- 1 Wiener AS, Peters HR. Haemolytic reactions following transfusions of blood of the homologous group, with three cases in which the same agglutinin was responsible. *Ann Intern Med* 1940;13:2306-21.
- 2 Levine P, Katzin EM, Burnham L. Isoimmunization in pregnancy, its possible bearing on the etiology of erythroblastosis fetalis. *JAMA* 1941;116:825-6.
- 3 Finn R, Clarke CA, Donohoe WTA, et al. Experimental studies on the prevention of Rh haemolytic disease. *Br Med J* 1961;i:1486-90.
- 4 Clark CA, Sheppard PM. Prevention of Rhesus haemolytic disease. *Lancet* 1965;iii:343.
- 5 Freda VJ, Gorman JG, Pollack W, Robertson JG, Jennings ER, Sullivan JF. Prevention of Rh isoimmunization. *JAMA* 1967;119:390-4.
- 6 Tovey LAD. Haemolytic disease of the newborn—the changing scene. *Br J Obstet Gynaecol* 1986;93:960-6.
- 7 Giblett ER. Blood group alloantibodies: an assessment of some laboratory practices. *Transfusion* 1977;17:299-308.
- 8 Beal RW. Non-rhesus (D) blood group isoimmunization in obstetrics. *Clin Obstet Gynaecol* 1979;6:493-508.
- 9 Hardy J, Napier JAF. Red cell antibodies detected in antenatal tests on Rhesus positive women in South and mid Wales, 1948-1978. *Br J Obstet Gynaecol* 1981;88:91-100.
- 10 Bowell PJ, Allen DL, Entwistle CC. Blood group antibody screening tests during pregnancy. *Br J Obstet Gynaecol* 1986;93:1038-43.
- 11 Bowell PJ, Brown SE, Dike AE, Inskip MJ. The significance of anti-c alloimmunization in pregnancy. *Br J Obstet Gynaecol* 1986;93:1044-8.
- 12 Griffith TK. The irregular antibodies—a continuing problem. *Am J Obstet Gynaecol* 1980;137:174-5.
- 13 Yoshida Y, Yoshida H, Tatsumi K, Asoh T, Hoshino T, Matsumoto A. Successful antibody elimination in severe M-incompatible pregnancy. *N Engl J Med* 1981;305:460-1.
- 14 Dacus JV, Spinnato JA. Severe erythroblastosis fetalis secondary to anti-K_b sensitization. *Am J Obstet Gynecol* 1984;150:888-9.
- 15 Astrup J, Kornstad L. Presence of anti-c in the serum of 42 women giving birth to c positive babies: Serological and clinical findings. *Acta Obstet Gynecol Scand* 1977;56:185-8.

Acute renal failure: diagnosis of cause needed within hours

Doctors may be so busy treating patients with acute renal failure with fluid and electrolyte replacement and, if necessary, dialysis that they may forget the importance of reaching an early diagnosis of the cause of the renal failure. This amnesia may cost patients their chance of having their renal failure reversed.

A recent prospective study of 250 patients with acute renal failure reminds us of the importance of making a histological diagnosis in those patients in whom specific treatment begun early might lead to reversal of acute renal failure.¹ The proportion of patients with medical causes of acute renal failure appears to be rising, and it is these patients who will benefit most from early diagnosis by renal biopsy.^{2,3} Immunosuppressive treatment may be indicated in those with necrotising or crescentic glomerulonephritis, and recovery of renal function seems more likely if specific treatment is started before oliguria or anuria occur.^{2,5}

The suggestion that early diagnosis of medical acute renal failure by renal biopsy may help in managing acute renal failure is not new,² but the recent finding that such an approach may lead to a histological diagnosis in up to a quarter of patients with acute renal failure presenting to a specialist unit suggests that the doctors responsible for the initial care of patients in a general ward should urgently determine whether a patient needs a biopsy.¹ Carefully examining the clinical, nursing, and anaesthetic records plus examining the urine and excluding obstructive uropathy (by ultrasonography if necessary) will identify the 40-50% of patients with surgical renal failure and the few with obstetric renal failure. Clinical clues to glomerulonephritis may be rare in the remainder, although dipstick tests and microscopic examination of the urine may show the proteinuria, haematuria, and casts that point to this possibility. Renal biopsy in these patients is urgent, and they should be transferred to a specialist unit.

