Hemodynamics in off-pump surgery: normal versus compromised preoperative left ventricular function

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Abstract

Objective: Off-pump coronary surgery (OPCABG), avoiding cardiopulmonary bypass and cardioplegic arrest, seems to be a better choice in patients with poor baseline cardiac function. Since cardiocirculatory collapse could be induced by heart displacement in this group of patients at high risk, a greater pathophysiologic understanding of the hemodynamic derangements occurring in such patients is needed. Methods: Twenty-eight elective OPCABG patients were evaluated for hemodynamic changes induced by heart displacement, using arterial thermodilution to measure cardiac output and global end-diastolic volume. Hemodynamic parameters were recorded: at baseline; during proper exposure and stabilization of each vessel; and at the end of surgery. Patients were divided into two groups, according to baseline ejection fraction (EF): group A (EF > 30%; N = 16), group B (EF ≤ 30%; N = 12). Results: Heart displacement induced a significant drop in the cardiac and stroke index, with a lesser decrease of mean arterial pressure because of raised systemic vascular resistance. Preload, measured as global end diastolic volume, significantly decreased in group A, while it remained unchanged or increased in group B. Linear regression between the preload index and left ventricular stroke work was significant only in group A. Conclusions: Patients with poor baseline cardiac function can well tolerate OPCABG. However, the pathophysiologic modifications underlying the hemodynamic changes are different compared to those in patients with good preoperative cardiac performance.

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Keywords: Beating heart; Coronary artery bypass surgery; Hemodynamics; Left ventricular function; Off-pump

1. Introduction

Off-pump coronary artery bypass surgery (OPCABG) by median sternotomy is becoming increasingly popular, as in most patients it allows complete myocardial revascularization with excellent short term results. The hemodynamic modifications induced by heart displacement are usually transient and reversible, and the technique seems to be safe even in patients with poor left ventricular function, improving myocardial preservation and leading to successful results [1]. However, some patients develop significant intraoperative hemodynamic instability requiring intra-aortic balloon counterpulsation (IABP) or cardiopulmonary bypass (CPB) [2], and recently Mishra [3] identified very low ejection fraction (<25%), recent myocardial infarction (<1 month), congestive heart failure and preoperative hemodynamic instability as risk factors for cardiocirculatory collapse during OPCABG. Because of the potentially catastrophic effects of an emergent CPB in a patient in acutely deteriorating conditions, an in-depth knowledge of the hemodynamics occurring during cardiac displacement, along with careful monitoring, is of paramount importance for the safe management of patients at risk.

Assessment of cardiac preload permits a better pathophysiologic evaluation of hemodynamic derangements. Arterial thermodilution, besides measurement of cardiac output (CO), allows calculation of the global end diastolic volume (GEDV), proven to be a reliable preload measurement [4]. The aim of the present study is to evaluate the hemodynamic changes induced by cardiac displacement for multivessel off-pump coronary grafting in patients with both normal and poor preoperative left ventricular function using arterial thermodilution for CO and preload assessment.

2. Material and methods

Twenty-eight patients scheduled for elective multivessel OPCABG were prospectively enrolled. Informed consent was obtained from all patients, and the study was approved by our ethics committee.
Exclusion criteria were: evolving myocardial infarction, preoperative hemodynamic instability and preoperative IABP. Unlike other studies [5], high risk patients, defined as patients with low ejection fraction (<30%) or recent myocardial infarction (<1 month), were not excluded.

According to the preoperative left ventricular function, patients have been divided in two groups:

- Group A: normal to moderately depressed left ventricular function (preoperative EF >30%).
- Group B: poor left ventricular function (preoperative EF ≤30%).

All patients were hemodynamically stable in the morning of surgery, with no need of IABP or other intravenous pharmacologic support but nitroglycerin and heparin. Patients received oral Diazepam 0.015 mg/kg 1 h before surgery as premedication. Preoperative β-blockers, nitrates and/or calcium-channel-blockers were continued until the morning of surgery. General anesthesia was induced with Thiopental (1–3 mg/kg), and maintained with Isoflurane. No patient was given intraoperative neuramnuscular blockade. Patients received oral Diazepam 0.015 mg/kg 1 h before surgery.

