STATIC AND DYNAMIC PROBLEMS IN SPASTIC CEREBRAL PALSY

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Postural abnormalities of joints and contractures of the surrounding muscles are recognised as important complications in cerebral palsy. Such postural abnormalities are due primarily to the cerebral disturbance, which causes varying degrees of paresis in the groups of muscles controlling the joints. This muscle imbalance is accentuated by the spasticity, which is usually most pronounced in the strongest muscle groups.

In the first stage there is a spastic or functional contracture which presents as a postural abnormality when the patient moves, whereas at rest there is a normal range of joint movement. In the absence of treatment, the strongest muscle groups become relatively too short on account of the absence of growth stimulation from the weaker antagonists, and an organic contracture develops with consequent limitation of movement (Sharrard 1967). If the organic contracture persists, an arthrogenic contracture may develop, for during growth the joint surfaces gradually become adjusted to the altered functional position.

The aim of treatment is to enable the patient to sit, stand and walk as naturally as possible. This is attempted by preventive conservative treatment, but when contractures and postural abnormalities develop, these need correction by operation.

Because maintenance of the sitting and standing positions requires that the centre of gravity of the trunk is vertically above the supporting surface, a change in one joint inevitably results in compensatory changes in the other joints. It is therefore all-important that the patient's static and dynamic problems should be analysed before treatment is begun (Melhove and Plum 1965).

The aim of the present study is to survey the static and dynamic relationships between the various postural abnormalities in the sagittal plane—in other words, as they are observed from the side—because these postural abnormalities illustrate to a special degree the functional relationships between the various joints of the lower limbs. The outcome of the most frequent contractures, whether isolated or in various combinations, is also examined.

Figure 1 shows schematically those muscle groups which have the greatest significance for the mechanical problems discussed in the next section. The abdominal muscles have been omitted for the sake of clarity.

EXAMPLES

ISOLATED CONTRACTURE OF THE HIP FLEXORS

A flexion attitude of the hips without other postural abnormalities is incompatible with the erect position, because the centre of gravity no longer lies vertically above the weight-bearing surface (Fig. 2 a and b). By flexing the knee so that the load falls on the forefoot, part of the pelvic tilt is corrected (Fig. 2c). However, the centre of gravity does not return to a position directly above the weight-bearing surface until the upper part of the spinal column moves backwards, causing a lumbar lordosis (Fig. 2d). With the same degree of contracture in the hip flexors, different patients have varying degrees of lordosis and knee flexion, depending on the function of the abdominal and gluteal musculature (Fig. 2 e and f).

Treatment—The postural abnormalities are reduced by lengthening the flexors of the hip.
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ISOLATED CONTRACTURE OF THE KNEE FLEXORS

A flexion attitude of the knee joint without postural abnormalities in other joints is again incompatible with the erect position (Fig. 3a). It produces flexion of the hips without lordosis, and the patient has to use the forefoot for support (Fig. 3b). The length of stride is short, and because of the short hamstrings the patient has to rotate the pelvis in order to bring the leg forward in the swing phase. The sitting position is compromised by lumbar kyphosis due to limitation of hip flexion (Fig. 4a) (Seymour and Sharrard 1968, Reimers, in press).

Treatment—By lengthening of the hamstrings, the patient once again can extend the knees (Fig. 3c). The sitting position returns to normal, because a normal lumbar curve is again possible when hip flexion becomes normal (Fig. 4b).

Faulty treatment—Primary lengthening of the hip flexors will not immediately improve the static picture when the postural abnormality is not due to their contracture. From the long-term point of view, weakening the hip flexors may indeed produce a contracture of the antagonists, the hip extensors, whereby the sitting position deteriorates still further.

Lengthening of the tendo calcaneus lowers the heel to the ground (Fig. 5a and b). The previous forefoot position has a purely mechanical effect favouring extension of the knee joint, but this effect ceases after tendon lengthening, and the quadriceps alone has to counteract the stronger and contracted hamstrings. The quadriceps being unable to do this, the patient sinks further at the knees (Fig. 5c). Because the knee flexion causes strain on the elongated tendon, the triceps musculature does not regain its strength after the operation. As a result
contracture may develop in the now relatively stronger dorsiflexors, causing a calcaneus deformity. If an attempt is made later to extend the knee by lengthening of the hamstrings, the patient's weight now comes on the heel, and the weak quadriceps being rarely able alone to extend the knee (Fig. 5d), the patient's weight continues to be transmitted through the flexed knee (Fig. 5e). The same argument as for lengthening of the tendo calcaneus also holds if the foot collapses into valgus during weight transmission, as the extending effect on the knee is missing when the tendon is relatively too long.

![Diagrams of knee extension and flexion](image)

**FIG. 5**
Diagrams to illustrate faults in the treatment of contracture of the hamstrings.

![Diagrams of knee extension and flexion](image)

**FIG. 6**
Diagrams to illustrate the effects of an untreated contracture of the hamstrings.

So-called quadriceps activation by shortening of the ligamentum patellae followed by a period in a plaster cast can at most produce transient extension of the knee (Fig. 6a and b). Because the hamstrings are not weakened and the quadriceps is still weaker than the knee flexors, there will be a recurrence due to muscle imbalance (Fig. 6c). If there are genuine indications for shortening the ligamentum patellae, this should only be done after lengthening and weakening of the hamstrings.

**ISOLATED CONTRACTURE OF THE TRICEPS SURAE**

This results in an equinus foot, with the supporting surface reduced and the balance poor (Fig. 7a). The foot is often deformed, either to a postural flat foot or to a rocker foot (Fig. 7b). Especially in hemiplegics, an equinus foot results in a tendency for the knee joint to hyperextend, permitting the heel to come into contact with the supporting surface (Fig. 7c).

