

The results are shown in the fig. The paired *t* test comparing the control concentration with the concentrations at 24, 48, and 72 hours showed significantly higher concentrations of plasma zinc for each time during the starvation period ( $P < 0.025$ ,  $< 0.0125$ , and  $< 0.0025$  respectively). The mean plasma concentrations ( $\pm$  SEM) were  $13.4 \pm 0.6 \mu\text{mol/l}$  ( $88.2 \pm 3.9 \mu\text{g}/100 \text{ ml}$ ) at the control reading and  $18.2 \pm 1.2$ ,  $18.0 \pm 1.2$ , and  $18.8 \pm 0.9 \mu\text{mol/l}$  ( $119.7 \pm 7.8$ ,  $118.4 \pm 7.8$ , and  $123.6 \pm 5.9 \mu\text{g}/100 \text{ ml}$ ) at 24, 48, and 72 hours respectively. There were individual variations but no subject failed to show a rise.

## Discussion

Plasma zinc concentrations vary diurnally and fall after a meal. This experiment was designed to try to eliminate these factors. Oral contraceptives lower plasma zinc concentrations, but the subjects in our study taking these preparations had unremarkable control values. They continued to take them throughout and the changes in their plasma zinc concentrations were similar to those of the other subjects. We do not know the origin of the zinc that raised the plasma concentrations in our subjects, but Spencer and Samachson<sup>3</sup> suggest that the zinc in the zincuria of prolonged starvation comes from the liver.

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<sup>1</sup> Pascoe, E, Arslanian, M, and Reinhold, J G, *Pahlavi Medical Journal*, 1971, 2, 29.

<sup>2</sup> Quarterman, J, 1974, personal communication.

<sup>3</sup> Spencer, H, and Samachson, J, in *Trace Element Metabolism in Animals*, ed C F Mills, p 312. Edinburgh, Livingstone, 1970.

<sup>4</sup> Peaston, R T, *Medical Laboratory Technology*, 1973, 30, 249.

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## Axillary artery occlusion and pulmonary tuberculosis

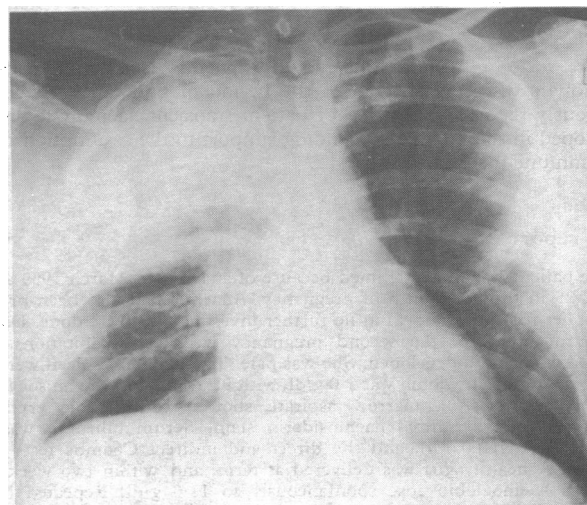
Axillary artery thrombosis may complicate various conditions, but its association with pulmonary tuberculosis has not to our knowledge been previously reported.

### Case report

A 45-year-old male Sikh was admitted with a ten-day history of productive cough and fever, together with pain of gradually increasing severity in the right arm. He was a non-smoker. On examination his temperature was  $38^{\circ}\text{C}$ , pulse 90/min, and blood pressure 120/90 mm Hg. The right hand was cold and cyanosed and all arterial pulses of the right upper limb were absent. No bruits were audible in the neck. There were signs of consolidation of the right upper lobe and this was confirmed by a chest radiograph (figure). An electrocardiogram was normal and urine testing showed no glycosuria. He was thought to have axillary artery thrombosis and pneumonia, possibly with an underlying bronchial carcinoma. Treatment was with heparin, analgesics, and antibiotics—initially ampicillin and later cephalothin and gentamicin. He remained febrile but the axillary and brachial pulses returned over the next few days and ischaemic changes were limited to dry gangrene of the finger tips.

One week after admission he deteriorated, becoming semicomatose with tachycardia, tachypnoea, hypotension, cyanosis, a rise in the jugular venous pressure, and crepitations in both lung fields. Despite the clinical appearance of heart failure the chest radiograph was essentially unchanged and an electrocardiogram showed no appreciable abnormality. Investigations showed that he had a haemoglobin of 4 g/dl and a blood glucose of 19.4 mmol/l (350 mg/100 ml). He was therefore transfused with packed cells and given

insulin, oxygen, frusemide, digoxin, and hydrocortisone. He was thought to have a septicaemia due either to tuberculosis or a Gram-positive organism; the antibiotic regimen was therefore changed to rifampicin, isoniazid, streptomycin, and cloxacillin.



Chest radiograph showing consolidation in right upper lobe.

He improved dramatically over the next 24 hours, becoming fully conscious and afebrile. Sputa were subsequently found to contain *Mycobacterium tuberculosis*; antituberculous therapy was continued and diabetic control achieved with oral agents. Six weeks after admission the tips of several fingers were amputated. His further progress has been satisfactory, with complete resolution of the radiological opacity.

## Discussion

It is unlikely that the arterial occlusion was embolic, as clinical examination and electrocardiography failed to show any source for an embolus. Axillary artery thrombosis is an uncommon lesion which may complicate trauma to the arm, a cervical rib,<sup>1</sup> radiotherapy,<sup>2</sup> or bronchial carcinoma<sup>3</sup>; there is one case report of its association with penicillin anaphylaxis.<sup>4</sup> Occasionally there is no obvious cause. The association with pulmonary tuberculosis has not been previously described, though we have been told about a similar case in which the arterial thrombosis occurred on the contralateral side to a tuberculous pneumonia. Possibly a generalised clotting tendency is present in tuberculosis, but this aspect has received little attention in published work. There was no evidence of a cervical rib or aneurysm of the axillary artery, but latent diabetes mellitus may have been a factor in our case.<sup>5</sup> The diagnosis of pulmonary tuberculosis was considered because of his race and because, as a life-long non-smoker, carcinoma of the bronchus was unlikely. He was in extremis and antituberculous therapy was started before bacteriological proof had been obtained.

Since treated tuberculosis has a good prognosis, this condition should be excluded in all patients who present with an arterial occlusion in the arm and an abnormal chest radiograph.

We wish to thank Mr A Clain and Dr J F Riordan for permission to report this case.

<sup>1</sup> Wood, P B, *British Journal of Surgery*, 1973, 60, 29.

<sup>2</sup> Mavor, G E, et al, *British Journal of Surgery*, 1973, 60, 983.

<sup>3</sup> Knox, W G, Nay, M R, and Blumenthal, J, *American Journal of Surgery*, 1966, 111, 752.

<sup>4</sup> Andrejevic, M, *Srpski Arhiv za Celokupno Lekarstvo*, 1964, 92, 793.

<sup>5</sup> Goldenberg, S, et al, *Diabetes*, 1959, 8, 261.

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