also recorded as palpable. In two of these patients splenic enlargement was confused with a massively enlarged left lobe of the liver and in another a large intra-abdominal tumour was subsequently shown to have caused the mass. Nevertheless, palpation gave false-positive results in 10·5% of cases and percussion in 16·6%.

Comment

Our findings show that the combination of splenic percussion and palpation will detect up to 88% of spleens considered to be enlarged on hepatic scintiscans.

In other studies the correlation between clinical detection of splenomegaly and radiography or radioisotope scanning has been poor. Although as little as a 40% increase in spleen size may be palpable, even massively enlarged spleens have occasionally gone clinically undetected. Perhaps the error is in the standard used for comparison. The borders of the spleen may not be identifiable on a radiograph and hepatic scans in which the peak count rate is set over the liver will often underestimate the actual size of the spleen. Some enlarged spleens may be undetected by both clinical and radiological techniques. Likewise, some spleens that appear enlarged by palpation or percussion, although not by radiography or scanning, may be enlarged. Nevertheless, not every palpable spleen is abnormal or enlarged, and up to 2·9% of healthy young people[4] and up to 5·6% of a general patient population[5] may have palpable spleens. In only slightly over half will an adequate explanation for the splenic palpability be found.

We can confirm that apparently normal spleens may be palpable but, in contrast to other studies, we have also shown that, with false-positives often due to hepatomegaly or abdominal masses, careful percussion and palpation will detect most enlarged spleens. Careful delineation of the left lobe of the liver may improve accuracy even further.

SS was supported by a grant from the University of Western Ontario, London, Ontario.

1 Castell, D O, *Annals of Internal Medicine*, 1967, **67**, 1265.
2 Nixon, R K, *New England Journal of Medicine*, 1954, **250**, 166.
3 Larson, S M, *et al*, *Journal of Nuclear Medicine*, 1971, **12**, 123.
4 McIntyre, O R, and Ebaugh, F G, *Annals of Internal Medicine*, 1967, **66**, 301.
5 Lipp, W F, Eckstein, E H, and Aaron, A H, *Gastroenterology*, 1944, **3**, 287.

Liver unit, King's College Hospital and Medical School, London SE5
STEPHEN SULLIVAN, MD, research fellow
ROGER WILLIAMS, MD, FRCP, director and consultant physician

Chronic sarcoid arthritis

We report here a patient with chronic sarcoid arthritis whose initial presentation resembled rheumatoid arthritis. Subsequent recognition of pulmonary and skin disease led to the correct diagnosis.

Case report

A 34-year-old Nigerian man was referred with a two-year history of chronic symmetrical polyarthritis, thought to be rheumatoid arthritis, which had not responded to salicylates or indomethacin. He had appreciable synovial thickening and chronic effusions in his knees, ankles, interphalangeal joints, and in the extensor tendon sheaths of the wrists. Four months previously he had first noted several small, well-demarcated plaques in the skin of his back, and small indurated nodules in the sites of old scars on his forehead and limbs. Mild superficial lymphadenopathy and splenomegaly were also noted on examination. His eyes were normal. There had been minimal systemic disturbance. He had no respiratory symptoms.

Erythrocyte sedimentation rate was 40 mm/h; haemoglobin 12·1 g/dl; IgG 34 g/l, other immunoglobulins normal; serum urate 0·44 mmol/l, calcium 2·4 mmol/l. Serum rheumatoid factor and antinuclear antibodies were absent. Joint radiographs were normal. On the chest *x*-ray film there was considerable diffuse shadowing throughout both lung fields. The hila were normal. A slight diminution of predicted lung volume and transfer factor was recorded in the respiratory function tests. No tubercle bacilli were found in sputum or gastric washings. The results of skin testing to tuberculin, streptokinase/streptodornase, *Candida albicans*, and mumps antigens were negative (the patient had received BCG some years previously). Surgical biopsies of tendon sheath synovium, a skin plaque lesion, and the Kveim test site all showed multiple non-caseating epithelioid granulomata consistent with sarcoidosis. Cultures of these tissues were sterile.

The initial diagnosis of seronegative rheumatoid arthritis was changed to chronic sarcoidosis with joint, skin, and lung disease. Prednisone 20 mg/day was started. There was an immediate improvement in joint pain, and a gradual reduction in joint swelling over one month. Six months later, while taking prednisone 7·5 mg/day, he had minimal joint symptoms or swelling. Joint and chest *x*-ray films were unchanged.

