Thorax (1966), **21**, 305.

Left atrial tamponade

M. H. YACOUB, W. P. CLELAND, AND C. W. DEAL

From the Brompton and London Chest Hospitals

Post-operative cardiac tamponade is a surgical emergency. The outcome depends on early diagnosis. The clinical picture of tamponade due to inflammatory pericardial effusion has been described by Stewart, Crane, and Deitrick (1938), Besterman and Thomas (1953), and Burch and Phillips (1962). Rapid accumulation of fluid or clot in the pericardium, as in post-operative tamponade, produces its own diagnostic problems, signs, and differential diagnosis. Unlike fluid blood, clot can produce local compression of the heart. We believe that some cases showing atypical features may be due to compression of the left atrium by clot behind the heart. Two cases are described, and the differential diagnosis of post-operative tamponade is discussed.

CASE REPORTS

CASE 1 A woman aged 53 was admitted to the London Chest Hospital on 19 October 1964. She was found to have severe aortic regurgitation and moderate mitral stenosis. The pulmonary vascular resistance was normal (1 unit).

On 25 November 1964 mitral valvotomy and aortic valve replacement were performed satisfactorily. The coronary arteries were perfused almost throughout the period of aortic occlusion.

In the immediate post-operative period the patient did not regain full consciousness, so it was decided to continue artificial ventilation through an endotracheal tube. At that time she was not anoxic and she had a good capillary circulation; the venous pressure was 6 cm. of water above the sternal angle. The blood-pressure was 140/90 mm. Hg. About 300 ml. drained from the tubes in the first hour, after which the rate slowed down. One hour after operation peripheral cyanosis appeared and the urinary output diminished. The blood-pressure and venous pressure remained unchanged and the pulse was not paradoxical. The arterial oxygen saturation at that time was 95% and the venous oxygen saturation was 35%. The arterial *p*H was 7·32 and the arterial P_{CO_2} was 38 mm. Hg. Fourteen hours later her condition was unchanged. Artificial ventilation was discontinued and bronchial secretions necessitated bronchoscopic aspiration. Although she was breathing adequately she remained confused and had peripheral cyanosis and a poor urinary output. She had a normal arterial oxygen saturation and arterial P_{CO_2}, but there was a moderate degree of metabolic acidosis, and the mixed venous oxygen saturation remained low. Thirty-six hours after operation the level of consciousness deteriorated, the peripheral cyanosis was profound, and she stopped secreting urine. The arterial oxygen saturation was 96% and the venous oxygen saturation was 30%. The arterial *p*H was 7·3 in spite of repeated doses of sodium bicarbonate intravenously. The blood-pressure was 120/80 mm. Hg and the venous pressure was 6 cm. of water. As she had all the signs of low cardiac output and there were bilateral basal crepitations, tracheostomy was performed and she was artificially ventilated. She did not improve and 20 minutes later she had a cardiac arrest. External cardiac massage and two attempts at external defibrillation failed. Exploration of the chest showed a big clot lying behind the left atrium with a small amount of fluid blood in the pericardium. Internal massage for a few minutes resulted in spontaneous reversion to sinus rhythm. She maintained a systolic blood pressure of 150 mm. Hg. The venous pressure was 5 cm. of water. The central venous oxygen saturation was 70% and this high level was maintained for the next few days. She regained full consciousness 12 hours after the episode of cardiac arrest. She remained anuric and died 15 days later from uraemia in spite of peritoneal dialysis.

COMMENT This patient developed the signs of a low cardiac output one hour after operation; this was corrected by evacuation of a clot from behind the left atrium. Cardiac tamponade was not suspected as there was no rise of venous pressure or pulse rate; the blood-pressure was maintained until very late and the pulse was not paradoxical.

CASE 2 A girl aged 3½ years was admitted to the Brompton Hospital on 29 May 1963. She had a ventricular septal defect with a small left-to-right shunt. The right ventricular pressure was 20/0 mm. Hg, and the pulmonary artery pressure was 20/10 mm. Hg.

