bacteriologist, Institute for Medical Research, Kuala Lumpur, for the personal attention he gave to all our requests for bacteriological examinations.

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CHARCOT'S ARTHROPATHY FOLLOWING INTRA-ARTICULAR HYDROCORTISONE

BY

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[WITH SPECIAL PLATE]

In 1868 Charcot described a group of cases of arthritis associated with locomotor ataxy and hemiplegia, and stressed the absence of the cardinal signs of inflammation in the involved joints. He attributed the absence of pain to joint anaesthesia ("L'absence de douleur dans ces cas pourrait dépendre de l'anesthésie articulaire"). Eloesser (1917) reproduced the pathological features of this condition by repeated joint trauma in cats whose limbs had been rendered anaesthetic by severance of the posterior nerve roots. This work was thought to confirm the theory of pathogenesis which attributes these neuropathies to the effect of single or repeated injuries to a joint which has lost its sensibility to pain. Soto-Hall and Haldeman (1940) studied 40 patients with neuropathic joint disease. These authors showed that the essential features, in order of development, were the appearance of joint effusion, followed by ligamentous relaxation leading to instability and deformity with thinning of the articular cartilage. Marginal fractures, subchondral bony sclerosis, patchy atrophy, loose-body formation, and new bone production completed the picture of severe destructive arthritis.

The use of hydrocortisone acetate by intra-articular injection in the local treatment of arthritis has become widely accepted since its introduction in 1951 by Hollander and others. Most reports stress the subjective benefit derived by patients. Side-reactions are few, and are limited to temporary local exacerbation of disease, occasional appearance of sensitivity, the introduction of sepsis, and the development of thrombophlebitis in the injected leg. Hollander (1953) reported an incidence of only 2.3% of adverse reactions after 8,696 hydrocortisone injections. Kendall (1958) had an even lower incidence (1.1%) of untoward effects in a series of 6,700 injections. There has been no suggestion that repeated intra-articular injections of hydrocortisone might seriously aggravate arthritis, yet such aggravation might be expected as a result of the relief of pain experienced from such treatment. Interference with the normal protective processes might well encourage undue weight-bearing and mobility, thereby accelerating the progress of joint destruction. This has, in fact, been our experience in 10 of 18 patients with rheumatoid arthritis given prolonged treatment during a controlled trial of intra-articular steroid therapy (Chandler and Wright, 1958).

The following report describes the rapid deterioration of an osteoarthritic hip following repeated hydrocortisone injections.

Case Report

A doctor's wife aged 66 was first seen in the Rheumatism Clinic of the General Infirmary at Leeds in May, 1956. She had had pain in the right hip with difficulty in walking since 1937. Apart from a course of physiotherapy given with temporary benefit in 1951, she had received no treatment. Radiography of the right hip at this time showed slight osteoarthritic change (Special Plate, Fig. 1).

Examination disclosed a thin, intelligent woman in obvious pain. Chest, heart, and abdomen were normal, and there was no neurological defect. The only abnormality was painful restriction of movement at the right hip-joint. There was no shortening of the limb. A radiograph of the right hip disclosed some progression of disease as compared with the earlier film. The differential agglutination test was negative and the erythrocyte sedimentation rate (E.S.R.) normal.

Treatment was begun with phenylbutazone, 200 mg. twice daily, and 50 mg. (2 ml.) of hydrocortisone acetate injected into the right hip-joint at approximately monthly intervals. After each injection she experienced great relief from pain, which lasted some three weeks on each occasion. Fig. 2 on the Special Plate shows the radiographic appearance six months after treatment was started. Injections were continued for a further 12 months, when 900 mg. (36 ml.) of hydrocortisone had been given. Re-examination then showed an extremely mobile, painless joint. There was 2 in. (5 cm.) of true shortening of the right leg. The rest of the clinical examination was negative. A radiograph of the right hip showed gross destruction of the femoral head and the roof of the acetabulum (Special Plate, Fig. 3). Blood Wassermann reaction was negative and E.S.R. normal.

