Acute Beneficial Effects of Muscular Counterpulsation in Patients with Coronary Heart Diseases

Larry V. Lapanashvili, MD, PhD¹ Michael D. Alshibaya, MD, PhD² Julia Veselova, MD²
Leo A. Bockeria, MD, PhD²

¹Department of Cardiology, Medical School of New Vision University, Tbilisi, Georgia

²Department of Anesthesiology, Bakoulev Scientific Centre for Cardiovascular Surgery, Moscow, Russia

Address for correspondence Larry V. Lapanashvili, MD, PhD, FICA, Medical Center, Marji Ltd., Tabukashvili str. 27, Tbilisi 0108, Georgia (e-mail: larry@marji.net).

Int J Angiol 2017;26:148–157.

Abstract

Keywords

- muscular counterpulsation
- external counterpulsation
- cardiac support device
- noninvasive assisted circulation
- cardiosynchronized stimulation
- coronary intervention
- systolic dysfunction

This prospective pilot study was designed to investigate the acute hemodynamic effects and clinical applicability of muscular counterpulsation (MCP), a one-shot procedure for biomechanical circulatory support. The study included 17 consecutive patients with coronary artery disease (CAD) and impaired ejection fraction ($EF \leq 45\%$) who underwent elective coronary artery bypass grafting (CABG). Patients were divided into control ($n = 7$) and treatment ($n = 10$) groups. MCP was applied through adhesive skin electrodes to the thighs and calves with a battery-powered, portable, ECG-triggered device for 15 minutes prior to general anesthesia. Standard ECG and invasive hemodynamic data were obtained from all patients. MCP was well tolerated in all patients, with no complications. Patients receiving MCP exhibited better cardiac function as indicated by reduced systemic vascular resistance and an augmented cardiac stroke index (+10%), which was maintained over time. After inducing general anesthesia via endotracheal intubation, the treatment group exhibited a reduced after-load (systemic vascular resistance index –28% and mean arterial pressure –10%) with increased left ventricular efficiency (stroke index/left ventricular stroke work index, +22%). Our findings indicate that MCP method was safe and easy to use in this patient population. In conclusion, a one-shot application of MCP prior to anesthesia was associated with an improvement in cardiac pump function and myocardial contractility.

Complication and mortality rates are known to increase considerably during invasive interventions (surgery or angioplasty) in cardiovascular patients with unstable hemodynamics, as the high oxygen demand of the weakened myocardium is increased even further by general anesthesia and the procedure itself.^{1,2} However, there is smoother intervention and less hazardous recovery period in a better-prepared patient.

The intra-aortic balloon pump (IABP) is a well-established invasive counterpulsation (CP) technique that provides me-

chanical circulatory support in patients with unstable hemodynamics due to cardiac failure, or as a preventive measure in high-risk patients with coronary artery disease (CAD) and systolic dysfunction.^{3–6} CP optimizes the oxygen consumption of the damaged myocardium and helps stabilize the patient's hemodynamics prior to surgery and during the critical postoperative period, when the cardiac oxygen demand is particularly high. The mechanism by which the IABP-assisted circulation (AC) system improves hemodynamics is twofold: first, inflation of a balloon inserted into the body up

Fig. 1 Biomechanics of muscular counterpulsation (arterial part). LV, left ventricular.

to the arcus aortae at the onset of diastole results in the aortal lumen occlusion and proximal blood displacement, leading to augmented pressure and improved coronary flow (increased oxygen supply); second, balloon deflation in presystole results in an open aortal lumen and a decrease in pressure and left ventricular systolic off-loading (decreased oxygen demand).^{7,8} Despite the beneficial properties of the IABP, it appears that up to 20% of vascular complications that occur (limb ischemia and embolization) are mainly related to the invasive nature of the device.^{9,10} This risk of severe complications has limited the indications of this therapy only to critical patients with severe hemodynamic compromise¹¹ and performance in a surgical theater or intensive care unit.