A repair of the defect was performed on 7 June 1963. A small infracristal defect was closed by direct suture through a transverse right ventriculotomy. The total bypass time was 44 minutes and the aorta was clamped for two short periods of 1·5 min. each at a body temperature of 31° C.

Post-operatively she was initially well with pink, warm extremities and a good urinary flow. The blood-pressure was 110/80 mm. Hg, and the venous pressure was 6–8 cm. of water above the sternal angle; the foot veins looked empty. Two hours later she developed signs of a low cardiac output, namely deterioration in the level of consciousness, peripheral cyanosis, and poor urinary output; the central venous oxygen saturation was 45%. The arterial and venous pressures remained unchanged. One hour later the blood-pressure fell to 75 mm. Hg and the venous pressure was 6 cm. of water with a sharp Y descent (diastolic dip). A diagnosis of cardiac tamponade was made due to the moderately raised venous pressure in spite of the fact that the jugular venous pressure showed a diastolic dip. Thoracotomy revealed no blood or clot in the pericardium or mediastinum. A rapid infusion of 250 ml. of blood resulted in a marked improvement of the circulation. This improvement was maintained for 12 hours, after which she developed the signs of a critically low cardiac output. The venous pressure was 7 cm. of water above the sternal angle, the central venous oxygen saturation was 12%, and the venous *p*H was 7·21. The absence of a paradoxical pulse and of a rise of the venous pressure were taken as evidence against a diagnosis of cardiac tamponade. Repeated blood transfusions over a period of four hours resulted in no improvement at all. The arterial blood-pressure was 75 mm. Hg and the rhythm changed to complete atrioventricular dissociation for the first time since operation. Re-exploration of the mediastinum showed a big clot behind the left atrium. Evacuation of the clot resulted in an immediate improvement of the circulation with a rise of the blood-pressure to 120 mm. Hg. The improvement was maintained for a few hours, after which she rapidly deteriorated and died.

COMMENT This is another example of cardiac tamponade produced by compression of the left atrium by a clot in the oblique sinus. The critically low cardiac output for four hours resulted in severe myocardial damage evidenced by complete heart block and final heart failure

DISCUSSION

Cardiac tamponade is a condition of low cardiac output due to impairment of cardiac filling by external compression of the heart. This may develop slowly as in cases of pericardial effusion or constrictive pericarditis. Rapidly developing tamponade is caused by acute haemopericardium (Cooley, Dunn, Brockman, and DeBakey 1955), or acute mediastinal haematoma (Endress, 1953; Vincent, Roche, and Michel, 1953; Laforet, 1955; Al-Naaman, 1959). After cardiac operations tamponade may be caused by an accumulation of liquid blood or clot in the pericardium; the latter can produce localized compression of the heart. Other possible causes of post-operative cardiac tamponade include acute mediastinal haematoma and compression of the heart by the pericardium in cases of acute dilatation of the heart due to failure or diastolic overload of one of the ventricles produced by the creation of valve regurgitation. Acute tamponade may occur after open-heart operations due to the increased bleeding tendency observed after cardiopulmonary bypass (Callaghan, Despres, and Benvenuto, 1961; Riker, DeBoer, Baffes, Grana, and Potts, 1962; McLean, Morris, and Stirling, 1963; Tice, Reed, Clauss, and Worth, 1963; Bentall, Smith, Al Omeri, Melrose, and Allwork, 1964). Patients suffering from congenital heart disease, especially those with cyanosis, tend to bleed more (Favre-Gilly, Bret, and Borel-Milhet, 1951; Hartmann, 1952; Bentall *et al.*, 1964) and so are more liable to develop post-operative tamponade. Children are more commonly affected due to the small available space and the comparatively large thymus.

