Discussion

Castell *et al*⁴ have determined normal liver size by percussion and derived tables based on age, sex, height, and lean body weight. Unfortunately the accuracy of their clinical estimates was not confirmed by radiography or scintiscans. Naftalis and Leevy¹ found an excellent correlation between liver size on scan and clinical estimation. However, our data and those of Peternel *et al*² and Halpern *et al*,⁵ who have also used scintiscans as a reference standard, suggest that the clinical assessment of liver size is frequently very inaccurate. Blendis *et al*⁶ came to a similar conclusion using abdominal radiography as a reference standard. When the data of Peternel *et al*² are reanalysed and presented in a similar way to ours (table I) there is a striking similarity between their results and ours. They also noted the difficulty in locating the upper liver border.

This study has shown that in approximately 50% of cases liver size will be underestimated by greater than 2 cm. Rarely will the liver size be overestimated. It is clear that the major difficulty is in percussing the upper border of the liver. The location of the upper border will be underestimated in approximately two-thirds of cases. This error will be even greater if Sherlock's suggestion⁷ of heavy percussion rather than light percussion is followed. While on theoretical grounds heavy percussion should be more accurate, it may be associated with a loss of fine discriminatory sense.⁸

In this study and in others it is assumed that liver scan size accurately reflects anatomical liver size, and, although scan size and post-mortem liver size may correlate closely,¹ a number of errors may be introduced by the scanning technique. The upper border is usually sharply defined by the air-filled lung, but respiratory movements may blur it and the lower border. Similarly, if scan sensitivity is reduced, the relatively thin anterior lower border may not be readily detected, in which case the clinically located lower liver border should lie below the scanning location. In this study the converse happened more frequently.

Error in the clinical determination of liver size may arise from three sources. The method of examination or the examiner may be inaccurate, or physical characteristics of the patient may make examination difficult. Part of the error in determining

liver span is undoubtedly intrinsic to the methods of physical examination. If a method of examination introduces a constant fixed error independent of observer variation or patient build, then as the liver sizes become larger the percentage error in clinical determination of their size will become smaller. No such inverse relationship was found in this study or that of Peternel *et al*,² which suggests that if the methods of examination are inaccurate they introduce errors which are variable and not constant. This study did not attempt to assess the variations of a single observer, but in other studies it has not been a major factor.

The other major variable is that of physical build. Insufficient patients were examined to assess accurately the effect of build, but for those with slight to medium build the average error in locating the upper border was less than in the obese patients. Interestingly, the errors for the two patients with chronic obstructive pulmonary disease and very flat diaphragms were minimal.

We must conclude, therefore, that the clinical estimation of liver size is frequently an inaccurate determination and that any studies which use clinical liver size as a diagnostic, prognostic, or therapeutic index must be considered suspect. A very large liver may be readily detectable but lesser abnormalities will usually be underestimated, so that more reliable methods of determining liver size must be used.

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SHORT REPORTS

Reliability of clinical techniques for detecting splenic enlargement

The detection of an enlarged spleen is important in clinical diagnosis but seems to be subject to considerable inaccuracies. We have compared the results of palpation and percussion techniques for detecting splenic enlargement with those of hepatic scintigraphy.

Patients, methods, and results

Sixty-five consecutive unselected patients who were undergoing 99m Tc-sulphur colloid liver scans were examined by one observer. Splenic palpation and percussion were performed on each patient in the supine and right lateral decubitus positions. On palpation the spleen was recorded only as palpable or non-palpable. In the first percussion technique¹ the patient was examined in the supine position. The lowest intercostal space in the left anterior axillary was percussed and if the note was dull or became dull on deep inspiration the spleen was considered enlarged. In the second² the patient was examined in the right lateral decubitus position. Percussion was performed along a line perpendicular to the left mid-costal margin. If dullness extended more than 8 cm above the costal margin the spleen was considered enlarged. The results for palpation and percussion were recorded separately and compared with the length and width of the outline of splenic radio-

activity on the liver scan record. The spleen was considered enlarged on the scan if it was longer than 12 cm or wider than 7 cm or both.³

In 17 of the 65 patients the hepatic scan showed an enlarged spleen. In 12 of these patients the spleen was recorded as palpable in the supine or lateral position and, with one exception, had an increased area of splenic dullness. There was a considerable range of sizes: the tip varied from being just palpable to reaching the umbilicus. In three patients in whom the spleen was not palpated increased dullness was detected on percussion. Thus only two of the enlarged spleens were not detected by any form of physical examination and in one of these, although the spleen was considerably enlarged on scan (19.5 × 9 cm), examination was complicated by ascites. In the other patient the spleen was only slightly enlarged (13 × 5 cm). In eight patients with no apparent splenic enlargement on scan the spleen was thought to be enlarged by percussion. In five of these cases the spleen was

Comparison of clinical methods and radioisotope scanning in detecting splenic enlargement

Spleen size on scan	No of patients	Spleen palpable in supine or lateral position	Spleen enlarged by percussion		Spleen enlarged by palpation or percussion
			Nixon ⁴ method	Castell ¹ method	
Normal	48	5	3	8	8 (16.6%)
Enlarged	17	12	10	14	15 (88%)

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 people⁴ and up to 5.6% of a general patient population⁵ may have palpable spleens. In only slightly over half 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.

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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.

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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

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 quadriplegia 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