Serial Semi-invasive Hemodynamic Assessment following Pericardiectomy for Chronic Constrictive Pericarditis

Abstract

Objectives: This study was designed to prospectively investigate the effects of pericardiectomy via median sternotomy on intra- and postoperative hemodynamics by a new semi-invasive device (Flotrac/VigileoTM monitor) using arterial pressure waveform analysis. Patients and Methods: Thirty consecutive patients aged 15 to 55 years (mean+SD, 31.73 + 13.53 years), who had undergone total pericardiectomy via median sternotomy underwent serial hemodynamic evaluation. FlotracTM Sensor – derived stroke volume, stroke volume variation, systemic vascular resistance index (SVRI), cardiac index and right atrial pressure were measured just before and after pericardiectomy, at 12 hours, 24 hours, 48 hours, 72 hours and at discharge postoperatively. Results: Majority of patients (73.33%) exhibited statistically significant reduction of right atrial pressure and SVRI along with improvement in cardiac index and oxygen delivery in the immediate and late postoperative period. However, the stroke volume and stroke volume variation did not increase proportionately on completion of surgery. Patients with low cardiac output syndrome exhibited persistently high central venous pressure with reduced cardiac index and echocardiographically abnormal diastolic filling characteristics. Conclusions: We conclude that there is early normalization of hemodynamics following pericardiectomy via median sternotomy and the adequacy of pericardiectomy can be accurately assessed by the new semi-invasive arterial pressure waveform analysis device. Stroke volume variation is a non-predictor of fluid requirement during and after pericardiectomy.

Keywords: Chronic constrictive pericarditis, hemodynamic assessment, pericardiectomy

Introduction

With the current operative techniques perioperative management. and the safety of pericardiectomy with long-term improvement in functional class has been well documented in the majority of patients undergoing the procedure.^[1-4] Nevertheless, some patients fail to improve after pericardiectomy.[5-7] In our previous study on 395 patients undergoing pericardiectomy between 1985 and 2004, we had demonstrated that total pericardiectomy is associated with lower mortality, less postoperative low cardiac output (CO) syndrome, early normalization of hemodynamics, and better long-term survival compared with partial pericardiectomy. In patients with constrictive pericarditis, this is more easily approached through median sternotomy.^[8,9]

Despite the wide use of cardiac catheterization in the preoperative evaluation of patients with constrictive pericarditis, there have been only scattered reports of postoperative hemodynamic studies.^[1,5-8] Moreover, the data obtained and conclusions reached have varied considerably, from the demonstration of normal findings^[1,2,5,6] to the conclusion that restitution of normal cardiac function seldom occurs.^[7] The postoperative evaluation of these patients has been complicated by disparate opinions regarding the proper incision for optimal surgical exposure and the extent of decortication necessary for adequate cardiac decompression.[1-10]

Assessment of patient's CO and other hemodynamic parameters usually involves placement of a pulmonary artery (PA) catheter and performing thermodilution assessment.^[11,12] This is an invasive procedure requiring balloon flotation of a catheter through the right heart and an elaborate protocol of intermittent PA injection for thermodilution calculation. Second, surgical manipulation of the heart during pericardiectomy can make thermodilution, PA, central venous

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monitoring, and transesophageal echocardiography unreliable as monitors.^[11,12]

The emergence of new modalities of noninvasive hemodynamic monitoring has opened up newer frontiers for evaluation of such patients without the risk of invasive cardiac catheterization.^[13-15] The FloTracTM sensor and VigileoTM monitor system introduced by Edwards Lifesciences allows continuous measurement of CO without requiring thermodilution or dye dilution. It bases its calculations on arterial waveform characteristics in conjunction with patients demographic data and does not require external calibration.^[16]

This prospective, nonrandomized study was performed to (i) determine serially, semi-invasively the stroke volume (SV), SV variation (SVV), systemic vascular resistance index (SVRI), CO, and tissue oxygen delivery and (ii) monitor the right atrial (RA) pressure serially in patients undergoing total pericardiectomy through median sternotomy for constrictive pericarditis using FloTracTM sensor and VigileoTM monitor (Edwards Lifesciences, Irvine, CA, USA).^[16]

Patients and Methods

Between January 2009 and December 2014, thirty consecutive patients undergoing pericardiectomy at All India Institute of Medical Sciences, New Delhi, operated by a single surgeon (UKC) were included in the study. All thirty patients (25 males) underwent total pericardiectomy through median sternotomy. Age ranged from 15 to 55 years (mean \pm standard deviation [SD], 31.73 \pm 13.53 years). Duration of symptoms ranged from 5 to 48 months (mean \pm SD, 16.33 \pm 11.9 months). Preoperatively, 18 (60%) patients were in New York Heart Association (NYHA) III and 12 (40%) patients were in NYHA IV.