Discussion

Two main types of joint disease occur in sarcoidosis.[1-3] The more common is an acute transient arthritis, predominantly affecting the ankles and knees, occurring with or sometimes before erythema nodosum, and bearing the same relation to the course of the disease. Less frequently a more persistent chronic form occurs, as in this patient, reflecting granulomatous disease of the synovium, which may lead to joint destruction. Active systemic disease in other organs is generally associated. Negroes appear to be more susceptible to this type.[4] Corticosteroids were successful in controlling troublesome symptoms of synovitis in this patient. We hope that active granulomatosis will be suppressed with this treatment, and further joint damage averted.[1]

It is reported that Nigerian patients with polyarthritis satisfying the American Rheumatism Association criteria for a diagnosis of rheumatoid arthritis differ from Caucasian patients with the disease in several important respects. Though the initial features of their joint disease are typical, nodules and vascular lesions are rarely seen; radiological erosions are generally mild; and most are seronegative. A chest *x*-ray film and careful search for skin lesions may help in distinguishing chronic sarcoid arthritis in this group.

We wish to thank Professor J G Scadding for his advice and Dr D N Mitchell, who performed the Kveim test.

1 Scadding, J G, *Sarcoidosis*. London, Eyre and Spottiswoode, 1967.
2 Spilberg, I, Siltzbach, L E, and McEwen, C, *Arthritis and Rheumatism*, 1969, **12**, 126.
3 Gumpel, J M, Johns, C J, and Shulman, L E, *Annals of the Rheumatic Diseases*, 1967, **26**, 194.
4 Morrison, J G L, *British Journal of Dermatology*, 1974, **90**, 649.
5 Greenwood, B M, *Annals of the Rheumatic Diseases*, 1969, **28**, 488.

Department of Medicine, Royal Postgraduate Medical School, London W12 0HS
R R GRIGOR, MB, FRACP, registrar
G R V HUGHES, MD, MRCP, consultant physician and lecturer in medicine

Nitrofurantoin crystalluria

Nitrofurantoin is widely prescribed in the treatment and prophylaxis of urinary tract infections. It is well absorbed, rapidly concentrated in urine, and its toxic effects are rare. One substantial advantage over sulphonamides is that crystalluria—with its dangers of renal colic, haematuria, and oliguria—is believed not to occur. Goodman and Gilman state that "even supersaturated solutions of nitrofurantoin do not cause crystalluria."[1] We report the first three described cases of nitrofurantoin crystalluria.

Case 1

An 84-year-old man sustained a traumatic quadriparesis from a "whiplash" injury during a car crash in March 1975. His resulting urinary incontinence required long-term catheterisation. During the next year he developed recurrent *Escherichia coli* urinary tract infections. Prophylactic nitrofurantoin

50 mg twice daily was started in April 1976 after urine sterilisation. No side effects were noted, and his renal function remained normal. However, 10 weeks later he developed crystalluria, with small irregular bright purple crystals, despite an excellent fluid intake. After two days the crystals blocked his Silastic urinary catheter and he required repeated bladder lavage. Nitrofurantoin, his only medication during the 10 weeks, was stopped, and the crystalluria disappeared within four days. Analysis showed a sterile urine with acidity 12×10^3 nmol/l (pH 4·9). The crystals showed solubilities typical of nitrofurantoin, and ultraviolet spectrophotometry[2] identified substantial quantities of nitrofurantoin in the crystals, with calcium and magnesium also present.

Case 2

After a transurethral resection for benign prostatic hypertrophy a 75-year-old man developed urinary incontinence requiring a permanent indwelling catheter. In the next six months he developed repeated urinary tract infections. After urine sterilisation prophylactic nitrofurantoin 50 mg twice daily was begun in April 1976. Six weeks later his Silastic catheter became blocked by small bright purple crystals. Nitrofurantoin treatment was stopped, and the crystals disappeared after three days of repeated bladder washouts. His only other medication was tetrabenazine 25 mg twice daily for Parkinsonism, which he continued unchanged through this episode.

Case 3

A 71-year-old woman required long-term urinary catheterisation after a severe right hemiparesis in June 1975. Repeated urinary tract infections occurred in the next nine months, and prophylactic nitrofurantoin was started in March 1976. Three months later her Silastic catheter became blocked by small irregular purple crystals. Her only medication, nitrofurantoin, was discontinued, and the crystals ceased after two days of bladder lavage.

Discussion

No previous cases of nitrofurantoin crystalluria have been reported either to the Committee on Safety of Medicines or to the manufacturers. Both the clinical and analytical evidence point very strongly to nitrofurantoin as being the cause of these three cases of crystalluria. All occurred in old people with urinary catheters who received modest doses of nitrofurantoin for long-term prophylaxis of urinary infections. These three cases all occurred during the recent heat wave, but the patients maintained a recorded urine output of at least 1800 ml per day throughout, and all had normal renal function. Since nitrofurantoin is used frequently in urinary chemoprophylaxis, crystalluria should be widely recognised as a potential problem, though probably an uncommon one.

