with oxygen. This can be judged accurately enough if the total bag capacity is not more than about 2 litres.

Stage 1—Preparation of the Bag.—The bag of oxygen is rebreathed until an increase is noted in the depth or rate of respiration. About 10 seconds' more rebreathing is encouraged and the first stage is terminated.

Stage 2—Fine Adjustment.—After at least two minutes the prepared bag is rebreathed for 20 seconds and its PCO_2 is measured. After a further two-minutes rest (about the time required to analyse the previous sample of gas), the 20-second rebreathing period is repeated. This procedure is continued until the bag PCO_2 remains unaltered by the rebreathing. This value is taken as the mixed venous PCO_2 .

Summary

The problems encountered in the measurement of mixed venous PCO_2 by the rebreathing method are discussed. An adaptation of the method is described which allows the use of small-bag volumes without distressing the patient. The time for the initial preparation of the bag is not fixed but depends

upon the observation of a change in ventilation which is thought to be a CO_2 response. The subsequent 20-second rebreathing periods are repeated to ensure more accurate results. The application of the method to a group of patients is described and the findings are discussed.

I am extremely grateful to Dr. E. J. M. Campbell for introducing me to the study of PCO_2 and for his helpful criticism and advice during this investigation. Professor J. G. Scadding, Dr. J. R. Bignall, and Dr. K. M. Citron kindly allowed me to study patients under their care and read the manuscript. Dr. L. H. Capel provided facilities for studies with a rapid CO_2 analyser and Dr. E. N. O'Brien for estimation of arterial PCO_2 .

REFERENCES

- Campbell, E. J. M. (1960). *Brit. med. J.*, 1, 457.
 — and Howell, J. B. L. (1960a). *pH and Blood Gas Measurements: Methods and Interpretations*, edited by R. Woolmer. Churchill, London.
 — (1960b). *Brit. med. J.*, 1, 458.
 — (1962). *Ibid.*, 2, 630.
 Collier, C. R. (1956). *J. appl. Physiol.*, 9, 25.
 Hackney, J. D., Sears, C. H., and Collier, C. R. (1958). *Ibid.*, 12, 425.
 Plesch, J. (1909). *Z. exp. Path. Ther.*, 6, 484.

Dissection of the Aorta with Pericardial Tamponade: Successful Relief of Tamponade

PETER SLEIGHT,* M.D., M.R.C.P.

Brit. med. J., 1965, 1, 1165–1167

Dissection of the aorta is fatal in over 75% of cases (De Bakey, Henly, Cooley, Crawford, and Morris, 1961). Rupture of the aorta is the commonest cause of death, and the usual site of rupture is into the pericardium. This caused death by cardiac tamponade in 86% of Hirst, Johns, and Kine's (1958) collected series of 505 cases, and in 89% of the 106 cases in Shennan's (1934) classic report. It was the cause of post-operative death in 3 out of 72 cases of aortic dissection treated surgically by De Bakey *et al.* (1961). Tamponade has usually been a rapidly fatal catastrophe revealed only at post-mortem examination; occasionally this has been diagnosed before death (Lownsbury, 1935; Lowell, 1940; Ellis, 1947), but relief of tamponade by aspiration has seldom been reported.

Campbell (1946) described survival for three months after aspiration of a haemothorax following rupture of a dissecting aneurysm into the left pleura. The following case is reported as the diagnosis of cardiac tamponade due to haemopericardium was made clinically, and the patient was relieved surgically, with dramatic improvement in her condition.

Case Report

A 79-year-old woman was on her way to play bridge when she developed severe central chest pain while struggling to open a lift door. She collapsed and was taken to the casualty department of this hospital. The pain was very severe and was also felt in the left side of the abdomen. Later she gave a history of a previous attack of severe chest pain one week earlier; this earlier chest

pain had been situated behind the xiphisternum and through to the back, but not below the diaphragm.

On first examination in the casualty department she was in severe pain and complaining of thirst; she was shocked and sweating, but had a blood-pressure of 110/70. The pulse rate was 96. The jugular venous pressure (J.V.P.) was raised to the angle of the jaw. The heart sounds were normal; no murmurs were heard. The arterial pulses were all present and equal. The liver was palpable with an enlarged right lobe, but the abdomen was soft and normal bowel sounds were heard. Neurological examination was negative.

An electrocardiogram (E.C.G.) (Fig. 1) showed minimal ST segment depression and T-wave inversion in leads I, II, AVL, V4–V7, but was otherwise normal. The systolic blood-pressure rapidly fell to 40–50 mm. Hg, but the pulse rate remained inappropriately low with a sinus rate of 80/min. The J.V.P. was measured (with an intravenous catheter) at +8 cm. of saline above the sternal angle.

She was given morphine (15 mg.) intravenously and continuous oxygen by mask. She became stuporous, with Cheyne-Stokes

FIG 1.—E.C.G showing non-specific T inversion V4–V6.

