Pre- and Postoperative Hemodynamic Assessments and Survival Rates in Patients with Postinfarction Ventricular Septal Perforation

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Pre- and postoperative hemodynamics were assessed in 14 consecutive patients who developed ventricular septal perforation (VSP) following acute myocardial infarction (AMI). Results were correlated with the surgical outcome and with postoperative clinical improvements. The patients were divided into 3 groups according to the time intervals between the onset of AMI and the operation; acute (within 2 weeks after AMI), subacute (between 2 and 4 weeks) and chronic (after 4 weeks). In the above groups, 6, 2 and 6 patients were included, respectively. Eleven patients had antero-septal infarction and 3 patients sustained inferior infarction. The survival rates were 33, 50 and 100% in the acute, subacute and chronic groups, respectively with an overall survival rate of 64%.

Hemodynamic comparisons between survivors and non-survivors revealed that the systolic aortic pressure and left ventricular stroke volume index were significantly higher and the right ventricular end-diastolic pressure was significantly lower in survivors than in non-survivors ($p < 0.05$). Although no statistical significance was obtained, left ventricular end-diastolic volumes and ejection fractions were higher in survivors. No difference was present between survivors and non-survivors in either Qp/Qs, Pp/Ps, Rp/Rs, systolic pulmonary pressure, left ventricular end-diastolic pressure or cardiac index. Patients with low arterial pressure and high right ventricular end-diastolic pressure under intensive medical regimens, indicating the presence of cardiogenic shock and/or associated right ventricular infarction or severe failure, had a high mortality and should be considered for emergency operation.

Postoperative hemodynamics improved significantly in all variables measured ($p < 0.05-0.01$). Patients with a VSP should all be considered for surgery unless a definite contraindication exists.

VENTRICULAR septal perforation (VSP) following acute myocardial infarction (AMI) is a grave complication with a high mor-

Key Words:

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ality rate and the survival rate is better in surgical patients than in medical patients.¹ Recently, early surgical intervention are recommended to improve the survival rate in patients with severe heart failure or cardiogenic shock.²⁻⁴ In 1969, Selzer and associates⁵ studied hemodynamic characteristics in postinfarction VSP and reported the occurrence of pulmonary hypertension with increased pulmonary flow,

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TABLE 1 POSTINFARCTION VENTRICULAR SEPTAL PERFORATION

	No.	Mean age	Hospital death	Survival rate
Acute phase (< 2W) (4 patients < 1W)	6	74	4	33%
Subacute phase (2W < 4W)	2	64	1	50%
Chronic phase (> 4W)	6	67	0	100%
Total	14	70	5	64%

(Anterior MI 11, Inferior MI 3)

Operative results in 14 patients with ventricular septal perforation following acute myocardial infarction. Survival rates are shown in 3 groups of patients classified according to the time of surgery after acute myocardial infarction. In the acute group in which operation was carried out within 2 weeks (W) after infarction, the mortality was highest. Also the patients' ages averaged highest among 3 groups.

elevated pulmonary wedge pressure and elevation of the right atrial pressure. Recently Radford and associates⁶ reported that surgical mortality is high in VSP patients who developed cardiogenic shock in association with right ventricular infarction or extensive septal infarction. Grose and associates⁷ also reported that one of the major factors contributing to a high mortality rate is right ventricular failure with increased right atrial and right ventricular end-diastolic pressures. However, studies regarding pre- and postoperative hemodynamic assessments and their relation to the survival rate are still limited. The purpose of this paper, therefore, is to report our pre- and postoperative hemodynamic studies and the survival rates in 14 consecutive patients with a postinfarction VSP managed by surgery.

PATIENT MATERIALS

Fourteen consecutive patients complicated by a VSP following AMI underwent surgery. There were 4 male and 10 female patients with a male-female ratio of 1 to 2.5. Their ages ranged from 62 to 79 years with a mean of 70 years. Eleven patients sustained anteroseptal infarction and the remaining 3 patients had inferior infarction. They were divided into 3 groups depending upon the time of surgery. In the acute group, there were 6 patients who required emergency surgery as a last resort for salvaging their lives within 2 weeks following AMI. Two patients required operation between 2 and 4 weeks after AMI (subacute group) and 6 patients were operated on the elective to semi-emergency basis later than 4 weeks after AMI (chronic group). Patients in the acute or subacute group had either cardiogenic

shock or profound heart failure with pulmonary edema, and in the patients of the chronic group, persisting congestive heart failure was the main reason for operation.

