Mobility, end-plate defects, and disc degeneration in the lower spine. By R. C. HILTON and J. BALL (*University of Manchester, Department of Rheumatology*)

Because of the paucity of data on the mobility and morbid anatomy of the lower spine, we have systematically studied the T10-S1 region in 50 post-mortem specimens covering the age range 13–96 years.

Patients with cancer, bone diseases, and disorders likely to cause the latter were excluded. The flexion-extension mobility of the fresh specimen was measured radiologically. Serial slab sections of vertebral bodies were examined radiologically, and selected blocks studied histologically, with particular reference to lesions at the disc-bone border (rim and cartilage end-plate) and disc degeneration. Spinal bone density was assessed by measuring the weight/volume ratio of a complete central slab of L3.

Mean mobility fell progressively from L5/S1 to T10/11, but this smooth pattern of movement was seen mainly in those aged <50. More often the pattern was irregular indicating that maldistribution of stress may be common in the lower spine *in vivo*. In the lumbar spine mean overall mobility was less in those aged 50+ than in the <50 age group, especially in females, and the difference was largely due to a fall in mobility at the L5/S1 level.

Defects of the cartilage end-plate (Schmorl's nodes) were found in 76% of cases with similar frequency in the <50 and 50+ age groups and some, but not significant, preponderance in males. They were characteristically localized in the posterocentral part of the end-plate and were usually found at more than one level. They were not related to bone density and were significantly more severe and more frequent in the upper (T10-L1) region than in the lower (L2-S1) region. In those aged 50+ there was a significant relation between end plate defects and disc degeneration in the upper region but not in the lower region.

Reference

SCHMORL, G., AND JUNGHANNS, H. (1971) 'The human spine in health and disease'. 2nd American edition translated by E. F. Besemann from the 5th German edition 1968. Grune and Stratton, New York and London

The musculoskeletal features of Wilson's disease: a clinical, radiological, and serological survey. By D. N. GOLDING and J. M. WALSHE (*Department of Rheumatology, Princess Alexandra Hospital, Harlow; Department of Clinical Investigations, Addenbrooke's Hospital, Cambridge*)

Twenty-six patients with established Wilson's disease (hepatolenticular degeneration) have been reviewed with regard to musculoskeletal abnormalities. This is a preliminary report of the clinical and radiological findings.

There were 12 males and 14 females, ages ranging from 7 to 36 years (median 20 years). The majority were having penicillamine therapy at the time of review. All but four had one or more locomotor abnormality considered to be significant. The following principle clinicoradiological syndromes were identified: (a) A syndrome resembling premature osteoarthritis of the knees, characterized by pain (sometimes severe), stiffness and 'gelling', and radiological evidence of loss of joint space and osteophytes (7 patients). (b) Spinal pain and stiffness, often with radiological appearances resembling active or healed vertebral osteochondritis (5 patients). (c) Episodes of acute or subacute polyarthritis (5 patients). (Some may relate to penicillamine therapy, which is known to cause occasionally an LE-like disturbance.) (d) Clinical and radiological features indistinguishable from rheumatoid arthritis (2 patients).

Other features of interest with regard to the musculoskeletal system were recurrent painful nodules about MCP joints 1, muscle cramps 8, tenosynovitis 3, painful feet 2, paraesthesiae of hands/feet 2.

Other radiological features of probable or possible significance were generalized osteoporosis 16, prominent periosteal reaction (fluffy spurs) in relation to trochanters 4, premature OA of hips 4, premature OA of wrists carpal joints 2, premature OA of spine 1, premature OA of ankles 1, 'squaring' of vertebrae 3, osteochondritis dessicans 3, multiple tongue-like osteophytic protrusions 2, and chondrocalcinosis 1.

Terminal phalangeal osteosclerosis. By W. HALIM, J. K. VAN DER KORST, H. A. VALKENBURG, and P. P. VAN ELTEREN (*Division of Rheumatology, Catholic University, Nijmegen, and Department of Epidemiology, Erasmus University, Rotterdam, The Netherlands*). Published in full in the *Annals*, 1975, **34**, 82.

Plasma zinc in rheumatoid arthritis. Its relationship to corticosteroid therapy and osteoporosis. By A. C. KENNEDY, P. LEE, W. CARSON DICK, and W. W. BUCHANAN. (*Centre for Rheumatic Diseases, the University Department of Medicine, Royal Infirmary, Glasgow*)

Plasma zinc concentrations were determined in 47 male and 68 female patients with definite or classical rheumatoid arthritis, including 17 males and 24 females who were receiving corticosteroid therapy.

The mean ± SD for the noncorticosteroid-treated groups was 85·7 ± 19 (females 93·6 ± 23·6 μg/100 ml; males 82·6 ± 15·2 μg/100 ml), which was lower than normal controls (99 ± 11 μg/100 ml). The corticosteroid-treated group had a lower mean zinc level (females 83·4 ± 11·5 μg/100 ml; males 78·9 ± 26·1 μg/100 ml), which was significantly lower than the nonsteroid-treated group (+ = 2·01; $P < 0·05$). In addition, 5 patients with confirmed vasculitic skin ulcers also had a lower mean plasma zinc level (74·8 ± 8·5 μg/100 ml) which was significantly lower than the corticosteroid-treated group (+ = 6·24; $P < 0·001$).

The plasma zinc levels were also correlated against other clinical and laboratory indices. It was found that there was a correlation coefficient between plasma zinc and an index of osteoporosis (metacarpal index) in the corticosteroid-treated group of 0·47 (+ = 2·15; $P = <0·05$) and also in the nonsteroid group of 0·45 (+ = 2·1; $P < 0·05$).

It is well recognized that collagen synthesis requires the presence of zinc, and the results of this study appear to confirm this. The results also suggest that zinc deficiency may play a role in the pathogenesis of osteoporosis in rheumatoid arthritis, especially that induced by corticosteroid therapy.

Single daily dose of allopurinol. By I. D. L. BREWIS, W. Y. LOEBL, and J. T. SCOTT (*The Mathilda & Terence Kennedy Institute of Rheumatology, Bute Gardens, Hammersmith, London, W.6*)

The most common dosage schedule for the xanthine oxidase inhibitor allopurinol is 100 mg taken 3 times a day,