Unfortunately, apart from pharmaceutical therapy, no bloodless or noninvasive simple device-based technology is available for use in routine clinical practice involving ordi-

nary, rather than critical, cases. In an attempt to apply the beneficial principles of IABP to a wider number of patients, muscular counterpulsation (MCP), a noninvasive method of biomechanical AC, has been developed.¹²⁻¹⁴ MCP is based on short muscular contractions opposed to the heartbeat that are provided by cardiosynchronous electrostimulation of skeletal muscles during diastole (Lapanashvili method of muscular electrostimulation. Patent of Georgia #366, 1993). MCP was created as a hybrid of AC and electrical muscular stimulation (EMS). Therefore, all positive clinical effects associated with AC and EMS can be expected with MCP. In addition, whereas EMS use may be dangerous in some cardiac patients, MCP is indicated for most patients with cardiovascular disease. On the other hand, whereas IABP is used only in sterile facilities to revive a patient from a critical state, MCP can be self-administered by patients for cardiovascular prevention even while at home.

The mechanism by which MCP, which incorporates the patient's own skeletal muscles "in situ," improves hemodynamics is twofold (► **Fig. 1**). First, it causes muscle contraction and blood vessel constriction at the onset of diastole, resulting in venous and arterial blood displacement that ultimately leads to an augmented early diastolic pressure (► **Fig. 2**) and contributes to improved coronary blood flow (increased oxygen supply). Second, muscular relaxation provokes short-term local vasoplegia with postcontraction active hyperemia^{15,16} and a drop in presystolic pressure with left ventricle systolic off-loading (decreased oxygen demand). Altogether, this hemodynamic improvement regulates the oxygen demand/supply ratio of the damaged myocardium and simultaneously increases locally stimulated tissue perfusion.^{17,18}

The goal of this pilot study was to evaluate direct changes in central hemodynamics induced by MCP in ischemic heart disease patients with systolic dysfunction and to assess the clinical applicability of MCP for acute preoperative preparation.

Fig. 2 A 66-year-old man. Ds: 2CAD, HD, CHF NYHA II. Electrocardiogram (ECG) and aortal pressure (AoP) traces at baseline and during the muscular counterpulsation (MCP) procedure. Stimulation interference is evident after the T-wave on the ECG trace, and a clearly defined diastolic notch appears with end-diastolic pressure reduction on the aortal pressure trace.

Table 1 Patient population, M (min–max)

	MCP group (n = 10)	Control group (n = 7)
Age (y)	56 (43–71)	53 (50–56)
Weight (kg)	79 (64–100)	83 (76–92)
Ejection fraction (%)	40 (35–44)	42 (35–45)
Cardiac output (L/min)	5.3 (4.2–7.4)	5.2 (4.0–5.8)
Hypertension cases	6	5

Abbreviation: MCP, muscular counterpulsation.

Materials and Methods

This study was approved by the Bakoulev Scientific Institutional Review Board (IRB) and performed without any financial support from commercial organizations or MCP producer. Because MCP is relatively novel invented and not yet widely known technic, all investigators were especially trained by MCP author to avoid any technical errors, which could negatively affect patients and falsified study data. The clinical investigation was performed in accordance with the European standard BS EN 540:1993, “Clinical investigation of medical devices for human subjects.” All patients were previously informed about MCP treatment and provided signed consent to participate.

Study Population

The trial inclusion criteria were elective coronary artery bypass grafting (CABG) and an impaired ejection fraction (EF ≤ 45%). Seventeen consecutive CAD patients undergoing

planned CABG were enrolled in this trial. The exclusion criteria were the presence of aortic insufficiency (> mild) and any cardiac arrhythmias. However, no patients were excluded from the study. Patients were divided into two groups: the first 7 patients comprised the control arm and 10 subsequent patients received MCP treatment according to the protocol. ▶ **Table 1** summarizes the demographic data of both groups.

Study Design

The investigation was performed in a surgical theater. The patients were awake and had been given standard premedication (intramuscular morphine, 10 mg and diazepam, 10 mg). No other medications such as β -blockers or angiotensin-converting enzyme inhibitors were used during this investigation. All catheters and electrodes were installed prior to recording the first baseline measurements in both groups. The treatment group received the MCP procedure for 15 minutes, after which measurements (MCP-on) were obtained and the stimulation was ended. Five minutes after MCP was stopped, further measurements (MCP-off) were obtained. The control group did not receive MCP. Finally, immediately after initiating artificial ventilation, final measurements were obtained in both groups. Therefore, the treatment group had four measurement points, whereas the control group had only two points because the intermediate measurement points 2 and 3 (MCP-on and MCP-off) were not relevant for the latter group (▶ **Fig. 3**). All recorded measurements were taken as averages over a few beats.

Study Methods

Standard ECG with invasive central venous, radial artery and pulmonary artery (Swan–Ganz) catheters monitoring was

Fig. 3 Study protocol schematic. MCP, muscular counterpulsation.

