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Influence of passive leg elevation on the right ventricular function in anaesthetized coronary patients

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Abstract

Introduction: The aim of the present study was to evaluate the haemodynamic effects of passive leg elevation on the right ventricular function in two groups of patients, one with a normal right ventricular ejection fraction (RVEF) and one with a reduced RVEF.

Methods: Twenty coronary patients undergoing elective coronary artery bypass grafting surgery were studied by a RVEF pulmonary artery catheter. The haemodynamic data reported were collected before the induction of anaesthesia (time point 1), just before (time point 2) and 1 min (time point 3) after the legs were simultaneously raised at 60°, and 1 min after the legs were lowered (time point 4). The patients were divided into two groups: group A, with preinduction RVEF >45%; and group B, with preinduction RVEF <40%.

Results: In group A ($n=10$), at time point 3 compared with time point 2, the heart rate significantly decreased (from 75 ± 10 to 66 ± 7 beats/min). The right ventricular end diastolic volume index (from 105 ± 17 to 133 ± 29 ml/m^2), the right ventricular end systolic volume index (from 61 ± 13 to 77 ± 24 ml/m^2), the systolic systemic arterial/right ventricular pressure gradient (from 93 ± 24 to 113 ± 22 mmHg) and the diastolic systemic arterial/right ventricular pressure gradient (from 58 ± 11 to 66 ± 12 mmHg) significantly increased. Also in group A, the cardiac index did not significantly increase (from 3.28 ± 0.6 to 3.62 ± 0.6 l/min/m^2), the RVEF was unchanged, and the right ventricular end diastolic volume/pressure ratio (RVED V/P) did not significantly decrease (from 48 ± 26 to 37 ± 13 ml/mmHg). In group B ($n=6$) at the same time, the heart rate (from 72 ± 15 to 66 ± 12 beats/min), the right ventricular end diastolic volume index (from 171 ± 50 to 142 ± 32 ml/m^2) and the RVED V/P (from 71 ± 24 to 39 ± 7 ml/mmHg) significantly decreased. The cardiac index and the diastolic systemic arterial/right ventricular pressure gradient were unchanged in group B, while the RVEF and the systolic systemic arterial/right ventricular pressure gradient did not significantly increase, and the right ventricular end-systolic volume index did not significantly decrease. All results are expressed as mean ± standard deviation.

Conclusions: We conclude that passive leg elevation caused a worse condition in the right ventricle of group B because, with stable values of cardiac index, of systolic systemic arterial/right ventricular pressure gradient and of diastolic systemic arterial/right ventricular pressure gradient (which supply oxygen), the RVED V/P (to which oxygen consumption is inversely related) markedly decreased. This is as opposed to group A, where the cardiac index, the systolic systemic arterial/right ventricular pressure gradient and the diastolic systemic arterial/right ventricular pressure gradient increased, and the RVED V/P slightly decreased. Passive leg elevation must therefore be performed cautiously in coronary patients with a reduced RVEF.

Keywords coronary patient, leg elevation, right ventricle

$\Delta p_{SSP/RVSP}$ = pressure gradient between systolic systemic pressure and right ventricular systolic pressure; $\Delta p_{DSP/mRVDP}$ = pressure gradient between diastolic systemic pressure and the mean right ventricular diastolic pressure (the mean value between diastolic and end diastolic right ventricular pressures); PLE = passive leg elevation; RVEDVI = right ventricular end diastolic volume index; RVED V/P = right ventricular end diastolic volume/pressure ratio; RVEF = right ventricular ejection fraction; RVESVI = right ventricular end systolic volume index.

Introduction

Passive leg elevation (PLE) is a common manoeuvre performed to prepare the sterile field in several surgical specialities (coronary surgery, vascular and orthopaedic surgery), to facilitate surgical exposure (gynaecologic and urologic surgery) or to treat acute hypotension. It is well known that this postural change induces some haemodynamic modifications represented by the shift of blood from the legs into the central circulation, by the increase in the venous return and by the improvement in systemic haemodynamics [1–4]. These effects are more pronounced under general anaesthesia because of the larger blood sequestration into the peripheral higher compliant venous system, as a result of the loss in the muscular and vascular tone induced by the anaesthetic drugs [1,2]. If the manoeuvre can be utilized to treat acute hypotension due to hypovolaemia, it could not be as beneficial in the patient with a compromised right ventricle, where acute volume loading may lead to a further deterioration of the right ventricular function [5–7].

