Deformities in the child with cerebral palsy have been ascribed to muscle imbalance (Sharrard 1961) and increased tone (Pollock 1959) or to the type of cerebral palsy (Bobath and Bobath 1975). As far as we know, the position in which the child is nursed, especially during the first year of life, has not been considered as a cause of deformity.

It is generally agreed that position in the postnatal period can be a cause of deformity in the normal baby. Paine (1961) suggested that plagiocephaly was caused by postnatal head posture, and Hay (1971) found that plagiocephaly was present in 10 percent of normal babies. Scott (1956) reported that infants commonly had lateral curvatures of the spine which could be seen on x-rays but not on clinical examination, all of which had resolved by the age of two years. Other asymmetries associated with plagiocephaly are unilateral fisting, asymmetrical groin creases, apparent shortening of one lower limb and asymmetry of gait (Robson 1968). We accept the asymmetrical deformities of plagiocephaly, unilateral bat ear, facial and thoracic asymmetry, pelvic obliquity and apparent shortening of one leg – some or all of which may be present in normal babies – as forming the ‘squint’ baby syndrome. Because asymmetrical deformities also occur in children with cerebral palsy, we thought it worthwhile to compare the pattern of deformity in a group of ‘squint’ but otherwise normal babies with a group of cerebral-palsied children with asymmetrical deformities to see if there is any relationship.

Commentary

Before the 1970s the cause of soft tissue deformity in cerebral palsy (CP) and poliomyelitis was seen as the same: an imbalance of opposing muscle strengths (equated, for CP, as ‘spasticity’) which affected tissue growth across a joint. For polio, a difference of two grades of the Oxford muscle strength scale was said to cause fixed deformity. However, an association between posture and deformity had been noted in infantile plagiocephaly by Paine; in infantile postural scoliosis by Lloyd-Roberts; and in CP.

A few physiotherapists, together with a Florentine paediatrician, Adriano Milani-Camparetti, disagreed vociferously at Ronnie Mac Keith’s meetings, asserting the cause to be an imbalance of posture rather than strength (otherwise we would all have equinus and wrist/finger flexion deformities). Such objections were swept aside since ‘normal agonists and antagonists are both strength 5’ (as true as it is irrelevant). A more surprising, but inadvertently helpful, dismissal was ‘that normal people are different’, for here we could agree as we saw the difference being that they had balanced posture. However, both shared the same physiological response to bone growth: to respond to the forces applied across the joint. Balanced posture = balanced muscle lengths; imbalanced (pathological) posture = fixed deformity.

Even today these deformities are referred to as ‘contractures’, emphasizing the shorter (movement restricting) tissues and ignoring the inevitably accompanying overlong antagonists which are an integral part of the deformity. ‘Contracture’ (which it is not) also implies a pathological mechanism, which this isn’t.

What marked this article out for me was its challenge. A paediatric neurologist and an orthopaedic surgeon, both well experienced with CP, threw down the gauntlet to a strongly entrenched view. From that time, the posture-rationalized acceptance of deformity and management of early deformity in CP moved from the tidy world of the lower motor neuron into the poorly mapped uplands of the central nervous system. It also helped to open up other, wider, discussions, e.g. the poor outcome of some bilateral heel cord lengthening in spastic diplegia: where subsequently overlong heel cords could be seen as a physiological response to a postural change, rather than necessarily a surgical over-lengthening.

Today’s reader might wonder why this article was chosen, for its message must seem obvious; but it wasn’t obvious then, as the directness of the title makes abundantly clear.

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References

Yet another example of the ubiquitous Le Chatelier-Braun principle.