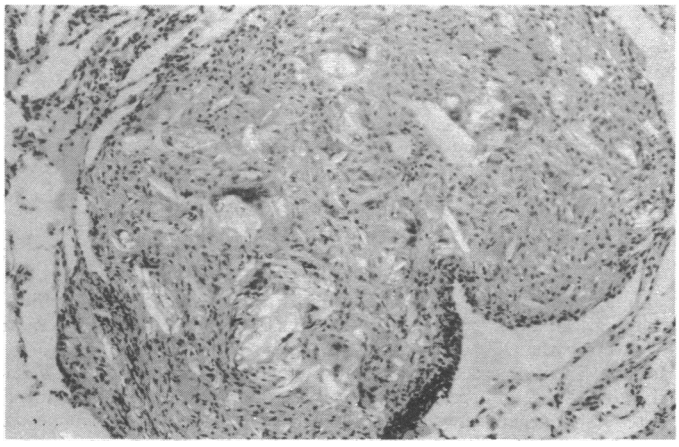

Lung biopsy specimen showing a foreign body granuloma containing many birefringent elongated particles. Section viewed with partly polarised light. Haematoxylin and eosin × 100.

patient must have been injecting himself intravenously. Subsequent investigation, however, gave more credence to the patient's claim that he had not injected himself. Examination of a deparaffinised 5 μm thick section of the lung biopsy specimen in a scanning electron microscope equipped for electron microprobe analysis with energy dispersive *x* ray diffraction apparatus failed to detect magnesium, silicon, or any other mineral constituents. On the other hand, the supposed "crystals" stained with periodic acid Schiff, silver methenamine, and Congo red, these reactions being compatible with their representing cellulose rather than talc.[3]

Comment

When the foreign material was recognised to be cellulose rather than talc we realised that the needle shaped particles represented organic fibre rather than plate like crystals seen edge on and that the aerodynamic properties of the material would consequently be quite different. If long inorganic fibres such as asbestos are capable of reaching the alveolar tissue, lighter organic fibres of similar length could be expected to behave in the same way and the patient's denial of intravenous injections appeared more credible.

We now believe that the cellulose filler damaging this patient's lungs might well have reached them via the airways rather than the blood stream. Cellulose granulomas have previously been described in the lungs of drug addicts but these patients injected their drugs.[3 4] Buchanan *et al* reported pulmonary granulomatosis in an addict who inhaled powdered drugs but denied intravenous injections: drill biopsy revealed "acicular refractile material, probably talc, though the specimen was too small for further analyses."[5] We excluded talc and identified cellulose using only five 5 μm paraffin sections of a fibreoptic transbronchial biopsy.

We thank Dr R P H Thompson for allowing us to report this case.

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Bursal fluid lactate determination and the diagnosis of bursitis

Bursitis in common is both general and hospital practice. The vast majority of cases are idiopathic but trauma and gout may be aetiological factors. Such patients respond well to simple treatment though some may require antibiotics and even drainage for infection, Not infrequently the clinical signs are misleading and sepsis is not recognised. Furthermore, microscopical appearances of an aspirate may be equivocal and the results of bacteriological culture may not be available for 48-72 hours. If in the interim corticosteroids are administered locally disastrous sequelae may ensue.

Attention has been drawn to the usefulness of cerebrospinal,[1] synovial,[2-4] and pleural and peritoneal[5] fluid lactate determinations for the rapid diagnosis of infection. We report the value of this test in the differentiation of septic and non-septic bursitis.

Methods and results

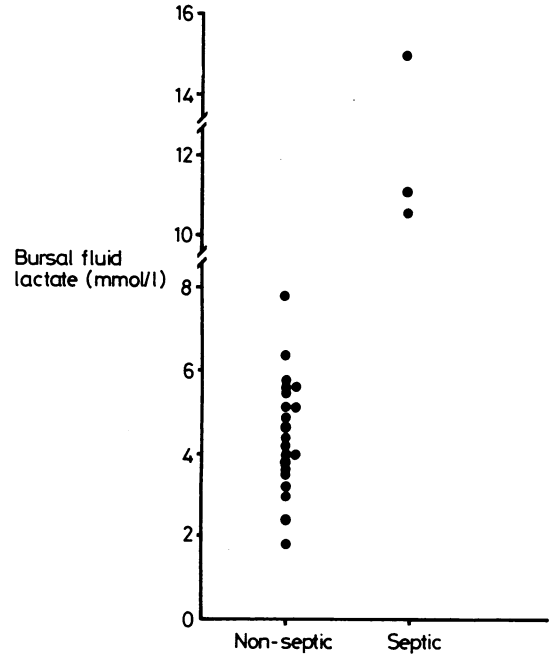
Twenty four aspirates from 23 acutely painful and clinically inflamed bursas (12 olecranon, 11 prepatella, and one tendo Achillis) were referred to this laboratory for analysis. The 23 patients (three women) were aged 22-86 years. Two cases were the result of repeated minor trauma, and in only one patient were there signs of a systemic arthropathy (chronic gout). In four cases the inflammation was a complication of a bursa that had been present for several months.

