Thorax, 1979, **34**, 559–560

Constrictive pericarditis resulting from the transpulmonary migration of an inhaled nail

RICHARD HAAS, RICHARD LEA, COSTAS SBOKOS, AND NEVILLE CONWAY

From the Wessex Regional Cardiac and Thoracic Centre, Western Hospital, Southampton, UK

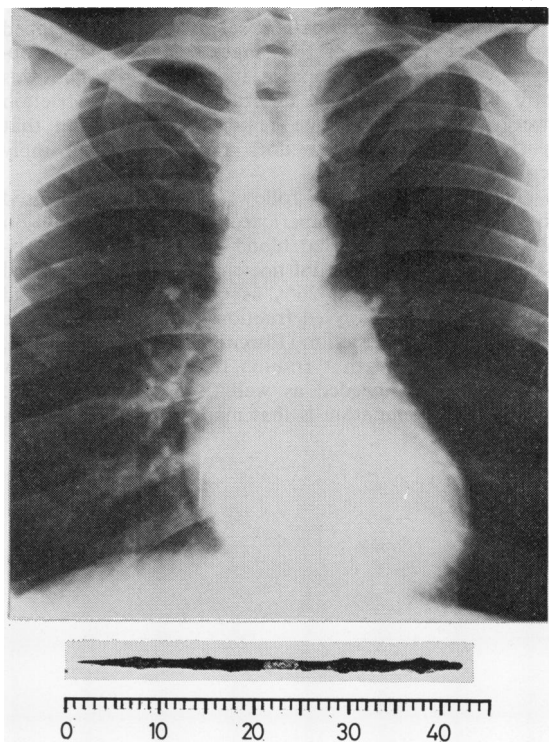
Post-traumatic constrictive pericarditis is uncommon. Its development after non-penetrating injury to the heart is well recorded (Rasaretnam and Paul, 1975), but there have been few reports of constriction after penetrating trauma (McKusick *et al*, 1955). The case presented here is a remarkable example in that the initial injury to the pericardium was the result of the migration of an inhaled carpenter's nail across the lung, constriction following many years later.

Case report

A carpenter aged 37 presented in 1958 with a history of aching low anterior chest pain for one year. The pain was worse on exertion but also occurred at night. He had noted an intermittent cough productive of white sputum, and nine months before admission this had been associated with a small haemoptysis. Like most carpenters he was in the habit of holding nails in his mouth while working but could not remember inhaling one. A chest radiograph at the time of the haemoptysis was initially reported as normal, but on reviewing it a nail was seen lying close to the right cardiac border (see figure). Screening showed the nail in position and, in addition, calcification in the pericardium both adjacent to it and over the left ventricular border. A barium swallow showed a small sliding hiatus hernia with intermittent oesophageal reflux; this was thought to account for his pain. At this stage there were no cardiovascular signs.

At thoracotomy the nail was found deep to the parietal pericardium, anterior to the inferior vena cava, and superficially embedded in the right atrial wall; there was calcification of the pericardium anterior to its point. The nail was removed and the hiatus hernia repaired, but pericardiectomy was not undertaken. The patient made an uneventful recovery and was lost to follow-up.

Sixteen years later he was admitted elsewhere complaining of persistent chest discomfort, effort dyspnoea, and orthopnoea. He was found to have atrial fibrillation, to which his symptoms were attributed. Shortly afterwards sinus rhythm returned. He was also noted to have a persistently high jugular venous pressure. When seen at the Regional Cardiac Centre he had the classic signs of pericardial constriction: pulsus paradoxus of 15 mmHg, a jugular venous pressure of 10 cm with deep "x" and "y" descents, a quiet cardiac impulse, and an early third heart sound. A chest



PA chest radiograph in 1958. Nail can be seen just inside right border of heart. Below: Nail after surgical removal.

radiograph now showed more extensive pericardial calcification. The electrocardiogram showed generalised T wave inversion and low voltage complexes; the P waves were bifid.

The diagnosis of constriction was confirmed at cardiac catheterisation, similar diastolic pressures being found in all four cardiac chambers.

At thoracotomy pericardial constriction was found to be associated with extensive calcification, particularly anteriorly and over the left ventricle. Pericardiectomy was performed, and immediately a more dynamic ventricular contraction was noted.

Histological examination of the excised pericardium showed chronic inflammation and dense hyaline

fibrosis. The patient recovered well and lost all signs of constriction.

Discussion

Constrictive pericarditis after penetrating cardiac trauma is rare. Paul Wood (1961) in a review of chronic constrictive pericarditis commented that in a series of 30 stab, bullet, or shrapnel wounds of the heart he had been unable to detect a single instance in which constriction followed. McKusick *et al* (1955), however, reported two cases and reviewed three others they were able to trace.

Calcification in our patient seems to have developed within ten months of the single episode of haemoptysis, when it is reasonable to assume the nail was still in lung tissue. No clinical signs of constriction were noted at that stage, emphasising the point that pericardial calcification does not necessarily imply constriction.

The constriction that follows trauma is not caused by the presence of intrapericardial blood alone. Wilson *et al* (1962) showed that blood injected into the pericardial cavity of dogs did not clot and was reabsorbed in three to six days, 26% as whole blood cells and the rest as a haemolysed fraction. Though lipids alone can produce constriction (Ehrenhaft and Taber, 1952), there is evidence that trauma to the pericardium or myocardium is needed as well (Sbokos *et al*, 1972). A possible explanation is that myocardial damage produces clot, which is necessary for constriction to occur. One interesting point in our case is the widespread pericardial involvement in such a localised injury.

References

Ehrenhaft, J L, and Taber, R E (1952). Haemopericardium and constrictive pericarditis. *Journal of Thoracic Surgery*, **24**, 355–368.

McKusick, V A, Kay, J H, and Isaacs, J P (1955). Constrictive pericarditis following traumatic hemopericardium. *Annals of Surgery*, **142**, 97–103.

Rasaretnam, R, and Paul, ATS (1975). Constrictive pericarditis following mild non-penetrating trauma. *Australian and New Zealand Journal of Medicine*, **5**, 57–62.

Sbokos, C G, Kontaxis, A, and Skalkeas, G (1972). Traumatic haemopericardium and chronic constrictive pericarditis. Presented at the International Cardiovascular Congress, European Chapter. June 28–30.

Wilson, J L, Saleh, S S, Yacoubian, H D, Dagher, I K, and Mnaymneh, L G (1962). The absorption of blood from the pericardium. *Journal of Thoracic and Cardiovascular Surgery*, **44**, 785–792.

Wood, P (1961). Chronic constrictive pericarditis. *American Journal of Cardiology*, **7**, 48–61.

Requests for reprints to: Dr Neville Conway, Wessex Cardiac and Thoracic Centre, Western Hospital, Oakley Road, Southampton SO9 4WQ.