Cohnheim (1889) found that the injection of oil into the pericardium of dogs produced a rise in venous pressure. Starling (1897) pointed out that compression of the heart interferes with its function as a pump, and so arterial and venous pressures approach an identical (and therefore 'flowless') level. These findings were confirmed both in experimental animals (Lewis, 1908; Kuno, 1917; Cooper, Stead, and Warren, 1944; Post, 1951) and in patients with pericardial effusion (Caughey, 1937; Stewart *et al.*, 1938; Besterman and Thomas, 1953). Stewart *et al.* (1938) found that there is a linear correlation between cardiac output and systemic venous pressure in cardiac tamponade produced by pericardial effusion. They concluded that the rise in systemic venous pressure can be taken as a measure of the decrease in cardiac output. According to all these workers, tamponade is caused mainly by interference with the flow of blood into the right side of the heart. Experimental studies in animals have shown that in tamponade there is a rise of left atrial pressure (Nerlich, 1951) as well as of pulmonary venous and pulmonary capillary pressures (Metcalfe, Woodbury, Richards, and Burwell, 1952). Nerlich (1951) stressed the importance of the diminution of the veno-atrial gradient in tamponade. He found that this gradient diminishes more rapidly on the left side; this, he thought, is probably due to the higher elasticity coefficient of the left atrium. These findings show that left atrial compression

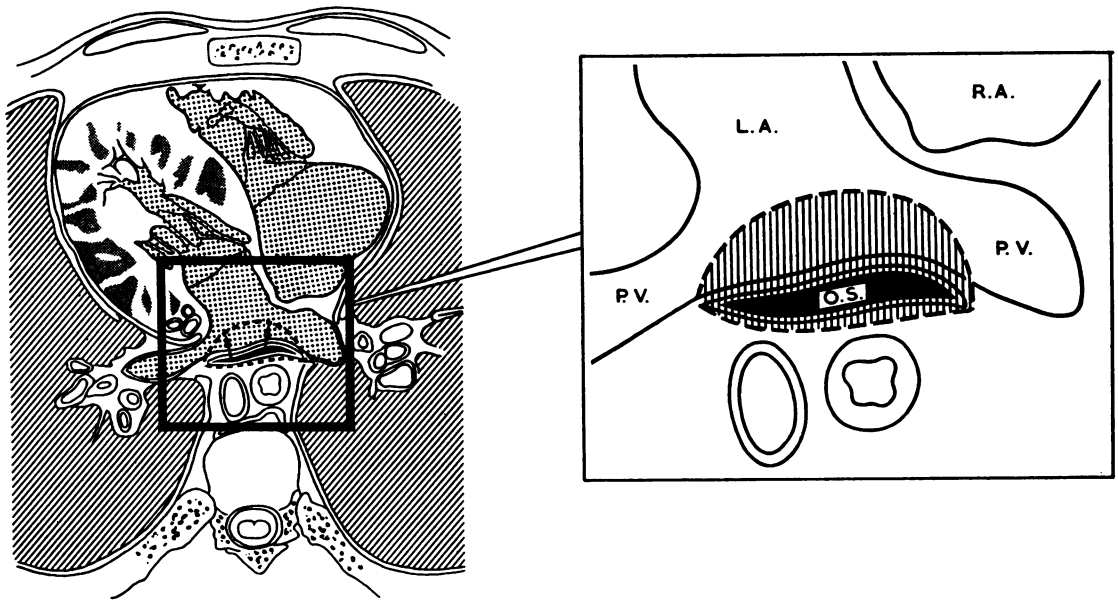

FIG. 1. *Transverse section of the chest at the level of the fourth thoracic vertebra showing compression of the left atrium and entrance of the pulmonary veins by a clot in the oblique sinus (O.S.). L.A., left atrium; R.A., right atrium; P.V., pulmonary vein.*

is an important feature of tamponade caused by a collection of fluid in the pericardial sac. As the left atrium is fixed to the parietal pericardium on both sides by the entry of the pulmonary veins, localized compression of that chamber by clot in the oblique sinus is rendered possible, especially as fluid blood or clot can readily become encysted in this situation (Fig. 1). The cases described here are examples of this special type of tamponade. They presented with signs of a low cardiac output and with no rise in the systemic venous pressure. The absence of a paradoxical pulse in these cases is probably due to the fact that the parietal pericardium is not under tension ; this sign is thought to be due to the descent of the diaphragm stretching the already tense pericardium and producing a further rise of the intrapericardial pressure (Wood, 1956).

