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Characteristics Postoperative Hemodynamics in Aortocoronary Bypass and Mitral valve Surgery

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Abstract To determine the characteristics of postoperative homodynamics following aortocoronary bypass surgery(CABG) and mitral valve surgery, twenty three patients (13 patients undergoing CABG, 10 patients undergoing mitral valve surgery) were studied. Sequential hemodynamic measurements during early postoperative period revealed the following : (1) Systemic hypertension caused by elevated systemic vascular resistance was seen immediately aftetr aortocoronary bypass surgery, and it induced a deterioration of cardiac performance. (2) In the patients undergoing mitral valve surgery. central venous pressure, pulmonary arterial pressure, pulmonary capillary wedge pressure and pulmonary vascular resistance were significantly higher than those in the patients undergoing CABG during 24 hours postoperatively. (3) Cardiac index and left ventricular stroke work in the CABG group showed better improvements in late study period than those in the mitral valve group, according to reduction in systemic vascular resistance. (4) Preoperative pulmonary hypertension associated with mitral valve disease decreased significantly after surgery, but there were some patients demonstrated no significant reduction in pulmonary vascular resistance. The results suggest that the homodynamic alterations during postoperative period were influenced by peripheral vascular resistance and preexisting disease.

Key Words : Cardiac surgery, Postoperative hemodynamics

Introduction

Cold potassium cardioplegia provides excellent myocardial protection and has reduced risks of open heart surgery. However, most surgeons have been encountered transient hemodynamic instability in the early postoperative period, despite apparently adequate cardioplegic protection(1).

These hemodynamic changes following open heart surgery have usually been attributed to alterations in myocardial performance. More recently. we have become aware of the role of noncardiac factors in determining cariac performance. These factors include the effect of anesthesia, extracorporeal circulation and 7, neural and hormonal changes in the early postoperative period. Furthermore, the differences of hemodynamic alterations according to the characteristics in the pre-existing cardiac lesion are also found in the postoperative period. The hemodynamic patterns developed in the postoperative period of certain cardiac

surgery is very important for the surgeons to manage their patients.

This study was designed to describe and analyze the patterns of postoperative hemodynamics in the patients underwent aortocoronary bypass and mitral valve surgery.

Patients and Methods

Thirteen patients with coronary artery disease and 10 patients with mitral valve disease. as outlined below, were operated on electively at the Yamaguchi University Hospital. The patients with coronary artery disease included 2 Women and 11 men, with a mean age of 55.7±8. 0 years. They underwent coronary artery bypass graft(CABG) operations. The patients with mitral valve disease included 8 women and 2 men, with a mean age of 49.1±12.7 years. This group consisted of 6 patients undergoing mitral valve replacement (MVR) and 4 patients undergoing open mitral commissurotomy (OMC). Two patients with MVR underwent combined aortic valve replacement, and a patient with MVR had tricuspid anuloplasty. A profile of clinical characteristics of the two study groups is shown in Table 1.

Surgical technique. Operations were performed with inhalation of euflurane or halothane supplemented with narcotic anesthesia during total cardiopulmonary bypass. Bypass was conducted with the use of moderate systemic hypothermia (Rectal temperature : 25-28℃). Cardioplegic arrest induced by intermittent infusions of cold crystalloid cardioplegic solution was employed and combined with a topical hypothermia for intraoperative myocardial protection. Left ventricular venting was not performed on any patient of CABG group, but left atrial venting was applied in the patients of mitral group. During bypass the mean arterial pressure was kept above 60mmHg by adjusting perfusion flow rate. The duration of cardiopulmonary bypass were 109.5±32.2 minutes in the CABG group and 107.5±32.5 minutes in the mitarl group. The ascending aorta was cross-clamped for 48.8±15. 3 minutes in the CABG group and 62.3±26.3 minutes in the mitrtal group.

No patients of CABG group required inotropic agents during the weaning from cardiopulmonary bypass. However, some patients of mitral group needed a small dose of Dopamine in this period.