Many experimental and clinical studies have shown, however, that PLE has no or little haemodynamic benefit in normovolaemic patients with a stable cardiocirculatory status [2,8–11]. Studying the haemodynamic effects of the manoeuvre in anaesthetized coronary patients with a rapid-response thermistor pulmonary artery catheter [12], Reich *et al*. concluded that PLE results in minor haemodynamic improvement, in right ventricular dilatation and in right ventricular ejection fraction (RVEF) decrease [13]. The authors, however, did not differentiate between the patients with a normal right ventricular function and those with a reduced right ventricular function. For this reason, we planned our study in order to evaluate the effects of PLE on the right ventricle with the same technique, comparing a group of patients with a preinduction RVEF $>45\%$ with a second group of patients with preinduction RVEF $<40\%$.

Methods

Twenty coronary patients scheduled for elective myocardial revascularization were studied after the study protocol was approved by the local Ethics Committee and the written informed consent was obtained from each patient. The admission criteria to the study were stable preoperative cardiocirculatory conditions without intravenous cardiovascular drugs, no evidence of valvular disease or pulmonary hypertension, a normal sinus rhythm, normovolaemia and no diuretic therapy.

Preoperative cardiac medications (β-blockers, calcium channel blockers, nitroglycerin) were continued until the day of surgery.

All patients were premedicated with intramuscular morphine (0.1 mg/kg) and scopolamine (0.005 mg/kg) 60 min before entering the operating room. They were monitored by electrocardiogram (D II, V5, ST segment analysis), by radial artery cannula, and by a thermodilution RVEF pulmonary artery catheter (model 93/A-432H-7.5F; Baxter Edwards Healthcare Laboratories, Santa Ana, CA, USA) positioned before induction under local anaesthesia.

General anaesthesia was induced with etomidate (0.15 mg/kg), fentanyl (5 μg/kg) and vecuronium (0.1 mg/kg), and was maintained during the study with a continuous infusion of midazolam (8–10 mg/hour). Mechanical ventilation was provided with an oxygen/air mixture, and was adjusted to maintain the arterial carbon dioxide tension between 35 and 40 mmHg and to maintain the arterial oxygen tension higher than 100 mmHg. No level of positive end-expiratory pressure was applied. During the study, 2–3 ml/kg fluids per hour were administered to all patients and no other drug was used. After endotracheal intubation, a two-dimensional transoesophageal echocardiography probe was introduced into the oesophagus. The probe was connected to an ultrasonograph device (Color Doppler, model SSD-830; Aloka Company, Tokyo, Japan) and videorecording of two-dimensional transoesophageal echocardiography images were made for later analysis.

A complete haemodynamic profile was performed before induction of anaesthesia (time point 1), just before (time point 2) and 1 min (time point 3) after the patients had their legs simultaneously raised at an angle of 60°, and 1 min after the legs were lowered (time point 4). The pressure data at each time were recorded (the right intraventricular pressures advancing the pulmonary artery catheter until the proximal port entered the ventricular chamber) and were then followed by injection of iced 5% glucose solution at the end of the expiratory time, until three values of cardiac output within 10% of each other were obtained.

Each haemodynamic profile consisted of data recorded by the Hewlett Packard (Palo Alto, CA, USA) monitor (model 7853 C) and registered on a five-channel Hewlett Packard strip chart recorder, and data recorded by the Explorer computer (Baxter Edwards Healthcare Laboratories).

The first profile measured the heart rate, the systolic systemic pressure, the diastolic systemic pressure, the mean systemic pressure, the mean pulmonary pressure, the pulmonary capillary wedge pressure, the central venous pressure, the right ventricular diastolic pressure, the right ventricular end diastolic pressure measured at the interception of the R point in the QRS complex with the intraventricular pressure trace [14], and the right ventricular systolic pressure. The right ventricular end systolic pressure was not measured because the dicrotic notch on the pulmonary artery pressure wave was very often not evident. All pressures were measured on the strip chart at the end of the expiratory time.

The other profile measured the right ventricular end diastolic volume, the right ventricular end systolic volume, the RVEF and the cardiac output.

The derived parameters then calculated were the cardiac index, the stroke volume index, the right ventricular end diastolic volume index (RVEDVI) and the right ventricular end systolic volume index (RVESVI), the right ventricular end diastolic volume/pressure ratio (RVED V/P), the right ventricular stroke work index, the pressure gradient between systolic systemic pressure and right ventricular systolic pressure ($\Delta p_{SSP/RVSP}$) and the pressure gradient between diastolic systemic pressure and the mean right ventricular diastolic pressure (the mean value between diastolic and end diastolic right ventricular pressures) ($\Delta p_{DSP/mRVDP}$).