[1] Goodman, L S, and Gilman, A, *The Pharmacological Basis of Therapeutics*, p 1008. New York, Macmillan, 1975.
[2] *British Pharmacopoeia*, p 321. London, HMSO, 1973.

City Hospital, Hucknall Road, Nottingham
J B MACDONALD, MB, MRCP, senior medical registrar
E T MACDONALD, MB, MRCP, medical assistant

Acute epiglottiditis requiring tracheostomy in an adult

Acute epiglottiditis—an inflammatory swelling of the epiglottis or arytenoepiglottic folds—usually occurs in children between 18 months and 4 years of age. In adults it rarely leads to respiratory obstruction, though when it does the mortality may be high unless promptly recognised and treated.[1-3] Such a case treated successfully by tracheostomy is reported here.

Case report

A 44-year-old man was admitted to hospital with a two-day history of sore throat, breathlessness, and minimal dysphagia. He was mildly dyspnoeic, flushed, and had a fever of 37·7°C. Pulse was 112/min, and blood pressure 150/105 mm Hg. The soft palate was injected and there were palpably enlarged submandibular and cervical lymph nodes. Bronchial breathing was heard at the left base with associated crepitations. Chest *x*-ray examination showed patchy consolidation in the left lower lobe. White cell count was 24×10^9/l (24 000/mm^3), with 90% neutrophils. Throat swabs were taken.

The patient was treated with humidified oxygen, ampicillin 500 mg six-hourly by mouth, and domiphen bromide lozenges. For 11 hours his condition remained unchanged, then rapidly deteriorated over half an hour with increasing stridor, altered level of consciousness, shock, and central cyanosis. A large, red, swollen epiglottis was noted and he was immediately given 200 mg hydrocortisone, 500 mg ampicillin, and 500 mg cloxacillin intravenously. An emergency tracheostomy was performed and he was transferred to the intensive care unit.

The throat swabs grew *Haemophilus influenzae* sensitive to ampicillin, and this and hydrocortisone were therefore continued in full therapeutic doses. Oxygen pressure remained low (62-74 mm Hg) the first day after tracheostomy, probably due to pneumonia. The epiglottis rapidly returned to normal, and the patient was extubated on the fifth day after admission. He was discharged on the ninth day, and 20 days later was fit with a well-healed tracheostomy scar.

Discussion

Epiglottiditis differs from other respiratory tract infections in often causing rapidly progressive or abrupt airways obstruction,[4] which may occur within two hours of onset.[2] Acute epiglottiditis rarely leads to respiratory obstruction in adults, however, because of the larger larynx, smaller amount of loose mucosa, and more spatular shape of the epiglottis.[5] It is more common in men aged under 45[1] and may be more frequent in winter and in polluted atmospheres.[2] Sore throat and dysphagia may be present for up to four days before obstruction occurs.[3] The degree of shock and prostration seems to be out of proportion to the severity of the infection. Signs of respiratory distress—namely, increasing dyspnoea, cyanosis, and stridor—may be followed by death from respiratory obstruction within six hours.[3]

The commonest causative organism in adults is *H influenzae*, followed in decreasing order of frequency by group A β-haemolytic streptococci, *Staphylococcus aureus*, and pneumococci.[4] Acute epiglottiditis is a severe infection in adults, tracheostomy being needed in 30-60% of cases.[1-4] Treatment consists in maintaining clear airway, by tracheostomy if necessary. The initial antibiotic of choice is ampicillin.[1 2 4] The place of steroids, however, is still controversial.[2 3] The possibility of acute epiglottiditis should be considered in any adult who presents with upper respiratory tract infection, disproportionately sore throat, dysphagia, or any degree of respiratory distress no matter how slight.

I thank Dr R Finn for permission to report this case, and Miss P Ralphs for secretarial help.

[1] Hawkins, D B, *et al*, *Laryngoscope*, 1973, **83**, 1211.
[2] Fearn, B W, *et al*, *Canadian Medical Association Journal*, 1975, **112**, 760.
[3] Gorfinkel, H J, Brown, R, and Kasins, S A, *Annals of Internal Medicine*, 1969, **70**, 289.
[4] Bass, J W, *et al*, *Journal of the American Medical Association*, 1974, **229**, 671.
[5] Scott Brown, W G, *et al*, *Diseases of the Ear, Nose, and Throat*, 3rd edn, p 316. Philadelphia. Lippincott, 1971.

Royal Southern Hospital, Liverpool
B D LINAKER, MB, MRCP, senior medical registrar (present appointment: senior medical registrar in gastroenterology, Broadgreen Hospital, Liverpool L14 3LB)