Homodynamic measurements. A Teflon catheter was inserted percutaneously into the radial artery before the induction of anesthesia. A 7 Fr. thermal-dilution Swan-Ganz catheter was placed into the pulmonary artery via the femoral vein for measurements of pulmonary arterial pressure.pulmonary capillary wedge pressure, central venous pressure and cardiac output after the termination of cardiopulamonary bypass. All pressures were monitered with a Statham P23Db transducer. Cardiac output was determined in triplicate by the Edward 9520A cardiac output computer. The study was conducted approximately 3 hours after termination of cardiopulmonary bypass, when the patients were in the

Table 1 Pre-operative catheterization data

	MV group(N=10)	CABG group(N=13)	Significance
mAP(mmHg)	82 ± 12	99 ± 10	$p<0.01$
LVSP(mmHg)	114 ± 15	137 ± 16	$p<0.01$
LVEDP(mmHg)	9 ± 32	10 ± 3	NS
mPAP(mmHg)	32 ± 8	12 ± 2	$p<0.01$
PCWP(mmHg)	23 ± 4	7 ± 2	$p<0.01$
CVP(mmHg)	5 ± 3	3 ± 2	$p<0.05$
CI(L/min/m²)	2.58± 0.44	2.85± 0.54	NS
LVSWI(gm-m/beat/m²)	33.7 ± 10.2	49.9 ± 13.1	$p<0.01$
SVR(dynes · sec · cm^{-5})	1683 ±417	1694 ±284	NS
PVR(dynes · sec · cm^{-5})	193 ± 96	99 ± 34	$p<0.02$
EF(%)	54 ± 9	53 ± 10	NS

mAP=mean arterial pressure, LVSP=ventricular systolic pressure, LVEDP=left ventricular end-diastolic pressure, mPAP=mean pulmonary arterial pressure, PCWP=pulmonary capillary wedge pressure, CVP=central venous pressure, CI=cardiac index, LVSWI=left ventricular stroke index, SVR=systemic vascular resistance, PVR=pulmonary vascular resistance, EF=ejection fraction.

intensive care unit. All measurements were performed at 3, 6, 9, 12 and 24 hours after termination of bypass. During the study period all patients were ventilated with a volume cycle venuilator, and blood pH, PaO2 and PaCO2 were kept within normal limits.

Hemodynamic measurements included cardiac index (CI), left ventricular stroke work index (LVSWI), right ventricular stroke work index (RVSWI), systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR), which derived using the following formulae :

Cardic index (liters/min/m2) =
cardiac output/body surface area

LVSWI (g · m/m2) =
(mAP−PCWP) ×13.6×CI/HR

RVSWI (g · m/m2) =
(mPAP−CVP) ×13.6×CI/HR

SVR (dynes · sec · cm^{-5}) =
(mAP−CVP) /CI×80

PVR (dynes · sec · cm^{-5}) =
(mPAP−PCWP) /CI×80

where mAP is mean arterial pressure (mmHg) ; PCWP is pulmonary capillary wedge pressure (mmHg) ; mPAP is mean pulmonary arterial pressure (mmHg) ; CVP is central venous pressure (mmHg).

The indirect index of myocardial oxygen consumption was caculated for the patients : the rate pressure product=systolic arterial pressure x heart rate (double products).

During this study period eleven patients of CABG group received vasodilater therapy for the treatment of systemic arterial hypertension or low output state, using intravenous nitroglycerin, nitroglycerin paste or intravenous nitroprusside, and 7 patients of mitral group received this therapy for low output state and/or pulmonary hypertension.

All results are expressed as mean ± SD. Statistical significance was evaluated by paired or unpaired Student's t test and an one way analysis of variance.

Results

The cardiac catheterization data before operation in the two groups are listed in Table 1. There were significant differences between the CABG group and the MV group in CVP, MPAP, left ventricular systolic pressure and LVSWI. Central venous pressure and MPAP in the CABG group were lower than MV group, and left ventricular systolic pressure and LVSWI in the CABG group were higher than in the MV group.

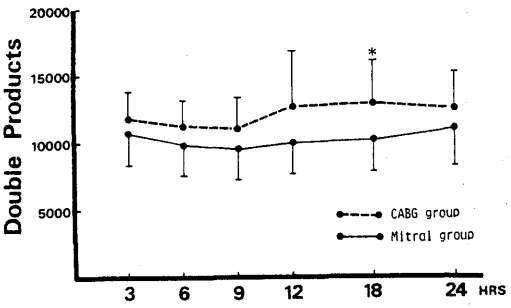

Fig. 1 Postoperative changes in double products (rate pressure product). Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05.

