Correspondence

Pneumococcal septic arthritis

SIR, I was interested in the paper on pneumococcal septic arthritis by Morley *et al.*¹ Although it is worthwhile being reminded of this association, it is not a new observation. Of 22 cases of rheumatoid disease with joint infection seen in Coventry hospitals in 1968-75, two were due to *Streptococcus pneumoniae* and both had had recent chest infections.²

Morley *et al* state that *S pneumoniae* is a rare cause of joint infection, but Goldenberg and Cohen found five of their 50 cases of acute infectious arthritis were due to the pneumococcus, and their analysis of selected major reviews of septic arthritis found 14 of 177 cases to be due to this organism.³ The series from Coventry showed four out of all 75 cases (rheumatoid and non-rheumatoid) to be caused by *S pneumoniae*.

Morley *et al* rightly highlight the difficulty in diagnosis, in that the infected joint may not be obviously acutely inflamed; other clues to acute infection are commonly absent, too. They feel that a recently raised erythrocyte sedimentation rate (ESR) to above 100 mm/h may be the main clue to diagnosis. Previous experience suggests this is an optimistic view; in Cooper and Cawley's series only 13 of 74 had an ESR of greater than 100 mm/h.⁴

In the Coventry series it was stated 'Laboratory investigations were unhelpful',² and the ESR was not considered further. Review of that series, however, shows the mean ESR in those with pre-existing rheumatoid disease went from 57 mm/h (range 5-110) before infection to 95 mm/h (range 29-150) during infection. Unfortunately these useful looking figures obscure the fact that the high ESR results were obtained on the whole in those with more obvious infection, while those with less obvious infection had lower ESRs. The Coventry paper also stated 'Suspicion should be aroused by a patient going off colour or developing extra pain or swelling of one or two joints only . . . suspicion should lead to aspiration'.² This view remains true and no other investigations should be relied upon to help.

Morley and coauthors discuss the need for reducing morbidity and mortality and quote Cooper and Cawley as avoiding the 35% chronic morbidity when the infection was recognised and treated 'early'. This conclusion does not appear in Cooper and Cawley's paper, though they do state that the mean delay to diagnosis and treatment was three times longer (21.5 days) for those with complications than in those without complication (7.5 days).⁴ In rheumatoid disease the situation appears to be somewhat different as the Coventry series found the mean time to appropriate treatment was very nearly the same for the three outcome groups of death (9.3 days), permanent joint damage (9.5 days), and unaltered joint (9.3 days).² Maybe very early diagnosis and treatment would make a substantial difference to outcome, but this early diagnosis would require a major change in levels of suspicion of infection leading to more ready aspiration of joints.

Finally, could Morley and coauthors define their term elderly? They describe their three cases as elderly, yet one was aged 48 years. Perhaps this is a reflection of the paediatric interest of their unit, but it is a little perturbing to find that middle age no longer exists.

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- 3 Goldenberg D L, Cohen A S. Acute infectious arthritis. *Am J Med* 1976; 60: 369-77.
- 4 Cooper C, Cawley M I D. Bacterial arthritis in an English health district: a 10 year review. *Ann Rheum Dis* 1986; 45: 458-63.

SIR, We read Dr Morris's letter with interest. The omission of the study from Coventry¹ is regretted, but we now acknowledge the contribution of this group and were particularly interested in the new data on the erythrocyte sedimentation rate (ESR). With respect to the diagnosis of sepsis in a joint we would entirely concur that if there is suspicion that a joint is infected then aspiration should be performed. In discussing the ESR,² we were stating the fact that our patients' ESRs has risen above previous values and that they were all above 100 mm/h. We feel an unexplained rise in ESR should alert the clinician to the possibility of sepsis. In Cooper and Cawley's paper most patients were stated to have a raised ESR, 60% had a value greater than 50 mm/h and 18% greater than 100 mm/h.³

It is well recognised that the spectrum of organisms in septic arthritis in the United States is different from that in the United Kingdom. We therefore compared our patients with accepted studies in this country. In the paper and review cited by Morris⁴ a major cause of septic arthritis, namely the gonococcus, was specifically excluded so that the true figures of the incidence of *Streptococcus pneumoniae* would be lower. In Morris and Eade's paper the Coventry experience¹ was also compared as similar to one from Oxford,⁵ where there were no cases of pneumococcal septic arthritis in a series of 134 patients.

