9</td>
<td>17</td>
<td>41</td>
<td>8</td>
<td>106</td>
</tr>
<tr>
<td>Bony operations</td>
<td>2</td>
<td>38</td>
<td>12</td>
<td>1</td>
<td>9</td>
<td>3</td>
<td>65</td>
</tr>
<tr>
<td>Total operations</td>
<td>80</td>
<td>190</td>
<td>45</td>
<td>28</td>
<td>64</td>
<td>26</td>
<td>433</td>
</tr>
</tbody>
</table>

In the 241 feet in this series in which there was deformity, 433 operations were performed. Tenotomies and soft-tissue divisions, with or without tendon transfer at the same operation, were done on 262 occasions; tendon transfer was the primary operation in 106 instances, and in sixty-five a bony procedure was performed. Table II shows the types of operation needed in the six main types of deformity.

**RESULTS**

**EQUINUS DEFORMITY**

In many children with equinus deformity there was complete paralysis of all muscles below the knee (Fig. 1). Although night splints can be used to prevent equinus deformity in a flail foot, correction was usually maintained by daily passive movements by physiotherapists...
or parents. When passive movements were not kept up, equinus deformity with a short tight tendo calcaneus developed. If the foot had not been allowed to lie in an equinus position for too long, simple subcutaneous tenotomy of the tendo calcaneus was usually adequate to correct deformity, the position being maintained by a double layer of crêpe bandage rather than by plaster splintage. After correction of the equinus the toes sometimes became flexed because of associated shortness of the flexor hallucis longus and flexor digitorum longus tendons. If necessary, the tendons of these muscles were divided in the sole of the foot or through a small incision behind the ankle. If this was not done, pressure sores could develop on the tips of the toes as soon as the child was allowed to bear weight in footwear.

In some patients equinus recurred rapidly after the tenotomy in spite of attempts to maintain the range of dorsiflexion. In some there was voluntary or reflex activity in the calf muscle. In others there was no voluntary action in the calf muscles and no evidence of reflex activity. Faradic stimulation showed a response in the triceps surae and there was evidence from biopsy of such muscles that they had retained motor innervation but had lost sensory innervation. In these instances hemitransplantation of the tendo calcaneus to the dorsum of the foot was indicated. The correction of equinus deformity that had been established for a considerable time was often difficult. When the anterior part of the talus had been outside the ankle mortise for a year or more it had grown too wide for restoration into the mortise, though sometimes this could be achieved by division of the inferior tibio-fibular ligament. In the last resort excision of the talus was needed.

Equinus must be corrected to avoid pressure necrosis under the metatarsal heads or on the toes. The provision of a surgical shoe moulded to the deformity will not prevent this complication. In one patient in this series three toes had to be amputated because of osteomyelitis secondary to pressure necrosis when the child was allowed to walk on a foot with uncorrected equinus deformity.

Fourteen per cent of the operations were revisions—that is, the same operation was repeated more than once in a foot to obtain correction (Table III). At the time of follow-up 11 per cent still required further correction (Table IV), and 89 per cent were corrected and fit to bear weight.

**Equinovarus Deformity**

This was the most common deformity requiring correction and was the most difficult to correct. For seventy-eight feet with this deformity a total of 190 operations were needed. A few could be corrected by one operation, using an extensive medial release, but many required two, three, and even four operations before the deformity was finally brought under control.

In most instances the tendo calcaneus, both long toe flexor tendons, the tibialis anterior and posterior tendons, the medial ligament of the ankle and the medial ligament of the subtalar joint had to be divided and the tibialis anterior tendon transferred laterally to the outer side of the dorsum of the foot. Skin closure was often difficult in a foot originally inverted and adducted 90 degrees or more (Fig. 2). The use of a V-Y incision (Sharrard 1967) made satisfactory skin closure possible in many, but skin breakdown occurred in seven out of
fifty-two medial release operations and in five patients superficial sepsis occurred, probably from excessive tissue tension and haematoma. Difficulty in correcting varus and equinus of the hindfoot was responsible for the high revision rate of 22 per cent (Table III), and even after repeated operation 22 per cent were still awaiting further correction at the time of review (Table IV). The impression was gained that, if adequate correction of the deformity by soft-tissue release without excessive skin tension could not be obtained at the first operation, it was better to complete the correction by calcaneal osteotomy to avoid having to operate a second time through scarred, contracted and adherent tissues. In more than half the patients medial release of soft tissues alone with tendon transfer was sufficient.