The interval between AMI and VSP was within 5 days in all patients, and in most of them it was less than 2 to 3 days after AMI. In the acute or subacute group, operative decision was preceded by maximal efforts for stabilization of hemodynamic conditions using catecholamine, vasodilator therapy, diuretics, and intraaortic balloon pumping (IABP). All patients in the chronic group were on digitalis and diuretics.

METHODS

a) Hemodynamic Evaluation

In all patients of the acute group and in one patient of the subacute group, a Swan-Ganz catheter was inserted at bedside to measure pulmonary arterial (PA) and wedge pressures, and the radial arterial pressure was monitored by direct puncture and was assumed to be the aortic pressure (AO). The PA wedge pressure was assumed equivalent to the left ventricular end-diastolic pressure (LVEDP). The right ventricular end-diastolic pressure (RVEDP) was also measured through a Swan-Ganz catheter. In two patients in the acute and one patient in the subacute groups and in all patients in the chronic group, right and left catheterizations were performed in a catheterization laboratory. The cardiac output was measured by a dye-dilution method and was estimated by a forward-triangle method, although automatic computer calculation of cardiac output was usually impossible because of the presence of the large shunt and the low output state. The cardiac output was divided by

Fig.1. Hemodynamic comparisons between operative survivors (S) and deaths (D).

Abbreviations: See text, $\bar{x}$ mean $\pm$ S.D.

Top: a), Middle: b), Low: c)

Fig.2. Hemodynamic comparisons before (B) and after (A) surgery in survivors.

Abbreviations: See text, $\bar{x} \pm S.D.$

Top: a), Low: b)

the patient's body surface area to calculate the cardiac index (CI). In only 7 patients, left ventricular and coronary angiography was performed and left ventricular volumes and ejection fractions were calculated from a single right anterior oblique view⁸. The pulmonary and systemic flow ratio (Q_p/Q_s) was calculated by oximetry and the pulmonary and systemic resistant ratio (R_p/R_s) was also calculated from the pressure and flow data. Statistical comparisons were carried out using a Student t-test between survivors and non-survivors, and a paired t-test was utilized for comparison between pre- and post-operative variables.

b) Operative Method

The VSP was approached through a left ventricular infarctectomy or aneurysmectomy. In the chronic group, the defect was closed directly with Teflon pledgets in anteroseptal infarction and the defect in inferior infarction was closed with a patch. In the acute and subacute groups, a large patch was used to create the septum after resection of the necrotic septum and the free walls of both the left and right ventricles. IABP was used in 7 patients. Concomitant coronary bypass surgery was performed in only one patient.

TABLE II POSTOPERATIVE HEMODYNAMIC EVALUATION AT REST AND DURING EXERCISE AT THE EARLY (1.5 MONTHS) AND LATE (9 MONTHS) POST-OPERATIVE PERIODS IN A 78-YEAR-OLD MAN IN THE ACUTE GROUP

		Rest		Exercise (50 watts 5 min)	
		P.O. 1.5M	9M	P.O. 1.5M	9M
HR	(/min)	80	61	96	78
RA	(mmHg)	(1)	(5)	(3)	(8)
RV	(mmHg)	16/~2	25/~6	30/~7	40/~12
RVEDP	(mmHg)	2	6	7	12
PA	(mmHg)	16/6 (9)	25/12 (16)	30/10 (17)	40/16 (27)
LVEDP	(mmHg)	5	12	12	20
AO	(mmHg)	124/68 (90)	115/58 (84)	144/65 (92)	140/64 (85)
COd	(l/min)	2.81	4.69	5.44	7.21
SVd	(ml)	35	77	57	92
LVEDV	(ml)	97	118		
LVESV	(ml)	50	51		
EF		0.49	0.57		
E _{max}	(mmHg/ml)	2.48	2.25		

Abbreviations: P.O. = postoperative; M = month(s); HR = heart rate; RA = right atrium; RV = right ventricle; RVEDP = right ventricular end-diastolic pressure; PA = pulmonary artery; LVEDP = left ventricular end-diastolic pressure; AO = aorta; COd = cardiac output by dye dilution method; SVd = stroke volume by dye dilution method; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; EF = ejection fraction; E_{max} = peak systolic pressure / ESV

RESULTS

a) Operative Results

Operative results in each group of patients are shown in Table I. Two of the 6 patients (33%) in the acute group, one of 2 (50%) in the subacute group and all 6 patients (100%) in the chronic group survived and were discharged from the hospital with an overall survival rate of 64%. The mean age was highest, 74 years old, in patients of the acute group and the survival rate of this group was the lowest.

