FEATURES RESISTING PRIMARY TREATMENT OF CONGENITAL CLUB FOOT

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The aims of this study were: (1) to see whether the number of relapses of hindfoot deformity in congenital club foot in the first year of life could be reduced by operative treatment, and (2) to try by surgery to pinpoint features of congenital club feet that make them resistant to treatment.

As compared to treatment of 95 feet with manipulation and plaster casts only or with heel cord tenotomy added, early tenotomy of both the heel cord and the tibialis posterior tendon in 23 feet markedly reduced the number of relapses of hindfoot deformity, and the need for additional treatment. The results indicate that the achilles and the tibialis posterior tendons, and their corresponding muscles, are the main dynamic features that need to be dealt with when treating the hindfoot deformity in congenital club foot.

Key words: club foot; muscles; primary treatment
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Primary closed treatment is the generally accepted method of choice for club foot deformities in the newborn; operations are not usually performed unless the correction is insufficient. However, the earlier the patient is operated on the better the results appear to be (Contargyris 1931 and 1952, Hirsch 1960, Pasila & Sulamaa 1961, Alldred 1966, Attenborough 1966, Somppi & Sulamaa 1971, Reimann & Becker-Andersen 1974, Main et al. 1977, Pous & Dimeglio 1978, Main & Crider 1978, Sudmann 1979).

We therefore wished to test whether the high proportion of relapses of the deformity in the first year of life could be reduced by primary operative treatment followed by closed treatment, instead of the usual primary closed treatment. Secondly, during neonatal surgery, especially in untreated club feet, we wanted to look for features that normally resist primary treatment of congenital club foot.

PATIENTS AND METHODS

From 1968 through 1978, 80 consecutive, very young babies with 118 congenital club feet were seen in Tromsø. The children were divided into three groups (Table 1). Group A. Forty-eight children, born in 1968–1974, with 71 club feet, were given primary closed treatment for about 1 month (Hauge 1959). Group B. Fifteen consecutive children born in 1975–1976, were also mainly treated closed, but for about 4 months. Group C. During the following 2 years, 1977–1978, 17 consecutive children were seen, of whom 15 were operated on, 13 during the neonatal period without any previous treatment.

The club foot deformity was bilateral in about half the cases. In unilateral club foot the proportions of unilateral right and left club feet were about the same in the groups, except in Group A.

* Bjørn Skandfer died in January 1982, before the completion of this study.
Table 1. Patients with club feet born in 1968–1978

<table>
<thead>
<tr>
<th>Group Year of birth</th>
<th>Sex</th>
<th>Patients</th>
<th>Bilateral</th>
<th>Right</th>
<th>Left</th>
<th>Per group</th>
<th>Total club feet</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A) 1968–1974</td>
<td>M</td>
<td>34</td>
<td>36</td>
<td>12</td>
<td>4</td>
<td>71</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>14</td>
<td>10</td>
<td>6</td>
<td>3</td>
<td></td>
<td>19</td>
</tr>
<tr>
<td>(B) 1975–1976</td>
<td>M</td>
<td>10</td>
<td>8</td>
<td>2</td>
<td>4</td>
<td>22</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>5</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td></td>
<td>8</td>
</tr>
<tr>
<td>(C) 1977–1978</td>
<td>M</td>
<td>13</td>
<td>10</td>
<td>5</td>
<td>3</td>
<td>25</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>1</td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>80</td>
<td>76</td>
<td>26</td>
<td>16</td>
<td></td>
<td>118</td>
</tr>
</tbody>
</table>

Other congenital anomalies were seen in 16%, and 19% had relatives with congenital club foot. There were no patients with arthrogryphosis or neurological deficits.