In many patients with equinovarus there was associated adduction of the forefoot that required division of soft tissues on the inner side of the foot and sometimes wedge resection of the tarsus. Excision of the cuboid bone alone always failed to correct adduction of the forefoot: to be successful, osteotomy had to be made completely across the mid-foot; if this was done, the correction was satisfactorily maintained. When it was necessary to excise the talus, satisfactory correction of the hindfoot was achieved but the forefoot often remained adducted and required further soft-tissue or bony corrective procedures.

Even when the position of the hindfoot and forefoot appeared to have been satisfactorily corrected there might still remain a medially rotated position of the foot relative to the knee. This sometimes seemed at first sight to be due to medial torsion of the tibia, but palpation of

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<td>28</td>
<td>64</td>
<td>26</td>
<td>433</td>
</tr>
<tr>
<td>Number of revisions</td>
<td>11</td>
<td>42</td>
<td>5</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>61</td>
</tr>
<tr>
<td>Incidence of revision (percentage)</td>
<td>14</td>
<td>22</td>
<td>11</td>
<td>0</td>
<td>1.5</td>
<td>8</td>
<td>14</td>
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<tr>
<th>Type of deformity</th>
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<td>20</td>
<td>42</td>
<td>13</td>
<td>241</td>
</tr>
<tr>
<td>Failures</td>
<td>7</td>
<td>17</td>
<td>9</td>
<td>4</td>
<td>9</td>
<td>0</td>
<td>46</td>
</tr>
<tr>
<td>Incidence of failure (percentage)</td>
<td>11</td>
<td>22</td>
<td>37</td>
<td>20</td>
<td>21</td>
<td>0</td>
<td>19</td>
</tr>
</tbody>
</table>
the malleoli showed that this was not the cause but that the deformity was due to a general adduction deformity of the whole of the foot. Correction of this by osteotomy of all the individual tarsal bones was not found to be a practical solution; lateral rotation osteotomy of the upper end of the tibia gave just as good results.

**EQUINOVALGUS DEFORMITY**

(Paralytic Vertical Talus)

This deformity was relatively uncommon but presented as a very striking abnormality in which there was equinovalgus of the hindfoot, the calcaneus being everted on the talus, the talo-navicular joint subluxated or dislocated, and the talus vertical with an appearance comparable with that of congenital vertical talus (Fig. 3). The paralytic mechanism responsible for this deformity is variable: it is often associated with innervation down to at least the fifth lumbar root or first sacral root, with strongly acting dorsiflexors and evertors and weak or paralysed long toe flexors and intrinsic foot muscles. Division of soft tissues on the outer side of the foot, reduction of the talo-navicular dislocation and fixation with a Kirschner wire, transplantation of dorsiflexors and peroneal tendons to various sites in an attempt to restore muscle balance, and calcaneal osteotomy, extra-articular subtalar fusion or other bony procedures were all tried. The failure rate at review was 37 per cent, the highest in any group. This deformity is difficult to reduce and keep reduced. Further study of the anatomy and of the paralytic patterns associated with the deformity is needed to improve results.

**CALCANEUS DEFORMITY**

Calcaneus deformities even when severe (Fig. 4) proved to be easier to manage than deformities associated with equinus. The muscle imbalance was usually well defined, with active dorsiflexor muscles and paretic or paralysed plantar-flexors. Transfer of two or more tendons, usually the tibialis anterior and peroneus tertius, through the interosseous membrane into the tendo calcaneus or into the calcaneus itself with elongation of the remaining dorsiflexor tendons and division of the anterior capsule of the ankle was sufficient to allow weight to be borne even if correction of the deformity was not complete. If the deformity was severe, V-Y skin closure was needed. The failure to obtain plantigrade feet in four out of twenty instances arose either because the correction was made too late and bony deformity had become established or because insufficient tendons had been transposed posteriorly.