The following hemodynamic parameters were analyzed: mean arterial pressure (MAP), central venous pressure (CVP), heart rate (HR), cardiac index (CI), stroke index (SI), global end-diastolic volume index (GEDVI), systemic vascular resistance index (SVRI), and left ventricular stroke work index (LVSWI).

Measurements were performed according to the following timing:

(a) Baseline, with the chest and pericardium open.
(b) Five minutes into the construction of each distal anastomosis, with heart properly exposed and stabilized.
(c) Final (with the heart back in its anatomical position).

Arterial thermodilution measurements were performed injecting boluses of 15 ml cool 5% glucose in water in the central venous catheter and recording the thermal dilution curves by the termistor-tipped catheter inserted in the femoral artery and connected to PICCO.

CO was calculated from the thermodilution curve by the Stewart-Hamilton equation.

Two other parameters were calculated from the analysis of the thermal dilution curve:

- Mean Transit Time (MTt), which is the mean difference between the time until the first indicator particle has arrived at the point of detection and the time of arrival of all the following particles
- Downslope Time (DSt), which is the time of the exponential decay of the thermodilution curve.

The product of CO and MTt is the thermodilution 'needle to needle volume', that is the volume between the point of injection and the point of detection of the thermal indicator. This volume represents the intrathoracic total volume (ITTV) [9]. The product of CO and DSt is the volume of the largest mixing chamber between the site of injection and the site of detection [10], that is, for temperature, the pulmonary total volume (PTV). The difference between ITTV and PTV is GEDV, the sum of blood volumes in both right and left cardiac chambers at end diastole.

LVSWI has been calculated by the formula: LVSWI (g m/m²) = MAP × SI × 0.0144

2.1. Statistical analysis

All values have been indexed to body surface area. All continuous data are presented as mean ± standard deviation.

A 2 Levels Between Group by 7 Levels Within Subjects Repeated Measures ANOVA with post-hoc Tukey’s testing for multiple comparisons was used to analyse the hemodynamic changes induced by heart displacement.

The relationship between GEDVI and LVSWI was analysed by linear regression. A P value of <0.05 was considered statistically significant.

3. Results

Preoperative and intraoperative characteristics of the two groups of patients are summarized in Table 1.
and DG anastomosis, increasing at the time of RI, OM and PDA grafting. Final values of GEDVI in patients of group B remained at a higher level compared to baseline, although this was not statistically significant.

Patients in group B received an overall amount of fluids significantly lower than patients in group A, and the two groups were comparable for fluid balance at the end of surgery.

No patient developed significant hypotension (MAP > 60 mmHg) prompting administration of vasconstrictor agents, and CI reduction rapidly resolved with the heart back in its anatomic position, never requiring inotropic infusion.

Both CI and MAP returned to baseline values at the end of surgery, except even though a moderate increase of HR caused a lower final SI especially in group A.

The linear regression between GEDVI and LVSWI (Fig. 5) was significant only in group A patients, with no significant correlation in patients of group B.

### 4. Discussion

Both groups of patients in our study show a significant drop of CI and SI during cardiac displacement, with a better preserved MAP due an increase of SVRI. These changes reversed promptly when the heart was back in its anatomic position. At the end of surgery, CI and MAP returned to baseline values, with a significantly increased HR which led to a slight final reduction of SI.

Most studies, both experimental and clinical, report a drop in CO and SV in response to heart displacement during OPCABG, which is usually more evident while grafting the posterior and inferior wall vessels. These hemodynamic changes are related to many factors (heart displacement, compression by the stabilizer, occlusion of the coronary artery), vary according to the location of the vessel to be...
grafted, and depend also on preoperative ventricular function, heart size and coronary anatomy [11]. However, the precise mechanisms compromising cardiac function are not fully understood.

