increase of the creatine phosphokinase (CPK) indicated myocarditis: CPK 2865 IU/l, CPKMB 288 IU/l. The CSF contained 24 white blood cells/mm³ ($24 \times 10^6/l$) (94% lymphocytes, 6% monocytes) with an increased level of protein: 1.50 g/l. A spirochaete began to grow in BSK II⁴ after the second week and a borrelia was identified. It had a diameter of 0.2 μ m and a length between 4 and 22 μ m. By immunofluorescent assay high titres of IgG antibodies against *Borrelia burgdorferi* were detected in serum collected three weeks after the beginning of the illness (1/160), and two months later (1/320). The patient's phenotype was A2, B5, DR5. He received oral penicillin therapy (10 million U/day) for 15 days, and recovered.

For the first time in France a borrelia has been isolated from the CSF during Lyme disease. This isolation strengthens the role of borrelia as the aetiological agent responsible for the clinical signs in the acute phase of the disease. The late clinical manifestations could be secondary to an immune reaction triggered by the spirochaete, especially among patients who have the B cell alloantigen DR2. In France the carrier is unknown, but *Ixodes ricinus* occurs worldwide and its involvement is suspected.

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References

- 1 Steere A C, Malawista S E. A cluster of arthritis in children and adults in Lyme, Connecticut. *Arthritis Rheum* 1976; **19**: 824.
- 2 Steere A C, Robert L, Grodzicki R L, *et al*. The spirochetal etiology of Lyme disease. *N Engl J Med* 1983; **308**: 733-40.
- 3 Preac-Mursic V, Wilske B, Schierz G, Pfister H W, Einhaupl K. Repeated isolation of spirochetes from the cerebrospinal fluid of a patient with meningoradiculitis Bannwarth. *Eur J Clin Microbiol* 1984; **3**: 564-5.
- 4 Barbour A. Isolation of a cultivatable spirochete from Ixodes ricinus ticks of Switzerland. *Curr Microbiol* 1983; **8**: 123-6.

Tendon rupture in systemic lupus erythematosus

SIR, I read with interest the letter by Hanly and Urowitz¹ on the spontaneous rupture of the long head of biceps tendon in a 35 year old woman with SLE on long term corticosteroid therapy and who had suffered a fracture of the neck of the left humerus some seven months previously. It is perhaps straining our credibility too much to ask us to accept this as spontaneous rupture of the tendon when presumably the cause of the fracture (we are not told) was traumatic in nature. Rupture of the long head of biceps can

remain undetected and is not apparent until the patient flexes the elbow or supinates the forearm against resistance, manoeuvres which would be impossible to carry out at the time of the fracture. I would surmise that the rupture of the tendon may have occurred at the time of the fracture and remained undetected particularly if at follow up she was not always seen by the same doctor, or that the weakened tendon snapped in the convalescent phase when she was performing shoulder and arm exercises. In either case the tendon would not have ruptured had it not been for the fracture and so can hardly be called spontaneous.

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Reference

- 1 Hanly J G, Urowitz M B. Tendon rupture in systemic lupus erythematosus. *Ann Rheum Dis* 1986; **45**: 349.

SIR, Although it is impossible to disprove Dr Richards' comments, we feel that they are unlikely. Our patient sustained a minimally displaced fracture which required supportive strapping only. She was seen in follow up on four occasions by the orthopaedic and rheumatology services before presentation with tendon rupture. This review was in a research clinic setting where musculoskeletal problems are specifically sought. The patient insisted that muscle swelling did not occur until several months after the fracture. We feel, therefore, that spontaneous tendon rupture is the most likely diagnosis in this case.

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Corticosteroids in rheumatoid arthritis

SIR, I read with interest the recent paper of Byron and Kirwan on the feasibility of conducting a study to determine whether low dose corticosteroids modify the course of rheumatoid arthritis.¹ The authors solicited the views of your readers regarding the acceptability of long term use of prednisone (7.5 mg) in relation to potential side effects.

An increasing body of evidence suggests that prolonged corticosteroid therapy accelerates the development of atherosclerosis.² Even low doses of corticosteroids may result in significant side effects such as hypertension, hyperlipidaemia, and impaired glucose tolerance.^{3,4} Each of these adverse effects is a recognised coronary risk factor.

Virtually every study of survival among patients with rheumatoid arthritis indicates that mortality is increased,^{5,6} and some studies have found that cardiovascular disease is a major contributor to the decreased survival.^{7,8} Since there has not been adequate attention paid to the possible link between corticosteroids and atherosclerosis, most mortality studies in rheumatoid arthritis provide insufficient data on corticosteroid use, or the subject is ignored entirely. In a mortality study by Prior *et al* at least half of the patients had taken corticosteroids for treatment of their disease, but information on dosage and duration of therapy was incomplete.⁷

There have been no comprehensive pathological studies evaluating the long term effects of corticosteroids on the cardiovascular system. Nevertheless, since corticosteroids may induce or exacerbate known coronary risk factors, the prudent course is to avoid prolonged corticosteroid use whenever possible. Any benefit corticosteroids might offer in modifying the course of rheumatoid arthritis may be far outweighed by the potential for accelerating atherosclerotic disease, especially when therapy is initiated in younger individuals.

