

### Patients and methods

The six patients studied had all had carcinoma of the breast that had been treated by simple mastectomy followed by a full course of radiotherapy to the chest wall and cervicoaxillary lymph nodes six months to 12 years earlier. The maximum calculated tissue dose was 3000-3400 rads at the lung apex and base. They were aged from 44 to 71 years, and none gave a history of tuberculosis or chest disease. At the time of scanning all had a chest radiograph on which the lung fields were reported normal. All patients gave informed consent.

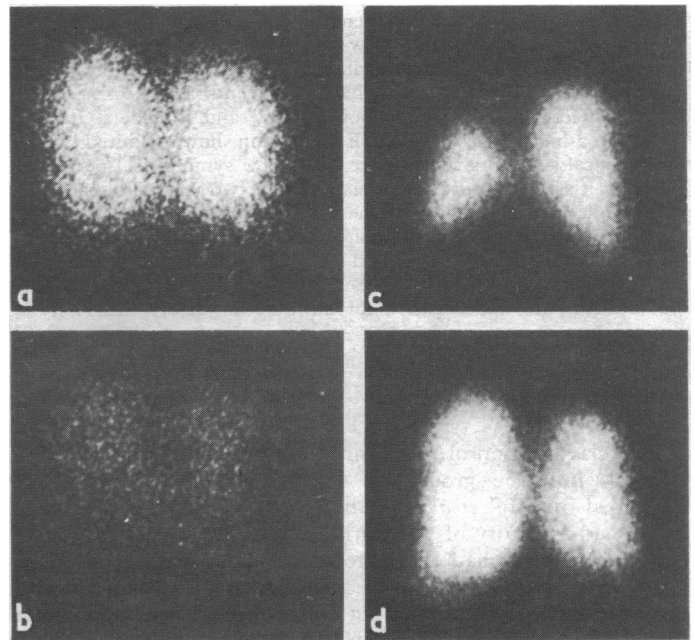
An Ohio Nuclear Series 100 gamma-camera equipped with a diverging collimator was used to record the scans on polaroid film. The ventilation scan was performed with  $^{133}\text{Xe}$  gas and the perfusion scan with  $^{99\text{m}}\text{Tc}$ -macroaggregated albumin.<sup>2</sup> Each scan was mixed with six other normal or abnormal scans before being reported on by two experienced observers who knew none of the patients.

### Results

Eleven of the 12 reports on the scans of the patients who had undergone radiotherapy noted abnormalities. Perfusion defects with ventilation-perfusion mismatch were noted at apex and base on six scans (three patients) and at the apex alone in the other five. One scan was reported normal. All the defects reported were on the irradiated side and corresponded to the areas of maximum calculated tissue dose. The figure shows mismatch (normal ventilation, impaired perfusion) in the scan of one of the patients.

### Discussion

The diagnosis of pulmonary embolism depends on a suggestive clinical history and clinical signs, a diagnostic or normal chest radiograph, and ventilation-perfusion mismatch on the combined lung scan.<sup>1-3</sup> False-positive scans are caused by the destruction of the pulmonary arterial bed while ventilation to the area is maintained. This occurs after radiotherapy. Radiotherapy may also cause breathlessness and non-specific chest pains in the presence of a normal chest radiograph,<sup>5</sup> thus clinically mimicking pulmonary embolism. In these circumstances the diagnosis will depend heavily on the lung scan and we have shown that radiotherapy will produce ventilation-perfusion mismatch on the combined lung scan. We therefore recommend that special care should be taken when interpreting the lung scans of patients who have received a significant tissue



Mismatch on ventilation-perfusion lung scan of one patient. (a)  $^{133}\text{Xe}$  ventilation, posterior view, first breath held. (b)  $^{133}\text{Xe}$  ventilation, posterior view, one-minute washout. (c)  $^{99\text{m}}\text{Tc}$  perfusion, anterior view. (d)  $^{99\text{m}}\text{Tc}$  perfusion, posterior view.

dose of radiation to the lung, as mismatch in affected areas may follow the radiotherapy and need not indicate pulmonary embolism.