**CALCANEOVARUS DEFORMITY**

This deformity (Fig. 5) proved easier to correct than equinovarus deformity. Soft-tissue release of structures on the inner side of the foot combined with transfer of the tibialis anterior tendon to the tendo calcaneus gave a good proportion of satisfactory corrections. In some older children additional secondary bony operations were needed and, as in equinovarus deformities, calcaneal osteotomy or excision of the talus might have given a better correction than soft-tissue release alone in some cases. Twenty-one per cent of feet still needed further correction at the time of review (Table IV).
CAVUS DEFORMITY AND CLAW TOES

Various combinations of forefoot and toe deformity were treated by a variety of operations such as detachment of the plantar fascia and intrinsic foot muscles from the calcaneus, medial and lateral plantar denervation (Garceau and Brahms 1956), flexor-extensor transplants in the lesser toes (Taylor 1951), arthrodesis of the interpha langeal joint of the great toe with or without transfer of the extensor hallucis longus to the first metatarsal, and occasionally metatarsal or midtarsal osteotomy. Many of these feet had good innervation, the main problem being paralytic imbalance between long toe flexors and extensors and the intrinsic foot muscles. Children with this level of paralysis are often very active and it was found to be important to correct toe and forefoot deformities before pressure sores could develop on the toes or beneath the metatarsal heads. This was the only group of deformities in which a satisfactory correction was obtained in every foot.

COMPLICATIONS

The complications are listed in Table V. The commonest complication, development of pressure sores beneath the plaster, emphasises the need for extreme care in the application of plasters. Attempts to obtain further correction of the deformity during the application of a plaster after an operation were responsible for some of these sores. The combination of anaesthetic skin and temporarily diminished circulation after an operation to correct deformity in the foot makes pressure necrosis more likely to occur. Pressure sores did not necessarily develop on the weight-bearing surface of the foot: several occurred on the dorsum of the foot or over the front of the ankle. These did not give a liability to recurrent ulceration on bearing weight but they did require extreme care in the fitting of footwear.

Pathological fractures and epiphyssial fracture-separations are common in children with myelomeningocele. They sometimes occurred while the child was still in plaster or soon after the plaster had been removed. The results of a study of vitamin C deficiency (McKibbin and Porter 1967) suggest that some of these might have been avoided by administration of vitamin C before operation. All the fractures united uneventfully and were seldom the cause of additional deformity of the foot.
Breakdown of skin has already been referred to in discussing the results of medial release for equinovarus deformities. All healed after skin grafting.

Division of vessels or nerves occurred in three instances in which dissection was being undertaken for the second time in varus deformities of the heel. None gave rise to any increased disability in the foot, because the nerves concerned were already functionless and the feet survived division of the posterior tibial vessels. Infections were never serious and all subsided with antibiotic therapy and drainage.

**TABLE V**

<table>
<thead>
<tr>
<th>Complications in 433 Foot Operations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td>Number</td>
</tr>
<tr>
<td>Pressure sores . . . 17</td>
</tr>
<tr>
<td>Fractures . . . 14</td>
</tr>
<tr>
<td>Infection . . . 6</td>
</tr>
<tr>
<td>Necrosis of incision edges (all required skin grafts) . . 7</td>
</tr>
<tr>
<td>Right neurovascular bundle cut 1</td>
</tr>
<tr>
<td>Right posterior tibial artery cut 1</td>
</tr>
<tr>
<td>Left medial plantar nerve cut 1</td>
</tr>
<tr>
<td>Total . . 47</td>
</tr>
</tbody>
</table>

**TIMING OF OPERATION**

One of the most vexed questions in the management of deformity in myelomeningocele is the timing of the operations. Correction of birth deformities by conservative or operative means during the first few weeks of life is difficult, interferes with the surgical management of the spinal defect and hydrocephalus, and poses considerable problems in splintage. The size of the foot tends to limit the age at which operations can be performed safely, and most of the operations were performed in children two years of age or more. Against this is the fact that, with increasing age, the deformity becomes increasingly rigid and bony deformity is a greater problem. Any deformity left beyond the age of four is likely to need an osteotomy or bony excision of some kind. A further difficulty lies in the proper estimation of muscle activity in the very young child. In making decisions to perform tendon transfers, it was often necessary to transfer a tendon although the power of its muscle was not precisely known because the child was not old enough to perform voluntary actions at request. This accounted for some of the revisions that had to be undertaken later, the most obvious tendon being transferred at an earlier age and a more definitive or additional transfer performed when the child was old enough to be able to cooperate. The general impression was that the best results were obtained when operation was performed between the age of one and two years.