All patients had chronic heart failure (CHF) as the predominant symptoms. Eighteen (60%) patients had chest pain, 2 (6.6%) patients had evidence of cardiac tamponade, and supraventricular arrhythmias were found in 11 (36.6%) patients. Ninety percent had distended jugular veins, 80% ascites, 86% hepatomegaly, 45% pleural effusion, and 30% had pulsus paradoxus [Table 1].

Two out of thirty patients with pericardial effusion required tapping and steroid therapy. All patients with tuberculosis (n = 29) received multidrug therapy (isoniazid, rifampicin, ethambutol, and pyrazinamide) for an initial 3 months followed by triple drug therapy to continue postoperatively for a period of 9 months. Before operation, all patients were on digitalis and diuretics. One patient (3.3%) was diagnosed to have recurrent chronic constrictive pericarditis (CCP) 5 years after pericardiectomy through left anterolateral thoracotomy at another center.

The etiology was considered tubercular if the histopathology of the excised pericardium showed granulomas, caseation,

Table 1: Demographic detail	s of the study group
Profile	n (%)
Number of patients	30
Males	25 (83.3)
Age in years, mean±SD,	31.73±13.53, 32.0 (15-55)
median (range)	
Age distribution (years)	
10-20	7 (23.3)
21-30	8 (26.6)
31-40	7 (23.3)
41-50	4 (13.3)
51-60	4 (13.3)
Body weight, kg (mean±SD)	51.93±9.32
Duration of illness in months,	16.33±11.9, 12.0 (5-48)
mean±SD, median (range)	
Orthopnea	12 (40)
Distended jugular veins in sitting	28 (93.3)
position	
Congestive heart failure	30 (100)
Preoperative-NYHA functional class	
NYHA III	18 (60)
NYHA IV	12 (40)
Peripheral edema	28 (93.3)
Pleural effusion	9 (30)
Hepatomegaly	26 (86.6)
Ascites	24 (80)
Renal derangement	11 (36.6)
Hepatic derangement	10 (33.3)
Pulsus paradoxus	9 (30)
ECG changes	30 (100)
Pericardial calcification on X-ray	9 (30)
Tuberculosis on culture or histology	29 (96.6)

NYHA: New York Heart Association Classification, SD: Standard deviation, ECG: Electrocardiography

and giant cells (n = 29, 96.6%) or if the fluid and debris removed at surgery were positive for acid-fast bacilli (n = 6). A history of pulmonary tuberculosis and lymph node tuberculosis was present in ten (33.3%) and four (13.3%) patients, respectively.

Laboratory investigation showed elevated erythrocyte sedimentation rate (range, 40–90 mm at 1 h) in 28 (93.3%), renal dysfunction (serum creatinine >2.0 mg/dl) in 11 (36.6%), and hyperbilirubinemia in 10 (33.3%) patients. Chest roentgenogram revealed pericardial calcification (n = 9, 30%), pleural effusion (n = 9, 30%), and pulmonary infiltrates (n = 3, 10%). The calcification was distributed over the anterior and inferior surfaces of the heart in 9 (30%) patients and all around the heart like a cocoon in 2 (6.6%) patients.

Electrocardiogram demonstrated sinus rhythm (n = 30), low voltage QRS complex (n = 30), flattening or T-wave inversion (n = 29), and premature ventricular contraction (n = 3). Although these changes were present in the majority of patients, they appeared nonspecific. Echocardiography revealed pericardial thickness >3 mm (n = 30), inferior vena cava (IVC) dilatation (n = 30), RA enlargement (n = 30), abnormal septal motion (n = 26), >25% increase in mitral inflow velocity with expiration compared with inspiratory phase (n = 29), moderate mitral regurgitation (grade 2+, n = 11), and moderate tricuspid regurgitation (grade 2+, n = 9).