In an experimental model on pig, using a suction-type stabilizer (Octopus), Gruendemann found that lifting the heart to expose the infero-lateral wall caused a decrease of MAP and CO, with markedly increased right ventricular end-diastolic pressure coupled to echocardiographic evidence of marked compression of the right ventricle and an elliptically shaped left ventricle. Interestingly, a 20° Trendelenburg position normalized MAP, CO and SV at the expense of a further rise in right and also left filling pressure, corresponding to an increased biventricular preload shown by echocardiography [7]. These findings suggest that a severe reduction in venous return to the folded and crumpled right ventricle is the main mechanism of hemodynamic impairment during inferolateral wall exposure. By increasing venous return, Trendelenburg pushes open the right ventricle, improving left ventricular filling. However, experimental findings on healthy animals should be applied cautiously in coronary artery disease patients, who often suffer from poor left ventricular function. Actually, in a clinical study on 44 OPCABG patients [8], which reported hemodynamic and TEE modifications similar to those in Gruendemann’s study, the Trendelenburg position did not reliably normalize CO. In another clinical study on 17 OPCABG patients, using transesophageal echodoppler, Biswas [12] showed a significant decline of regional left ventricular function during circumflex anastomosis compared to LAD and right coronary artery grafting, with a significant reduction of left ventricular compliance (a restrictive diastolic filling pattern on transmitral and pulmonary venous flow velocimetry).

A similar decrease of all measured indices of left ventricular systolic function in response to heart displacement was reported by Torracca [20], using an intraventricular conductance catheter to evaluate hemodynamics in eight OPCABG patients, two of whom had an EF < 30%. In this study exposure of the inferolateral vessels, especially after stabilizer positioning, caused a drop in CI with an unchanged left ventricular end-diastolic volume and a clear-cut, although not statistically significant, left ventricular end-systolic volume increase (from 48 ± 22 to 61 ± 26 ml/m²). Such a disagreement between an unchanged preload and a reduced SI further point out the role of reduced systolic function in the hemodynamic compromise due to cardiac manipulations.

All these findings suggest that probably hampered diastolic filling is not the sole cause of hemodynamic compromise during heart displacement and stabilization, and various mechanisms, such as right ventricular failure [13], left ventricular regional wall motion abnormalities

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**Fig. 1.** Change of the stroke volume index during and after coronary artery anastomosis in groups A and B. Abbreviation are the same as defined in Table 2. *P < 0.05 between group.

**Fig. 2.** Change of the systemic vascular resistance index during and after coronary artery anastomosis in groups A and B. Abbreviation are the same as defined in Table 2. *P < 0.05 between group.

**Fig. 3.** Change of the mean arterial pressure during and after coronary artery anastomosis in groups A and B. Abbreviation are the same as defined in Table 2.

**Fig. 4.** Change of the end-diastolic global volume index during and after coronary artery anastomosis in group A and group B. Abbreviation are the same as defined in Table 2. *P < 0.05 between group, **P < 0.01 between group, ***P < 0.001 between group.
pulmonary occlusion pressure in critically ill [17] and CABG. ITBV is a more reliable indicator of cardiac preload than patients [4], and it correlates to thermodilution CO and SVI. The approach to the estimation of cardiac preload [16]. The double-dilution technique, has been described as a new distensibility, due to compression and possibly ischemia, because markedly influenced by changes in ventricular distensibility, due to compression and possibly ischemia, and by hydrostatic effect, due to cardiac verticalization. The intrathoracic blood volume (ITBV), measured by distensibility, due to compression and possibly ischemia, and by hydrostatic effect, due to cardiac verticalization. The intrathoracic blood volume (ITBV), measured by double-dilution technique, has been described as a new approach to the estimation of cardiac preload [16]. The ITBV is a more reliable indicator of cardiac preload than pulmonary occlusion pressure in critically ill [17] and CABG patients [4], and it correlates to thermodilution CO and SVI during acute experimental hemorrhage [18]. Recently, GEDV has been shown to be linearly related to ITBV, allowing preload assessment by simple thermal dilution in a peripheral artery [19].