Fig. 4 “CardioLa,” the fourth-generation MCP device, which is synchronized by a polar HR transmitter and operated by the physician/patient.

performed in both groups. Cardiac output (thermodilution method), systolic and diastolic arterial pressures (SAP and DAP), systolic and diastolic pulmonary arterial pressures (SPAP and DPAP), central venous pressure (CVP), and heart rate (HR) were monitored in all patients. The mean arterial pressure (MAP), mean pulmonary arterial pressure (MPAP), cardiac index (CI), stroke index (SI), left ventricular stroke work index (LVSWI), systemic vascular resistance index (SVRI), pulmonary vascular resistance index (PVRI), rate pressure product (RPP), and left ventricular effectiveness (SI/LVSWI, amount of blood volume delivered per joule expended on outside work) were calculated using the appropriate formulas.¹⁹

Treatment Protocol

MCP, in the form of cardiosynchronized peripheral muscular pulsation in “systole-to-diastole” mode, was performed using an MK-4 “CardioLa” (–Fig. 4) portable, battery-powered device (Cardiola AG, Winterthur, Switzerland). This device was individually adjusted for each patient before commencing the protocol and a standard ECG was used to set the timing of the stimulation to just after the T wave. The muscle contraction intensity was determined by bursts of tetanizing (~40 Hz) biphasic electrical impulses (1 millisecond width, 10% of the R-R duration) at an amplitude up to 25 V. RPP dynamic was a simple criterion of stimulation setting (timing and intensity) adequacy.

Skin-adhesive electrodes (four active and two passive) were placed on the thigh and calf muscles of both lower extremities. The entire stimulation area was divided into four zones that were activated sequentially with each heartbeat; this carousel-type muscle pulsation allows the heart to be supported beat-by-beat (AC 1:1) while each muscle group contracts only once every fourth beat (EMS 1:4). Accordingly, each muscle group works for one cardiac cycle and relaxes

during the others, enabling the muscles to thoroughly recover their energy potential and assisting the heart in a 1:1 mode by the sequential activation of each of the four muscle groups working in 1:4 mode. Stimulation was initiated at a sub-threshold level, followed by a slow increase in amplitude that eventually reached a visible but light suprathereshold level of muscular contraction (not exceeding 25% of maximum tetanic tension [www.MuscularCounterpulsation.Jimdo.com]). The metabolism of the stimulated muscles should not change excessively and should not affect oxygen consumption, as the mass of simultaneously working muscle is not great and the power of the induced contractions is quite low.

Statistical Analysis

First, the MCP-on and MCP-off results of the treatment group were compared with the baseline values by taking the absolute and relative changes of each patient and calculating the means of those changes (Δ abs, Δ %). Second, the six treatment group patients for whom intubation (INT) measurements were available were compared with the control group. No INT measurements were obtained for four patients in the treatment group (see “Limitations”). Therefore, in each group, the INT parameters were compared with the corresponding baseline values by taking each patient’s absolute and relative changes and calculating the means of the changes (Δ abs, Δ %). In addition, the two groups’ baseline measurements and INT measurements were compared.

All statistical analyses were performed using NCCS 2000 software (NCCS, LLC., Kaysville, UT). The significance of differences between the means at different time points (e.g., from baseline to MCP-on) within groups was tested using a paired *t*-test (p_p) analysis. A two-sample *t*-test analysis (p_2) was used for comparisons of data from two groups at the same time points (e.g., at baseline). Statistical significance was assumed when a two-tailed *p* value < 0.05 was achieved.

Results

Both patient groups were similar with respect to demography and clinical status (–Table 1). MCP was well tolerated in all patients; no procedure-related complications occurred, and no pain or discomfort was observed either locally (in the stimulated area) or in the chest.

As shown in –Tables 2 and 3, acute MCP application improved cardiac performance as indicated by increases in the CI and SI of 7% and 10%, respectively ($p = 0.03$), together with an elevation of the CVP by 1.7 mm Hg ($p = 0.008$). These changes occurred along with a slight decrease (–7%, $p < 0.05$) in the SVRI and a nonsignificant increase (7%) in LVSWI. No notable changes were observed in HR, MAP, and MPAP or PVRI. A more detailed two-dimensional plot analysis of the individual traces of CI against SVRI (–Fig. 5) revealed a convincing trend toward the most central areas of normal parameter ranges.