Severity and relapse of club foot deformity

No specific grading of the severity of the club foot was used prior to 1977. In Groups A and B, a non-resistant club foot was therefore defined as a foot clinically correctable by closed methods in a few weeks, irrespective of later relapses. Five boys and six girls in Group A fulfilled these criteria, while 37 out of 48 children were classified as having a resistant club foot. In Group B, 10 out of 15 children had a resistant club foot. Since most of the children in Group C were operated on in the neonatal period, this grading could not be used for them. In this group a non-resistant club foot was defined as a foot in which the inversion of the hindfoot could be completely corrected by gentle eversion. Such correction was possible in only two out of 17 patients, while the equinus of the ankle could not be corrected by gentle means in any club foot. Thus, 15 out of 17 patients had a resistant club foot.

In Groups A, B and C, a relapse was defined as a club foot with a recurrent deformity needing treatment after the initial period of treatment in plaster was over (Laaveg & Ponseti 1980).

Primary treatment in Groups A and B

The club feet were treated by manipulation without anesthesia during the first week of life. The correction so obtained was maintained in a padded encircling plaster cast reaching from the toes to the groin, with the knee at right angles, and the foot pointing forward. The patients were usually treated weekly at first; and later, at intervals of up to 4 weeks. In addition, in one foot (Group A) and in nine feet (Group B) that were resistant to closed treatment, tenotomy of the heel cord was done as part of the primary treatment (Table 2).

After the primary treatment as above from 14 to 300 days (median 41 days), relapse of the deformity was often seen within weeks in Group A. Therefore, in Group B the period of closed treatment was increased to about 4 months (Somppi & Sulamaa 1971); it varied from 49 to 221 days (median 110 days). After primary treatment in plaster, day and night splints were usually used in Group A, but only irregularly in Group B.

Primary treatment in Group C

Closed treatment was used when the inversion of the hindfoot was correctable by gentle eversion of the foot (without concurrent correction of equinus). Since such correction was possible in two feet only, 23/25 club feet were operated on (Table 2). The operations were performed in the neonatal period of the 4th to 11th day after birth except in two babies, who were operated on when 5 and 16 weeks old.

The operation was usually performed in a bloodless field. The first seven children (nine club feet) were given general anesthesia and a longitudinal medial incision was made in the foot. The tibialis posterior tendon was divided near its insertion in the navicular bone, i.e. distal tenotomy. The joint capsule in the talonavicular joint was cut medially to such an extent that the medially luxated navicular bone could easily be repositioned. The wound was closed in layers. The achilles tendon was divided near its insertion through a separate, small wound.

A different approach was used on the following eight operatively treated children (14 club feet). Under local anesthesia, the tibialis posterior tendon was divided, through a small longitudinal skin incision, just proximal to and behind the medial malleolus, i.e. proximal tenotomy. The achilles tendon was cut through the same incision. The incision was closed in layers. In addition, in six club feet with marked cavus and/or marked forefoot adduction, the abductor brevis tendon was cut through a small separate skin incision.

After the operation, all the club feet were im-
Table 2. Primary treatment in 118 club feet

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Type of treatment (no. of feet)</th>
<th>Total club feet</th>
<th>Days in plaster</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Closed only</td>
<td>Closed, then tenotomy</td>
<td>Closed only</td>
</tr>
<tr>
<td>(A)</td>
<td>48</td>
<td>70²</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>(B)</td>
<td>15</td>
<td>13³</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>(C)</td>
<td>17</td>
<td>2</td>
<td>2</td>
<td>21</td>
</tr>
</tbody>
</table>

Days are given as median values with ranges in parentheses.

1. Duration of initial immobilization in plaster could not be ascertained in one patient (Group B).
2. Fifteen (21%) and 3 (23%) respectively were not operated on in the follow-up period. There was no significant difference ($P > 1.0$) in the duration of treatment between Groups B and C, but a marked difference ($P = 0.00008$) between the closed treated groups A and B.

Immediately immobilized by generously padded plaster slabs with the knee bent at right angles and with gentle everting pressure on the foot during setting. The stitches were removed about 10 days later and new slabs applied. The plaster bandages were thereafter changed once a week, then every 2 weeks, and thereafter at a maximum interval of 4 weeks. At each session the foot was gently rotated outward relative to the lower leg, and at the same time the equinus of the ankle was gently corrected.