Preoperative cardiac catheterization was performed in 18 patients. The rest (n = 12) did not have catheterization because of their Class IV symptoms with renal dysfunction. All demonstrated the findings consistent with constrictive pericarditis: an elevated RA pressure (RAP), usually with a M- or W-shaped contour; an abnormally high right ventricular (RV) end-diastolic pressure with a characteristic dip-plateau diastolic configuration; equalization of end-diastolic pressure in all cardiac chambers; and a ratio of RV end-diastolic to RV systolic pressure >0.30.

Surgical techniques

All patients in this study underwent pericardiectomy through median sternotomy. After sternotomy, the thymus and pleural reflections were mobilized laterally to obtain a wide width of the pericardium. Both pleural cavities were widely opened to remove the pleural fluid and to identify the phrenic pedicles on either side.

An I-shaped incision was made in the midline over the pericardium up to the level of the PA superiorly and diaphragm inferiorly. The dissection of the pericardium of the heart was done using cautery until the fibrous pericardium along with its serous layer. When it is done properly, there is a clear visualization of the epicardial fat and the coronary arteries. Inability to visualize the coronaries indicates that the dissection plane is not deep enough.

It is important to set the cautery between 8 and 10 mV during the process of dissection to avoid cautery-induced ventricular fibrillation. Multiple silk stay sutures were then placed on the cut edges of the incised pericardium. The pericardium was initially divided at the bottom portion close to the diaphragmatic reflection over the right ventricle, and the lateral pericardial flap was raised superiorly and laterally. If calcified plaques or spicules penetrating the epicardium were present, these were left behind with islands of calcified pericardium, making numerous scores over the patch. Circumferential patches of calcified pericardium were crushed with a thick hemostat and/or bone cutter and were removed avoiding injury to the underlying vascular structures, coronaries, and phrenic nerves. We have not used cavitational ultrasonic surgical aspiration system for removal of calcium or nerve stimulator for identification of the phrenic nerve on any patient in this study.

The pericardium covering both ventricles, the great vessels, the venae cavae, and the RA was excised 1 cm anterior to the phrenic nerve on either side. The pericardium over the venae cavae and RA was resected last. The pericardial and pleural cavities were irrigated with normal saline. None sustained any injury to major vessels or cardiac chambers requiring cardiopulmonary bypass.

Serial semi-invasive hemodynamic monitoring

FloTrac[™] sensor and Vigileo[™] monitor, Edwards Lifesciences, Irvine, CA, USA; monitor was used to monitor hemodynamics semi-invasively starting from the time arterial and central venous lines were inserted in the operating theater to the time when the arterial lines were removed when shifting from the Intensive Care Unit (ICU). The readings were taken just after the insertion of arterial and central venous lines through the procedure till the time when the patient was shifted to cardiac ICU, at 12, 24, 48, and 72 h postoperatively.

Statistical analysis

Data were analyzed with SPSS software version 10.0 statistical package (SPSS, Chicago, IL, USA). Continuous and interval-related data are presented as the mean \pm SD for normally distributed variables, whereas median (range) for other variables. Categorical variables are presented as frequency distribution and percentages. Wilcoxon signed-rank test was performed to analyze statistically the difference from baseline values. P < 0.05 was considered statistically significant.

Results

Early results

There were two (6.6%) early deaths due to low CO syndrome, septicemia, and hepatorenal syndrome culminating in multisystem failure.

One patient had undergone pericardiectomy through left anterolateral thoracotomy at another center and presented with recurrent constrictive pericarditis. Preoperatively, she was in NYHA Class IV with CHF. The mean RAP decreased from 23 mmHg (preoperative) to 17 mmHg (postoperative). After surgery, there was a reduction of SV and cardiac index (CI) from a baseline of 36 mL and 2.4 L/min/m²–28 mL and 1.8 L/min/m², respectively. Despite using inotropes and vasodilators (dopamine, dobutamine, epinephrine, and milrinone), she continued to suffer from low CO and expired on the 6th postoperative day due to severe biventricular failure.

The second patient was in NYHA Class IV and had massive ascites, preoperative azotemia, and severe biventricular dysfunction. After total pericardiectomy, he developed low CO with hepatic failure in the immediate postoperative period. The mean RAP decreased from 28 mmHg (preoperative) to 18 mmHg (postoperative). Six liters of pleural and ascitic fluid was removed after surgery. The SVV decreased from 12% (preoperative) to 8% (postoperative). Despite using inotropes and vasodilators, the SV and CI decreased from a baseline of 49 mL and 2.7 L/min/m² (preoperative) to 29 mL and 1.8 L/min/m², respectively. Postoperative echocardiography revealed increased biventricular dimensions, abnormal diastolic filling pattern with raised RAP. Despite all resuscitative measures with inotropes and vasodilators, he expired on 9th postoperative day.

