Closure of pericardium after open heart surgery

A way to prevent postoperative cardiac tamponade

P. Nandi, J. S. M. Leung, and K. L. Cheung

From Hong Kong University Surgical Unit, Queen Mary Hospital, Hong Kong

Between July 1968 and December 1975, 821 patients underwent open heart operations. In 596 cases the pericardium was left open and in 225 the pericardium was closed. Forty-one patients in the open pericardium group required reoperation and 23 of these had tamponade. Four patients in the closed pericardium group had reoperation but there was not a single case of tamponade. In most cases that required reoperation the bleeding was from extrapericardial sources. Absence of tamponade in the closed pericardium group can be explained by the fact that blood from extrapericardial sources of bleeding cannot collect round the heart because the pericardium is closed. Thus closure of pericardium helps to prevent tamponade.

Reoperations some months or years after the original operation are technically easier and less hazardous if the pericardium has been closed because the closed pericardium prevents the heart from becoming adherent to the back of sternum and also because there are fewer adhesions in the pericardial cavity.

Cardiac tamponade is a serious complication after open heart surgery and much attention has been paid to this problem in the past decade or so. The usual practice has been to leave the pericardium widely open in communication with one of the pleural cavities, so that blood from the pericardial sac and the mediastinum could drain into the pleural cavity. This practice of leaving the pericardium open has not decreased the incidence of postoperative cardiac tamponade. We find that by closing the pericardium after open heart surgery the incidence of postoperative tamponade can be drastically reduced if not completely eliminated. In recent years another great disadvantage of leaving the pericardium open has come to light. This is evident when such a patient requires reoperation some months or years after the original operation. Reoperation is technically more difficult because of adhesions. The right ventricle and the aorta become adherent to the back of the sternum and while reopening a previous sternotomy the right ventricle or the aorta may be inadvertently entered causing fatal haemorrhage. A closed pericardium will prevent the aorta and the right ventricle from adhering to the sternum. A closed pericardial cavity is also clean and consequently adhesion formation is much less.

Received 21 June 1976.

Subjects and methods

Between July 1968 and December 1975 a total of 821 patients underwent open heart operation in Hong Kong University Surgical Unit. The age of the patients ranged from 8½ months to 61 years. There were 527 cases of congenital heart disease and 278 cases of acquired heart disease (Table 1). In 596 (72.6%) cases the pericardium was left wide open and in 225 (27.4%) cases the pericardium was closed.

TABLE 1 *Types of original operation*

<i>Operation</i>	<i>No.</i>	<i>One reoperation</i>	<i>Second reoperation</i>
Tetralogy of Fallot	194	20	1
Atrial septal defect	143	6	2
Ventricular septal defect	120	5	
Pulmonary stenosis	36	1	
ASD and PS	29	2	
ASD and VSD	5	1	1
Mitral valve replacement	148	1	
Double valve replacement	47	5	1
Aortic valve replacement	36	3	
Open valvotomy (aortic and mitral)	47	1	
Others	16		
Total	821	45	5

Operative technique

In all cases the chest was opened through a vertical sternotomy avoiding the pleural spaces as and when possible. The anterior part of the pericardium was opened from the level of the left innominate vein to the diaphragm, with an extension of the incision to the left along the diaphragm for a short distance. This makes it easier to lift the heart for insertion of the left ventricular vent. The thymus and the fat in the upper mediastinum were carefully dissected. The edges of the pericardium were lifted to the level of the sternum with the aid of three stay sutures on each side and the sternal spreader was applied inside the pericardial edges. This lifts the heart up and also probably prevents the pericardium from shrinking (Cunningham *et al.*, 1975). At the end of operation heparin was neutralized with protamine and an intravenous injection of epsilon amino-caproic acid (EACA) was given. All cardiac incisions and sites of aortic cannulation and aortotomy were carefully checked for adequate haemostasis.

(a) Closed pericardium group The pericardium was closed with interrupted silk sutures inserted about 1.5 cm apart, leaving the lower inch of the pericardium open. Two drains were then inserted in the mediastinum, an upper reaching the level of the back of the manubrium sterni and a lower lying behind the lower part of the body of the sternum. Mayon plastic surgical tubing was used for drainage (internal diameter 0.9 cm for adults, and 0.6 cm for children). No drain was inserted inside the pericardial cavity; our experience showed that this was not necessary, as the lower mediastinal tube drains the pericardial cavity adequately. If the pleura was inadvertently opened, it was sutured after thorough suction of the pleural cavity and the top end of the upper mediastinal drain was left in the upper pleura instead of behind the manubrium sterni.