Our interpretation of the Southampton paper³ that morbidity could be avoided by early recognition and treatment of infection we feel was a fair assessment of the paper and would support this with the following quotations 'Our results suggest that early aspiration and administration of systemic antibiotics influence outcome favourably' and 'It seems likely that this morbidity (viz 35%) could be reduced by earlier diagnosis and treatment'. This concurs with an accepted view over many years.⁶

Finally, we might have been wiser to use the term infirm

rather than elderly. Our lady of 48 years had a physical state considerably more advanced than her years and not typical of others of her age. Although our unit has a paediatric interest, we continue to have a practice involving all ages, and two of the authors consider themselves 'middle aged'.

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Treatment of Raynaud's phenomenon with large doses of triiodothyronine: a pilot study

SIR, The use of triiodothyronine (T3) in doses sufficient to induce a hyperthyroid state has been advocated as a novel

form of treatment for Raynaud's phenomenon.¹ An extensive search of the English language literature failed to uncover a single descriptive account of the use of such therapy, however. This apparent lack of recorded data prompted us to report our experience with T3 in the treatment of Raynaud's phenomenon accompanying various connective tissue diseases.

The study group comprised nine subjects, this being the total number of patients with Raynaud's phenomenon under our care when the trial was begun and who had no clinical evidence of cardiovascular disease and were not taking vasoactive drugs. A summary of the relevant personal characteristics and clinical features of these patients is presented in Table 1. All were non-smokers and none consumed alcohol.

The trial was open and uncontrolled. At the time of enrolment, and after giving informed consent, each patient was asked the average number of attacks they were currently experiencing each week and the average duration of the attacks. For duration, figures of 5, 10, 15, 20, or more than 20 minutes were offered as examples. Measurements of (sitting) blood pressure and pulse rate were also recorded. Triiodothyronine was prescribed at a dosage of 80 µg/day (in accordance with recommendations¹), and each patient was reassessed at intervals of four weeks for the next 12 weeks. At every visit the patients were questioned about the development of side effects of therapy; specifically, the occurrence of nervousness, palpitations, or heat intolerance. Also, the blood pressure and pulse rate were measured. Compliance was assessed by regular estimations of a battery of thyroid function tests.

All the patients experienced remission of their Raynaud's symptoms (Table 1). During the trial period the mean minimum daily temperature fell from 12.3 to 4.6°C (Pretoria Weather Bureau). One patient (No 3) experienced heat intolerance and palpitations during the seventh week of therapy, which resolved when the dosage was decreased to 40 µg/day. A further patient (No 4) reported episodic palpitations at the first follow up visit; these remitted after a dosage reduction to 60 µg/day. Neither patient experienced a relapse while taking the lower

Table 1 Patient characteristics, clinical features, and responses to therapy

Patient No	Age (years)	Sex	Race	Time since onset of symptoms (years)	Before treatment		After 12 weeks' treatment		Underlying disease
					Number of attacks (per week)	Average duration of attack (min)	Number of attacks (per week)	Average duration of attack (min)	
1	41	F	B	4	4	10	0	—	SLE
2	44	F	B	3	21	20	7*	5	SS
3	35	F	B	4	7	20	0	—	SLE
4	37	F	B	2	4	15	0	—	OS
5	42	F	B	5	3	10	0	—	SS
6	21	F	B	1	2	15	0	—	SLE
7	41	F	W	1	12	20	0	—	SS
8	43	F	B	5	3	15	0	—	RA
9	41	F	B	5	2	15	0	—	SLE

B=black; W=white; SLE=systemic lupus erythematosus; SS=systemic sclerosis; OS=overlap syndrome; RA=rheumatoid arthritis.
*No longer painful.