All patients were routinely started on dopamine $(4 \mu g/kg/min)$ to increase renal perfusion on operation table after completing excision of the thickened pericardium. Patients considered to have low output syndrome (n = 8)μg/kg/min), required dopamine (4 - 10)epinephrine (0.01–0.1 µg/kg/min), and milrinone (50 µg/kg intravenous bolus followed by 0.375–0.75 µg/kg/min) either isolated or in combination. Median duration of inotrope requirement was 4 days (range 2-7 days) in these patients. Patients with normal renal function were administered oral angiotensin-converting enzyme inhibitors before weaning from inotropic agents.

Echocardiographically, diastolic filling characteristics remained abnormal in eight (26.6%) patients of the study group in the immediate postoperative period, including the two patients expired after surgery. At closing interval, two (7.14%) of the survivors continued to have abnormal diastolic filling pattern on Doppler echocardiogram. All survivors were in normal sinus rhythm after pericardiectomy.

Postoperatively, digoxin, diuretics, and angiotensin-converting enzyme inhibitors were weaned at varying time intervals. There were no late deaths.

Follow-up

Survivors (n = 28) underwent clinical examination, electrocardiogram, and echocardiogram every 3 months. Follow-up was 100% complete (range 3–26 months). At their last follow-up, 27 (96.4%) patients were in NYHA Class I and 1 (3.6%) patient was in NYHA Class III. Reoperation was not required for any patient.

Data analyses and study interpretation of hemodynamic variables

The pertinent pre- and post-operative hemodynamic data are summarized in Tables 2, 3 and Figures 1-3.

- Postoperatively, there were no significant differences in heart rate, mean partial pressure of oxygen in blood, mean hemoglobin, mean pH, mean base deficit, mean blood sugar, and mean blood lactate levels as compared to the preoperative values
- The mean (\pm SD) systemic arterial blood pressure in the postoperative period was 86.5 \pm 11.23 mmHg as compared to the preoperative values of 85 \pm 12.9 mmHg and was not statistically significant
- Twenty-two (73.3%) patients exhibited a significant reduction of mean RAP from a median of 17 (range,



Figure 1: Changes in cardiac output, cardiac index, and stroke volume with stroke volume index of patients undergoing pericardiectomy in this study



Figure 2: Changes in systemic vascular resistance and index of patients undergoing pericardiectomy in this study



Figure 3: Changes in tissue oxygen delivery of patients undergoing pericardiectomy in this study

- 12–28) mmHg to a median of 5.5 (range, 4–16) mmHg in all patients (P < 0.05). Postoperatively, these patients had normal RAP with disappearance of M- or W-shaped contour and no gradients between RA and venae cavae. Eight (26.4%) patients had persistently high central venous pressure (CVP) ranging between 14 and 16 mmHg
- Preoperatively, the CI was subnormal in all patients. Following pericardiectomy, there was an instantaneous improvement of cardiac index (CI) on operation table.

