An ECG confirmed the presence of sinus bradycardia in all three patients; the deterioration in their general health consisted of withdrawal, listlessness, refusal of food, and general apathy. The pulse reverted to normal and the symptoms disappeared within two days of ending the drug.

Discussion

Hydergine contains 1·5 mg of mesylates of dihydrogenated alkaloids of dihydroergokryptine. The substance is thought to have a profound alpha-blocking action. It has also been suggested that it may have beta-adrenergic blocking effects.[4] The bradycardiac effect of Hydergine is mentioned in earlier European literature particularly in relation to the use of Hydergine in peripheral vascular disease, but we have been unable to find a mention in the more recent literature in this country referring to its use in cerebrovascular insufficiency. Further research into the action of Hydergine on the heart rate would be of interest. We feel the development of this complication in three out of eight patients treated with Hydergine associated with severe systemic symptoms is of sufficient importance to report these cases.

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[1] Rehman, S A, *Current Medical Research and Opinion*, 1973, **1**, 8.
[2] McConnachie, R W, *Current Medical Research and Opinion*, 1973, **1**, 8.
[3] Gedye, J C, in *Aspects of Educational Technology 1*; *the Proceedings of the Programmed Learning Conference*, held at Loughborough, 15-18 April 1966, ed D Unwin and J Needham, p 369. London, Methuen, 1967.
[4] *Pharmacological Basis of Therapeutics*, ed L S Goodman and A Gilman, 4th edn. New York, Macmillan, 1970.

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SLE precipitated by antibodies in a patient with Sjøgren's syndrome and rheumatoid arthritis

Typical rheumatoid arthritis and systemic lupus erythematosus (SLE) are often considered as separate diseases with different prognoses and treatments. Sjøgren's syndrome has been reported in association with both diseases.[1] It has been suggested, however, that SLE and rheumatoid arthritis may well represent opposite ends of a spectrum of connective tissue diseases and that overlap cases combining features of both diseases may occur.[2] This latter concept is illustrated by the patient reported here, in whom a disease with the typical, clinical, and serological features of SLE was precipitated by antibiotics on two occasions but who had otherwise typical rheumatoid arthritis with Sjøgren's syndrome in the intervening period.

Case History

Mrs AB, aged 42 in April 1973, developed polyarthritis, fever and a chest infection two weeks after treatment for a sore throat with a 12 day course of co-trimoxazole. She had a history of respiratory tuberculosis treated with chemotherapy in 1961, while her mother had had Sjøgren's syndrome and rheumatoid arthritis. After further treatment elsewhere with co-trimoxazole and rifampicin, ethambutol, and isoniazid she became ill with worsening of her polyarthritis and pericarditis. LE cells were seen in the blood film; the titre of serum antinuclear factor was 1/1000, and that of rheumatoid factor 1/256, while DNA binding 28% (borderline abnormal). SLE was diagnosed, all therapy stopped, and high dose prednisolone started. She improved clinically and steroids were tailed off. During follow-up over the next year at the Centre for Rheumatic Diseases she developed symptomatic Sjøgren's syndrome with typical ocular and oral manifestations. She continued to have intermittent peripheral joint pain and stiffness and developed synovial hypertrophy of the metacarpophalangeal and proximal interphalangeal joints of the hands and *x*-ray changes of periarticular osteoporosis and juxta-articular erosions.

She was managed with non-steroidal anti-inflammatory drugs and by December 1974 her serum antinuclear factor had become negative, DNA binding 0%, and the titre of rheumatoid factor 1/1025. She remained generally well otherwise until April 1975, when she again was treated with co-trimoxazole at home for a sore throat. After this she remained generally unwell for a month until she was admitted to hospital in May with fever and signs of a left basal pneumonia. She was then treated with co-trimoxazole initially and rapidly became worse with confusion, pyrexia, and extreme weakness. Laboratory tests showed DNA binding of 60% with an antinuclear factor titre of 1/1000, complement C3 160% (normal 75-150%), complement C4 69% (normal 31-55%). Co-trimoxazole was stopped and she was again treated with high dose steroids and improved clinically in parallel with a change in her abnormal serological indices.

Discussion

This patient developed the multi-system features of SLE on two occasions after the administration of antibiotics, including co-trimoxazole. Sulphonamides, like penicillin, have been reported to precipitate SLE in those predisposed,[3] and it seems likely that they, rather than the trimethoprin component of co-trimoxazole, were responsible in this case. Antibodies to native DNA[4] were demonstrated during this patient's second illness and, as they are not usually found in drug-induced SLE[5] their presence in the patient may represent true SLE activated in a susceptible patient by drugs.

This case history emphasises the care that must be taken in considering giving antibiotics not only to patients with a previous history of SLE but also to those known to have Sjøgren's syndrome. It also supports the present concept that SLE and rheumatoid arthritis exist as different poles of a spectrum of disorders, with patients with Sjøgren's disease perhaps occupying an intermediate position. We think that further studies of patients like ours with similar overlap syndromes might give a greater understanding of the various disease processes concerned.

[1] Whaley, K, *et al*, *Quarterly Journal of Medicine*, 1973, **167**, 513.
[2] Dubois, E L, in *Lupus Erythematosus*, p 471, 2nd edn. Los Angeles, University of South California Press, 1974.
[3] Gould, S, *Lancet*, 1951, **2**, 268.
[4] Hughes, G R V, *Lancet*, 1971, **2**, 861.
[5] Harpey, J P, *Adverse Drug Reaction Bulletin*, 1973, **43**, 140.

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Acute rhabdomyolysis and acute renal failure after intravenous self-administration of peanut oil

Nontraumatic rhabdomyolysis with myoglobinuria may cause acute renal failure (ARF). Several agents may initiate rhabdomyolysis and another one is reported.

Case Report

A 27-year-old labourer was admitted to hospital unconscious. During the preceding 12 hours he had taken alcohol, cannabis, nitrazepam, Palfium (dextromoramide), and cocaine in unknown quantities and methadone by intravenous injection. In addition he had injected into his veins the contents of ten Hemineurin capsules. Each capsule contained 192 mg of chlormethiazole base and 384 mg of peanut oil. He was drowsy and complained of severe pain in the lower back and legs. The thighs and buttocks were appreciably swollen, painful, and tense, and there was a flaccid paraplegia with loss of pain and tactile sensation below L2 with sacral sparing. Vibration sense was absent in the legs.

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