ANAESTHESIA FOR MITRAL VALVOTOMY COMPLICATED BY HYPOTENSION DUE TO PERICARDIAL EFFUSION

Report of Two Cases

BY

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SUMMARY

Two cases are described in which unexpected arterial hypotension complicated the management of anaesthesia for surgical correction of mitral stenosis. In each case dramatic improvement followed the release of a blood-stained pericardial effusion. There is often an increased amount of clear pericardial fluid in cases of this type, but this is normally insufficient to cause cardiac embarrassment. Intrapericardial haemorrhage secondary to cardiac catheterization may be a cause of hypotension during anaesthesia. It is important to recognize and anticipate the risks associated with pre-operative catheterization and needle puncture of the heart.

Cardiac tamponade is an uncommon complication of anaesthesia, outside accident surgery. Two cases are described in which undiagnosed fluid in the pericardial sac led to difficulty during anaesthesia, prior to its release on incision of the pericardium.

CASE REPORTS

Case 1. Mrs. L. W., aged 57 years:

This lady, with mitral stenosis following rheumatic fever in childhood, had been seen for 3 years in the Outpatient Department following the onset of mild symptoms about 10 years ago. She was admitted for review and cardiac catheterization. Right heart catheterization was performed on August 2, 1963, but, as the effects of sedation were profound and the patient became hypotensive, the procedure was abandoned and simultaneous left heart catheterization was not undertaken. Soon afterwards atrial fibrillation developed and the patient became drowsy. The anti-coagulants were stopped thirteen days prior to mitral valvotomy which was performed on September 9. After premedication with pethidine 75 mg and hyoscine 0.3 mg the patient was awake and co-operative though drowsy. Anaesthesia was induced with thiopentone 100 mg, and endotracheal intubation with a No. 9.0 oral cuffed tube followed the injection of suxamethonium 50 mg and topical spray of 4 per cent lignocaine. On the return of spontaneous respiration, anaesthesia was maintained with nitrous oxide, oxygen and halothane (0.5—1 per cent). The systolic blood pressure at this time was 80—90 mm Hg and this fell to 70 mm Hg when intermittent positive pressure ventilation was started with the aid of hyperventilation but without muscle relaxants.

Case 2. Mrs. J. W., aged 42 years:

She was first seen in the Outpatient Department in February 1963. Mitral valve disease had been previously diagnosed and showed no improvement. The patient was still able to do a full day's work. In September 1963 a sudden attack of giddiness preceded a left hemiplegia and a speech difficulty. Cerebral embolism was diagnosed and the patient was digitalized and anticoagulants given. Recovery was complete. On November 6 a left-sided cardiac catheterization was carried out—one week after anticoagulants had been stopped. The left atrium was approached by the posterior percutaneous route and the left ventricle catheterized through the mitral orifice with a PE60 catheter. There were no complications and preparations were made for mitral valvotomy on November 20.

Premedication with papaveretum 20 mg and hyoscine 0.3 mg was satisfactory and anaesthesia was induced with thiopenitone 200 mg. Intubation followed suxamethonium 50 mg using a No. 9.0 cuffed oral endotracheal tube. Anaesthesia was maintained with nitrous oxide, oxygen and halothane, 0.5 per cent. The systolic blood pressure at this time was 80—90 mm Hg and this fell to 70 mm Hg when intermittent positive pressure ventilation was started with the aid of hyperventilation but without muscle relaxants.
Methylamphetamine on two occasions had little effect and there was no rise in the pressure until an incision had been made in the pericardial sac and an unsuspected collection of fluid released. There was about 200 ml of dark bloodstained fluid and as in the previous case, the initial thought was that a major vessel or a heart cavity had been damaged but this was not the case. The operation thereafter followed an uneventful course.

**DISCUSSION**

In both these cases the main anaesthetic problem was one of hypotension prior to incision of the pericardial sac, particularly after the onset of intermittent positive pressure ventilation.

Hypotension in such cases may be due to any of several causes. The mitral valve lesion may be severe enough to prevent a rise in cardiac output sufficient to maintain an adequate systemic blood pressure in the presence of vasodilatation associated with general anaesthesia. Neither of these patients was greatly incapacitated by her disease and the cardiac reserve should have allowed adequate compensation to occur.