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References

- 1 Byron M A, Kirwan J R. Corticosteroids in rheumatoid arthritis: Is a trial of their 'disease modifying' potential feasible? *Ann Rheum Dis* 1986; **45**: 171-3.
- 2 Nashel D J. Is atherosclerosis a complication of long-term corticosteroid treatment? *Am J Med* 1986; **80**: 925-9.
- 3 El-Shaboury A H, Hayes T M. Hyperlipidaemia in asthmatic patients receiving long-term steroid therapy. *Br Med J* 1973; **ii**: 85-6.
- 4 Thomas T P L. The complications of systemic corticosteroid therapy in the elderly. *Gerontology* 1984; **30**: 60-5.
- 5 Uddin J, Kraus A S, Kelly H G. Survivorship and death in rheumatoid arthritis. *Arthritis Rheum* 1970; **13**: 125-30.
- 6 Allebeck P. Increased mortality in rheumatoid arthritis. *Scand J Rheumatol* 1982; **11**: 81-6.
- 7 Prior P, Symmons D P M, Scott D L, Brown R, Hawkins C F. Cause of death in rheumatoid arthritis. *Br J Rheumatol* 1984; **23**: 92-9.
- 8 Abruzzo J L. Rheumatoid arthritis and mortality. *Arthritis Rheum* 1982; **25**: 1020-3.

years ago.² Extension or rupture of the suprapatellar pouch into the thigh is rare compared with the relatively common 'pseudo-thrombophlebitis syndrome' caused by the rupture of Baker's cyst into the calf.³ This fact is confirmed by the scarcity of reports in the literature.¹⁻⁷ The fact that nearly all the patients were suffering from rheumatoid arthritis does not seem to be accidental.^{1-3 5-7} In rheumatoid arthritis the extent of fluid overproduction and the presence of a hypertrophied synovium or a fibrotic joint capsule limiting the joint capacity lead to a rapid and important rise in intra-articular pressure.^{8,9}

Studies in cadaveric knees including rheumatoid arthritis showed the suprapatellar pouch to be the place most vulnerable to rupture.⁸ It is possible that in some cases the rupture is potentiated by the presence of clinically undetected antefemoral cysts originated at the apicomedial side of the suprapatellar bursae.⁷ During life the contraction of the quadriceps and especially of the vastus medialis protects the suprapatellar pouch during high intra-articular pressures and prevents its rupture, the pressure of the fluid being directed backwards, often to an existing Baker's cyst. In rheumatoid arthritis the sometimes severe quadriceps wasting and atrophy leave the suprapatellar pouch unprotected, and its rupture or extension into the thigh tissue can occur. We suppose that in the case presented in your journal¹ the previous rupture of a Baker's cyst in the same knee, probably followed by fibrosis and obstruction of the communication between the cyst and the knee joint, caused the accumulation of high pressure fluid in the suprapatellar pouch and its extension into the thigh.

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References

- 1 Coulton B L, Popert A J. Massive extension of the suprapatellar pouch into the thigh tissues in rheumatoid disease. *Ann Rheum Dis* 1986; **45**: 174-5.
- 2 Nahir M, Scharf Y, Kleinhaus U, Besser M, Rosenbaum M. Suprapatellar pouch rupture—a rare complication of rheumatoid arthritis. *Rheumatol Rehabil* 1980; **19**: 161-2.
- 3 Katz R S, Zizic T M, Arnold W P, Stevens M B. The pseudo thrombophlebitis syndrome. *Medicine (Baltimore)* 1977; **56**: 151-64.
- 4 Raman D, Dawes P T, Haslock J. Acute knee joint rupture—a report of two unusual cases. *Br J Rheumatol* 1983; **22**: 99-101.
- 5 Duncan A M. Arthrography in rupture of the suprapatellar bursa with pseudocyst formation. *Am J Roentgenol* 1974; **121**: 89-93.
- 6 Pastershank S P, Mitchell D M. Knee joint bursal abnormalities in rheumatoid arthritis. *J Can Assoc Radiol* 1977; **28**: 199-203.
- 7 Seidl G, Scherak O, Hofner W. Antefemoral dissecting cysts in rheumatoid arthritis. *Radiology* 1979; **133**: 343-7.
- 8 Dixon A S, Grant C. Acute synovial rupture in rheumatoid arthritis. Clinical and experimental observations. *Lancet* 1964; **i**: 742-5.
- 9 Jayson M I, Dixon A S. Intra-articular pressure in rheumatoid arthritis of the knee. III. Pressure changes during joint use. *Ann Rheum Dis* 1970; **29**: 401-8.

Suprapatellar pouch rupture or extension into the thigh tissue in rheumatoid disease

SIR, We read with great interest the letter on the massive extension of the suprapatellar pouch into the thigh tissues in rheumatoid disease.¹ We described a similar case several