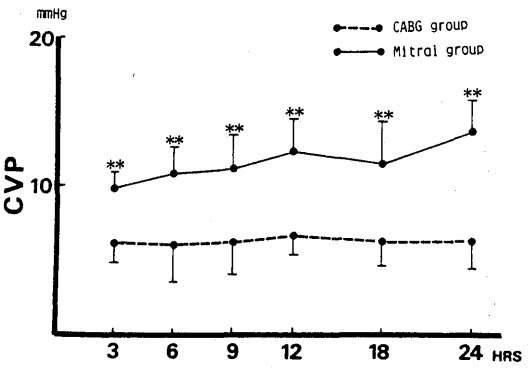

Fig. 2 Postoperative changes in central venous pressures. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : **p<0.01.

Sequential homodynamic measurements in the early postoperative periods were analyzed and compared between the two groups. Double products (rate pressure product) showed no significant changes throuh postoperative 24 hours in both groups (Fig. 1). Gentral venous pressure showed no significant changes postoperatively in the CABG group, but significant increases in the MV group at 12, 18 and 24 hours comparing to immediate postoperative level (3 hours). CVP in the MV group kept higher than in the CABG group through 24 hours postoper-

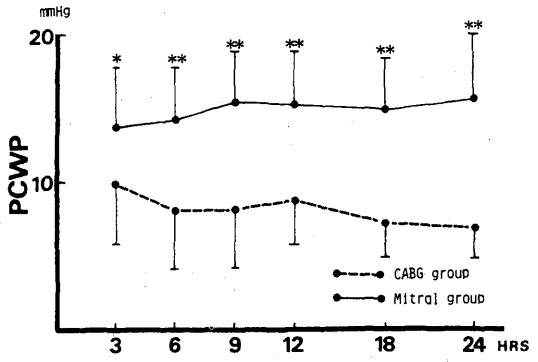

Fig. 3 Postoperative changes in pulmonary capillary wedge pressures. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05 ; **p<0.01.

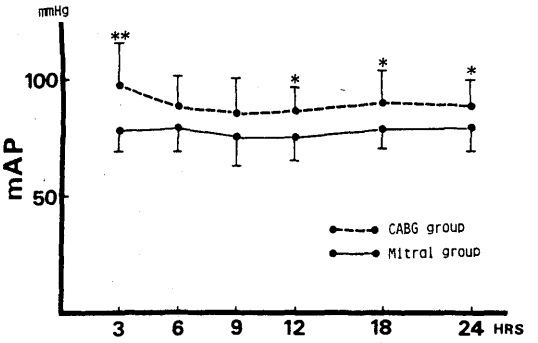

Fig. 4 Postoperative changes in mean arterial pressures. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05 : **p<0.01.

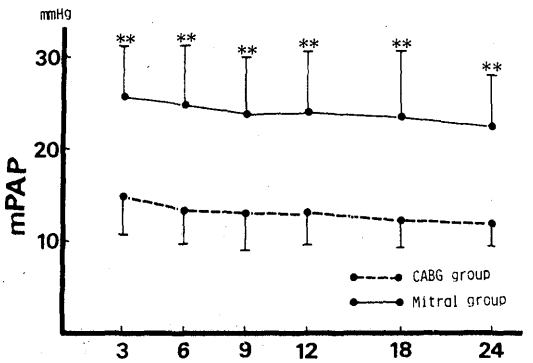

Fig. 5 Postoperative changes in mean pulmonary arterial pressures. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : **p<0.01.

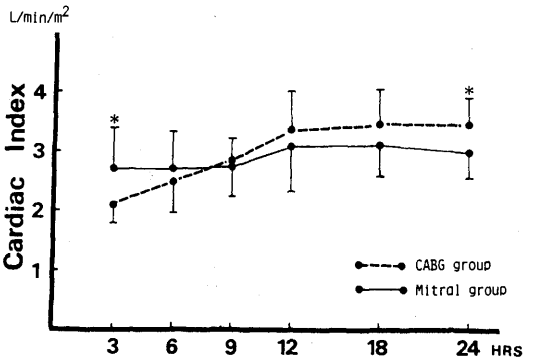

Fig. 6 Postoperative changes in cardiac indices. Results are expressed as mean± standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05.

atively (Fig. 2). No significant changes in PCWP were observed in the both groups. PCWP in the MV group were significantly higher than in the CABG group throughout the study period (Fig. 3). Mean AP in the CABG group were significantly higher than in the MV group as same as preoperative values (Fig. 4). Mean PAP in the MV Group were higher than in the CABG group throughout the study period, despite MV group had significant reductions as compared with the preoperative value (25.8±5.6 versus 31.8± 8.3mmHg, Fig. 5). The sequential increase in cardiac index were apparently observed in the CABG group, despite the CI in the MV group remained unchanged through 24 hours. At three hours postoperativly, the CI in the MV group was significantly greater than in the CABG group. By a gradual improvement in cardiac function, however, the CI in the CABG group increased to 163% of its level at 3 hours postoperatively and became greater than in the MV group at 24 hours (Fig. 6). The two groups showed significant increases in CI after 12 hours postoperatively. LVSWI in the CABG group increased at 12 hours and remained significantly greater than 3 hours value through 24 hours. Whereas, LVSWI in the MV group remained unchanged and lesser than in the CABG group at 12, 18 and hours (Fig. 7). RVSWI remained unchanged