Primary closed treatment was done without anesthesia in two club feet only, exactly as in the rest of Group C, but without any operation.

A postoperative period of closed treatment of about 4 months was used in Group C; the duration of immobilization in the plaster bandage varied from 41 to 150 days.

Additional treatment

Groups A and B. When a relapse was noted, closed treatment under general anesthesia was the treatment of choice in Group A (Hauge 1959), while operative treatment was used in Group B (Sudmann 1979). Except for achillotomy, operative treatment was not used before the age of 1 year in Group A, in older children relapses were treated by posterior and/or medial soft tissue release. In three club feet such a release operation was combined with an arthrodasis of the calcaneocuboid joint (Evans 1961), and in 22 feet the lateral bone column was shortened by resecting the middle part of the cuboid bone without any arthrodasis of the calcaneocuboid joint (Sudmann 1979). Intoeing was occasionally treated by rotational osteotomy of the lower leg (Hauge 1959). In Group B, relapses were treated by soft tissue release. In addition, in six club feet the lateral column was shortened, as above (Sudmann 1979).

Group C. Soft tissue release included plantar release in cases with marked cavus deformity. Marked anterior adduction of the forefoot was treated by operative mobilization of the tarsometatarsal and intermetatarsal joints (Heyman et al. 1958, Kendrik et al. 1970).

Follow-up and radiographs

All the children were seen in the out-patient department during 1978–1979, except seven children in Group A and one in Group B. A few of the patients refused to be radiographed and others had moved from the district. All but two of the children in Group C were reexamined in 1981; the latest available notes and radiographs were used in those not reexamined. Thus, the observation period varied from 5 to 12 years (median 8 years) in Group A, from 2 to 5 years (median 4 years) in Group B, and from 18 months to 4 years (median 4 years) in Group C.

The torsion of the lower leg was defined as the angle between the intermalleolar axis and the frontal plane with the patient in a sitting position. Further, the passive motion in the subtalar joint was estimated by rotating the foot outward relative to the lower leg toward (angle negative) or away from (angle positive) a parasagittal plane (Sudmann 1979). From standardized radiographs of both feet anteroposterior (ATC) and lateral talocalcaneal angles (LTC) and the talocalcaneal index (TCI) were calculated (Beatson & Pearson 1966). In the anteroposterior radiographs talocalcaneal overlap was defined as the ratio between the distance from the lateral part of the head of the talus to the
medial side of the calcaneus and the breadth of the anterior part of the calcaneus; a negative ratio indicates no overlap at all. This ratio was assessed in Groups B and C only.

**Statistics**

Subdivision of the groups into unilateral and bilateral cases did not alter the statistical results. Unilateral and bilateral cases were therefore pooled, and differences between the two groups A and B and B and C, respectively, were tested by the Wilcoxon rank sum test for unpaired data (the two-sample test). Contingency tables were tested by the Chi-square test (Swinscow 1976). The P values were found by two-tailed tests and differences were considered significant when \( P < 0.05 \).

**RESULTS**

During *distal* tenotomy and capsulotomy of the talonavicular joint (Group C, first nine feet), the navicular bone was found to be luxated medially onto the ventral surface of the medial malleolus. The articular surface of the head of the talus was flattened and medially positioned. After distal tenotomies and capsulotomy, the inversion of the hindfoot was correctable by the force of gravity. The equinus of the ankle could usually be corrected manually. To suture the wound without tension, these nine feet had to be repositioned in the equinovarus position. After *proximal* tenotomy of a taut tibialis posterior tendon and distal tenotomy of a taut achilles tendon (without capsulotomy) (Group C, 14 feet), the resistance to hindfoot correction was markedly reduced. The tendons of the other two muscles on the back of the lower leg were also taut.