**Treatment**—Lengthening of the tendo calcaneus before the development of further deformities.

**Faulty treatment**—Avoidance of treatment.
Instead of lengthening the tendo calcaneus, in other words all three components of triceps surae, the origin of the gastrocnemius muscles can be transposed from the femur to the tibia (Fig. 8 a and b) (Hagberg, Lemperg and Lundberg 1968). However, there is a risk of hyperextension developing in the knee joint if a functional or organic contracture of the soleus muscle is present or develops later. The resulting equinus foot will mechanically press the knee backward, and with the bellies of the gastrocnemius no longer resisting hyperextension of the knee, this occurs more easily than before the transposition (Fig. 8c).

**COMBINED CONTRACTURE OF HIP AND KNEE FLEXORS**

These contractures result in hip flexion, lumbar lordosis, flexion of the knee and overloading of the forefoot. When the patient stands, the position can be erroneously interpreted as due to isolated contracture of the hip flexors (Fig. 9a). It is not until the patient walks that the length of stride is seen to be short and the pelvis is seen to rotate when the leg is swung forward, as in the case of contracture of the knee flexors.

*Treatment*—Primarily, lengthening of the hip flexors. When these are lengthened, the pelvis tilts to the normal position and the lumbar lordosis disappears, provided that contractures have not already developed in the long extensors of the spine. With a level pelvis the distance between the origin and insertion of the hamstrings decreases. The hamstrings being now relatively longer, the knee is better able to extend (Fig. 9b). If after the operation there is still significant knee flexion, the hamstrings must be lengthened so that the knee can extend and the heel reach the ground.

*Faulty treatment*—Primary lengthening of the knee flexors certainly may enable the knee to be extended (Fig. 10 a and b), but because the contracture of the hip flexors has been retained, the pelvis tilts still more forward and the lumbar lordosis increases (Fig. 10c). If the patient is unable to increase the lordosis, he instead sinks once more at the knees in order to retain his balance (Fig. 10d).

If the contracted hip flexors are then lengthened as a secondary procedure, the patient will only be able to straighten up at the pelvis provided the gluteal muscles are strong enough to function as hip extensors by themselves, because the hamstrings become longer and hence weaker when the inclination of the pelvis decreases (Fig. 10e). As a rule the gluteal muscles
are too weak, so that the patient still stands and walks with hip and knee flexion (Fig. 10f) (Anthonsen 1966, Bleck 1971).

If instead of primary lengthening of the hamstrings, a modified Eggers operation is performed with transposition of some of the hamstrings from the tibia to the femur under tension (Eggers and Evans 1963), the contracture of the hip flexors is not eliminated (Fig. 11 a and b). After a “successful” transposition it is true that the knee can be extended, but because the range of flexion of the hip is diminished, the length of stride becomes less and the sitting position becomes further compromised (Fig. 12 a and b).

![Fig. 10](image)
Diagrams to illustrate the conduct of combined contractures of hip and knee.

![Fig. 11](image)
Figure 11—Diagrams to show the effects of an Eggers operation in the presence of contracture of the hip flexors. Figure 12—Diagrams to show how the sitting position may be affected by an Eggers operation.

**COMBINED CONTRACTURE OF KNEE FLEXORS AND TRICEPS SURAE**

These contractures produce flexion of the hip, flexion of the knee and equinus of the foot (Fig. 13a). With moderate degrees of contracture of the hamstrings, it is occasionally seen that when the patient attempts to extend the knees, the knees pass through the neutral position into hyperextension. Contracture of the triceps surae is a stimulus to this, and the hamstrings accentuate the extension when the knee is slightly past the extended position (Fig. 13b).

*Treatment*—Lengthening of the knee flexors and of the calcaneal tendons simultaneously. *Faulty treatment*—Elongation of the knee flexors alone results in a weakening of these muscles. The length of stride no doubt becomes greater and the sitting position better, but there is a risk of hyperextension of the knee joint when there is still an equinus position of the foot, particularly after an Eggers operation or distal elongation of the hamstrings (Fig. 14 a, b and c). Elongation of the tendo calcaneus alone results in the patient sinking further at the knees (Fig. 5 a, b and c).
COMBINED CONTRACTURE OF THE FLEXORS OF THE HIP AND KNEE AS WELL AS OF TRICEPS SURAE

This produces lordosis, flexion of the hip, flexion of the knee and equinus of the foot with a short stride and a poor sitting position.

Treatment—Lengthening of the contracted muscles, beginning at the hip and soon afterwards at the knee and foot.

Faulty treatment—Correction of the contractures in the reverse sequence, or correction of a single contracture. In the latter case, the least harm is done by correction of the hip flexion deformity.

SUMMARY

1. Analysis of the static and dynamic conditions in spastic cerebral palsy leads to the conclusion that contractures of the hip, knee and ankle should be released from above downwards, and that the full benefit is obtained only when all contractures have been corrected.
2. Only when these joints are mobilised by removal of the significant contractures is the spastic patient able, despite other handicaps, to sit, stand and walk with the least hindrance and the least cosmetic fault.
3. In our Danish experience, inadequate or poor results from the orthopaedic surgery of cerebral palsy are mainly due to the fact that patients with contractures are operated on too little and too late, or not in the correct sequence.

REFERENCES

MØLHAVE, A., and PLUM, P. (1965): Clinical Analysis of Static and Dynamic Patterns in Cerebral Palsy with a View to Active Correction. Archives of Physical Medicine and Rehabilitation, 37, 480.