b) Hemodynamic Comparisons Between Survivors and Deaths

As shown in Fig. 1 a, b and c, patient age, systolic aortic pressure (AOs), systolic PA pressure (PAs), RVEDP, LVEDP, Qp/Qs, Pp/Ps, Rp/Rs, CI, left ventricular end-diastolic volume (EDV), left ventricular stroke volume index (LVSVI) and left ventricular ejection fraction (EF) were compared between survivors and non-survivors. In comparisons among these variables, a significant difference was obtained in AOs, RVEDP and

LVSVI ($p < 0.05$). The AOs averaged 104.8 ± 14.7 (SD) mmHg in 9 survivors compared to 86.6 ± 9.4 mmHg in 5 non-survivors ($p < 0.05$). The mean RVEDP was 10.9 ± 5.1 mmHg in 8 survivors compared to 16.4 ± 3.0 mmHg in 5 non-survivors ($p < 0.05$). The mean LVSVI was 56.8 ± 8.0 ml/m² in 4 survivors and 34.5 ± 12.9 ml/m² in 3 non-survivors ($p < 0.05$). The Qp/Qs, EDV and EF tended to be lower in non-survivors, although the difference was not statistically significant. The Qp/Qs was 3.21 ± 1.57 in 9 survivors in comparison with 2.71 ± 1.25 in 5 non-survivors. The EDV averaged 150 ± 28 ml/m² in 4 survivors compared to 115 ± 20 ml/m² in 3 non-survivors and the EF averaged 0.38 ± 0.06 in 4 survivors in contrast to 0.29 ± 0.08 in 3 non-survivors. There were no significant differences in PAs, CI or LVEDP. The PAs was 47.0 ± 15.6 mmHg in 9 survivors and 41.2 ± 8.3 mmHg in 5 non-survivors. The CI averaged 1.48 ± 0.35 L/min/m² in 5 survivors and 1.22 ± 0.26 in 5 non-survivors. The LVEDP was slightly higher in non-survivors, 19.8 ± 4.1 mmHg compared to 16.3 ± 8.0 mmHg in survivors.

Postoperative (1.5M) LV-graphy

Postoperative (9M) LV-graphy

Fig.3. Postoperative quantitative biplane left ventriculograms in a 78-year-old man in the acute group. Studies were repeated twice at the early (1.5 months) and late (9 months) periods after surgery. At the late postoperative period, the end-diastolic volume (EDV) increased while the end-systolic volume (ESV) remained unchanged, resulting in an increase in the ejection fraction (EF) from 0.49 to 0.57. The left ventricular stroke volume increased from 47 ml to 67 ml at the late postoperative period.

c) Hemodynamic Comparisons Before and After Operation in Survivors

Pre- and postoperative hemodynamics were compared in 6 survivors. No residual shunt was found in any patients who survived the operation. As demonstrated in Fig. 2 a and b, all variables compared showed significant improvements after surgery ($p < 0.05-0.01$). The cardiac index was 1.48 ± 0.35 L/min/m² preoperatively and postoperatively 2.52 ± 0.63 L/min/m², showing a significant increase ($p < 0.01$). The PAs was elevated to 48.8 ± 18.2 mmHg preoperatively and was normalized to 24.5 ± 6.9 mmHg after surgery ($p < 0.05$). The RVEDP was elevated to 12.3 ± 5.8 mmHg preoperatively and was normalized to 4.8 ± 2.3 mmHg postoperatively ($p < 0.01$). The LVEDP was also normalized to 7.5 ± 3.4 mmHg after surgery from 14.7 ± 3.5 mmHg before operation ($p < 0.05$). The increased EDV before operation was reduced from 151 ± 34.0 to 87 ± 15.3 ml/m² after surgery ($p < 0.05$). The EF increased from 0.40 ± 0.06 preoperatively to 0.59 ± 0.08 postoperatively ($p < 0.05$).

A 78-year-old man in the acute group who had had severe pulmonary edema before surgery survived the operation and was studied twice 1.5 months (early) and 9 months (late) after surgery both at rest and during exercise. At the late postoperative period, the cardiac output increased markedly both at rest and during exercise compared to that at the early postoperative period. The resting cardiac output of 2.81 L/min increased to 4.69 L/min at the late period. The exercise cardiac output also increased from 5.44 L/min to 7.21 L/min at the late period. The stroke volume increased markedly to 77 ml at rest and 92 ml during exercise at the late postoperative period compared to 35 ml at rest and 57 ml during exercise at the early postoperative period. Concomitant increases in RVEDP, LVEDP and PA pressure both at rest and during exercise were observed at the late postoperative period. Changes in hemodynamics at the 2 different periods are shown in Table II. Quantitative biplane left ventriculography was performed in this patient at the early and late postoperative periods as demonstrated in Fig. 3. Interestingly, the left ventricular EDV increased at the late postoperative period to 118 ml compared to 97 ml at the early postoperative period. However, the ESV remained the same, resulting in an increase in EF from 0.49 to 0.57. The change in left ventricular size appeared to be the main cause of an increased cardiac output

and stroke volume at the late postoperative period.