The fluids were collected into plain sterile containers and analysed within six hours. All specimens were centrifuged and the deposit cultured aerobically and anaerobically on blood agar and aerobically on "chocolate" agar. A cooked meat enrichment broth was inoculated and a Gram stained film of the deposit examined. The lactate estimation was performed on the supernatant using the Boehringer-Mannheim (BCL, Lewes, Sussex) enzymatic UV (ultraviolet) system according to the manufacturer's instructions. A sample of only 0·1 ml was required, and a Vitatron MPS colorimeter with a mercury lamp and 366 nm interference filter was used. The coefficient of variation of this method was 3·5%.[2]

The results (figure) showed a clear separation between culture positive fluids (mean 12·2 (SD 2·4) mmol/l; 110·0 (SD 21·6) mg/100 ml) and culture negative fluids (mean 4·5 (SD 1·3) mmol/l; 40·5 (SD 11·7) mg/100 ml). *Staphylococcus aureus* was isolated from all the infected samples.

In one non-septic specimen the results fell outside the statistically normal range, but this specimen had been aspirated from a clinically infected bursa in a diabetic patient three days after beginning flucloxacillin. Culture at that time was negative.

One patient had received Magnapen (ampicillin and flucloxacillin) for seven days before aspiration. Culture at that time was sterile and the lactate concentration was 4·7 mmol/l (41·9 mg/100 ml).



Bursal fluid lactate concentrations in cases of septic and non-septic bursitis.

Conversion: *SI to traditional units*—Lactate: 1 mmol/l ≈ 9 mg/100 ml.

Comment

Our findings show that higher concentrations of lactate occur in septic bursal fluid when compared with non-septic samples. The series also included a non-infected specimen with an unexplained high lactate value, and this parallels the results of other workers[1-5] with other body fluids. For example, in a recent study of over 300 synovial fluid samples[2] raised concentrations were recorded in 13 out of 56 cases of rheumatoid arthritis and four out of 58 cases of osteoarthrosis. Normal values were not found in any untreated septic specimen, and this was also true in our study.

The UV lactate test is simple to perform and gives a result in 15-20 minutes at a cost of 40p per test. It is thought that its value lies in the rapid exclusion rather than the diagnosis of sepsis, and it can be usefully performed before the administration of local treatment.

We thank the physicians and surgeons of the Nuffield Orthopaedic Centre, Oxford, for allowing us to study patients under their care. We also thank Professor R B Duthie and Dr A G Mowat for advice in preparing the manuscript, and BCL for providing the UV lactate kits.

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Ineffectiveness of haemodialysis in atropine poisoning

Poisoning with atropine is uncommon but has recently been reported in children given atropine methonitrate drops[1] and may occur after use of eye drops, ingestion of plants containing belladonna alkaloids, or errors in prescribing or dispensing and in cases of deliberate self poisoning. The anticholinergic effects of atropine are evident both centrally and peripherally, and individual susceptibility is extremely variable, death having been recorded after as little as 100 mg and recovery after as much as 1 g.[2] We recently treated a patient who took about 300 mg atropine as a result of a series of errors and who presented with signs of severe atropine poisoning. In view of the considerable overdosage that had occurred and the florid features of atropine poisoning we attempted to remove the drug by haemodialysis. We report here the outcome.