The patients were divided into two groups on the basis of the RVEF measured at time point 1: the patients of group A had RVEF > 45%, and the patients of group B had RVEF < 40%.

Statistical analysis was performed by two-way analysis of variance test for repeated measures with a Bonferroni correction, $P < 0.05$ was considered significant. Results are expressed as mean ± standard deviation.

Results

Sixteen patients completed the study protocol. Four patients (two from each group) were excluded because of arrhythmias (one patient), because of intravenous administration of nitroglycerin for angina (two patients) and because of moderate tricuspid regurgitation at time point 3 (one patient).

The general characteristics of the patients are reported in Table 1. There are no significant differences between the two groups regarding age, left ventricular ejection fraction, preoperative therapy and the number of right and left coronary stenoses.

Comparing the basal values between the two groups, the bedside RVEF, the heart rate ($P < 0.01$), the RVEDVI, the RVESVI ($P < 0.007$) and the RVED V/P ($P < 0.01$) were significantly higher in group B than in group A (time point 1, Table 2). This statistical difference persisted at time point 2 only for the RVEF, the RVEDVI and the RVESVI.

The PLE induced similar changes in heart rate, which decreased significantly, in both groups and induced similar changes in the majority of the pressure values (central venous pressure, right ventricular diastolic pressure, right ventricular end diastolic pressure, pulmonary capillary wedge pressure), which increased significantly at time point 3 compared with time point 2 (Table 2). On the contrary, the systolic systemic pressure, the diastolic systemic pressure, the mean systemic pressure and the mean pulmonary pressure increased significantly only in group A at the same time point.

The main differences between the two groups after the legs were raised are represented, however, by the changes in the data regarding volumes (Table 2). In group A, at time point 3 versus time point 2, in the face of a slight and not significant increase in cardiac index, we observed a significant increase in the stroke volume index ($P < 0.003$), the RVEDVI ($P < 0.008$) and the RVESVI ($P < 0.006$). However, the RVEF was unchanged and the RVED V/P slightly decreased.

Table 1

General characteristics of the patients studied, concerning age, left ventricular ejection fraction (LVEF), preoperative therapy and the distribution of the right and left coronary artery stenoses

	Group A	Group B
Age	56 ± 8	67 ± 9
LVEF	48 ± 15	50 ± 13
Preoperative therapy		
Nitroglycerin	8	6
β-Blocker	6	4
Calcium antagonist	3	2
Right coronary stenosis		
100%	5	4
90%	4	1
80%	1	1
Left coronary stenosis		
Left anterior descending artery (80–100%)	10	6
Circumflex artery (70–100%)	6	3

There are no significant differences between the two groups regarding age and LVEF.

Moreover, the right ventricular stroke work index ($P < 0.01$), the $\Delta p_{SSP/RVSP}$ ($P < 0.01$) and the $\Delta p_{DSP/mRVDP}$ ($P < 0.009$) significantly increased at the same time. At time point 3, the $\Delta p_{DSP/mRVDP}$ change was statistically significant between the two groups.

An opposite haemodynamic behaviour was observed at time point 3 in group B. In fact, while the cardiac index remained stable and the stroke volume index increased slightly, the RVEDVI ($P < 0.04$) and the RVED V/P ($P < 0.02$) significantly decreased. The RVEF increased and the RVESVI decreased, but not significantly. No significant change was recorded in the right ventricular stroke work index, the $\Delta p_{SSP/RVSP}$ and the $\Delta p_{DSP/mRVDP}$.

The graphic representation of the relationship between right ventricular end diastolic volume and right ventricular end diastolic pressure shows the upward and left side movement of this ratio in the patients of group A, and shows the upward and right side movement in the patients of group B (Fig. 1). After the legs had been lowered, the haemodynamic parameters (time point 4) nearly returned to the same values recorded at time point 2.