DISCUSSION

At present, few surgical problems remain in VSP patients who survived more than a month after AMI. All 6 patients of the chronic group in our series survived the operation. However, not a few patients require emergency surgery in the early phase of AMI as a last resource for survival, having been unresponsive to maximal medical treatments including IABP. Surgical results in this group of patients have not yet been satisfactory in our series. Only 2 of the 6 patients who required operation within 2 weeks after AMI survived. These 2 survivors were operated upon within a week after AMI. The overall mortality in our series was 36%. Daggett and associates² reported in 1977 that the surgical mortality was 52% in 21 patients who required operation within 21 days after AMI. After 21 days, the mortality was reduced to 7%. The overall mortality was 34% in their series. Gaudiani and associates⁴ reported their surgical results in 1981. The mortality was 90% for patients requiring surgery within a day after septal rupture, 27% for patients after 1 day and 11% after 30 days, giving an overall mortality of 42%. Although their overall mortalities are more or less similar to ours, our surgical result in the acute group needs improvement. It is true that early surgery carries a high risk because of the severest and poorest preoperative conditions but merely awaiting time in these critical patients will further increase the mortality with progressive multiple organ failures. Multiple organ failures such as renal, pulmonary and cerebral dysfunctions develop and progress rapidly in the elderly patients, and the mean age of 74 years appeared to be responsible for poor results in our acute group of patients. Our present policy for surgery, as well as the policy of others^{3,9,10} is that the patient who deteriorates rapidly despite intensive medical treatments should be operated upon early on an emergency basis before profound cardiogenic shock or multiple organ failures develop.

Among the hemodynamic variables measured, AOs, RVEDP and LVSVI were significantly related to the patient's final outcome. The low arterial pressure and low left ventricular stroke volume may represent the presence of cardiogenic shock. Low postoperative survival rates in patients with cardiogenic shock are now apparent

from our results and the results of others^{2,6}. The markedly elevated RVEDP was also suggestive of severe right ventricular failure not only secondary to right ventricular overload due to the acute onset of a large amount of shunt, but also secondary to right ventricular infarction. Grose and associates⁷ reported that the important cause of death following VSP was a right ventricular infarct and that the elevated RVEDP or mean right atrial pressure over 12 mmHg was a common finding. In our patients, the RVEDP of a non-survivor group was 16.4 ± 3.0 mmHg, in contrast to 10.9 ± 5.1 mmHg in survivors ($p < 0.05$). We consider that relatively good right ventricular function is important for the compromised left ventricle to maintain cardiac output before and soon after surgery. The PAs, Qp/Qs, Pp/Ps, Rp/Rs and LVEDP were all irrelevant to survival after surgery and this finding is consistent with that of Radford and associates⁶. Our hemodynamic results are well suggestive of the fact that cardiogenic shock, severe right ventricular dysfunction and low left ventricular output are all important causes of patients' deaths.

We have previously reported that intraventricular conduction disturbances frequently occur in VSP patients who require early surgery.¹¹ The development of a bifascicular block is the sign of poor prognosis. This finding suggestive of extensive septal involvement is also consistent with the autopsy findings reported by Radford and associates⁶. They reported that extensive septal infarction contributed significantly to the development of cardiogenic shock resulting in poor survival.

Considering the significance of hemodynamic assessments in surgical decision for VSP patients, a low arterial pressure and high RVEDP under intensive medical regimens including the use of IABP should be considered important signs for early operation. In predicting the patient's outcome, PAs, LVEDP or Qp/Qs had less significant values in our study. Postoperative hemodynamic studies showed remarkable improvements in all variables. Also interestingly, the cardiac output increased significantly at the late postoperative period due to an increased left ventricular EDV with an unchanged ESV resulting in an increased EF. This phenomenon seems to be the adaptation of the heart to exercise and can likely occur following resection of left ventricular

aneurysms. Based upon these excellent hemodynamic improvements, we believe that patients with a VSP should all be considered for surgery unless a definite contraindication exists.

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