The reduced proportion of surgical and obstetric patients with acute renal failure probably reflects the recognition by anaesthetists of factors, such as fluid depletion, that

predispose to acute renal failure. Further encouragement about the prognosis in this group of patients comes from evidence that the survival of patients with acute renal failure after repair of abdominal aortic aneurysms can be almost as good as that of all patients with surgical acute renal failure.¹ The mortality from acute renal failure is appreciably higher in patients requiring mechanical ventilation and in those with sepsis and cardiorespiratory failure. Searching diligently for, and treating, foci of infection and avoiding measures that may precipitate cardiorespiratory failure early in the course of acute renal failure may help to reduce mortality.

The overall survival from acute renal failure has changed little in the past 20 years, although the range of underlying disease has changed appreciably. The increasing proportion of patients with medical acute renal failure calls for close collaboration between general physicians and specialist units. Failure to begin this collaboration within one or two hours of admitting a patient with acute renal failure to a general ward may deny him the chance of reversing his acute renal failure. The recent evidence that the mortality from medical acute renal failure may be lower than that from surgical acute renal failure provides strong support for management that places an emphasis on early diagnosis.¹

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- 1 Beaman M, Turney JH, Rodger RSC, McGonigle RJS, Adu D, Michael J. Changing pattern of acute renal failure. *Q J Med* (in press).
- 2 Wilson DM, Turner DR, Cameron JS, Ogg CS, Brown CB, Chantler C. Value of renal biopsy in acute intrinsic renal failure. *Br Med J* 1976;iii:459-61.
- 3 Beaufils M, Morel-Maroger L, Sraer J, Kanfer A, Kourilsky O, Richet G. Acute renal failure of glomerular origin during visceral abscesses. *N Engl J Med* 1976;295:185-8.
- 4 Heaf JG, Jorgensen F, Nielsen LP. Treatment and prognosis of extracapillary glomerulonephritis. *Nephron* 1983;35:217-24.
- 5 Courser WG. Idiopathic rapidly progressive glomerulonephritis. *Am J Nephrol* 1982;2:57-69.

Tenosynovitis in industry: menace or misnomer?

Tenosynovitis affecting the hand or forearm is now the second commonest prescribed industrial disease in Britain (the commonest is dermatitis).^{1,2} It is not always appreciated, however, that there are two distinct conditions commonly described as tenosynovitis that differ in their incidence, symptoms, signs, treatment, and prognosis.^{3,5} The first of these could be called true tenosynovitis—that is, inflammation of the synovial lining of a tendon sheath—and is rare and potentially serious. The second, which is known generally as peritendinitis crepitans, is inflammation at the musculotendinous junction that often extends well into the affected muscle.^{3,6} It is more common and much more benign. Assembly line workers who repeat the same movements up to 25 000 times a day are vulnerable to both conditions.^{7,9} The incidence is difficult to establish as many studies do not state their diagnostic criteria, but most accept that true tenosynovitis is much less common than peritendinitis crepitans.^{2,5,9} The incidence of the latter varies considerably between different occupations from under 1% to over 50%.³

Both conditions are painful (particularly on resisted movement), and there is usually localised swelling, tenderness, and audible or palpable crepitus. The distinguishing feature is the site of the lesion: true tenosynovitis affects the synovial sheaths and is thus confined to the wrist and hand, where

such sheaths are found; peritendinitis crepitans occurs well above the upper limit of the tendon sheaths in the muscles and musculotendinous junctions of the forearm.

The treatment of true tenosynovitis is well described in most standard texts.¹⁰ Once infection has been excluded, non-steroidal anti-inflammatory drugs, local injections of steroids, and plaster splintage are effective in most cases.¹¹ If conservative treatment fails or if the carpal tunnel syndrome develops then surgical release may become necessary. A few patients fail to respond to an operation and are then permanently incapacitated for repetitive work, but this is unusual.¹²

At one time rigid immobilisation was also advocated for peritendinitis crepitans,⁶ but this is now thought to be unnecessary as most cases either rapidly improve spontaneously⁵ or respond to ultrasound treatment.⁴ Most patients can resume work after four or five days.