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	Table 2: Cardiac h	hemodynamic parameters	measured semi-invasively	in patients undergoing per	ricardiectomy (n=30)	
Haemodynamic			Time of mea	surement		
parameters	Preoperative	Immediate postoperative (on operation table)	24 h postoperative	48 h postoperative	72 h postoperative	At discharge
Pulse rate (beat/min)	106±16.24 (82-140)	112±12.15 (90-133)	116±20.66 (87-162)	115 ± 19.51 (90-170)	115.92±19.21 (90-162)	107.08±15.87 (78-130)
Ρ		0.041	0.004	0.061	0.033	0.81
Mean arterial	85±12.9 (68-117)	79.6±18.8 (45-118)	84.92±13.35 (53-112)	89.23±14.17 (54-118)	88 ± 13.10 (54-110)	86.5±11.23 (54-95)
pressure (mmHg)						
Ρ		0.38	0.97	0.33	0.44	0.72
Central venous	Median 17 (12-28)	Median 12 (8-23)	Median 7 (4-17)	Median 7 (4-10)	Median 6 (4-8)	Median 6 (4-8)
pressure (mmHg)						
P		0.004	0.001	0.001	0.002	0.002
CO (L/min/m ²)	4.7±0.95 (3-6.6)	5.8±1.82 (3.1-8.6)	6.16 ± 1.30 (4-8.2)	6.65±1.60 (4.1-9.6)	11.9±19.2 (4.2-7.6)	6.8±1.32 (5.1-5.4)
Ρ		0.008	0.001	0.001	0.17	0.001
CI (L/min/m ²)	2.9±0.79 (1.5-4.2)	3.7±1.30 (1.9-6.2)	3.9±0.76 (2.8-5)	4.2±0.92 (2.6-5.6)	4.2±1.00 (2.5-6.1)	4.5±0.977 (3.1-6.4)
P		0.001	0.001	0.001	0.001	0.001
SV (mL/beat)	52.30±12.62 (31-72)	46±15.0 (24-70)	54.46±14.59 (28-92)	52±16.2 (29-88)	54.15±1875 (31-97)	56.08±18.30 (31-97)
Ρ		0.17	0.57	0.94	0.69	0.39
SVI (mL/beat/m ²)	35.07±10.02 (20-56)	32.92±9.71 (19-53)	34.76±8.91 (20-50)	$31.23\pm10.24~(19-48)$	32.46 ± 13.16 (19-66)	33.75±3.02 (19-86)
Р		0.49	0.90	0.17	0.36	0.66
SVV (%)	Median 18 (12-55)	Median 14 (11-42)	Median 11 (9-40)	Median 10 (8-34)	Median 10 (8-17)	Median 9.5 (7-17)
Ρ		0.83	0.88	0.18	0.11	0.09
SVR (dynes/s/cm ⁵)	1221.08±329.37 (730-1684)	950.08±265.92 (559-1684)	1033.66±3222.97 (385-1520)	1034.58±368.51 (356-1612)	1019.41±319.17 (556-1580)	1003 ± 335.93 (356-1580)
Р		0.001	0.001	0.010	0.008	0.002
SVRI (dynes/s/cm5/m2)	1762.167±678.89 (946-2826)	1410.83 ± 549.80 (680-2283)	1282.66±418.54 (551-2474)	1344.58±555.20 (542-2474)	1318.58 ± 504 (542-2146)	1225.45±462.18 (542-2063)
P		0.001	0.001	0.010	0.008	0.002
DO ₂ (mL/min)	841±246 (471-1266)	980.61±377 (422-1907)	1069.38±237.92 (748-1580)	1163.38±244 (817-1640)	1166.46±273.41 (753-1708)	1167.75±255.87 (860-1708)
P		0.20	0.009	0.001	0.001	0.001
DO ₂ I (mL/min/m ²)	Median 537 (280-699)	Median 635 (278-1386)	Median 734 (481-1362)	Median 803 (534-1214)	Median 803 (448-1418)	Median 804 (527-1418)
Ρ		0.17	0.006	0.002	0.002	0.003
Data are presented as I SVV: Stroke volume v	nean±SD (range). CO: Carc ariance, DO₂: Tissue oxyge	diac output, CI: Cardiac inde in delivery, DO ₂ I: Tissue oxy	ex, SVR: Systemic vascular ygen delivery index, SD: Sta	resistance, SVRI: Systemic indard deviation, SVI: Strok	vascular resistance index, se volume index	SV: Stroke volume,