Table 2

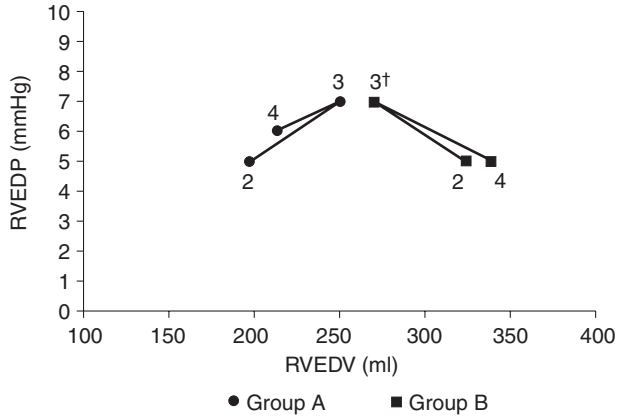
The main haemodynamic data recorded at the four times of the study

		Time point 1	Time point 2	Time point 3	Time point 4
Heart rate (beats/min)	Group A	63 ± 7	75 ± 10	66 ± 7†	69 ± 7†
	Group B	74 ± 6*	72 ± 15	66 ± 12†	65 ± 13†
SSP (mmHg)	Group A	136 ± 22	108 ± 25	133 ± 19†‡	111 ± 18
	Group B	136 ± 26	112 ± 22	115 ± 21	105 ± 23
DSP (mmHg)	Group A	67 ± 12	62 ± 10	71 ± 11†‡	62 ± 10
	Group B	63 ± 16	52 ± 11	55 ± 8	52 ± 9
MSP (mmHg)	Group A	88 ± 14	76 ± 11	90 ± 14†‡	77 ± 12
	Group B	84 ± 20	67 ± 14	71 ± 10	66 ± 12
MPP (mmHg)	Group A	15 ± 3	13 ± 2	15 ± 2†‡	12 ± 2†
	Group B	16 ± 4	13 ± 2	14 ± 2‡	12 ± 2
PCWP (mmHg)	Group A	9 ± 1	6 ± 3	8 ± 2†‡	6 ± 2
	Group B	11 ± 4	6 ± 2	8 ± 3†‡	6 ± 2
CVP (mmHg)	Group A	4 ± 2	4 ± 2	6 ± 1†‡	4 ± 2
	Group B	4 ± 3	4 ± 1	6 ± 1†	5 ± 1
RVEDP (mmHg)	Group A	6 ± 3	5 ± 2	7 ± 2†‡	6 ± 2
	Group B	5 ± 2	5 ± 1	7 ± 1†‡	5 ± 1
RVDP (mmHg)	Group A	3 ± 2	3 ± 1	4 ± 2†‡	3 ± 2
	Group B	2 ± 1	2 ± 1	4 ± 1†‡	3 ± 1
RVSP (mmHg)	Group A	24 ± 4	22 ± 3	24 ± 3	21 ± 3
	Group B	29 ± 7	23 ± 3	25 ± 4	22 ± 4
Cardiac index (l/min/m^2)	Group A	3.60 ± 0.4	3.28 ± 0.6	3.62 ± 0.6‡	3.01 ± 0.6
	Group B	3.90 ± 0.6	3.16 ± 0.5	3.16 ± 0.6	2.80 ± 0.4
SVI (ml/beat/m^2)	Group A	58 ± 9	45 ± 10	56 ± 13†‡	44 ± 9
	Group B	54 ± 11	46 ± 11	50 ± 14	45 ± 10
RVEF (%)	Group A	0.54 ± 0.06	0.43 ± 0.08	0.43 ± 0.09	0.38 ± 0.08
	Group B	0.29 ± 0.07*	0.28 ± 0.05*	0.35 ± 0.07	0.29 ± 0.09
RVEDVI (ml/m^2)	Group A	110 ± 22	105 ± 17	133 ± 29†	114 ± 15
	Group B	196 ± 44*	171 ± 50*	142 ± 32†	180 ± 91
RVESVI (ml/m^2)	Group A	51 ± 15	61 ± 13	77 ± 24†	71 ± 15
	Group B	142 ± 42*	124 ± 45*	91 ± 22	134 ± 87
RVSWI (g m/m^2)	Group A	9 ± 2	6 ± 2	7 ± 2†‡	4 ± 1
	Group B	9 ± 3	6 ± 2 #	6 ± 2‡	4 ± 2
RVED V/P (ml/mmHg)	Group A	42 ± 22	48 ± 26	37 ± 13	41 ± 15
	Group B	83 ± 27 *	71 ± 24	39 ± 7†‡	68 ± 27
$\Delta p_{SSP/RVSP}$ (mmHg)	Group A	112 ± 22	90 ± 25	110 ± 22†‡	89 ± 20
	Group B	107 ± 29	84 ± 28	90 ± 23	83 ± 25
$\Delta p_{DSP/mRVDP}$ (mmHg)	Group A	63 ± 13	58 ± 11	66 ± 12†‡	58 ± 11
	Group B	59 ± 17	50 ± 12	50 ± 10*	48 ± 10