In group C, there was only one relapse of the hindfoot deformity during the first year of life after *operative* treatment. In contrast, among the club feet that had primary *closed* treatment in Groups B and C, relapse of the hindfoot deformity was noted in 14 out of 24 feet (Table 3). In Group A, a relapse of the hindfoot deformity was recorded in 54 out of 71 club feet (Table 3).

While relapse of the hindfoot deformity was the main therapeutic problem in Groups A and B, cavus deformity and marked anterior adduction of the forefoot were the main problems in Group C. These midfoot and forefoot deformities did not respond to primary treatment, including tenotomy of the abductor hallucis. However, in the first 2 years of life, additional treatment was needed less often in the operatively treated Group C than in Groups A and B (Table 4).

At reoperation in four club feet (Group C), the proximal and distal ends of the tibialis posterior tendon had not grown together. No postoperative complications were noted in either group.

In Group C, after the plaster bandages had been discontinued (after which no day and night splints were used), passive dorsal motion in the ankle decreased markedly during the first year of life in four operated children (seven club feet) and in one child (one club foot) given closed treatment. This trend was more marked in Groups A and B. At follow-up, the median passive dorsal motion in the ankle joint and rotation in the sub-

Table 3. Relapse of hindfoot deformity in first year of life

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>No. of club feet</th>
<th>Without relapse</th>
<th>Relapse</th>
<th>No data</th>
<th>Total club feet</th>
</tr>
</thead>
<tbody>
<tr>
<td>(A)</td>
<td>48</td>
<td></td>
<td>12 (17%)</td>
<td>54 (76%)</td>
<td>5 (7%)</td>
<td>71 (100%)</td>
</tr>
<tr>
<td>(B)</td>
<td>15</td>
<td></td>
<td>9 (41%)</td>
<td>13 (59%)</td>
<td>0</td>
<td>22 (100%)</td>
</tr>
<tr>
<td>(C)</td>
<td>17</td>
<td></td>
<td>23 (92%)</td>
<td>2 (8%)</td>
<td>0</td>
<td>25 (100%)</td>
</tr>
</tbody>
</table>

1. Four feet (two patients) operated on for midfoot and forefoot deformities.
2. Equinus of 10 degrees in one foot, given primary closed treatment and restricted dorsiflexion in one foot given primary operative treatment. Thus, only 4% relapses in operatively treated club feet.

There was a significant difference \( (P = 10^{-9}) \) between the groups.
Table 4. Additional treatment in first two years of life

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>No. of feet given additional treatment</th>
<th>Total club feet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>(A)</td>
<td>48</td>
<td>17(^1) (24%)</td>
<td>54 (76%)</td>
</tr>
<tr>
<td>(B)</td>
<td>15</td>
<td>6 (27%)</td>
<td>16 (73%)</td>
</tr>
<tr>
<td>(C)</td>
<td>17</td>
<td>13 (60%)</td>
<td>12(^2) (40%)</td>
</tr>
</tbody>
</table>

1. Operative treatment later in five markedly deformed feet.
2. Reoperation of five patients because of: forefoot adduction (four feet), restricted dorsal ankle motion (five feet) and cavus deformity (two feet). Dynamic splint in one closed treated patient (one foot) because of equinus of ankle.

There was a significant difference (\(P = 0.03\)) between the groups.

Table 5. Anterior adduction of foot in normal feet and in treated club feet

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of feet</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–5 (degrees)</td>
</tr>
<tr>
<td>Normal foot (B+C)</td>
<td>17</td>
</tr>
<tr>
<td>Club feet</td>
<td></td>
</tr>
<tr>
<td>(A)</td>
<td>71</td>
</tr>
<tr>
<td>(B)</td>
<td>22</td>
</tr>
<tr>
<td>(C)</td>
<td>25</td>
</tr>
</tbody>
</table>

There was a significant difference (\(P = 0.0004\)) between the groups.