In the present study, GEDVI, used as an index of preload, underwent significantly different modifications in patients with poor baseline left ventricular function (group B), compared to patients with normal or moderately depressed left ventricular function (group A). This finding gives possible insights about different pathophysiologic mechanisms underlying hemodynamic modifications induced by heart displacement. First of all, patients in group B show a significantly higher GEDVI compared to group A, as a reflection of their lower EF. A far more important finding is the behaviour of preload, measured as GEDVI, inside each group, in response to cardiac displacement. Actually, facing a similar CI and SI reduction, GEDVI is significantly reduced in group A but remains unchanged or significantly increases (at the time of inferolateral and inferior wall exposure) in group B, in agreement to what reported by Torracca [20]. Such a different behaviour is not the expression of the lower EF in patients of group B. In fact, patients with different EF should respond in a fairly similar way to similar preload changes, whereas our two groups of patients respond in a similar way (reduction of SV) to opposite changes of preload. These findings suggest a different mechanism underlying CI reduction induced by heart displacement in the two groups. In other words, cardiac displacement behaves as a challenge to ventricular pumps, and different hearts adapt to this challenge in a different way, according to their baseline function. In our study, patients of group A, with a fairly good myocardial performance, suffer only the effects of the reduced venous return to the restricted ventricles, and show a reduced SV as a consequence of the decreased preload. On the other hand, in patients of group B, in whom a recent acute myocardial infarction was complicated by a severely compromised ventricular performance, the restrictive effect on venous return is in some way overwhelmed by the ventricular dilatation due to a further decrease of an already low EF. That is to say, cardiac displacement acts not only hindering venous return by an increase of biventricular rigidly; in some cases its effect might be a (further) reduced systolic performance and/or hampered ventricular ejection resulting in a decreased SV along with a dilated ventricle.

To better assess cardiac performance in our two groups of patients, we evaluated the relationship between preload, measured as GEDVI, and LVSWI. This relationship, called preload recruitable stroke work (PRSW), has been shown to be linear and independent of loading, geometry and heart rate [21]; its slope has been proposed as a potential measure of myocardial performance [21]. In our study, linear regression between GEDVI and LVSWI was significant only in group A patients, whereas the regression points were widely scattered of in our group B patients (Fig. 5). This finding confirms that in patients of group A heart displacement causes mainly a preload reduction with an unchanged myocardial performance, mimicking the effects of veno caval occlusion used in experimental models. The lack of correlation between the index of preload and LVSWI in group B is more difficult to explain. The most obvious explanation is a depressed left ventricular contractility during cardiac manipulations, changing the slope of PRSW relationship, so that individual points cannot be aligned on the same curve. Actually, a non linear PRSW relationship has been hypothesized [21] in ventricles with a baseline depressed function due to a greater afterload sensitivity, and Ryan [22] reported simultaneous changes both in slope and in x-axis intercept of PRSW line in ischemic ventricles, due to the so called ‘creep phenomenon’, making the evaluation of such ventricles difficult by this model. Moreover, a decreasing LVSWI together with a rising preload could also be the expression of deficient length-dependent activation related to exhaustion of the physiologic preload recruitment mechanism in dysfunctional ischemic left ventricles, as reported by De Hert [23].

All these findings seemingly point to a different pathophysiologic mechanism of the hemodynamic changes induced by heart displacement in the two groups of patients. Our findings should prompt different considerations when treating low CO during OPCABG. In fact, if postural maneuvers and volume expansion seems to be all what is needed in patients with a good baseline cardiac performance (REF), this could be less than optimal, or even deleterious, in subgroups of patients whose baseline myocardial performance is severely compromised by a recent acute myocardial infarction. These patients require more strict hemodynamic monitoring by techniques allowing ventricular volume
evaluation. The aim is to early detect a trend towards progressive worsening of ventricular function, with reduced CI and heart chamber dilation during cardiac displacement. Such a pump failure should prompt some therapeutic intervention, which could be, in individual cases, the use of inotropic support or conversion to on-pump. Low doses of dobutamine, improving length-dependent regulation of myocardial function [24], increase CI and reduce end-systolic and end-diastolic ventricular volumes without worsening post-ischemic ventricular dysfunction [25]. If dobutamine should be ineffective, a conversion to an on-pump beating heart technique might be the best choice. In our study, we did not use inotropic drugs in any group B patient because CI, after the initial drop, remained stable throughout the time of displacement, not showing a trend towards progressive deterioration, and promptly reversed when the heart was back in its anatomic position.

In conclusion, our data point out that patients undergoing OPCABG respond not uniformly to hemodynamic challenge of cardiac displacement. A subgroup of patients with poor baseline myocardial function due to a recent myocardial infarction, showing a trend towards a further reduced myocardial performance coupled to ventricular dilatation, demands strict hemodynamic evaluation to early detect signs of progressive deterioration requiring, in individual cases, inotropic support or even on-pump conversion. Arterial thermodilution, allowing intermittent evaluation of preload with the same catheter used to monitor continuous CO and invasive blood pressure, proves to be an useful tool for intraoperative evaluation of such patients.

Reference