SSP, systolic systemic pressure; DSP, diastolic systemic pressure; MSP, mean systemic pressures; MPP, mean pulmonary pressure; PCWP, pulmonary capillary wedge pressure; CVP, central venous pressure; RVEDP, right ventricular end diastolic pressure; RVDP, right ventricular diastolic pressure; RVSP, right ventricular systolic pressure; SVI, stroke volume index; RVEF, right ventricular ejection fraction; RVEDVI, right ventricular end diastolic volume index; RVESVI, right ventricular end systolic volume index; RVSWI, right ventricular stroke work index; RVED V/P, right ventricular end diastolic volume/pressure ratio; $\Delta p_{SSP/RVSP}$, pressure gradient between systolic systemic pressure and right ventricular systolic pressure; $\Delta p_{DSP/mRVDP}$, pressure gradient between diastolic systemic pressure and mean right ventricular diastolic pressure.
* $P < 0.05$ versus group A within the same time; † $P < 0.05$ versus time point 2 within the same group; ‡ $P < 0.05$ versus time point 4 within the same group.

The transoesophageal echocardiographic images did not show any episode of tricuspid regurgitation, and no apparent displacement of the interventricular septum in the patients who completed the study protocol.

Figure 1



Change of relationship between right ventricular end diastolic volume (RVEDV) and right ventricular end diastolic pressure (RVEDP), by raising and lowering the legs. Time point 2, before leg raising; time point 3, 1 min after leg raising; time point 4, 1 min after leg lowering. † $P < 0.05$, compared with time point 2 and time point 4 within group B.

No ST segment change was observed in any patient at the four times of the study.

Discussion

Many investigators have examined the haemodynamic effects of passive leg raising with different monitoring techniques. Wong *et al*., using thoracic bioimpedence, showed a small but significant increase in cardiac index after PLE in awake patients (American Society of Anaesthesiologists score index II and III) undergoing elective operations [3]. Rutlen *et al*., using nuclear scintigraphy, reported that 150 ml or less of blood transferred to the intravascular space after leg elevation [15]. Kyriakides *et al*., employing a Doppler echocardiographic technique, showed that the postural change increases preload and cardiac performance in normovolaemic coronary patients [10]. However, studying the effects of PLE on anaesthetized coronary patients by a rapid-response pulmonary artery catheter for measurement of RVEF and volumes [12], Reich *et al*. found no improvement in cardiac performance [13].

Our results are in agreement with the conclusions drawn by the previous authors; in fact, a slight increase or no increase in cardiac index was seen in all the patients studied. The haemodynamic response of the right ventricle to the postural change was quite different in the two groups of patients, however, according to the basal right ventricular function recorded before the induction of anaesthesia.

In the patients with a higher basal RVEF and lower right ventricular volumes, the haemodynamic behaviour was similar to that described by Reich *et al*. [13]. The right ventricular end diastolic volume increased more than the right ventricular end systolic volume and, as the RVEF did not change, the stroke volume increased. The cardiac index increased only slightly because of the reduction in the heart rate.

Moreover, the rise in right ventricular end diastolic pressure did not significantly affect right ventricular compliance [16], to which myocardial wall stress and oxygen consumption are inversely related [5,17], because the pressure increase was proportional to that of the end diastolic volume. The significant increase in the pressure gradient between radial artery pressures (generally equal to aortic pressures) and the right intraventricular pressures, whose gradient is considered a good index of coronary perfusion pressure [6], may have improved the right coronary driving pressure. All these findings lead us to conclude that leg elevation induced a favourable condition in the right ventricle of these patients.

On the contrary, with the right ventricles dilated and a lower basal ejection fraction, the manoeuvre of raising the legs was followed by a decrease in end diastolic volume index and end systolic volume index, and by a small increase in ejection fraction and stroke volume, while the cardiac index was unchanged because of the reduction in the heart rate. The reduction of the ventricular size seemed to be advantageous because, according to the Frank–Starling relationship, the right ventricle accomplished the same work with a smaller end diastolic volume and a shorter fibre length [18,19]. The concomitant increase in the right ventricular end diastolic pressure, however, led to a marked reduction in the right ventricular compliance, with adverse effects on ventricular wall stress and oxygen consumption [5,16,17].

Another difference in group B was the lack of increase in the pressure gradient between the radial artery and the right ventricular cavity after the legs were raised. This haemodynamic event, combined with stability in the cardiac index, did not offer a significant gain to the right ventricular oxygen supply.