Secondly, the choice of anaesthetic agents and technique may be responsible. Halothane is known to cause hypotension and also to be a myocardial depressant drug. However, in neither case was a high concentration used and there was no response to vasopressor drugs. This anaesthetic technique has been used frequently on similar cases without any significant change in systolic blood pressure. Intermittent positive pressure ventilation is a necessary part of the anaesthetic technique for open chest surgery and, though it may cause some fall in venous return, such a fall in systemic pressure is uncommon. Until the chest is opened, spontaneous respiration may be continued or, alternatively, intermittent positive pressure ventilation, supplemented by a negative phase during expiration in order to lower the mean intrapulmonary pressure and to improve the venous return, may be used.

In neither of these cases could the profound and unresponsive fall in blood pressure be accounted for until release of the unsuspected pericardial effusion. Pre-operatively the condition of both patients had been good and there were no symptoms to suggest the presence of an effusion, though at operation, in the first case, the pericardial sac was apparently tense.

Pericardial effusions under 500 ml are difficult to detect (Davidson, 1962). It is assumed that this may not be the case under anaesthesia and that smaller amounts of fluid may be sufficient to prevent adequate diastolic filling of the ventricles, thus diminishing the stroke and cardiac output with a fall in systemic pressure.

Other factors possibly contributing to the hypotension, included vasodilatation occurring under general anaesthesia, and the rise in mean intrapulmonary pressure associated with IPPR, directly impeding venous return and exerting direct pressure on the heart, thus adding to the problem of ventricular filling (Brecher, 1956). Neither effusion was large, but the release of this fluid was responsible for the immediate improvement and must have been the main complicating factor.

Pre-operative assessment of mitral valve disease can be most fully accomplished with the help of cardiac catheterization. In Case 1, only a right heart catheterization was accomplished as the procedure had to be abandoned prior to the left atrial puncture. Right heart catheterization is achieved through the right basilic vein in the antecubital space and, therefore, the pericardial sac is not crossed. It was thought in this case that the bleeding must have occurred into the pericardial sac from damage to the coronary sinus. This was only a retrospective diagnosis as no suspicion of bleeding was ever present and the chest radiograph never showed any increase of heart size.

In the second case the left atrium was punctured by the posterior percutaneous route, passing across the pericardial sac (Bjork, Malmström and Uggla, 1953). This was obviously sufficient reason for the haemopericardium. Brock, Milstein and Ross (1956) described the percutaneous route for direct left ventricular puncture, and in this paper a number of unpleasant complications following left atrial puncture are noted. These included haemopericardium which was in some cases sufficient to cause a significant increase in heart size. This complication was also noted by Brockenbrough et al. (1961), following left ventricular puncture and was sufficient on one occasion to require immediate aspiration.

Both these patients received anticoagulants, although it is unlikely that they were responsible for the degree of haemorrhage because in the first
case treatment was not started until after the catheterization, and in the second case treatment was stopped one week before the catheterization.

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REFERENCES

DEUX CAS D'HYPOTENSION PAR SUITE D'EFFUSION DANS LE PERICARDE COMPLIQUANT L'ANESTHÉSIE PENDANT DES VALVOTOMIES MITRALES

SOMMAIRE
Description de deux cas dans lesquels une hypotension artérielle inattendue compliqua le maintien "sous contrôle" de l'anesthésie pendant valvotomie pour sténose mitrale. Dans chacun des deux cas une amélioration dramatique suivit l'écoulement du liquide sanguinolent d'un épanchement pericardial. Dans ce type de cas il existe souvent une quantité accrue de liquide pericardial limpidé, mais sa quantité est normalement insuffisante pour causer des embarras cardiaques. Il est possible que l'hémorragie intrapericardiale après un sondage intracardial serait la cause première d'hypotension pendant l'anesthésie. Il est important pour l'anesthésiste de connaître (et d'en prévoir les risques éventuels) des sondages intracardiaux — et surtout, de savoir si un sondage vient d'être fait avant une intervention intracardiale.

ANÄSTHESIE FÜR DIE VALVOTOMIE DER MITRALKLAPPE ERSCHWERT DURCH HYPOTONIE INFOLGE EINES PERIKARDIALEN ERGUSSES

ZUSAMMENFASSUNG