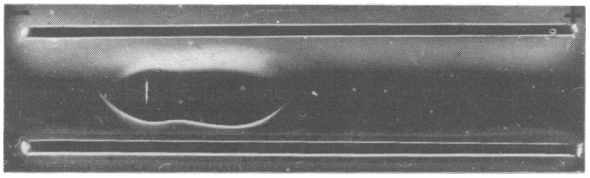
neutrophils, 5% lymphocytes, 6% monocytes, and 3% eosinophils. Chest radiography showed a mass 6 × 5 × 5 cm occupying the apical segment of the left dorsal lobe of the lung. Liver scan showed a space-occupying mass 5-6 mm in diameter in the posterior-dorsal region of the right lobe near the upper pole of the right kidney. This combination of masses in lung and liver in a farm worker suggested hydatid disease. Within 24 h of incubating a serum sample a broad and intense precipitin band with *E granulosus* cyst fluid antigen developed in the IEP test where the diagnostic hydatidosis arc 5 is located. After a further 48 h of incubation, however, the edges of this precipitin band failed to join with those of the arc 5 given by the reference positive serum placed in the opposite trough of the same IEP plate (figure).



Immunoelectrophoresis test for hydatidosis after 72 h incubation, using concentrated ovine *Echinococcus granulosus* cyst fluid antigen. Upper trough contained serum from patient with pulmonary carcinoma. Lower trough contained arc 5 positive serum from sheep-infected with *E granulosus*.

The mass removed by radical left upper lobectomy was a squamous cell carcinoma with a necrotic core. On further laboratory tests, eight weeks after the lung operation and before exploratory surgery to investigate the mass in the liver area, the white blood cell count had dropped to $7{\cdot}1 \times 10^9$/l (7100/mm$^3$). The peripheral differential cell count had not altered. The erythrocyte sedimentation rate had also dropped to 26 mm in 1st h. A serum sample taken at this stage did not give any precipitin bands in the IEP test for hydatidosis. The mass was found to be a hard, calcified benign adrenal tumour with no histologically recognisable tissue.

## Comment

The cyst fluid of *E granulosus* contains a number of antigenic molecules which are apparently shared with a variety of parasites.[3] Nevertheless, reports on cross-reactions between hydatid antigens and sera from patients with confirmed neoplastic growth are rare.[1] Recently a patient with multiple cysticercosis and myeloma was found to have circulating antibodies against the *E granulosus* arc 5 antigen.[4]

The precipitin band that we observed did not dissolve in 5% trisodium acetate solution,[3] indicating that it was the product of an antigen-antibody reaction. Because of its diffuse nature we could not definitely say that it did not join up with the arc 5 precipitin band in *E granulosus* cyst fluid precipitated with hydatid-infected sheep serum. We have seen similar diffuse bands produced by serum from hydatid-infected sheep. Circumstantial evidence that the band was not stimulated by an *E granulosus* infection was that the patient's serum eight weeks after the removal of the pulmonary carcinoma did not precipitate with *E granulosus* cyst fluid. Since the patient had always worked on farms he had been exposed to hydatid organisms or other taeniid tapeworms, which are endemic in New Zealand. Nevertheless, careful palpation of the liver and lungs during surgery and further organ and bone scans revealed no other lesions. Therefore the antigens released by the pulmonary squamous cell carcinoma are apparently similar to antigens in hydatid cyst fluid.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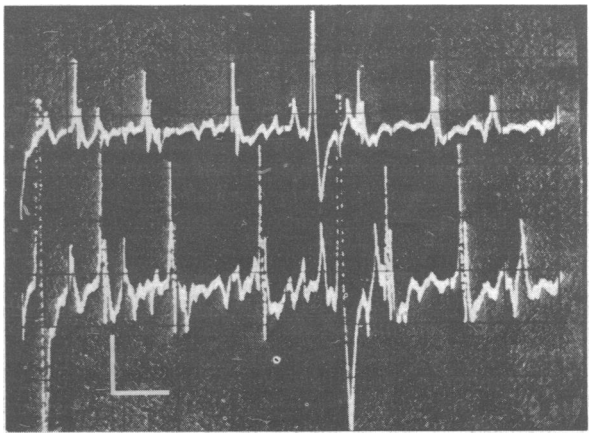
# Penicillamine-induced neuromyotonia

Treatment of rheumatoid arthritis with penicillamine[1] has been associated with various unwanted effects,[2] including polymyositis and myasthenia gravis. We have recently encountered an additional neuromuscular complication of penicillamine treatment—neuromyotonia.

