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Osteogenesis imperfecta (OI) represents a spectrum of condi tions linked by a qualitative and/or quantitative abnormality of collagen production. Most identified mutations a ff ect the collagen genes. The bone may break easily but it heals promptly and well. All structures containing collagen may be a ff ected, accounting for the ligamentous laxity , blue sclerae and poor teeth in some phenotypes. Cyclical bisphosphonate treatment decreases bone resorp tion and turnover. This reduces bone pain and the fracture rate, promoting weight-bearing mobility and bone strength (Figure 44.12). Following fracture, treatment options range from simple casting techniques to mor e specialised surgical procedures to correct/maintain bony alignment while allowing growth. Intramedullary techniques f or stabilisation of fractures or osteotomies are preferred to plate fixation. Rehabilitation should start promptly . Summary box 44.4 Metabolic bone disease /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Thomas Geo ff rey Barlow , 1915–1975, orthopaedic surgeon, Salford Royal Hospital, Salford, UK. Marius Ortolani , 1904–1987, orthopaedic surgeon, Instituto Provinciale Per L’Infanzia di Ferrara, Italy , described this test in 1937.

Rickets, from nutritional or other causes, is characterised by a failure of bone mineralisation X-linked hypophosphataemic rickets is a dominant condition, affecting boys and girls; in some countries, treatment is with monoclonal FGF23 antibodies In OI: There is defective type I collagen production In severe forms frequent fractures lead to progressive deformity, which in turn increases fracture risk Systemic treatment with bisphosphonates reduces the fracture rate Figure 44.12 Radiograph of a child with osteogenesis imperfecta who has been treated with cyclical bisphosphonates. Multiple growth lines are visible in addition to intramedullary devices in both the femur and tibia.

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Revision #1
Created 2025-12-31 15:16:55 UTC by Omar Ayman
Updated 2025-12-31 15:16:55 UTC by Omar Ayman