Table 6. Flat-topped talus in radiographs in club feet

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of patients</th>
<th>Not flat-topped</th>
<th>Flat-topped data</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>Yes</td>
<td></td>
</tr>
<tr>
<td>(A)</td>
<td>48</td>
<td>12</td>
<td>52(^3)</td>
<td>7</td>
</tr>
<tr>
<td>(B)</td>
<td>15</td>
<td>6</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>(C)</td>
<td>17</td>
<td>20</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

1. Peroperatively gross ankle joint deformation in nine feet (six patients).

There was a significant difference (\(P = 10^{-8}\)) between the groups.

Groups A and B, but the follow-up period was much longer in Group A than in Groups B and C (Table 5). Gross joint deformation of the ankle and/or talonavicular joints was noted peroperatively in nine feet in Group A, whereas no obvious pressure necrosis of articular cartilage (Salter & Field 1960) was seen in Groups B and C. No flat feet were seen among the treated club feet.

In children with unilateral club foot (all groups), the torsion of the deformed lower leg was equal to or greater than the torsion of the undeformed, presumably normal, lower leg, except one.

Radiographs

The proportion of club feet with a flat-topped talus as seen in lateral radiographs of the ankle was much lower in the operatively treated Group C than in the closed treated Groups A and B (Table 6, Figures 1–2). Flattening of the superior surface of the talus appeared at the age of 16–131 months (median 66 months) in Group A, at the age of 17–44 months (median 23 months) in Group B, and at the age of 13 months in Group C. In Group C, a flat-topped talus was only observed in one child with two club feet. Some of the children in Group A had not been radiographed for 5–10 years. This may contribute to the relatively high median value of 66 months for the flattening of the superior surface of the talus.
There was no significant difference in the anteroposterior (medians 24–30 degrees) and the lateral TC angles (medians 28–32 degrees) between Groups A and B, and B and C. A negative LTC angle was seen in one primary closed treated club foot only (Group C); this foot responded to additional closed treatment. There was no significant difference in the TC index (medians 54–64 degrees) between Groups A and B, and B and C, nor the TC overlap (median 0.17) between Groups B and C. In children with one club foot (Groups B and C) the median ATC angle in the presumably normal feet was 44, the LTC angle 46, the TC index 86 degrees and the TC overlap zero.

**DISCUSSION**

In congenital club foot, inversion of the heel and equinus of the ankle are the deformities most detrimental to the patient’s ability to walk (Beatson & Pearson 1966). Any method of primary treatment should therefore take into account those features that are likely to resist primary correction – and cause relapse – of the hindfoot deformity. Our results show that such relapses are often noted during the first year of life (Table 3). On the other hand, when the number of relapses during the first year of life was reduced, our results show that the need for additional treatment during the first 2 years of life was also significantly reduced (Table 4). These findings suggest that relapse of the hindfoot deformity during the first year of life may be a bad prognostic sign.

Inversion and subluxation of the cal-
caneo-cuboido-navicular bone complex, deformed bones, contracted joint capsules and ligaments, taut tendons, and taut skin on the medial side of the ankle, can separately or together resist correction of the hindfoot deformity.

As shown in a few children, eversion of the hindfoot was possible by the force of gravity when the skin incision on the medial side of the foot was wide open, and the tibialis posterior tendon, the achilles tendon and the medial part of the joint capsule in the talonavicular joint were cut. Further, the equinus of the ankle could then usually be gently corrected. Finally, resistance to the initial correction of the hindfoot deformity in children primarily treated by tenotomies only was markedly reduced. These results indicate that the soft tissues — and not the bones — were the main features resisting hindfoot correction in our patients.

Marked fibrosis of joint capsules, ligaments and tendon sheaths was always seen during release operations in club feet given closed treatment (Sudmann 1979). But, in untreated club feet such fibrosis was not seen at neonatal surgery. Although fibrosis has been found in club feet in embryos (Ippolito & Ponseti 1980), our results indicate that the fibrosis seen in relapsed club feet is iatrogenic. Further, our results suggest that fibrosis of joint capsules, ligaments and tendon sheaths is not the central feature of resistance to hindfoot correction in the newborn.