We have to underline, however, that the conclusions drawn on the right ventricular oxygen supply/demand ratio in the two loading conditions are mainly speculative. This is because they are deduced from indirect indicators (intraventricular volumes and pressures) of the myocardial metabolic balance, and they do not allow a direct measurement of the oxygen supplied or extracted by the heart [5,6,16,17]. Nevertheless, we believe that the variations of the parameters measured in our patients can help the anaesthesiologist and the intensivist to understand or to predict the physiopathological changes that occur in the right ventricle after the legs are raised, also because the haemodynamic data used can be easily available in the clinical anaesthesiological and intensive care setting.

Our protocol included the ejection fraction as a tool to divide the patients on the basis of the right ventricular function. This parameter shows important limitations as an index of ventricular function. In fact, ejection fraction is preload and afterload dependent, and it correlates poorly with myocardial contractil-

ity. It is therefore not a specific parameter, since its variation is due to the changes of one or more of the related factors (preload, afterload or contractility) [20,21]. Ejection fraction, more than a measurement of ventricular performance, can consequently be considered as a measurement of the integrated cardiovascular system in dealing with a pathological process [20]. However, in spite of these limitations, we used the ejection fraction to define the basal right ventricular function of our patients, because this parameter is measured by the pulmonary catheter we used and because it is the one we use in our clinical department for this purpose.

The attempt to correlate the haemodynamic response of the right ventricle to leg elevation with the degree and the number of right coronary artery stenoses was unsuccessful because the coronary obstructions were equally distributed in the two groups. It would therefore not have been possible to predict from the coronarographic data the haemodynamic changes of the right ventricle that followed the passive leg raising manoeuvre.

When analysing the haemodynamic data, a difficulty can arise in interpreting the pulmonary capillary wedge pressure increase; whether this change is due to an increase in left ventricular preload that follows the increase in venous return, or to a decrease in left ventricular compliance as a result of the leftward shift of the interventricular septum for right ventricular dilatation [16,22]. As the wedge pressure also increased in the patients of group B, whose right ventricular volume decreased after PLE, and the transoesophageal echocardiographic images did not show any apparent shift of the interventricular septum in all patients, we conclude that the increase in preload may have been the probable explanation for this haemodynamic change.

The medication drugs given to the patients before surgery (nitroglycerin, β-blockers, calcium antagonists) and the anaesthetic treatment (drugs, mechanical ventilation) may have interfered significantly with the cardiovascular function and the haemodynamic data recorded at the four time points of the study [23–27]. However, as the drugs administered before surgery were distributed equally in the two groups and the anaesthesiological treatment was the same for all patients, we believe that this interference can be considered negligible in the evaluation of the haemodynamic differences observed in the patients studied.

Concerning the data recorded by the thermodilution right ventricular ejection fraction pulmonary artery catheter, the absence of episodes of tricuspid regurgitation (a condition that can lead to underestimating cardiac output and RVEF [6,12,28]) made those parameters reliable and comparable with the parameters recorded before the elevation of the legs.

Another limitation of the present study is represented by the number of patients studied, which should be larger to have more statistical weight and to provide additional support for definitive conclusions. However, in spite of the limitations described and the expectation of further investigations confirming our results, we conclude that the manoeuvre of leg elevation, necessary to prepare the sterile field in several surgical specialities (coronary, vascular and orthopaedic surgery) or to position the patient for some gynaecologic and urologic procedures, must be performed slowly and progressively in normovolaemic coronary patients with a reduced right ventricular function because it could decompensate the already poor balance of oxygen supply/demand in the right ventricle.

Key messages

- PLE brings a slight benefit in coronary patients with a normal RVEF, but causes no benefit or adverse effects (decreased right ventricular compliance, unchanged radial artery pressure–right intraventricular pressure gradient and cardiac index) in right ventricles with a reduced ejection fraction
- PLE must be performed slowly in anaesthetized coronary patients with a reduced right ventricular basal function
- The same caution should also be taken in administering fluids to such coronary patients

We finally suggest that, as the main physiopathological changes of PLE are caused by the increase in venous return to the right ventricle and they are common to those induced by the administration of fluids, the manoeuvre of fluid loading should be performed cautiously and progressively in such coronary patients. Even if this statement seems obvious and other authors have drawn the same conclusions [5], further work needs to be carried out in this area to confirm such a hypothesis.

Competing interests

None declared.

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