Gas-liquid chromatography in diagnosis of pyogenic arthritis

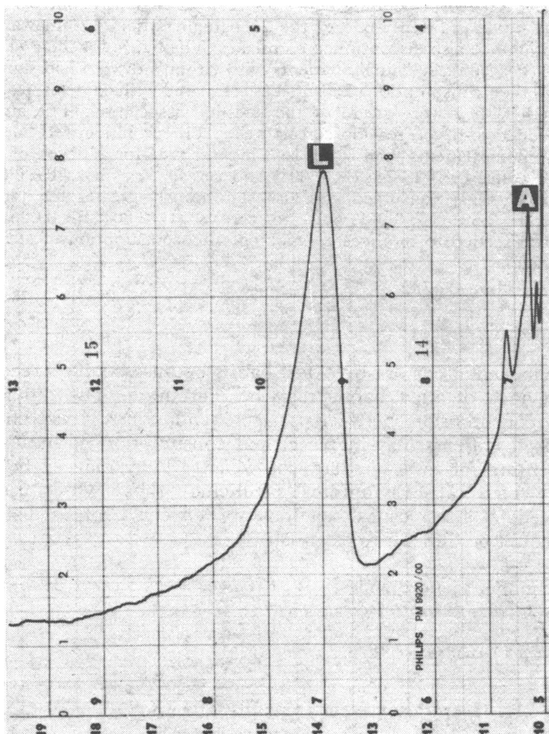
Pyogenic arthritis must be diagnosed early if joint destruction is to be avoided. Differentiation from other arthropathies is crucial, and immediate aspiration and culture of synovial fluid is essential when infection is suspected. Synovial fluid contains pus cells in all inflammatory arthropathies and, since Gram-staining is of limited reliability, a further rapid screening test for infection would be of great help. The end products of in-vivo bacterial metabolism can be detected in clinical samples by gas-liquid chromatography (GLC).[1] This technique has been applied to joint fluids in Reiter's disease and gonococcal arthritis.[2] The published methods are complex, entailing extraction and derivatisation of the metabolic products in the sample. We decided to evaluate a simplified, rapid technique, avoiding both extraction and derivatisation, for identifying volatile fatty acids in joint fluid.

Patients, methods, and results

Patients were included in the study if they had appreciable joint effusion. All were examined clinically, radiologically, and by laboratory investigation. Of the 73 patients included, 20 had Reiter's disease, seven benign gonococcal arthritis (*Neisseria gonorrhoeae* not being isolated from the joint fluid itself), five had infected joints (three with *Staphylococcus aureus*, one *N gonorrhoeae*, one non-haemolytic streptococcus), 10 seronegative rheumatoid arthritis, five seropositive rheumatoid arthritis, six degenerative arthrosis, and two crystal synovitis. Eighteen patients had an assortment of arthropathies, mostly unclassified, but including psoriatic and colitic arthropathy.

Joint fluids were collected aseptically without local anaesthesia, which might have interfered with GLC. Specimens were divided into two aliquots and either examined immediately or stored at −20°C with or without citrate as an anticoagulant. The joint fluids were injected, without extraction or derivatisation, into a Pye-Unicam series 104 chromatograph operating isothermally at 200°C with columns 1·5 m long and 4 mm bore packed with Chromosorb 101. Results were displayed graphically with a Philips PM8220 pen recorder. This technique allows the detection of a wide range of volatile organic fatty acids including acetic acid in concentrations above 1 mmol/l (5·9 mg/100 ml) and lactic acid above 10 mmol/l (90 mg/100 ml). Three specimens of joint fluid from patients with degenerative arthrosis were inoculated, one each with *Staph aureus*, *Streptococcus pyogenes*, and *N gonorrhoeae*, and then analysed after overnight incubation.

All samples produced traces, the first three minutes of which could be reproduced with serum and whole blood, with an acetic acid peak at six minutes (figure). The five culture-positive fluids also produced very high lactic acid peaks (c 50 mmol/l (c 450 mg/100 ml)) (figure). Two patients with a clinical diagnosis of gonococcal arthritis but with negative joint-fluid cultures produced lactic acid peaks, but these were not found in fluids inoculated with bacteria or samples containing many pus cells but no bacteria.



Chromatograph showing, from right to left, acetic acid peak A and high lactic acid peak L associated with joint infections.

Comment

With this simple technique of GLC examination we detected high lactic acid concentrations in all culture-positive joint fluids and in two patients with gonococcal arthritis but culture-negative joint fluid. Lactic acid was not detected in other equally purulent joint fluids, or fluids where organisms were introduced in vitro. These findings are largely supported by the brief report of Reza *et al*.[3] Detection of lactic acid therefore seems to be a useful screening test for the presence of infection. Even simpler techniques could make this a bedside procedure.

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1 Mitruka, B M, *Gas Chromographic Applications in Microbiology and Medicine*. New York, John Wiley, 1975.
2 Brooks, J B, *et al*, *Journal of Infectious Diseases*, 1974, **129**, 660.
3 Reza, M J, *et al*, in a Communication to the XIV International Congress of Rheumatology, 1977, Los Angeles.

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Myasthenia gravis and red cell aplasia

Myasthenia gravis and pure red cell aplasia (PRCA) are two rare diseases that are often associated with thymic tumours. This report describes a patient with myasthenia in whom red cell aplasia responded dramatically to corticosteroid therapy.

Case report

A 51-year-old blacksmith presented in January 1972 with a month's history of double vision, ptosis, and difficulty in prolonged talking. These features were dramatically relieved by intravenous edrophonium, confirming the diagnosis of myasthenia gravis, and subsequently his condition was well controlled by neostigmine and pyridostigmine. He remained well for the next six years, apart from long-standing chronic obstructive airways disease. In November 1977 he was admitted as an emergency with congestive cardiac failure secondary to severe anaemia. Investigation showed haemoglobin 3·7 g/dl; leucocytes $7{\cdot}3 \times 10^9/l$ (7300/mm^3) (neutrophils $4{\cdot}8 \times 10^9/l$ (4800/mm^3)); platelets $56 \times 10^9/l$ (56 000/mm^3); ESR 175 mm in 1 h; reticulocytes 0·0%; direct antiglobulin test positive (cells IgG coated); serum IgG 2700 mg/100 ml; serum IgA 540 mg/100 ml; serum IgM 220 mg/100 ml; antibodies to striated muscle were detected. Bone marrow aspiration produced hypocellular fragments with a decrease in granulopoietic and megakaryocyte elements, but was most notable for the complete absence of red cell precursors. Chest *x*-ray examination and tomography showed no evidence of thymoma. An EMI scan of the thorax later showed a nodule 0·5 cm in cross section and 3 cm long in the anterior mediastinum, which could have been in the thymus but equally well could have been a lymph node.

Red cell aplasia was diagnosed and the patient was treated with diuretics and transfusion of six units of red cells. Corticosteroid therapy was started initially with hydrocortisone 200 mg six-hourly and subsequently with prednisolone 40 mg daily. After the transfusion the haemoglobin concentration continued to rise and eight months later was stable at 13·8 g/dl; leucocytes $10{\cdot}7 \times 10^9/l$ (10 700/mm^3); platelets $282 \times 10^9/l$ (282 000/mm^3). The dosage

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