Biopsy of the right hip (Mr. A. B. Pain) showed no unusual features in the portions of tendon and joint capsule examined (Dr. D. Harriman) beyond some non-specific connective-tissue proliferation and occasional small foci of foreign-body reaction. Muscular atrophy was advanced. Biopsy of bone examined by Professor C. E. Lumsden showed features of resorption (Special Plate, Fig. 4) and new bone formation (Special Plate, Fig. 5). One section of biopsy was sent to Professor D. H. Collins, who reported: "There are really no diagnostic features in this biopsy. Most of the bone in the section is of compact or cortical pattern and appears normal, but at its junction with the periosteal tissues there are certain progressive changes of resorption and regeneration such as one may encounter in any slow remodelling process. Rather large, thin-walled vascular spaces and some islets of new metaplastic bone are seen in part of the outlying connective tissues. There is no evidence of tuberculosis or other infection. The changes that we see may be encountered around an osteoarthritic joint or, indeed, a Charcot's joint, but there is nothing to

say which. The inflammatory reaction in the tissues is of a very low order such as may accompany any reconstruction of bone."

Culture from the right hip and guinea-pig inoculation showed no evidence of tuberculosis.

Discussion

This patient has been the subject of wide interest, and a number of opinions have been obtained in respect of the aetiology of her arthritis. All observers agree that osteoarthritis was the primary joint disease. The slow progression from onset in 1937 to the inception of treatment in 1956 contrasted with the florid advance of joint destruction culminating in complete disorganization during the period of therapy with intra-articular injections of hydrocortisone acetate. The final picture, both clinical and radiographic, was indistinguishable from the condition described by Charcot. There was no evidence of neurological disease. Rheumatoid arthritis, tuberculosis, and other infective processes were excluded by serological tests and the results of biopsy.

The striking similarity of this picture to that of neuropathic arthritis suggests that temporary suppression of pain of local origin by frequent intra-articular injection encourages a damaging degree of movement and weight-bearing. This in turn permits the development and rapid progression of joint destruction. The deterioration observed in this patient during treatment with locally injected hydrocortisone suggests that the procedure is potentially dangerous. This is particularly so since it has been our experience that symptomatic benefit from injections may lead to a vicious circle of dependence despite worsening disease. Careful radiological supervision is desirable if prolonged treatment by intra-articular injections of hydrocortisone and related compounds is undertaken.

Summary

The development of a severe destructive and relatively painless arthritis (Charcot joint) is reported during prolonged treatment of an osteoarthritic hip with intra-articular injections of hydrocortisone acetate. The need for careful radiological supervision during such treatment is emphasized.

We are indebted to Professor C. E. Lumsden for his helpful criticism.

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Remotivating the Mental Patient, by Otto von Mering and Stanley H. King (Russell Sage Foundation, New York, 1957; 216 pp., $3), is based on a survey of in-patient care in 30 mental institutions in the United States. In spite of crowded conditions and shortage of staff it was found that courageous and promising programmes of rehabilitation had been developed in some places. The authors consider that such experiments as the elimination of restraint, the encouragement of the patient's interest in his personal appearance, transformation of his environment into a more normal atmosphere, and the promotion of social and recreational activities, among other things, are justified by the results they produce. Guidance is given in carrying out these and other measures of rehabilitation.

SYMPTOMLESS MYELOMATOSIS

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[WITH SPECIAL PLATE]

Myeloma commonly presents with pain in the bones. Out of 97 patients with myeloma examined by Snapper *et al.* (1953), 92 suffered from bone pain. Other common presenting features are pathological fractures, vertebral collapse with associated neurological complications, renal impairment, anaemia, and recurrent pulmonary infections.

The patient whose history is here recorded has been under continuous observation for 5½ years. For four years she was free from any symptoms or signs, except that she had a persistently raised erythrocyte sedimentation rate (120–130 mm. in one hour by the Westergren technique), a moderate orthochromic anaemia (Hb 10.7 g. per 100 ml.), and a very high serum globulin (7–8 g. per 100 ml.). Sternal puncture showed some increase in plasma cells, but no abnormal forms.