(b) Open pericardium group In these patients the pericardium was not sutured and the right pleura was left open in communication with the

TABLE 3 Indications for reoperation

Indication	No.	Pericardium
Cardiac tamponade	23	Open
Excessive blood loss	18	Open
Persistent bleeding	4	Closed

pericardial cavity; one mediastinal and one pleural drain was used.

Vacuum suction (10 to 15 cm water) was applied to the drains separately in the intensive care unit. In addition the drains were milked every few minutes.

Results

Forty-five (5.5%) of the total of 821 cases required reoperation for bleeding or tamponade or both; 41 of these cases were in the open pericardium group and 5 of these required second reoperation; only 4 cases in the closed pericardium group required reoperation. In the open pericardium group, 23 patients showed evidence of cardiac tamponade. In contrast to this there was not a single case of cardiac tamponade in the group of patients in whom the pericardium was closed (Table 2).

Indications for reoperation (Tables 3 and 4)

Cardiac tamponade Twenty-three patients were reopened with a diagnosis of cardiac tamponade and this was confirmed at operation. All these cases were in the open pericardium group. All but one case developed tamponade in the early postoperative period. There was only one case of delayed tamponade in the series (Table 4) and this was related to anticoagulant therapy. The clinical picture of tamponade was one of 'low output failure' with pale cold extremities, falling blood pressure, tachycardia, rising central venous pressure, and a fall in urine output. The decrease in urine output was a very valuable sign and appeared early. There was no case of cardiac tamponade among patients in whom the pericardium was closed.

Continued excessive bleeding Eighteen patients were reopened because of continued excessive blood loss and in all these the pericardium was left wide

TABLE 2 Cases requiring reoperation

Pericardium	No.	No. of cases with tamponade
Open 596 (72.59%)	41 (6.87%)	23 (3.85%)
Closed 225	4 (1.77%)	0
Congenital 154		
Acquired 71		
Total 821	45 (5.48%)	23

TABLE 4 Time of reoperation

Pericardium	2-6 h	7-12 h	13-24 h	25-48 h	72 h	1 wk
Open	9	13	7	10	6	1
Closed				3	1	

TABLE 5 Amount of drainage in reoperated patients

Pericardium	Total volume to time of reoperation (mean)	Amount per hour (average)
Open	1225 ml	224 ml
Closed	850 ml	36 ml

open. The average blood loss in these patients was 224 ml per hour. The details of the amount of drainage are given in Table 5.

Persistent bleeding Four patients required reoperation for persistent bleeding but the amount of drainage was not excessive and all of them were in the closed pericardium group. At operation little or no blood could be found in the pericardial cavity. In one case there was bleeding from the entry site of the upper mediastinal drain and in the others there was bleeding around the sternal wires and the thymus.

Amount of drainage (Table 5)

The comparison of the amount of drainage in the two groups requiring reoperation is given in Table 5. The amount of drainage was much greater in the open pericardium group than in the closed pericardium group.

Source of bleeding at reoperation (Table 6)

The common sites of bleeding were in the anterior mediastinum where there may be numerous small oozing points. One frequent source of bleeding was the back of the sternum where the self-retaining sternal spreader lay in contact with it; the spreader stripped the pleura and other soft tissues including the periosteum from the back of the sternum and the costal cartilages. The cases with bleeding from the cardiac incisions or the aorta were mostly encountered earlier in the series.