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Variables			Time of mea	surement	3	
	Preoperative	Immediate postoperative (on operation table)	24 h postoperative	48 h postoperative	72 h postoperative	At discharge
Hemoglobin level (g/dl)	12.64±0.93 (10.9-14)	12.32±1.89 (7.8-15)	12.26±1.66 (8.9-14.9)	12.13±2.27 (8.4-15.2)	12.12±2.09 (8.4-15)	12.44±1.87 (8.7-15)
P		0.61	0.45	0.51	0.40	0.62
PO_2 (mmHg)	Median 181 (104-419)	Median 206 (136-406)	Median 178 (89-408)	Median 182 (88.7-484)	Median 182 (82.8-484)	Median 173 (82.8-484)
P		0.97	0.38	0.91	0.6	0.69
pH	7.39±0.5 (7.29-7.46)	7.34±0.10 (7.12-7.46)	7.41±0.006 (7.3-7.51)	7.41±0.051 (7.34-7.49)	7.42±0.037 (7.34-7.49)	7.41±0.047 (7.34-7.49)
P		0.08	0.32	0.22	0.14	0.40
Blood lactate (mmol/l)	Median 1.3 (0.7-2.5)	Median 1.3 (0.6-2.3)	Median 1.1 (0.7-3.4)	Median 1.2 (0.7-3.2)	Median 1.2 (0.7-2.4)	Median 1.1 (0.7-2.4)
P		0.91	0.50	0.32	0.75	0.93
Base deficit	-2.86±2.9 (-10.3-1.1)	−3.65±3.07 (−11.1-1)	0.0084±3.47 (-5.6-5.09)	-1.00±4.13 (-12.8-4.8)	0.59±2.9 (−4-6.6)	0.83±2.78 (-4-6.6)
P		0.388	0.021	0.177	0.001	0.001
Blood sugar (g/dl)	102.08±31.55 (58-159)	130.16±43.41 (70-204)	121.25±28.30 (86-189)	103.41±39.9 (61-195)	113.33±35.54 (61-186)	113.41±25.68 (80-180)
P		0.02	0.11	0.92	0.42	0.36
Data are presented as me	an±SD (range). SD: Standa	ard deviation, PO ₂ : Partial pre	ssure of oxygen in blood			

Twenty-two patients in the study group demonstrated statistically significant improvement in CI in all phases of measurement. The mean preoperative values were 2.9 ± 0.79 L/min/m² and the values at discharge were 4.5 ± 0.97 L/min/m² (P < 0.001). Eight (26.4%) patients had reduced CI ranging between 1.6 and 1.8 L/min/m²

- Immediately following surgery, the SVRI decreased from a mean preoperative value of $1762.17 \pm 678.89-1410.83 \pm 549.90$ dynes/s/cm⁵/m² (P = 0.001). The SVRI continued to decline over time to a mean value of 1225.45 ± 462.18 dynes/s/cm⁵/m² at discharge (P = 0.002)
- Preoperatively, the study group demonstrated a mean SV of 52.30 (±SD, 12.62 mL). Immediately following surgery, there was a reduction in SV in all patients of the study group. Although the mean SV demonstrated an increase over time to a mean value of 56.08 ± 18.30 , the degree of rise did not achieve statistical significance (P = 0.39)
- There was marked elevation of the SVV (median 15%, range 4%–55%) in all patients of the study group in the preoperative period and during the process of decortication. Subsequently, there was a reduction of SVV throughout the postoperative period in all patients of the study group.

Discussion

Assessment of cardiac performance is of paramount importance in the management of patients undergoing pericardiectomy for constrictive pericarditis.^[2,5-8] Despite the accuracy of thermodilution technique for measuring CO, it is invasive, and there is an unclear risk–benefit ratio.^[16,17] Recently, less invasive techniques such as transthoracic bioimpedance, pulse dye densitometry, LiDCO system, and PiCCO-system (Paulson SG) have been developed for hemodynamic assessment.^[11-14] However, the validity, practicability, and accuracy of these techniques are not uniform.^[14]

The Flotrac[™]/Vigileo[™] device calculates continuous CO from an arterial pressure waveform characteristics and does not require external calibration. Individual demographic data including height, weight, age, gender, and real-time arterial pressure waveform analysis are used to estimate arterial compliance.^[16]

As far as we are aware, there are no studies addressing specifically the usefulness of semi-invasive hemodynamic monitoring during surgery for CCP. As yet, there are no specific physiologic and/or hemodynamic criteria that can be used in deciding an optimal selection of exposure and extent of pericardial resection for a given patient.^[1-10]

The principal finding of this investigation includes the occurrence of low CO in 26.4% (n = 8) of patients in spite of performing total pericardiectomy through median sternotomy.

The second important finding of this investigation includes large SVV (>10%) in all patients in the operating room during decortication despite being intravascularly hypervolemic.

The third important finding was marked reduction of RAP and SVRI in the majority immediately after pericardiectomy.

The fourth major finding includes immediate improvement in CO and tissue oxygen delivery in the majority of patients following pericardiectomy.