Case History

The patient, a housewife aged 43, had been in good health until April, 1952, when she developed a small right pleural effusion following an attack of "bronchitis," and in May she was admitted to the Pleural Effusion Unit of Queen Mary's Hospital, Sidcup. The effusion resolved spontaneously, and she was discharged in August, 1952, when her chest was radiologically normal apart from pleural thickening. It was assumed that she had a primary tuberculous effusion, although no tubercle bacilli were isolated, but the Mantoux reaction was positive. The only unusual feature was that her erythrocyte sedimentation rate (E.S.R.) was persistently raised and remained in the range 117–131 mm. in one hour (Westergren). Estimation of her serum proteins showed a total protein value of 11.2 g. per 100 ml. (albumin 3.65 g., globulin 7.55 g.). The urine was normal and contained no Bence Jones protein.

Electrophoretic analysis: pH 8.0; μ=0.2; protein concentration 2 g. per 100 ml.

Ultracentrifugal analysis: protein concentration 1 g. per 100 ml.

FIG. A.—Electrophoretic and ultracentrifugal analysis, December 14, 1952.

On return home she attended the Croydon Chest Clinic for surveillance, and at the end of December, 1952, she came under our observation on account of her very high E.S.R. She stated that she felt well and had no complaints. She looked fit and no abnormal signs were found, apart from slight pallor. Blood count was normal except for a mild orthochromic anaemia (Hb 10.7 g. per 100 ml.). E.S.R. 125 mm. in one hour. Sternal marrow from a puncture in February, 1953, appeared to be normal, but a differential

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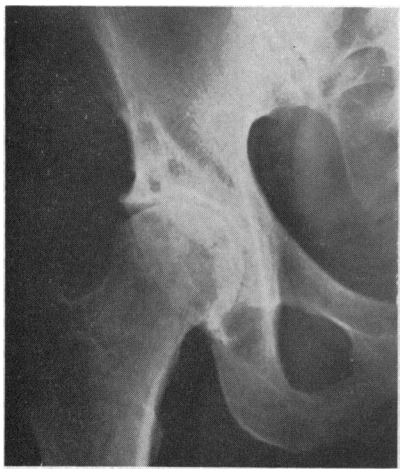

FIG. 1.—Radiograph of right hip in 1951. Note moderate osteoarthritic changes.

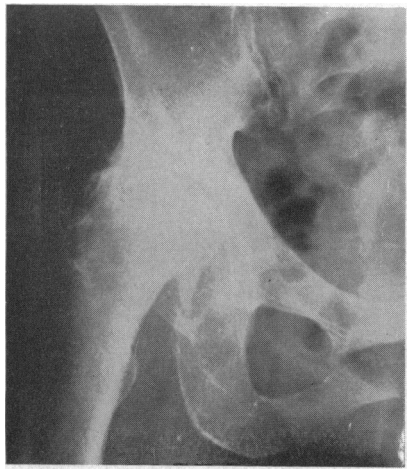

FIG. 2.—Radiograph of right hip after six months' treatment. Note marked osteoarthritic change with loss of joint space, sclerosis, and flattening of femoral head.

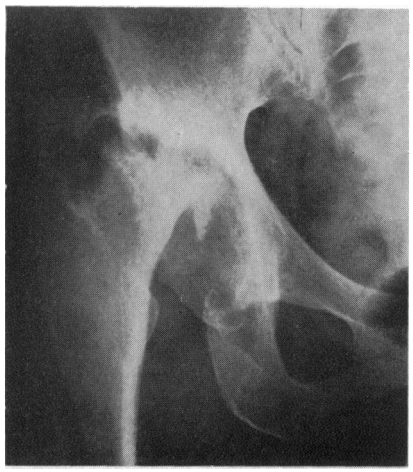

FIG. 3.—Radiograph of right hip after 18 months' treatment. Note gross destruction of femoral head and roof of acetabulum.

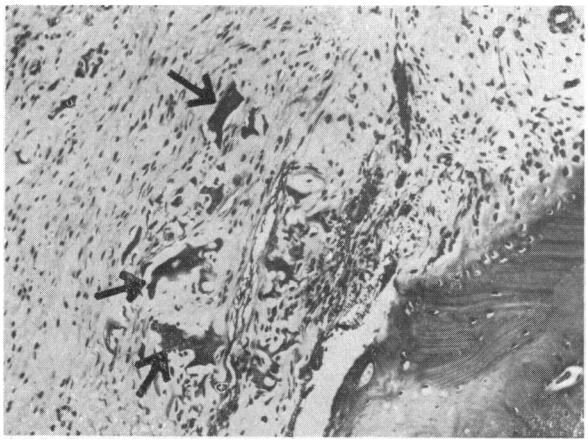

FIG. 4.—Field showing focus of bone resorption, with angulated fragments of necrotic bone indicated by arrows. There is some perivascular cellular reaction. (H. and E. ×80.)