Although our patients showed signs of a low cardiac output, the blood pressure was maintained until late. Stewart *et al.* (1938) showed that in man there is no significant change in the arterial blood pressure with the rise in cardiac output produced by aspiration of pericardial effusion. Nerlich (1951) described three phases of progressive experimental tamponade in dogs. The first stage was characterized by a low cardiac output and a normal arterial blood pressure ; in stage 2 there was a precipitous decline of blood pressure following additional small increments of pericardial fluid ; in stage 3 the blood pressure was critically low.

It is very important to differentiate post-operative tamponade from circulatory failure due to other causes, since tamponade is an eminently curable condition. In cases with a raised systemic venous pressure the wave form of the jugular venous pressure or a central venous pressure tracing helps to differentiate heart failure from tamponade (Gibson, 1965). In heart failure there is a steep Y descent (diastolic dip) (Fig. 2) which is caused by rapid filling of the right ventricle and is usually accompanied by a third heart sound which is due to the same cause. In tamponade due to generalized compression of the heart, the intrapericardial pressure falls during the ventricular ejection phase, and this causes a rapid X^1 descent (systolic dip) (Fig. 3). When heart failure and tamponade co-exist the venous pressure shows a systolic as well as a diastolic dip (Fig. 4). The electrocardiogram (E.C.G.) may be of some help in differentiating the two conditions. Total electrical alternans has been described in some cases of cardiac tamponade and is thought to be diagnostic (Traut, 1950 ; McGregor and Baskind, 1955 ; Colvin, 1958 ; Goley and Schwartz, 1958 ; Littmann

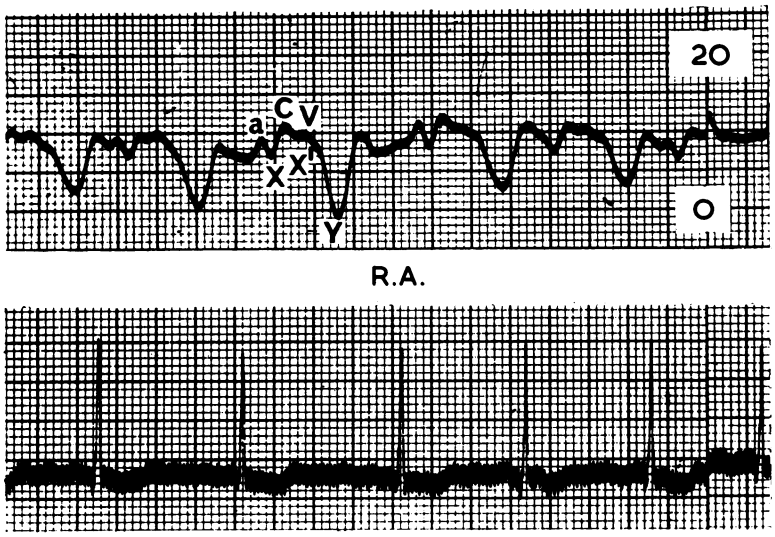

FIG. 2. *Right atrial pressure tracing in a case of heart failure showing the prominent 'Y' descent (diastolic dip).*

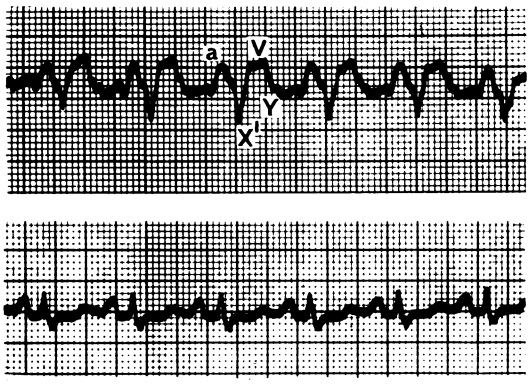

FIG. 3. *Right atrial pressure in a patient with tamponade showing the prominent X^1 descent (systolic dip).*

