Skeletal maturation in cerebral palsy

Commentary

Skeletal health in children is made up of a number of factors including the overall pattern of the skeleton, the appropriate size and shape of individual bones, bone mass and internal architecture of both tubular and plate-like bones, and bone metabolism and turnover. Where among this group of attributes does skeletal maturity fit in?

Skeletal maturity has been assessed either alone or in association with the variables indicated above for many years. A markedly advanced or delayed skeletal maturity may raise questions as to hormonal or other systemic disturbances that require further investigation in the individual patient. Such disturbances are often associated with other effects on bone that require attention. For example, corticosteroid-induced delay in skeletal maturation may be associated with bone loss, microarchitectural deterioration, and increased fracture risk.

Altered skeletal maturity does not of itself, however, appear to confer any particular risk in terms of immediate harm to the skeleton. There have been concerns that delayed bone age might result in a reduced final height and bone mass. Some of the results obtained in other studies of delayed bone age and adult bone mass might reflect the uncritical use of bone density measurements by dual energy X-ray absorptiometry (DXA). Studies in which measurements are adjusted for bone size indicate that volumetric bone density is probably normal.[1]

The study in this issue of DMCN[2] also used DXA to assess bone density and, as the authors acknowledge, the results obtained are a reflection both of bone mass and bone size. The correlation of reduced skeletal maturity with reduced height and bone density is not surprising. An alternative interpretation of the data would be that the reduction in bone density is more a reflection of the small bone size. A well-reported feature of reduced muscle activity is reduced bone size; reduced stature and overall bone dimensions in cerebral palsy (CP) would, therefore, be expected. One element of the overall picture that is unclear to me is the extent to which reduced bone density might impact on the assessment of skeletal maturation using the Fels method.

The use of anticonvulsant medication, as indicated by the authors, is associated with a reduction in bone mass in children who are not ambulant. Anticonvulsant medication may accelerate the metabolism of vitamin D, and vitamin D intake from diet and sunlight exposure may be reduced in this population, increasing the risk of fracture.[3] Assessing vitamin D status in children who are immobilized with CP should be a routine event with appropriate supplements being given to all affected children. The American Academy of Paediatrics recently suggested that all members of the population should receive 200IU/day of vitamin D, irrespective of race or age.[4] Such a dose would be unlikely to accelerate any immobilization-induced calciuria.

There is no doubt that exercise increases bone size in proportion to an increase in muscle cross-sectional area. Physiotherapy is routinely used to maintain range of movement in children with severe CP and studies using vibrating plates suggest that the action of such micro-strains directly on bone can increase bone mass in the appendicular skeleton.[5] There are suggestions, however, that over-vigorous physiotherapy could put patients at risk for fractures at the ends of long bones and, certainly, physiotherapy regimes should be tailored to the state of the individual patient.

In the short term, skeletal health in children with CP might be judged in terms of propensity to fracture and, on the metabolic side, the development of osteomalacic or rachitic changes. This being so, it is not clear to me that skeletal maturity needs to be assessed regularly in these patients for assessment of skeletal health in the short term. An exception might be when, during the planning of limb lengthening procedures, information on growth potential in individual limbs is required.[6] In the longer term, bone age might give some information about final height but I would be unlikely to prescribe a medication to increase bone length if bone width, which confers most of the strength in long tubular bones, were not expected to benefit as well.

Although Henderson and colleagues provide an interesting set of observations, I would not expect the information to change my clinical practice for a given individual.

Nick Bishop
Professor of Paediatric Bone Disease, University of Sheffield

DOI: 10.1017/S0012162205000435

References

1. Bertelloni S, Baroncelli GI, Ferdeghini M, Perri G, Saggese G. (1998) Normal volumetric bone mineral density and bone turnover in young men with histories of constitutional delay of puberty. *J Clin Endocrinol Metab* **83:** 4280–4283.
2. Henderson R, Gilbert SR, Clement ME, Abbas A, Worley G, Stevenson RD. (2005) Altered skeletal maturation in moderate to severe cerebral palsy. *Dev Med Child Neurol* **47:** 229–236.
3. Bischof F, Basu D, Pettifor JM. (2002) Pathological long-bone fractures in residents with cerebral palsy in a long-term care facility in South Africa. *Dev Med Child Neurol* **44:** 119–122.
4. Greer FR. (2004) Issues in establishing vitamin D recommendations for infants and children. *Am J Clin Nutr* **80** (Suppl. 6)**:** 1759S–1762S.
5. Caulton JM, Ward KA, Alsop CW, Dunn G, Adams JE, Mughal MZ. (2004) A randomised controlled trial of standing programme on bone mineral density in non-ambulant children with cerebral palsy. *Arch Dis Child* **89:** 131–135.
6. Erickson T, Loder RT. (2003) Bone age in children with hemiplegic cerebral palsy. *J Pediatr Orthop* **23:** 669–671.