### References

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## SHORT REPORTS

### Traumatic division of pharyngeal branch of vagus causing dysphagia

Trauma to the neck produced dysphagia thought to be due to division of the pharyngeal branch of the vagus nerve and consequent failure of relaxation of one side of the cricopharyngeus.

#### Case report

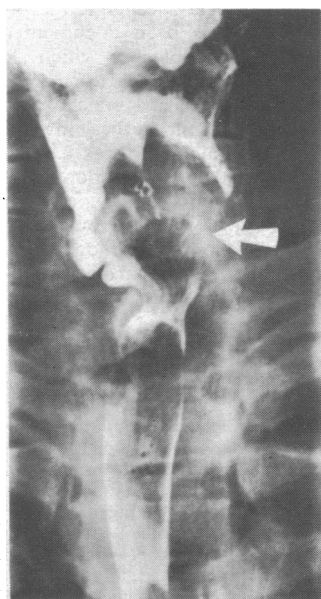
A 55-year-old foreman supervising the pouring of concrete was knocked off a platform 6 m high by a crane. His fall was partly broken by a ledge 2 m above the ground and when he landed he impaled his neck on one of several upright iron rods forming the reinforcing structure. The rod was 3 cm in diameter and 76 cm high. He did not lose consciousness and received no other injuries in the fall. When seen he had a jagged triangular wound below the left mandible measuring  $6 \times 10 \times 12$  cm. There was little bleeding but the submandibular gland was dislocated and lay almost free on the skin of the neck. Examination revealed a left hypoglossal nerve palsy. The soft palate was also partially paralysed on the left so some degree of vagal damage was likely. The wound extended behind the pharynx to beyond the midline but the large vessels of the neck were intact. The submandibular gland

together with some devitalised skin and muscle was removed and healing was obtained by first intention.

The only subsequent problem was dysphagia. Oesophagoscopy established that there was no stricture or extrinsic pressure but the cricopharyngeus failed to relax. Barium swallows showed pooling of barium on the left above the cricopharyngeus—the appearance of cricopharyngeal achalasia.<sup>1</sup> Interestingly, the patient was able to describe food “sticking” on the left and taught himself a curious trick of rocking his head over to the right to empty this pocket (see fig). Further neurological examinations during his recovery confirmed persistence of paralysis of the left side of the tongue and soft palate. There was full movement of both vocal cords. Sensibility was lost from the left side of the soft palate and the left tonsillar fossa. It was preserved over the posterior third of the tongue and the oropharynx. The gag reflex was not obtained by stimulation to the left of the midline.

#### Discussion

The neurological deficit is interesting and its production by this type of trauma would appear to be unique. The sensory changes imply damage to the left glossopharyngeal nerve. The palatal palsy implies an injury to some part of the left vagus although as the left cord moves the recurrent laryngeal nerve and therefore the left vagal trunk must be intact. It therefore appears that the pharyngeal branch of the vagus



Barium swallow showing contracted left cricopharyngeus (arrowed) obstructing flow on this side and barium passing down on right side.

has been divided. There has been some debate about the innervation of the cricopharyngeus. Asherson<sup>2</sup> states that the recurrent laryngeal nerve is responsible and blames "achalasia of the cricopharyngeal sphincter" on trauma to recurrent laryngeal nerves.<sup>3</sup> None of the patients described, however, had symptomatic dysphagia.

More recent work,<sup>4</sup> including dissections, has failed to support this view and suggests that the cricopharyngeus is innervated unilaterally by the pharyngeal branch of the vagus via the pharyngeal plexus. The active side will push barium into the paralysed and therefore atonic side of the pharynx.<sup>1</sup> The failure of relaxation or incoordination of cricopharyngeus on the denervated side causes formation of a pocket and thus the appearances of achalasia of the cricopharyngeus.

Some improvement was achieved in this patient's swallowing by performing cricopharyngeal myotomy and subsequent dilation. He was then able to take a reasonable diet and maintain a somewhat reduced weight. Further surgical interference was not attempted.

I wish to thank Dr Stuart Field and Mr Alan Ardouin for their help and Mr Michael Williams for permission to report this case.

<sup>1</sup> Seaman, William B, *Alimentary Tract Roentgenology*, ed A R Margulis and H J Burnhenn, Saint Louis, The C V Mosby Company, 1967.