The tibialis posterior muscle is the main inverter of the foot and the triceps surae muscle the main plantar flexor. In congenital club feet the tendons of these muscles are taut. When these tendons were cut, the resistance to hindfoot correction was markedly reduced, especially before the skin incision in the taut skin was sutured. Although the tendons of the flexor hallucis longus and the flexor digitorum longus were also taut, these findings suggest that the posterior tibialis tendon, the achilles tendon — and their muscles — and the skin on the inside of the ankle are the main features resisting primary treatment of the hindfoot deformity. This is in accord with anatomical and histochemical studies of muscle in club foot (Wiley 1959, Gray & Katz 1981).

Our results show that passive dorsiflexion decreases in operated feet as the children — and their lower legs — grow in length. Since a cut achilles tendon heals quickly, these results support the contention that the triceps surae muscle is an important dynamic feature in relapse of the hindfoot deformity (Wiley 1959, Gray & Katz 1981). Since the relative increase in lower leg length is large in the first year of life, external splinting (first, e.g., plaster bandages, later day and night splints) during this year appears to be essential.

When the tibialis posterior tendon was not initially cut, relapse of the inversion of the hindfoot in the first year of life was often seen, but when it was initially cut no such relapse was noted. As the tibialis posterior is the only muscle able to produce club foot by its contraction or shortening alone (Barr 1958, Fried 1959), our results suggest, first that the tibialis posterior is an important feature to resist primary treatment of the hindfoot deformity and of its relapse, and second that a cut tibialis posterior tendon may not heal spontaneously in club feet responding to treatment. No flat feet were seen in club feet after tenotomy of the tibialis posterior. These results suggest that club feet may have and/or develop during treatment some tarsal joint stiffness.

No specific grading of severity of the club foot had been used in Groups A and B, whereas in Group C the hindfoot deformity was graded according to whether or not the inversion of the hindfoot could be completely corrected by gentle eversion; only two out of 25 club feet could so be corrected and thus not operated on. But in Groups A and B, 21–23% of the club feet did not need operative treatment at all during the first years of life (Table 2). These data may suggest that in Group C more than one tenth of the club feet were operated on unnecessarily. On the other hand, these data may also suggest that the club feet in Group C were more deformed than those in Groups A and B. In another heterogeneous group of 31 babies (51 club feet), correction of inversion by gentle eversion was possible in about 16% of the club feet (Sudmann, unpublished data). These data support the latter contention.

A club foot cannot be treated by operation alone. Thus, the deformity cannot be corrected without applying external forces greater than the
static and dynamic features resisting correction. Opposing forces can do permanent damage to the bones and joints of the foot, and occasionally to the lower leg (Harrenstein 1933, Salter & Field 1960, Keim & Ritchie 1964). During primary treatment, the pressure created on the delicate joint cartilage and the bone tissue can be much relieved by reducing the main internal forces in the foot itself, that is by tenotomy of the tibialis posterior tendon and of the achilles tendon. Our results indicate that the proportion of flat-topped taluses can be very significantly reduced by such neonatal tenotomies (Table 6). On the other hand, when tenotomies markedly reduced the external forces necessary for the correction of the hindfoot deformity, midfoot and forefoot deformities might thereby be undercorrected. In Group C, cavus deformity and marked anterior adduction of the forefoot present at birth needed later additional operative treatment in four out of six feet. However, in the present study there was nothing to indicate whether or not such treatment could have been prevented by primary closed treatment only.

Our findings in unilaterally affected children show that the torsion of the lower leg on the affected side is the same as (or greater than) on the normal side. These findings suggest that intoeing in treated club feet should not be corrected by a rotational osteotomy on the lower leg (Hauge 1959), but by correcting the deformity of the foot (Sudmann 1979).

REFERENCES

Harrenstein, R. J. (1933) Über die Beschädigung der Epiphysen der Tibia und Fibula bei der Behandlung des Klumpfusses. Z. Orthop. 59, 115–125.

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