## Case report

A 61-year-old woman developed seropositive, erosive rheumatoid arthritis in 1966. She was treated with analgesics and anti-inflammatory agents. In 1974 her arthritis became more active and penicillamine 750 mg daily was started. In 1975 her platelet count fell slightly and the penicillamine was reduced to 500 mg daily. Since then her rheumatoid arthritis has remained quiescent. For two months she had complained of feelings like "electric shocks" in the arms and legs with a "pulling" feeling in her muscles. She also noted some tingling round her mouth and had difficulty in opening her eyes. When attempting to move, her hands or toes would curl and she would momentarily be unable to grip objects. She was seen on one occasion to become rigid on standing.

On examination, she had difficulty in relaxing muscles after contraction. For example, her fingers after clenching remained flexed for up to 30 seconds before the forearm flexors suddenly relaxed. Relaxation could be induced by gently extending the fingers. She had similar prolonged contraction in her periorbital facial muscles with difficulty in opening her eyes after blinking. This was mistaken at first for ptosis. Extraocular movements were normal. There was no myasthenic weakness or percussion myotonia; sensory examination was normal; ankle jerks were diminished; joint deformities were only slight. Concentric needle electromyography (EMG) in the small hand muscles, biceps, and deltoids showed prominent spontaneous discharges (figure). These consisted of one or more motor units firing repetitively at



EMG: spontaneous firing of three motor units. Calibration: 100 $\mu$V and 10 ms.

10 to 30/s. Electrode movement induced bursts of motor unit activity lasting for between 21 and 26 seconds. After brief voluntary muscular contraction similar activity was seen, continuing for the same time. Rare pseudomyotonic discharges were seen. Motor unit action potentials during voluntary activity were normal, and the interference pattern was full. Single fibre EMG showed no jitter or blocking, and the fibre density was normal. Nerve conduction velocity measurements were normal. The evoked response to repetitive stimulation of the ulnar nerve at the wrist, recording from the abductor digiti minimi, showed no decrement of the 4th potential at 2 Hz, but at 10 Hz and 20 Hz there was a 25% decrement. A needle biopsy of the right quadriceps muscle was normal; the blood creatine kinase concentration was also normal.

Neuromyotonia was diagnosed and phenytoin 300 mg daily was given. The patient noted improvement within three days. EMG studies six weeks later showed much reduction in the neuromyotonia. The penicillamine was stopped and the neuromyotonia disappeared. It did not recur when phenytoin was also discontinued.

## Comment

The neuromyotonia in this patient was associated with penicillamine. There was no EMG evidence of neuropathy, although neuromyotonia has been described in patients with both congenital and acquired neuropathies.[3] The pathogenesis of neuromyotonia is uncertain. It is probably due to spontaneous firing in the distal part of the axonal tree,

since ischaemia or procaine block of peripheral nerves usually abolishes it.[3 4] Toxic reactions to penicillamine, especially haematological, renal, and gastrointestinal disturbances, occur in patients with rheumatoid arthritis. Neurological complications are relatively uncommon. Polymyositis and myasthenia gravis have been reported, but both were excluded in our patient. The decremental EMG response found only at higher stimulation rates, in the absence of single fibre EMG abnormalities, is unlike that found in myasthenia gravis or Eaton-Lambert syndrome.[5] To recognise neuromyotonia requires careful clinical and EMG examination since it is easily mistaken for myasthenia gravis.

We thank Dr C G Barnes for referring this patient.

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# Age and prognosis in breast cancer

Contrary to previously stated opinion,[1] it has recently been suggested that breast cancer is more rapidly lethal with increasing age at presentation[2] and that this might be explained by an age-related difference in tumour biology or host response.[3] We did an actuarial analysis of the relationship between age at presentation with breast cancer and survival.