* From the Cardiac Department, St. George's Hospital, London. Present address: Department of the Regius Professor of Medicine, Radcliffe Infirmary, Oxford.

respiration. A differential diagnosis was made of aortic dissection or cardiac infarction, but the lack of E.C.G. changes did not correlate with the severity of the shock. She remained in this state of hypotension and stupor with a pulse rate of 80 for three hours. Noradrenaline (10–20 $\mu\text{g./min.}$) was given by intravenous infusion, and the blood-pressure was easily restored to normal. The noradrenaline had a striking effect on the pulse pressure but little effect on the heart rate. There was little evidence of peripheral vasoconstriction, and the forearm and cervical veins remained over-filled. The lungs were free from crepitations. At this time pulsus paradoxus was noted, the radial pulse almost disappearing with the onset of inspiration. After a further four hours the patient was conscious and rational, but dependent on the noradrenaline infusion for maintenance of a systolic blood-pressure of 90 mm. Hg, which fell to 40–50 mm. Hg if the drip was stopped. At this time arterial-pressure tracings (Fig. 2, courtesy of Professor A. C. Dornhorst) confirmed that there was true diminution of pulse amplitude on inspiration, and not “dislocation” of the pulse-pressure as seen in large intrathoracic pressure swings due to respiratory obstruction or left ventricular failure (Dornhorst, Howard, and Leathart, 1952). The venous pressure at this time was +20 cm. of saline.

FIG. 2.—Upper trace=blood-pressure (radial artery). Lower trace=pneumogram, inspiration downwards; calibration of pressure trace=0.100 mm. Hg. Record A, after noradrenaline infusion stopped. Record B, during infusion of noradrenaline, 20 $\mu\text{g./min.}$ intravenously, showing increase in pulse-pressure and blood-pressure. Both records show marked pulsus paradoxus with an inspiratory decrease in pulse amplitude.

It was noted that her circulation could also be maintained, but with a higher heart rate, by intravenous isoprenaline (10 $\mu\text{g./min.}$) in place of noradrenaline (Fig. 3); again the pressure fell when the isoprenaline infusion was stopped. The reason for the inappropriately slow pulse was not clear, but Professor Dornhorst suggested that the aortic dissection might have stimulated aortic baroreceptors, and reflexly increased the vagal tone. Atropine (1.2 mg.)

FIG. 3.—Chart of blood-pressure and pulse rate during infusion of noradrenaline (20 $\mu\text{g./min.}$) and isoprenaline (10 $\mu\text{g./min.}$) and after the intravenous injection of 1.2 mg. of atropine.

was given intravenously, and the pulse rate rose to 96/min. It was then possible to stop the noradrenaline infusion for the first time, the systolic blood-pressure remaining at 90 mm. Hg.

A chest x-ray film (portable, supine, Fig. 4) showed widening of the upper mediastinal shadow, with irregular angulation of the ascending aorta and calcification in the aortic knuckle.

FIG. 4.—Portable chest x-ray film taken on admission, showing angulation of the ascending aorta and a wide upper mediastinal shadow.

The patient was transferred to the theatre, and by an epigastric incision, under local anaesthesia, Mr. Charles Drew opened the pericardium and removed 200 ml. of fluid blood and a small amount of clotted blood. The blood-pressure immediately rose to 180 mm. Hg and then gradually settled to 140/90. On her return to the ward it was noted that the venous pressure was normal. The patient made an uninterrupted recovery and showed no neurological or renal sequelae from the three to four hours of profound hypotension. There were no signs of aortic regurgitation. She was discharged from hospital, symptom-free, four weeks after admission.

Discussion

The patient's history was classically that of aortic dissection, but the intrapericardial rupture was perhaps unusually slow in its progression. Severe shock occurs in 25–30% of cases of aortic dissection, but even in these cases the blood-pressure is usually over 100 mm. Hg, and a low blood-pressure should suggest the possibility of cardiac tamponade due to haemopericardium (Hirst *et al.*, 1958). As in this reported case, the electrocardiographic changes in aortic dissection are often minimal, usually showing mild ST and T-wave changes, interpreted as left ventricular “strain,” though when the dissection involves the coronary arteries the diagnosis is made more difficult by the changes of myocardial infarction on the E.C.G. (Bourne and Mills, 1946; Levinson, Edmeades, and Griffith, 1950). Levinson *et al.* describe 4 out of their series of 58 cases of aortic dissection where the E.C.G. showed ST segment elevation compatible with a diagnosis of pericarditis; in these cases the dissection was slowly progressive, and at post-mortem examination a fibrinous pericarditis was present.

The dramatic improvement with noradrenaline infusion was unexpected, and it is possible that the positive inotropic effect of the drug caused a decrease in heart size and thus partially

relieved the tamponade. In support of this was the action of isoprenaline, which has no vasoconstrictor action but which also maintained the pressure. Although it might be thought that an increase in arterial pressure would predispose to further bleeding, a high arterial pressure after the dissection does not appear to influence the prognosis (Hirst *et al.*, 1958).