The fifth major finding is the observation that despite decrease in RAP, SV resistance (SVR), and improvement in CO, the SV did not increase proportionately on completion of surgery. On the contrary, these parameters (i.e., SV, SVI) decreased from the preoperative levels immediately following surgery. Subsequently, the indexed SV continued to improve in the postoperative period and returned above the preoperative values at discharge. This transient depression of the SV parameter in these patients could be multifactorial. It is well known to the clinicians that the hemodynamic hallmark of CCP is impairment of ventricular diastolic compliance.[1-3,8-10] On completion of a successful pericardiectomy, there are major fluid shifts from extravascular to intravascular compartments. In addition, due to repeated compression during the process of pericardial mobilization, there is myocardial edema, which subsides over time.

The sixth major finding is persistently high CVP and reduced SV along with depressed CI values after pericardiectomy in a subset of patients with low CO syndrome.

Fluid responsiveness in peri- and postoperative period in patients undergoing pericardiectomy may not be possible with CVP monitoring alone. Since there are massive fluid shifts with autotransfusion in this subset of patients monitoring of cvp alone (which is a static preload indicator) may not suffice for hemodynamic assessment.^[8,18] SVV is the beat-to-beat change in SV around the mean in one respiratory cycle. Previous investigators have demonstrated that a large SVV (>10%) in a mechanically ventilated patient indicates that the patient is likely to respond to fluid administration.^[16,19]

In this study, there was marked elevation of SVV (>10%) in all patients at presentation in the operating room and during decortication. This possibly is due to decreased compliance of both ventricles secondary to generalized pericardial compression, dissociation between intrathoracic and intracardiac pressures, and an interventricular "coupling" phenomenon, resulting in a septal shift.^[2,4,20] Subsequently, despite restriction of fluid administration, there was only mild reduction of SVV throughout the postoperative period. Ideally, once constriction is relieved, SVV should have immediately dropped and become more dependent on blood volume. The above findings of SVV could be explained by the above explanations as well as our findings of statistically significant alterations in vasomotor tone as reflected by low SVRI following pericardiectomy. The third possibility of residual postoperative constriction in patients undergoing pericardiectomy through median sternotomy posterior to the phrenic nerves cannot be ruled out since the data show borderline high SVV values in some patients even after surgery.

Reports addressing the issue of surgical approach, the extent of pericardiectomy, and postoperative hemodynamics are limited and controversial.^[1-4] The terms "radical," "total," "near total," "subtotal," and "partial" pericardiectomy have been variably used in the literature. For uniformity with other studies including ourselves, we have maintained the definition of "total pericardiectomy" as used in the text.

The culprit pathophysiological mechanisms responsible for low CO syndrome immediately following pericardiectomy are not well understood. Diastolic filling characteristics remained abnormal in a subset of these patients. Results of this study and a review of published reports indicate that regardless of the operative approach or extent of pericardial resection, a subset of these patients with CCP will develop low CO syndrome.^[1-5,8,19,20]

Despite following an uniform intraoperative and postoperative management protocol of the extent of pericardiectomy and postoperative administration of cardioactive medications, 8 (26.4%) patients continued to have persistently high CVP ranging between 14 and 16 mmHg, reduction of SV and CI, and abnormal diastolic filling characteristics on echocardiography. Despite all measures, two (6.6%) patients died of early postoperatively of low CO syndrome.

In our previous investigation on 395 patients, we had demonstrated the occurrence of low CO syndrome and hospital mortality in 34.4% and 7.6% of patients, respectively, before the use of FlotracTM/VigileoTM device.^[8] In this study, although the extent of pericardial resection through median sternotomy remained the same, the use of this semi-invasive device has helped to decrease the hospital mortality to 6.6% by early recognition and timely intervention of low CO syndrome (n = 8, 26.4%).

Poor results with persistent elevation of ventricular filling pressures have been variously attributed by previous investigators to imperfect or incomplete decortication, fibrous invasion of the myocardium, myocardial atrophy, remodeling of the ventricle, worsening tricuspid regurgitation, and postoperative mitral regurgitation secondary to papillary muscle elongation.^[20-22] These myocardial pathologic changes bring about changes in SVV perioperatively, and once the myocardial dysfunction settles down, the svv returns to normal in the postoperative period at varying time intervals in the majority of

patients. Therefore, contrary to other postoperative clinical situations, despite fluid intake restrictions, an exaggerated SVV do not occur in postoperative patients following pericardiectomy.