## Patients, methods, and results

Up to 31 December 1977, 807 patients with primary breast cancer had presented to our unit. Of these, 214 (26·5%) were aged 21-49, 382 (47·3%) were aged 50-59, and 211 (26·2%) were aged 70-93. All were screened on presentation for metastatic disease, by methods we have previously described,[4] and staged according to the TNM classification. Survival data for the three age groups were determined by actuarial analysis. Thirty (3·7%) patients were excluded from the study because they died of causes other than breast cancer. A $\chi^2$ test was used to establish whether there was any linear trend for the incidence of advanced local disease or metastatic disease at presentation and to establish statistical significance between the estimated 10-year survival in the three age groups.

*Correlation of age at presentation with breast cancer and incidence of advanced local disease, incidence of metastatic disease, and estimated 10-year survival*

| Age at presentation (years) | Advanced local disease (%) | Metastatic disease (%) | Estimated 10-year survival (%) |
|---|---|---|---|
| 21-49 | 34·1 | 7·5 | 53·0 |
| 50-69 | 48·7 | 13·6 | 42·5 |
| 70-93 | 59·7 | 16·6 | 33·9 |

Patients had more advanced local disease with increasing age at presentation. The incidence of T3 and T4 tumours in the three age groups is shown in the table ($P < 0·001$). The incidence of metastatic disease at presentation also significantly increased with age ($0·001 < P < 0·01$). Actuarial analysis confirmed that the estimated 10-year survival was significantly reduced with increasing age at presentation (table). But when the analysis was repeated excluding patients with evidence of metastatic disease at presentation there was no statistical difference between the actuarial curves of the three age groups ($P = 0·59$).

## Comment

With increasing age at presentation of breast cancer the patients had more advanced local disease, a higher incidence of metastatic disease, and a lower estimated 10-year survival. But when patients with metastatic disease at presentation were excluded there was no statistical difference in estimated survival between the three age groups. This evidence suggests that the deteriorating prognosis with increasing age was due to a more advanced stage of disease at presentation and not to age-related tumour or host factors.

This work was supported by the Breast Cancer Research Trust.

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# Diverticular disease of the colon in Africans

Diverticular disease of the colon has been thought to be almost unknown in Africans.[1] Personal knowledge of six cases,[2] however, suggested that the condition might not be so uncommon, and so a search for further cases was instituted.

## Materials, methods, and results

The findings of 226 barium-enema examinations performed on adults at this hospital between January 1976 and February 1978 were reviewed. The table gives the results.

A total of 15 of the 226 patients had diverticular disease. This was the commonest single diagnosis made on barium-enema examination, as the "non-specific colitis" group almost certainly contained several different disease entities. The age range of patients with diverticular disease was 29-80 (mean 41) years and that of all patients in the series 18-80 (mean 40) years. Five patients with diverticular disease were aged 40 or under. This age incidence is lower than would be expected in a developed country.[3]

*Results of 226 barium-enema examinations performed on adult Africans over two years*

| | No of patients | | No of patients |
|---|---|---|---|
| Normal | 155 | Granuloma | 6 |
| Diverticular disease | 15 | Ulcerative colitis | 4 |
| Non-specific inflammatory disease | 21 | Other | 5 |
| Carcinoma | 7 | Unsatisfactory examinations | 13 |

## Comment

Diverticular disease of the colon was the commonest barium-enema diagnosis made at this hospital during the two years. Since the condition is considered to be rare in Africans[1] the finding suggests either an increasing incidence or lack of recognition in the past. The rarity of diverticular disease in Africans has been attributed to their high-fibre diet.[1 4] Dietary histories could not be obtained in this retrospective survey, but general observations and discussions with African colleagues suggest that the diet of even urban Kenyan Africans is still high in fibre by Western standards, although there has been an increasing use of sifted maize flour and refined sugar in recent years. If as Burkitt[4] suggests the greater part of a lifetime or about 40 years in a responsible environment is required to produce