TABLE 6 Source of bleeding at reoperation

Source	No.
Extrapericardial	
Mediastinal pleura, sternum, thymus, pericardium, drain sites	32 (71.11%)
Intrapericardial	
Atrial incisions, right ventriculotomy, aortic incisions	9 (20%)
No definite source	4 (8.88%)

TABLE 7 Results of reoperation

	Pericardium	No.
Mortality	Open	8 (17.77%)
	Closed	0
Tracheostomy	Open	10 (22.22%)
	Closed	0
Wound infection	Open	4 (8.88%)
	Closed	1

Results of reoperation (Table 7)

Eight (17.8%) patients died between 1 and 3 weeks after operation, but in none was death directly related to reoperation. No patient in the closed pericardium group died after reoperation. Ten patients required tracheostomy and were in the open pericardium group. There were cases of wound infection after reoperation in both groups. Craddock, Logan, and Fadali (1968) reported a death rate of 36 per cent in those requiring reoperation, and in the series of Nelson, Jensen, and Smoot (1969) the mortality was 7 per cent.

Discussion

In this series of 821 cases, 23 patients developed postoperative cardiac tamponade. This is an incidence of 2.8 per cent and compares favourably with the results of Craddock *et al.* (1968) and Nelson *et al.* (1969), who reported incidences of 5.7 and 3.4 per cent, respectively. Cardiac tamponade is a surgical emergency and the outcome depends on early diagnosis and quick decompression. The diagnosis of postoperative cardiac tamponade is often delayed because of the difficulty of differentiating it from other causes of low cardiac output, especially in the absence of reliable radiological signs. The use of 'juxtaposed epicardial-pericardial clips' (Meckstroth and Cattaneo, 1974) in diagnosing postoperative tamponade is of limited value. We found it difficult to obtain standard positions for the clips for all patients; in addition their position tends to vary with phases of respiration and with cardiac systole and diastole. Furthermore, erosion of the metal clip into the heart remains a potential danger. Measurement of pericardial or mediastinal pressure to diagnose tamponade (Frater, 1970) also has its limitations. The best solution, therefore, appears to be not to allow postoperative cardiac tamponade to develop at all; closure of the pericardium after open heart surgery seems to be able to achieve this. This becomes evident from comparison of the two groups in our series. There was no incidence of tamponade in the 225 cases in whom the pericardium was closed, as

opposed to 23 cases of tamponade in the 596 cases in whom the pericardium was left wide open in communication with the right pleural cavity. Cunningham *et al.* (1975) have recently reported a similar experience. In their series of 100 cases in whom the pericardium was closed after open heart surgery there was not a single case of cardiac tamponade. As shown in Table 6, in most cases the postoperative bleeding occurred from extrapericardial sources. This was also the experience of others (Bentall *et al.*, 1964; Craddock *et al.*, 1968; Nelson *et al.*, 1969; Cunningham *et al.*, 1975). In most reported series haemopericardium was the cause of tamponade and in all of these patients the pericardium was left wide open. Blood from extrapericardial sources collects around the heart, and while most of it drains out some invariably clots causing tamponade. This has also been the cause of left atrial tamponade (Yacoub, Cleland, and Deal, 1966), and delayed tamponade (Nelson *et al.*, 1969; Ellison and Kirsh, 1974). The anterior surface of the closed pericardium acts as a collecting receptacle, where blood from the extrapericardial sources of bleeding collects and is immediately drained through mediastinal drainage tubes, thus preventing the collection of blood around the heart. After decannulation and neutralization of heparin and while carrying out haemostasis, blood quickly collects in the pericardial sac. After this has been removed by suction, there is rarely any bleeding from any of the cardiac or aortic sites; the blood usually gravitates from the back of the sternum, the mediastinal pleura, or the pericardium. This to a lesser degree may continue even after careful haemostasis and closure of the sternum. For several hours after open heart surgery the patients lie supine, and with the pericardium open blood will collect in the posterior part of the pericardial sac; before all can be drained out by suction some will no doubt clot. This seems the probable explanation for the high incidence of postoperative tamponade in the open pericardium group in this series and others (Craddock *et al.*, 1968; Hill *et al.*, 1969; Nelson *et al.*, 1969; Engelman *et al.*, 1970). In cases with open pericardium who required reoperation for continued excessive bleeding we found clots around the heart. Similar experience was also reported by others (Craddock *et al.*, 1968; Hill *et al.*, 1969; Engelman *et al.*, 1970). Surprisingly no blood was found in the pericardial cavity of the patients in the closed pericardium group who required reoperation for persistent bleeding. Cunningham *et al.* (1975) reported a similar experience in a group of 100 patients, 5 of whom required reoperation for excessive blood loss (2 litres or more). Therefore, it appears that postoperative cardiac tamponade can

be prevented by closure of the pericardium after open heart surgery. This theory is completely opposed to the usual practice of leaving the pericardium wide open in the attempt to avoid tamponade. Before closing the pericardium, it is important to make absolutely certain that none of the cardiac or aortic wounds is bleeding.