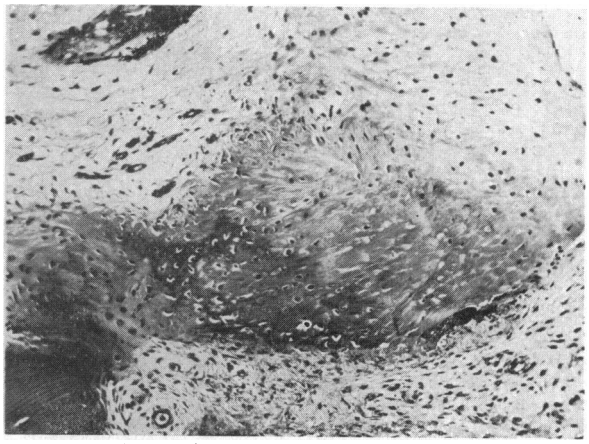

FIG. 5.—Field showing focus of new metaplastic bone formation spreading out from old bone seen (striated) in top left-hand corner. (H. and E. ×80.)

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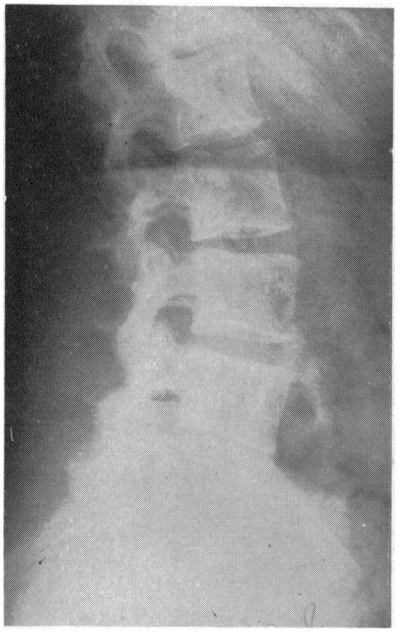

FIG. 1.—Radiograph of lumbar spine, February 9, 1953.

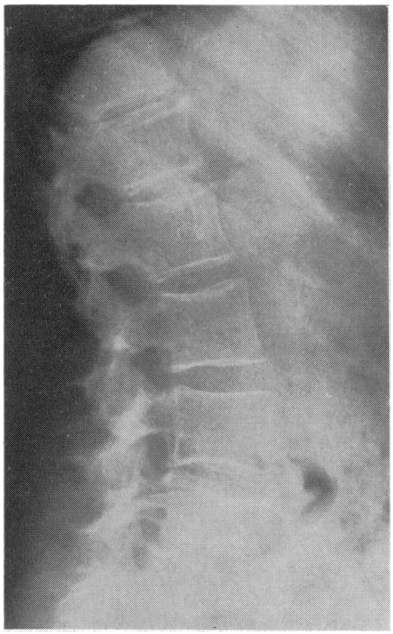

FIG. 2.—Radiograph of lumbar spine, February 7, 1957.

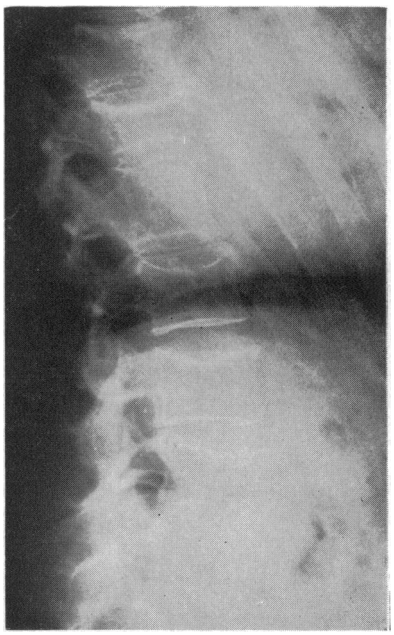

FIG. 3.—Radiograph of lumbar spine, August 10, 1957.