The nature of many jobs predisposes workers to tenosynovitis or peritendinitis crepitans, and methods of prevention must be explored. Several factors seem to be important: badly designed tools,⁸ awkward working positions,⁹ lack of job variation, inadequate rest breaks, and bonuses for a high work rate or overtime.¹² Mechanisation might be expected to reduce physical work, but many industries are in a semimechanised phase in which people have to undertake repetitive work at a rate over which they have little or no control.⁹

The trade unions in Britain, taking a lead from the Australian unions,¹² have now begun to encourage their members to claim compensation from their employers for

tenosynovitis. A recent trade union publication presents "teno" as an intractable problem that typically causes long term pain and disability.¹³ This is an alarmist view, particularly as the definition of tenosynovitis used is broad and covers other less serious problems including peritendinitis crepitans. Trade union pressure might lead to industry being unfairly penalised with the costs of compensation for minor or even naturally occurring disorders.² Nevertheless, industrialists should take note of the rising tide of public opinion on this issue and take effective steps to prevent these conditions arising.

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- 1 Anonymous. *Notes on the diagnosis of occupational diseases—prescribed under the Social Security Act of 1977*. London: HMSO, 1983.
- 2 Semple JC. Tenosynovitis. *J Hand Surg [Br]* 1986;11:155-6.
- 3 Thompson AR, Plewes LW, Shaw EG. Peritendinitis crepitans and simple tenosynovitis: a clinical study of 544 cases in industry. *Br J Ind Med* 1951;8:150.
- 4 Lanfear RT, Clarke WB. The treatment of tenosynovitis in industry. *Physiotherapy* 1972;58:128-9.
- 5 Wilson RN, Wilson S. Tenosynovitis in industry. *Practitioner* 1957;173:612-5.
- 6 Howard NJ. Peritendinitis crepitans—a muscle effort syndrome. *J Bone Joint Surg [Br]* 1937;19:447-59.
- 7 Silverstein BA, Fine LJ, Armstrong TJ. Hand wrist cumulative trauma disorders in industry. *Br J Ind Med* 1986;43:779-84.
- 8 Armstrong TJ, Foulke JA, Joseph BS, Goldstein SA. Investigation of cumulative trauma disorders in a poultry processing plant. *Am Ind Hyg Assoc J* 1982;43:103-16.
- 9 Luopajarvi T, Kuorinka I, Virolainen M, Holmberg M. Prevalence of tenosynovitis and other injuries of the upper extremities in repetitive work. *Scand J Work Environ Health* 1979;5:48-55.
- 10 Verdan C. *Tendon surgery of the hand*. Edinburgh: Churchill Livingstone, 1979.
- 11 Anonymous. Traumatic tenosynovitis of the wrist [Editorial]. *Br Med J* 1978;i:528.
- 12 Browne CD, Nolan BM, Faithful DK. Occupational repetition strain injuries. Guidelines for diagnosis and management. *Med J Aust* 1984;140:329-32.
- 13 General, Municipal, Boilermakers, and Allied Trades Union. *Tackling teno*. London: College Hill Press, 1986.

Regular Review

Nuclear magnetic resonance imaging

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The first clinical reports on using magnetic resonance imaging were published more than five years ago.¹⁻⁵ Much experience has accumulated and there are now probably over 600 magnetic resonance whole body scanners operating throughout the world.

Magnetic resonance imaging does not use ionising radiation but strong magnetic fields and radiofrequency pulses. The hydrogen protons of water and fat are imaged, and their concentration determines the contrast in the images. The technique is non-invasive, well tolerated by patients, and seems to be harmless.⁶ The crucial question is whether magnetic resonance imaging is of greater diagnostic value than established non-invasive techniques, such as computed tomography, ultrasonography, and isotope imaging.

The soft tissue contrast and spatial resolution with magnetic resonance imaging are often better than with computed tomography, but in some parts of the body where motion is an important problem—for example, in the thorax and abdomen—the spatial resolution of computed tomography is

still better.⁷⁻⁹ This is largely because of the faster image acquisition time of computed tomography. The specificity of both magnetic resonance imaging and computed tomography remains limited, although great efforts are being made to improve it.¹⁰⁻¹¹ The multiplanar facility of magnetic resonance imaging is an advantage over computed tomography, which can achieve multiplanar images only by image reconstruction. The diagnostic advantages of a three dimensional facility have been shown in investigating the midline structures of the brain, the craniocervical junction, the neural canal,¹²⁻¹⁴ the mediastinum,¹⁵⁻¹⁶ the heart and great vessels,¹⁷⁻¹⁸ and some abdominal and pelvic organs.¹⁹⁻²¹

Investigating the central nervous system

Magnetic resonance imaging has several advantages over computed tomography for studying the central nervous system. The high degree of contrast between grey and white