<sup>2</sup> Asherson, N, *Proceedings of the Royal Society of Medicine*, 1962, **55**, 117.

<sup>3</sup> Asherson, N, *Journal of Laryngology*, 1950, **64**, 747.

<sup>4</sup> Lund, W S, and Ardran, G M, *Annals of Otolaryngology and Rhinology*, 1964, **73**, 599.

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## Erythema nodosum and brucellosis

Erythema nodosum is a distinct clinical entity which may be an important cutaneous sign of systemic disease. Its uniform appearance gives no indication of the underlying cause nor does the characteristic histopathology.<sup>1</sup> It is thought to represent a hypersensitivity phenomenon<sup>2</sup> but not the classic Arthus type; its close association with conditions having delayed hypersensitivity suggests a cell-mediated mechanism.<sup>3</sup> The commonest cause of erythema nodosum in Britain

up to 20 years ago was primary tuberculosis, but is now probably sarcoidosis; whereas in the USA streptococcal pharyngitis is the predominant cause on the east coast and coccidioidomycosis in the west.<sup>3</sup> Other recognised associations include drug hypersensitivity, leprosy, and ulcerative colitis. An association between erythema nodosum and brucellosis has not been recorded in the past 10 years and is not mentioned in the standard textbooks of medicine. The following case history is reported because of the importance of diagnosing brucellosis, for which there is effective treatment.

### Case report

A 38-year-old man was admitted to hospital in January 1974 with pain which started four weeks previously in the ankles and knees and spread to spine, hips, wrists, and hands. He had had drenching night sweats for 10 days. Five days before admission he developed typical erythema nodosum on the front of both legs below the knees. The only drug he had taken was two tablets of naproxen prescribed a few days earlier. His occupation involved metal weighing, principally gold and silver but also brass, nickel, and bronze. He had had no contact with livestock or unpasteurised milk, nor had he recently left the city. He had a low-grade fever but was otherwise fit. The typical rash on the legs was accompanied by swelling of soft tissues around the joints. He continued to have night sweats and fever over the next few days.

The results of investigations showed: haemoglobin 13.2 g/dl, white cell count  $7 \times 10^9/l$  ( $7000/mm^3$ ), and erythrocyte sedimentation rate (ESR) 40 mm in 1 hour; chest radiograph and liver function were normal; Mantoux test reaction (1/1000) was negative; sheep red cell agglutination test result was normal; and he was negative for antinuclear factor; *Brucella abortus* agglutination titre  $> 1/3200$ . He was treated for six weeks with tetracycline and all his symptoms and signs disappeared. The ESR fell to normal and the *Br abortus* agglutination titre was  $< 1/25$  at the end of treatment and six months later when he appeared fit.

### Comment

Brucellosis is still endemic in Britain and may be difficult to diagnose unless suspected. It should be considered in patients with erythema nodosum in whom the usual investigations do not reveal the underlying cause.

<sup>1</sup> Cecil-Loeb *Textbook of Medicine*, ed P B Beeson and W McDermott, 13th edn, p 846. Philadelphia, Saunders 1971.

<sup>2</sup> Samter, M, *Immunological Diseases*, ch 28, 29. London, Churchill Livingstone, 1971.

<sup>3</sup> Sherman, W B, *Hypersensitivity, Mechanisms and Management*, p 370. Philadelphia, W B, Saunders, 1968.

<sup>4</sup> James, D Geraint, *British Medical Journal*, 1961, **1**, 853.

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## Extramedullary plasmacytomas, renal failure, and intermittent haemodialysis

Chronic renal failure in multiple myeloma is associated with a poor prognosis, and treatment by dialysis rarely prolongs life.<sup>1</sup> Extramedullary plasmacytomas, however, may be associated with prolonged survival, and we have been unable to find any report of renal failure complicating the disease. We describe here a patient with extramedullary plasmacytomas who developed irreversible renal failure and who was maintained on regular dialysis.

### Case report

A 56-year-old plumber was admitted to hospital in October 1974 with a four-month history of bilateral loin pain, nocturia, weight loss, and feeling unwell. He was pale with periorbital oedema, and had tenderness in his right