picture in a case of childhood dermatomyositis. A five-year-old girl was first hospitalised in 1971 with muscular pain and weakness accompanied by mild malar erythema and an erythematous/violaceous maculopapular eruption over the extensor surfaces of the interphalangeal joints, elbows, and knees; the electromyogram (EMG) was consistent with myositis. Without treatment the patient presented spontaneous remission during the following months.

In 1975, at 9 years of age, similar symptoms affecting the skin and proximal muscle groups recurred, and a diagnosis of dermatomyositis was confirmed by increase of muscle enzymes (creatine phosphokinase 410 U/l) and typical EMG findings. Again the symptomatology disappeared without treatment.

The child led a normal life and played sports. In January 1984 the skin lesions recurred accompanied by periorbital lilac discoloration, cutaneous arteritic lesions on the medial aspect of the thighs, mild proximal muscular weakness, and moderate increase of muscle enzymes. The symptomatology resolved rapidly with a short period of low dose steroids. Generally, childhood dermatomyositis follows a uniphasic course. The activity of the disease may last from only a few months to two or three years followed by recovery;² during this period recurrence of the symptomatology is generally the consequence of premature discontinuation of treatment. On the contrary in our patient, as in the adult reported by Foley and Payne,¹ the clinical course of the disease was polyphasic with recurrences separated by long periods of spontaneous remissions.

Department of Pediatrics
University of Pavia,
Policlinico S. Matteo,
27100 Pavia, Italy

ALBERTO MARTINI
ANGELO RAVELLI

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The lupus anticoagulant, pulmonary thromboembolism, and fatal pulmonary hypertension

SIR, The article by Anderson and Ali¹ is an important contribution which may provide insights into the development of pulmonary hypertension in patients with systemic lupus erythematosus (SLE). While decreased synthesis/release of prostacyclin² or diminution of its activity³ may be responsible for the paradoxical association of the lupus anticoagulant with thrombotic tendencies or states, other mechanisms may be significant. Relative deficiencies of protein C or antithrombin III (ATIII) have been associated with development of a thrombotic diathesis.^{4,5} Depression of ATIII levels has been noted in SLE, correlating with disease activity.⁶ Perhaps central to the

question is the cogent observation by Anderson and Ali¹ that warfarin therapy was ineffective in controlling the thrombotic diathesis in their patient, in spite of maintenance of the prothrombin ratio in the therapeutic range. Oral anticoagulants have been generally noted to augment levels of both protein C⁷ and ATIII.⁸ One exception has been noted. A subgroup of individuals was identified, by Penner and Hunter,⁹ in whom recurrent thrombotic phenomena were noted, even when the prothrombin time was prolonged to the normally 'acceptable' range. That group was categorised by failure of the anticoagulant to produce the expected increase in antithrombin III concentration. As ATIII deficiency-associated phenomena are responsive to ATIII transfusion,¹⁰ identification of deficiency states would appear to be of more than academic interest. It would be of interest to study plasma⁸ from such patients as described by Anderson and Ali further, to determine if such potentially treatable phenomena were present.

Rheumatology Division,
Menorah Medical Center, and
Kansas University School of Medicine,
USA.

B M ROTHSCHILD

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Bufexamac crystals in synovial fluid analysis

SIR, Bufexamac, a non-steroidal drug for intra-articular injection, was recently introduced. Its pharmacology and efficacy have been studied by several authors.¹⁻⁴ On

microscopic examination some batches appear as squares or rectangles with rounded angles, but some appear as sticks with sharp or square ends measuring between 3 and 12 µm. On compensated polarised microscopy they are negatively birefringent and must not be confused with monosodium urate crystals.

Our purpose was to determine what chances there are of encountering such crystals in synovial fluid after an intra-articular injection. We also compared the result of a buprenorphine injection with the result of a corticosteroid intra-articular injection.

We selected 94 patients (59 females and 35 males) with chronic exudative gonarthrosis, who had a normal blood analysis and no crystals whatsoever in their synovial fluid.

Forty patients received an intra-articular (IA) injection of 7 mg betamethasone acetate, 36 received an IA injection of 20 mg triamcinolone hexacetonide, and 18 patients received 20 mg buprenorphine. All were asked to return after two days. If at that time there was at least 5 ml synovial fluid effusion, a new synovial fluid analysis was performed; if not, we asked the patients to return after five additional days for this analysis.

From the 40 patients who had received IA betamethasone acetate 35 came back for follow up: 14 synovial analyses were performed after two days and in three of them we found betamethasone crystals. Twenty one patients were seen after seven days, and no crystals were found in their synovial fluid.

From the 36 patients who had received IA triamcinolone hexacetonide 31 returned: 11 fluids were aspirated after two days and triamcinolone crystals were found in two. In

the synovial fluid of the 20 patients who were seen after one week no crystals were found.

In the last group 17 of 18 patients who had received buprenorphine were seen in follow up: nine fluids were aspirated after two days and buprenorphine crystals were found in two; eight patients were seen after seven days and no crystals were found.

All crystals were extracellular. We conclude that if a synovial fluid examination is performed two days after an intra-articular injection, there is about one chance in five of encountering crystals if a crystal suspension drug had been previously injected. After seven days there is not one chance in 50 of finding such crystals.

Centre Médico-Social de Bruxelles,
69 rue des Chartreux,
B-1000 Brussels, Belgium

C H R MOENS

D MOENS

P H MOENS

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Book review

Textbook of Orthopaedic Medicine. Vol. 2. Treatment by Manipulation, Massage and Injection. By J Cyriax and M Coldham. Pp. 266. £14.50. Baillière Tindall: London. 1984.

For those familiar with previous editions of this book of practical procedures, there is little new. If you are new to it, and feeling a bit therapeutically nihilistic, try it for a tonic! The dogmatic, optimistic style encourages you to do something positive. One quarter of the procedures are injections: this aspect can be compared with Allan Dixon

and Graber's *Eular Monograph Local Injection Therapy in Rheumatic Diseases*. Cyriax gives better detail on shoulder and back injections, while Dixon covers finger and toe joint injections well because of more emphasis on rheumatoid arthritis. In both, exact details of positioning and injection technique are given. Dixon's line drawings and x-rays are an added help; in this book, even a recent photograph of Mrs Cyriax is reproduced somewhat indistinctly by the publisher. With controlled trials now showing efficacy of manipulation, the half of the book exactly describing spinal and peripheral joint manipulation is a useful reference for those who have been physically taught the rudiments of the art.

R M ELLIS

Note

Volvo awards for low back pain research

To encourage research in low back pain the Volvo Company of Göteborg, Sweden, has sponsored three prizes of US \$6000 each. Awards will be made competitively on the basis of scientific merit in the following three areas: (1) clinical studies; (2) bioengineering studies; (3) studies in other basic science areas. Papers submitted for the contest must contain original material, not previously

published or submitted for publication. Multiple authorship is acceptable. The manuscripts should be in the form of a complete report, not exceeding 30 typewritten pages, double-spaced, and in a form suitable for submission to a scientific journal. Five copies of each paper submitted in full should reach the address given below not later than 21 January 1986. One of the authors should be prepared, at his own expense, to come to Dallas, Texas, USA, at the time of the meeting of the International Society for the Study of the Lumbar Spine, 4-8 June 1986, to present the paper and to receive the award. Further details from Professor Alf L. Nachemson, Department of Orthopaedic Surgery I, Sahlgren Hospital, S-413 45 Göteborg, Sweden.