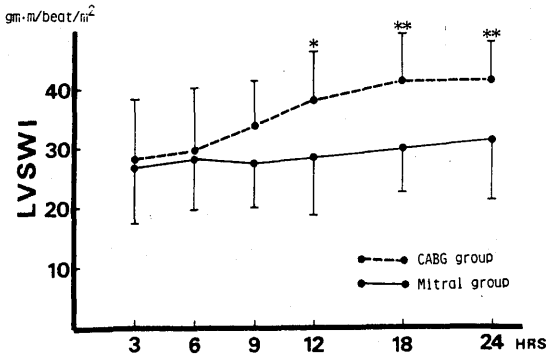

Fig. 7 Postoperative changes in left ventricular stroke work indices. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05 : **p<0.01.

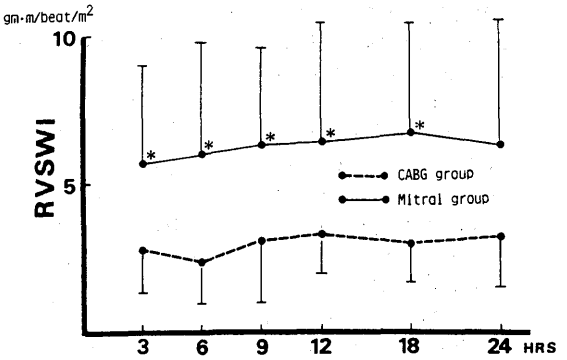

Fig. 8 Postoperative changes in right ventricular stroke work indices. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05.

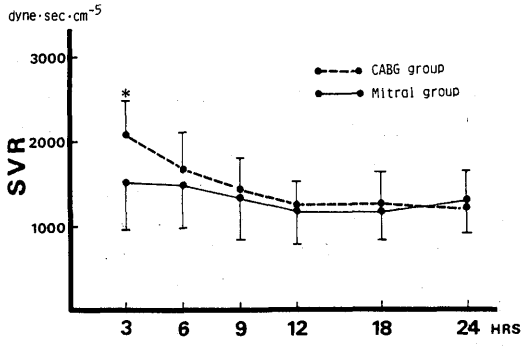

Fig. 9 Postoperative changes in systemic vascular resistances. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.05.

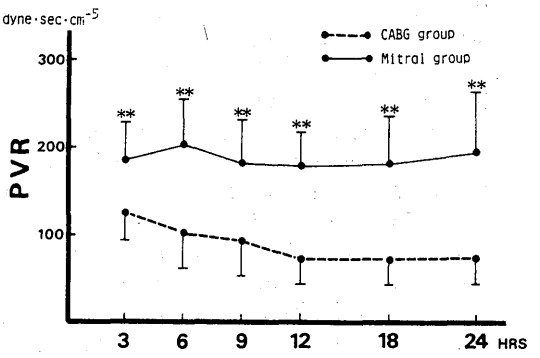

Fig. 10 Postoperative changes in pulmonary vascular resistances. Results are expressed as mean±standard deviation. Significant differences between CABG group and MV group are noted as follows : *p<0.01.

in both groups throughout the study, and the MV group kept RVSWI higher than the CABG group reflecting high pulmonary pressures (Fig. 8). A significant reduction in SVR was observed in the CABG at 6 hours postoperatively and remained significantly low comparing to the 3 hours. There were no significant differences between the two groups, except that the value at 3 hours in the CABG was significantly higher than in the MV group (Fig. 9). PVR in the MV group remained unchanged and were higher than in the CABG group throughout the study. No significant reduction in PVR was observed in the MV group even comparing to the preoperative value. In the CABG group, PVR decreased at 12, 18 and 24 hours comparing to the high level at 3 hours (Fig. 10).