**DISCUSSION**

Eighty-one per cent of the feet in this series had been satisfactorily corrected at the time of review. The criteria for success were strict. In all the successful patients a plantigrade foot, free of deformity and requiring a minimum of external splintage, was obtained. These results in our hands were much better than those of conservative management. No pressure sores have developed in any children in whom the deformity has been successfully corrected, provided satisfactory footwear has been used. Shoes were found to be preferable to boots,
which tend to cause pressure sores over the malleoli. If there was need to provide additional
stability at the ankle this was better obtained by the use of light Exeter-type spring appliances.

Nineteen per cent of feet were in need of further correction at the time of review and
represented failed surgical treatment up to that time. It is expected that further operation
should render most of these feet plantigrade and safe to bear weight.

Often, when a very severe deformity was tackled, particularly of equinovarus, there was
a tendency to be pleased to have gained an almost complete correction. It was these incompletely
corrected feet that formed a high proportion of the failures and, in retrospect, one must not
be content with other than total correction by whatever means are necessary to achieve this.
Closure of the skin has provided a difficult problem in some instances and this has only been
partly solved by the use of plastic types of approach. Sometimes it may be possible to obtain
correction only by a two-stage procedure because of skin tension and, in any particular
operation, the viability of the skin flap needs to be watched with great care.

Tenotomies and division of soft tissues generally were found to give much greater
 correction than the severity of the original deformity suggested would occur. The circumstances
in myelomeningocele are quite different from those in poliomyelitis or cerebral palsy in that
wholehearted division of paralysed tendons can be performed without anxiety. The anterior
or posterior tibial vessels, though not immune from damage, have been found to be
surprisingly amenable to mobilisation and alteration of position of the foot and ankle, and
circulatory problems after operation have not been as worrying as might have been expected.

The results of tendon transfers, particularly in such young children, as many in this
series were, are very difficult to assess. Among the feet in which there was a recurrence of
deformity it was notable that there were several in which a potentially deforming tendon had
not been transferred because it was thought to be very weak or paralysed. In these feet
deformity sometimes recurred within six months of an adequate correction. In making
decisions about which muscles to transfer a common-sense judgement based on as adequate
an assessment of muscle activity as possible before operation seems to have succeeded in at
least 80 per cent of patients.

Most of the children in this series were too young to have bony operations such as triple
arthrodesis with correction of deformity. As compared with paralytic conditions not involving
anaesthesia of the foot, it was not safe to leave a bony deformity uncorrected until a child
was old enough (over the age of twelve years) to have an adult type of operation. Operations
such as extra-articular subtalar arthrodesis, calcaneal osteotomy, metatarsal osteotomy, wedge
resection of the foot and excision of the talus were used in such a way as to cause as little
disturbance as possible to growth in the foot. Of sixty-five bony procedures, fifty-three gave
satisfactory results. Among the failures should be noted the development of pseudarthrosis
above the level of a satisfactory arthrodesis of the ankle that subsequently developed into a
type of Charcot joint. Others have had similar experience (Hayes, Gross and Dow 1964)
and it seems that this operation should be avoided in myelomeningocele for this reason.

A policy of operative management of foot deformity in myelomeningocele was instituted
because it was felt to be the safest way of correcting deformity without producing pressure
sores. Although pressure sores did occur as a complication in slightly under 4 per cent of
operations none was in the weight-bearing area of the sole and, in retrospect, all of them
could have been avoided by curbing one's enthusiasm to obtain the last degree of correction
in the plaster cast. The results of operation in the correction and maintenance of correction
of deformity without production of pressure sores in the weight-bearing area of the foot have
been very much better than rigid splints of metal or plaster or the application of inflexible
strapping, but there may be room for the limited use of carefully applied elastic adhesive
strapping in the partial correction of deformities during the early weeks of life to minimise
the extent of operative surgery that will be needed to complete correction of the deformity
in later months.
THE MANAGEMENT OF DEFORMITY AND PARALYSIS OF THE FOOT IN MYELOMENINGOELE

SUMMARY

1. Deformities of the foot in children with myelomeningocele are described and classified. The results of a policy of operative correction of deformity in 148 patients all of whom had had at least one operation on the foot between 1947 and 1965 are described.

2. In 241 feet in which there were deformities 433 operations were performed, including tenotomies, soft-tissue divisions, tendon transfers and bony procedures. At the time of review successful correction of deformity had been obtained in 81 per cent with a plantigrade foot that could bear weight safely, and with a distribution of muscle activity that required minimal external support and presented the least liability to recurrent deformity.

3. The management of individual deformities is described and the causes of failure are analysed and discussed.

REFERENCES