There are other advantages in closing the pericardium. For example, the right ventricle is protected from any damage by the sharp posterior edge of the split sternum during closed heart massage in the postoperative period, especially in patients with dextrocardia. Another advantage of closing the pericardium is apparent in cases that require reoperation some months or years later. As we have shown, when the pericardium is closed after open heart surgery the pericardial cavity is fairly clean. Because of this adhesion formation is much less in these cases. This is well supported by the experimental work of Cliff, Grobety, and Ryan (1973). Their conclusion was that dense adhesions form when blood collects in the injured pericardium as so often happens in patients in whom the pericardium is left open after open heart surgery. Repeat operations may be required in certain patients for a variety of reasons some months or years after the original operation and are technically more difficult when adhesions are present. The right ventricle and the aorta may become adherent to the back of the sternum, and while a previous sternotomy is being reopened the right ventricle or aorta may rupture (Hysten *et al.*, 1970). Reoperation would be technically easier and less hazardous if the pericardium had been closed, because the heart is then not adherent to the back of the sternum and there are fewer adhesions in the pericardial cavity.

We thank Professor G. B. Ong, Head of the Department of Surgery, for his advice in preparing this paper.

References

- Bentall, H. H., Smith, B., Omeri, M. A., Melrose, D. G., and Allwork, S. (1964). Blood-loss after cardiopulmonary bypass. *Lancet*, **2**, 277.
- Cliff, W. J., Grobety, J., and Ryan, G. B. (1973). Postoperative pericardial adhesions—the role of mild serosal injury and spilled blood. *Journal of Thoracic and Cardiovascular Surgery*, **65**, 744.
- Craddock, D. R., Logan, A., and Fadali, A. (1968). Reoperation for haemorrhage following cardiopulmonary bypass. *British Journal of Surgery*, **55**, 17.
- Cunningham, J. N., Jr., Spencer, F. C., Zeff, R., Williams, C. D., Cukingnan, R., and Mullin, M. (1975). Influence of primary closure of the pericardium after openheart surgery on the frequency of tamponade, postcardiotomy syndrome, and pulmonary complications. *Journal of Thoracic and Cardiovascular Surgery*, **70**, 119.
- Ellison, L. H., and Kirsh, M. M. (1974). Delayed mediastinal tamponade after open heart surgery. *Chest*, **65**, 64.

- Engelman, R. M., Spencer, F. C., Reed, G. E., and Tice, D. A. (1970). Cardiac tamponade following open-heart surgery. *Circulation*, **41**, Suppl. II, 165.
- Frater, R. W. M. (1970). Intrapericardial pressure and pericardial tamponade in cardiac surgery. *Annals of Thoracic Surgery*, **10**, 563.
- Hill, J. D., Johnson, D. C., Miller, G. E., Jr., Kerth, W. J., and Gerbode, F. (1969). Latent mediastinal tamponade after open-heart surgery. *Archives of Surgery*, **99**, 808.
- Hylan, J. C., Kloster, F. E., Starr, A., and Griswold, H. E. (1970). Aortic ball variance: diagnosis and treatment. *Annals of Internal Medicine*, **72**, 1.
- Meckstroth, C. V., and Cattaneo, S. M. (1974). Juxtaposed epicardial-pericardial clips: an adjunct to the differential diagnosis of hemopericardium, pericardial tamponade and primary low cardiac output syndrome after open-heart surgery. *Journal of Thoracic and Cardiovascular Surgery*, **68**, 447.
- Nelson, R. M., Jenson, C. B., and Smoot, W. M., III (1969). Pericardial tamponade following open-heart surgery. *Journal of Thoracic and Cardiovascular Surgery*, **58**, 510.
- Yacoub, M. H., Cleland, W. P., and Deal, C. W. (1966). Left atrial tamponade. *Thorax*, **21**, 305.

Requests for reprints to Dr. P. Nandi, Department of Surgery, Queen Mary Hospital, Kong Kong.