Discussion

The present study demonstrated clearly the characteristics of postoperative hemodynamics in the patients underwent aortocoronary bypass and mitral valve surgery. Considering the group as a whole, mean values of CVP, PCWP, mPAP, PVR and RVSWI were significantly higher in the

mitral group when compared with the CABG group throughout 24 hours postoperatively. These findings might be reflected by the pre-existing pulmonary hypertention observed in the patients of mitral valve disease. Cardiac indeces, expressed as a cardiac preformance, remained at relatively low level of 2.5 to 3.0 L/min in the mitral group during the study period, in contrast to a gradual improvement of cardiac indices in the CABG group . In the CABG group, low cardiac output observed at 3 hours after surgery was apparantly related to an elevation of systemic vascular resistance.

Postoperative systemic hypertension associated with cardiac surgery has been recognized recently as a frequent complication of aortocoronary bypass surgery and other cardiac surgery[1~5]. Hypertension occurs during the first 3 hours after aortocornary bypass in 30% to 60% of patients(3, 5, 6). Five of 13 patients in our CABG group(38%) had hypertension greater than 160 mmHg in the early postoperative period. One of ten patients in the MV group(10%) had hypertension. Systemic hypertention appeared to develop somewhat frequently after aortocoronary bypass aortic valve replacement than after mitral valve replacement. Our impression was that this hypertension occured more frequently among patients with well preserved myocardial function.

The role of non-cardiac factors in this systemic hypertension during early postoperative period has become more apparent ; potential predisposing factors include hypothermia, elevated plasma renin and angiotensin activity[4], elevated serum catecholamines activity[6] or pressor reflexes originating from the heart and great vessels[7,8]. These humoral and neurogenic mechanysms may be stimulated chiefly by extracorporeal circulation and cardiac manipulation during surgery. Several studies[3,6] have established that this paroxysmal hypertensive response is due to an increase in systemic vascular resistance (SVR). Deterioration of cardiac performance in the early postoperative period manifests by low cardiac output, often in association with elevated sustemic vascular resistance. The factors contributing to the impaired ventricular function during this period remain unclear, but elevated SVR may result in an increase in metabolic demand and worsen metabolic recovery of the postischemic myocardium in the early postoperative period[9]. Patients with left ventricular dysfunction exhibit improved performance with afterload reduction. In our study, vasodilator therapy demonstrated a significant improvement in cardiac output in the patients of the CABG group. Postoperative hypertension should be treated aggressively during this vulnerable period to prevent progressive ischemic injury. Regulation of mAP between 90 and 100 mmHg may reduce ventricular work while maintaining coronary perfusion pressure[5].

In the mitral group, a prominent finding in the postoperative hemodynamics was high pulmonary arterial pressure associated with high PCWP even after completing a repair of mitral valve disease. A number of hemodynamic studies regarding the postoperative changes of pulmonary artery hypertension in mitral valve disease have been reported[10~15]. Several studies demonstrated a striking reduction in pulmonary arterial pressure following surgical repair when the patients are studied at some time during the follow-up period[11~13]. However, immediate postoperative changes in pulmonary hypertension are still uncler. Mcllduff and associates[4] documented that patients with mitral valve disease and severe pulmonary artery hypertension exhibit a significant decrease in pulmonary vascular resistance as well as a decrease in pulmonary arteial perssure and increase in cardiac output following valve replacement. They emphasized a vasoreflex, reversible mechanism responsible for the pulmonary artery hypertension seen in patien with mitral valve disease. The present study, in contrast, demonstrated a significant decrease in pulmonary arterial pressure while pulmonary vascular resistance did not change. Although it is widely known that a passive, retrograde transmission elevated left atrial pressure produces pulmonary artery hypertension mitral valve disease, to some extent, there can exist irreversible morphological changes in the pulmonary vascu-

lar bed[15,16].

In early postoperative period, elevated pulmonary arterial pressure with increased pulmonary vascular resistance may cause low cardiac output. Austen and coworkers[10] reported deaths in patients with persisted elevation in pulmonary vascular resistance throughout 48 hours postoperatively. Therefore, it is essential for postoperative management in patients with mitral valve disease and pulmonary artery hypertension to moniter pulmonary arterial pressure. Persisted pulmonary hypertension during early postoperative period should be treated to stabilize hemodynamic with some vasodilators effective to the pulmonary vasculature.

Results from this study suggest that postoperative courses are influenced by postoperative hemodynamic status, non-cardiac factors including anesthesia, sugical mainpulation and extracorporeal circulation, and preexisting disease. This study can describe the patterns of postoperative hemodynamics in the patients undergoing aortocoronary bypass surgery and mitral valve surgery, but the results need to be corroborated with prospective studies.

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