After the initial period of shock the peripheral vessels did not appear unduly constricted, despite the low arterial pressure; it is possible that aortic baroreceptor stimulation by the dissection led to peripheral vasodilatation in addition to inappropriate vagal slowing of the heart, and thus aided tissue survival, despite the low perfusion pressure, by preventing extreme vasoconstriction. Bradycardia is unusual in dissection of the aorta and occurred in only 4% of the cases of Hirst *et al.* (1958). The increase in pulse rate caused by atropine suggests that a vagal mechanism was involved.

In Sherman's series of 143 recent dissections death occurred within 12 hours in 77 patients. The cause of death in 89% of these acute cases was pericardial tamponade. If anything surgical is to be attempted in this grave condition (and the results of De Bakey *et al.* (1961) are encouraging in this respect), then a close watch should be kept for signs of pericardial tamponade, and facilities should be available for its relief. In the present case a direct surgical approach under local anaesthesia was considered preferable to attempted aspiration. This had the advantage of unequivocal confirmation of the diagnosis, and it was possible to leave a drain in position in case of further bleeding.

Summary

A case of dissecting aneurysm presenting with acute circulatory collapse due to pericardial tamponade is described. The diagnosis was made clinically and confirmed by arterial pressure recordings. The beneficial effects of noradrenaline and isoprenaline are reported, and the uncomplicated recovery from three to four hours' severe hypotension is discussed. Surgical relief of the pericardial tamponade appeared to be life-saving.

I am grateful to Dr. Alastair Hunter and Mr. Charles Drew for permission to publish this report, to Professor A. C. Dornhorst for the arterial-pressure records and much helpful advice, to Dr. R. G. D. Lowe for suggesting the trial of isoprenaline, and to Dr. A. G. Leatham for criticizing the manuscript.

REFERENCES

- Bourne, G., and Mills, P. J. W. (1946). *Brit. Heart J.*, 8, 181.
Campbell, M. (1946). *Ibid.*, 8, 200.
De Bakey, M. E., Henly, W. S., Cooley, D. A., Crawford, E. S., and Morris, G. C. (1961). *Circulation*, 24, 290.
Dornhorst, A. C., Howard, P., and Leatham, G. L. (1952). *Lancet*, 1, 746.
Ellis, G. M. (1947). *Amer. Heart J.*, 33, 860.
Hirst, A. E., Johns, V. J., and Kime, S. W. (1958). *Medicine (Baltimore)*, 37, 217.
Levinson, D. C., Edmeades, D. T., and Griffith, G. C. (1950). *Circulation*, 1, 360.
Lowell, W. H., jun. (1940). *Conn. med. J.*, 4, 724.
Lownsbury, J. B. (1935). *Yale J. Biol. Med.*, 7, 209.
Shennan, T. (1934). *Spec. Rep. Ser. med. Res. Coun. (Lond.)*, No. 193.

Preliminary Communications

Mediastinoscopy

Brit. med. J., 1965, 1, 1167-1169

"Les ganglions bronchiques sont comme le miroir du poumon"—Parrot (1876).

Many of the pathological processes inside the chest involve the lymph nodes. The lymphatic drainage of the lungs passes from the bronchopulmonary nodes to those in the hilum of the lung and from there to the tracheobronchial nodes. It then proceeds upwards to the scalene nodes, via the paratracheal lymph nodes. Daniels (1949) showed the diagnostic value of scalene-node biopsy. Mediastinoscopy, as first practised by Carlens (1959), enables us to obtain a biopsy very much nearer to the lung root than a scalene-node biopsy. This procedure avoids many unnecessary thoracotomies in cases of bronchial carcinoma, and for this reason has gained considerable popularity on the Continent (Palva and Viikari, 1961; Reynders, 1963). The purpose of this paper is to report my experience with this method.

SURGICAL TECHNIQUE

The operation is carried out under general anaesthesia. A 1½-in. (4-cm.) incision is made in the suprasternal notch, as for a low tracheostomy. The dissection is carried down to the pre-tracheal fascia, which is incised. With blunt finger dissection a tunnel is made downwards into the mediastinum, keeping the dorsum of the finger all the time in contact with the anterior

surface of the trachea. The pleura is separated on either side.

The plane of dissection is depicted in the diagram by an arrow. The pulsation of the innominate artery in front and the arch of the aorta on the left can be felt quite distinctly. The diagram also shows why anterior mediastinal tumours are not really accessible by this method, as they lie anterior to the large vessels.

The origin of each main bronchus can be palpated by feeling for the superior tracheobronchial angles. Much information

Diagram showing the relation of the mediastinal structures to plane of dissection (arrow) and the close association of the vena azygos to the right superior tracheobronchial nodes.