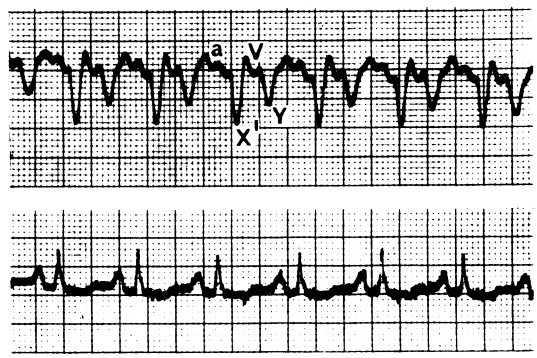

FIG. 4. *Right atrial pressure in a patient with heart failure and tamponade showing both X^1 and Y descents (systolic and diastolic dips).*

and Spodick, 1958 ; Ström, 1959 ; Curren, 1961 ; Lawrence and Cronin, 1963). This differs from the usual type of electrical alternans, which classically occurs with myocardial disease, in that it involves both the P and QRS vectors, causing a shift of both vectors in the same direction every second beat. The alternans is not pronounced in all leads and may only appear in one or more of the chest leads. The cause of these changes is not known : the most probable explanation is that they are secondary to anatomical changes in the cardiac position due to an increased freedom of movement (Littmann and Spodick, 1958 ; Lawrence and Cronin, 1963). London and London (1962) studied the E.C.G. of eight necropsied cases of acute haemopericardium secondary to rupture of the heart or aorta and found that all had high peaked T waves in the precordial leads regardless of the shape of the T wave before the onset of haemorrhage. They thought that this might be due to the effect of potassium released by haemolysis on the subepicardial layer or alternatively due to compression of the subendocardial layers.

It is here stressed that in patients with a severe post-operative low cardiac output without an obvious cause, such as uncorrected valve lesions, hypovolaemia or high pulmonary vascular resistance, tamponade should be excluded even if the

venous pressure is not significantly raised. A fall in arterial blood pressure can be a late sign. The oblique sinus is a potential space which can be shut off from the rest of the pericardium and it should be adequately drained.

SUMMARY

Two cases of cardiac tamponade due to left atrial compression presenting with signs of a low cardiac output without a rise of venous pressure are described. The differential diagnosis of post-operative tamponade is discussed. Drainage of the oblique sinus is stressed.

We should like to thank Mr. J. R. Belcher for his permission to publish the details of case 1 and Dr. M. Honey for his help. Our thanks also are due to the Medical Art Department of the Royal Marsden Hospital for the preparation of the diagrams and photographs.

REFERENCES

Al-Naaman, Y. D. (1959). Acute compression of the mediastinum by a hematoma. *Amer. J. Surg.*, **98**, 735.

Bentall, H. H., Smith, B., Al Omeri, M., Melrose, D. G., and Allwork, S. (1964). Blood-loss after cardiopulmonary bypass. *Lancet*, **2**, 277.

Besterman, E. M. M., and Thomas, G. T. (1953). Radiological diagnosis of rheumatic pericardial effusion. *Brit. Heart J.*, **15**, 113.

Burch, G. E., and Phillips, J. H. (1962). Methods in the diagnostic differentiation of myocardial dilatation from pericardial effusion. *Amer. Heart J.*, **64**, 266.

Callaghan, J. C., Despres, J. P., and Benvenuto, R. (1961). A study of the causes of 60 deaths following total cardiopulmonary bypass. *J. thorac. Surg.*, **42**, 489.

Caughey, J. L. Jr. (1937). A case of pericarditis with effusion: studies of venous pressure changes. *Bull. N.Y. Acad. Med.*, **13**, 1.

Cohnheim, J. (1889). *Lectures on General Pathology*, translated by A. B. McKee, Vol. 1, p. 26. New Sydenham Society, London.

Colvin, J. (1958). Electrical alternans: case report and comments on the literature. *Amer. Heart J.*, **55**, 513.

Cooley, D. A., Dunn, J. R., Brockman, H. L., and DeBakey, M. E. (1955). Treatment of penetrating wounds of the heart: experimental and clinical observations. *Surgery*, **37**, 882.

Cooper, F. W. Jr., Stead, E. A. Jr., and Warren, J. V. (1944). The beneficial effect of intravenous infusions in acute pericardial tamponade. *Ann. Surg.*, **120**, 822.