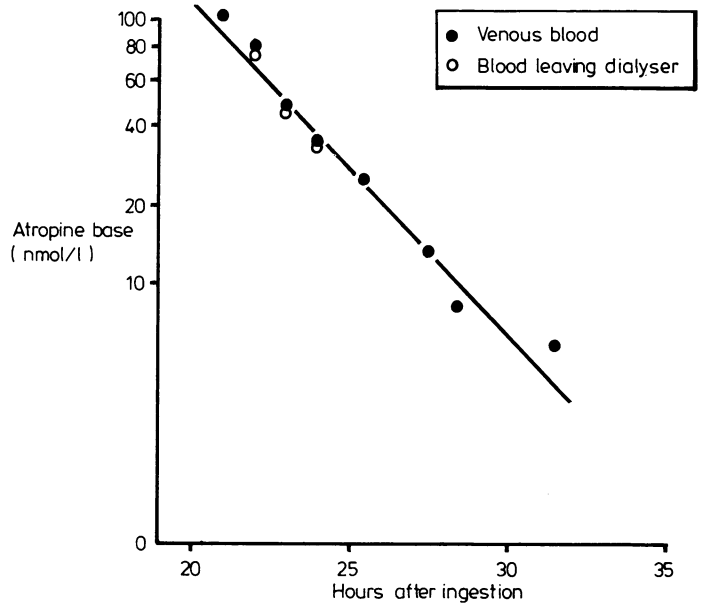
Case report

A 27 year old woman who had had a duodenal ulcer confirmed by barium meal three months previously had been prescribed magnesium trisilicate mixture and atropine sulphate for abdominal pain. As a result of an error in dispensing and her own overtreatment she ingested about 300 mg atropine. She rapidly developed symptoms and was admitted to hospital, where she was found to be agitated, feverish, and hypertensive with a dry mouth and dilated pupils. She developed acute retention of urine and required catheterisation. Initial treatment consisted of sedation (with diazepam and paraldehyde) and neostigmine 2·5-5·0 mg every three hours, after which she was transferred to be considered for haemodialysis.

On admission she was conscious and coherent, although speech was slurred and slow. There was occasional muscle clonus with generalised hyperreflexia, bilaterally upgoing planter reflexes, and fixed, dilated pupils. Blood pressure was 130/90 mm Hg and pulse 92 beats/min. Her mouth was very dry, but there were no abnormal abdominal findings other than infrequent bowel sounds. Initial investigations showed haemoglobin concentration 7·9 g/dl with normal white cell count and platelets, and normal renal function (creatinine concentration 80 μmol/l (0·9 mg/100 ml)). She was treated with physostigmine 2 mg intramuscularly or intravenously every one to two hours, forced diuresis, and haemodialysis (four hours with a Gambro Lundia 1 m^2 dialyser and blood flow rate of 192 ml/min; started 21 hours after ingestion of atropine).

There was no striking improvement immediately after dialysis, but the next day the muscle clonus had improved and plantar responses were equivocal, although her pupils remained fixed and dilated and the reflexes brisk. Improvement continued over the next three days, and she was free of the effects of atropine 96 hours after ingestion.

The plasma atropine concentrations in the first two hours of dialysis showed a gradient across the dialyser of 10%, corresponding to a clearance of about 20 ml/min. Dialysis did not, however, appear to have an appreciable effect on the plasma rate of decay of atropine since all the results, including the predialyser values taken during dialysis, had a correlation coefficient of 0·99, as shown on a semilog plot (figure).



Plasma decay curve on semilog plot.
Conversion: SI to traditional units—Atropine base: 1 nmol/l ≈ 289 ng/l.

Comment

Atropine sulphate has a molecular weight of 695 and is well absorbed from the gastrointestinal tract, rapidly distributed throughout the body, and both metabolised by the liver and excreted in the urine (94% of an injected dose appears in the urine within 24 hours, 33-50% as the parent drug and the remainder as metabolites).[3 4] The drug's poor clearance may be related to the fact that 50% is protein bound,[4] but the ineffectiveness of dialysis in the overall elimination of atropine is probably a result of the drug's large apparent volume of distribution (2-4 litre/kg)[4 5] and rapid metabolism and excretion by liver and kidneys.[4] Indeed, in this patient the half life of atropine was roughly two and a half hours, which contrasts with other reported values of 13-38 hours.[3] The only other report of haemodialysis in atropine poisoning, albeit in a moribund patient,[5] also showed the procedure to be ineffective, and we would therefore conclude that haemodialysis has no place in the management of atropine poisoning. Other patients with atropine poisoning after ingesting between 200 and 700 mg have been successfully treated with physostigmine,[2] and it was probably adequate treatment with a centrally acting cholinesterase inhibitor together with other routine supportive measures, rather than haemodialysis, that resulted in the successful outcome in this patient.

We thank Helen Remington, of the pharmacy department, for her help; Dr R F Metcalf, Chemical Defence Establishment, Porton Down, for measuring the atropine concentrations; Dr D G Leitch, of Scunthorpe General Hospital, for referring the patient; and the Yorkshire Kidney Research Fund for its support.