The above observations have important clinical implications in the goal-directed management of patients undergoing pericardiectomy. Although most patients undergoing total pericardiectomy through median sternotomy have an uneventful postoperative period, intensive care specialists are often confronted with a group of patients with borderline clinical situations. Serial hemodynamic monitoring using this semi-invasive Vigileo[™] device could help identify those patients at an early stage who, because of preexisting cardiac damage, could benefit from timely intervention and prolonged monitoring in an ICU.

Study limitations

The study included only a small number of patients, and all of them underwent total pericardiectomy through median sternotomy. Hence, the hemodynamic variables could not be compared with anterolateral thoracotomy approach.

Second, SVV needs to be measured after at least 1 min period of hemodynamic stability to avoid misleading values that may have been induced by any acute change in heart rate or mean arterial pressure, as its algorithm relies on mean pulse pressure. These issues are of particular importance in the perioperative period when dynamic fluctuations in arterial tone may result in erroneous estimation of CO. Therefore, it is important to observe a steady hemodynamic state before accepting the value of SVV. More number of patients are needed to identify whether SVV is really a "gold standard" to predict fluid responsiveness in these "sick" patients undergoing pericardiectomy with large fluid shifts.

Third, since surgical manipulation of the heart during pericardiectomy can make thermodilution, PA, and transesophageal echocardiographic techniques unreliable as monitors, we have not validated our results with these techniques.

Fourth, other potential weaknesses of this system include possible inaccuracy of the data in the presence of arterial wave artifact, compromise of the arterial catheter, heart rate, altered SVR, and supraventricular arrhythmias. These problems are, however, existent with other arterial waveform CO devices. Since the system uses a full 20 s of data and calculates the overall pulsatility of the wave, it is likely to be robust in cases of artifact, underdamping, and dysrhythmia.^[16]

Moreover, finally, the system currently is unable to provide advanced volumetrics, i.e., systolic and diastolic cardiac volumes and ejection fraction.

Further studies are underway to compare these semi-invasively determined hemodynamic indices achieved

by median sternotomy and anterolateral thoracotomy approaches on a larger number of patients.

Conclusions

Semi-invasive arterial pressure waveform analysis using $FloTrac^{TM}/Vigileo^{TM}$ device is an useful test for serial hemodynamic monitoring of patients undergoing pericardiectomy. It can be performed serially with a high degree of reproducibility. It may be used for late postoperative assessment, obviating the need for frequent cardiac catheterization.

In addition, we propose that routine and serial utilization of this semi-invasive modality may be the investigation of choice for hemodynamic monitoring of these patients undergoing pericardiectomy.

FloTrac[™] sensor, Vigileo[™] monitor; Edwards Lifesciences, Irvine, CA, USA

The system consists of a FlotracTM sensor and a processing/display unit (Vigileo, Edwards LLC). The sensor is a transducer that preprocesses and sends a signal to both the cardiovascular monitor (for real-time waveform display) and Vigileo monitor.^[16] The Vigileo is a small instrument, weighing 2.1 kg, and can be mounted on an intravenous pole. The processing unit applies a proprietary algorithm to the digitized arterial pressure wave and reports CO, CI, SV, SVI, and SVV. If a CVP catheter has been placed, its signal can be interfaced with the Vigileo, allowing for the calculation of SVR and SVRI. When used with a central venous oximetry catheter, the Vigileo also provides continuous central venous oxygen saturation. The rear panel of the Vigileo allows interfacing with CVP and oximetry, external video, and a printer (USB). The Vigileo reports hemodynamic parameters at 20 s intervals, performing its calculations on the most recent 20 s of data.

The system calculates the arterial pressure using arterial pulsatility, resistance, and compliance, according to the following equation: $SV = K \times pulsatility$. Where K is a constant quantifying arterial compliance and vascular resistance, and pulsatility is proportional to the SD of the arterial pressure wave over a 20 s interval. K is derived from patient characteristics (gender, age, height, and weight), as well as waveform characteristics. This calibration constant is recalculated every 10 min.

This advanced technology assesses all hemodynamic variables without requiring external calibration allowing its use in emergency room, medical/surgical ICU, and operation theater. It also reports SVV, i.e., the variation in the beat-to-beat SV around the mean during a respiratory cycle. Patients suffering from hypovolemia exhibit an exaggerated SVV (>10%). However, SVV may be affected by other factors such as vasodilator therapy, lung disease, and mechanical ventilation.^[16,17,19]

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Conflicts of interest

There are no conflicts of interest.

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