Curren, R. L. (1961). Electrical alternans in association with haemorrhagic pericardial effusion. *Amer. J. Cardiol.*, **8**, 453.

Endress, Z. F. (1953). Traumatic mediastinal hematoma. Report of two cases. *Amer. J. Roentgenol.*, **70**, 576.

Favre-Gilly, J. Bret, J., and Borel-Milhet, J. (1951). Un trouble inattendu de la coagulation dans la maladie bleue: l'hypoprothrombinémie; son importance en clinique et dans le problème de l'hémostase au cours des opérations de Blalock et de Potts. *Sang*, **22**, 278.

Gibson, R. V. (1965). Personal communication.

Goley, A. F., and Schwartz, R. (1958). Disappearance of electrical alternans following pericardiocentesis. *Arch. intern. Med.*, **101**, 577.

Hartmann, R. C. (1952). A hemorrhagic disorder occurring in patients with cyanotic congenital heart disease. *Bull. Johns Hopk. Hosp.*, **91**, 49.

Kuno, Y. (1917). The mechanical effect of fluid in the pericardium on the function of the heart. *J. Physiol. (Lond.)*, **51**, 221.

Laforet, E. G. (1955). Traumatic hemomediastinum. *J. thorac. Surg.*, **29**, 597.

Lawrence, L. T., and Cronin, J. F. (1963). Electrical alternans and pericardial tamponade. *Arch. intern. Med.*, **112**, 415.

Lewis, T. (1908). Studies of the relationship between respiration and blood-pressure. Part 1. The effect of changes of intrapericardial pressure on aortic pressure. *J. Physiol. (Lond.)*, **37**, 213.

Littmann, D., and Spodick, D. H. (1958). Total electrical alternation in pericardial disease. *Circulation*, **17**, 912.

London, R. E., and London, S. B. (1962). The electrocardiographic sign of acute hemopericardium. *Ibid.*, **25**, 780.

McGregor, M., and Baskind, E. (1955). Electric alternans in pericardial effusion. *Ibid.*, **11**, 837.

McLean, J. A., Morris, K. N., and Stirling, G. R. (1963). Direct blood-transfusion in severe haemorrhage after heart surgery, *Lancet*, **1**, 27.

Metcalfe, J., Woodbury, J. W., Richards, V., and Burwell, C. S. (1952). Studies in experimental pericardial tamponade. Effects on intravascular pressures and cardiac output. *Circulation*, **5**, 518.

Nerlich, W. E. (1951). Determinants of impairment of cardiac filling during progressive pericardial effusion. *Ibid.*, **3**, 377.

Post, R. S. (1951). Decrease of cardiac output by acute pericardial effusion and its effect on renal hemodynamics and electrolyte excretion. *Amer. J. Physiol.*, **165**, 278.

Riker, W. L., DeBoer, A., Baffes, T. G., Grana, L., and Potts, W. J. (1962). Experiences with extracorporeal circulation. *Surg. Gynec. Obstet.*, **115**, 757.

Starling, E. H. (1897). Some points in the pathology of heart disease. *Lancet*, **1**, 652.

Stewart, H. J., Crane, N. F., and Deitrick, J. E. (1938). Studies of the circulation in pericardial effusion. *Amer. Heart J.*, **16**, 189.

Ström, O. (1959). Hemopericardium with electric alternans. *Acta med. scand.*, **164**, 367.

Tice, D. A., Reed, G. E., Clauss, R. H., and Worth, M. H. (1963). Haemorrhage due to fibrinolysis occurring with open heart operations. 43rd Annual Meeting of the American Association for Thoracic Surgery.

Traut, E. F. (1950). Alternans: Report of a case associated with acute pericarditis. *Postgrad. Med.*, **8**, 439.

Vincent, E., Roche, L., and Michel, R. (1953). L'hémomédiastin traumatique. *Ann. méd. lég.*, **33**, 107.

Wood, P. (1956). *Diseases of the Heart and Circulation*, 2nd ed. Eyre and Spottiswoode, London.