Therefore, MCP optimized cardiac pump capacity by increasing left ventricular performance without any change in efficiency (SI/LVSWI). The improved output rate occurred as a result of afterload reduction with preload activation, but not

Table 2 Changes in the measured hemodynamic parameters ($M \pm \delta$) at baseline, during the procedure, and 5 minutes after ceasing stimulation in the MCP group ($n = 10$)

Moment (U)	HR (beats/min)	SAP (mm Hg)	DAP (mm Hg)	CVP (mm Hg)	SPAP (mm Hg)	DPAP (mm Hg)
BL	70.0 $\pm$ 15.9	152.8 $\pm$ 21.7	80.8 $\pm$ 5.8	7.2 $\pm$ 2.1	28.5 $\pm$ 3.6	13.8 $\pm$ 5.1
MCP-on	67.9 $\pm$ 15.0	154.7 $\pm$ 23.5	79.2 $\pm$ 5.8	8.9 $\pm$ 2.2	30.2 $\pm$ 6.1	14.6 $\pm$ 5.6
Pp				0.008		
Δ , abs	-2.1	1.8	-1.6	1.7	1.8	0.8
Δ , %	-3%	1%	-2%	28%	6%	0%
MCP-off	69.8 $\pm$ 16.8	157.8 $\pm$ 28.4	81.0 $\pm$ 6.9	9.4 $\pm$ 1.5	30.3 $\pm$ 6.0	15.7 $\pm$ 5.2
pp				0.01		0.04
Δ , abs	-0.2	5.0	0.2	2.3	1.8	1.9
Δ , %	-0.2%	3%	1%	41%	6%	34%

Abbreviations: BL, baseline; CVP, central venous pressure; DAP, diastolic arterial pressure; DPAP, diastolic pulmonary artery pressure; HR, heart rate; MCP, muscular counterpulsation; SAP, systolic arterial pressure; SPAP, systolic pulmonary artery pressure.

at the expense of a myocardial inotropic increase: the LVSWI step-up was equivalent to the SVRI step-down, whereas the SI/LVSWI ratio remained unchanged. This is beneficial because an increase in myocardial inotropism would negatively affect the oxygen demand of the heart and would be counterproductive in these patients.

An assessment of hemodynamic parameters measured 5 minutes after shutting off the device indicated further changes from the baseline values ($\rightarrow$ **Tables 2** and **3**). The CI and SI continued to increase significantly by an additional 5% while the CVP and MPAP increased up to 9.4 ± 1.5 mm Hg ($p = 0.01$) and 21.5 ± 4.7 mm Hg ($p = 0.03$), respectively. Again, this latter preload increase did not overload the lesser circuit or affect the myocardial oxygen demand. Although the LVSWI increased by 13% ($p = 0.006$), the SVRI and PVRI dropped by 12% ($p = 0.03$) and 10% ($p = \text{NS}$), respectively. Thus, the cardiac efficiency (SI/LVSWI) remained unchanged.

Assuming that the hemodynamic changes observed in the control group were "normal" or reflective of a normal occurrence after the induction of anesthesia and INT, a positive effect of the MCP procedure was clearly noted after INT. The average changes in both groups from baseline to post-INT values, along with the intergroup differences, are presented in $\rightarrow$ **Tables 4** and **5** and illustrated in $\rightarrow$ **Figs. 6** and **7**. Endotracheal INT stress reduced the cardiac pump capacities in both patient groups; specifically, CI and SI decreased by 14% and 21%, respectively ($p = 0.03$), in the control group, compared with 11% and 12%, respectively ($p = 0.03$), in the MCP group. However, the post-INT levels of these parameters did not differ significantly between the groups.

There were no statistically significant changes in the HR or the arterial and pulmonary arterial blood pressures either within or between the groups. Although MAP decreased by 7% ($p = \text{NS}$) after INT in the MCP group to a value of

97.3 ± 9.9 mm Hg versus 104.6 ± 14.4 mm Hg in the controls, the difference between the groups was not significant.

Regarding venous return after the basic induction of anesthesia and endotracheal INT, the CVP did not change ($p = \text{NS}$) in the control group but notably exceeded the "normal" level in the treatment group, reaching 10.2 ± 2.0 mm Hg ($p = 0.03$). In contrast to the significant difference in CVP between the MCP and control groups, there was no statistically significant difference between the groups in MPAP after INT.

Compared with the baseline values, SVRI and PVRI increased by 24% ($p = 0.05$) and 2% ($p = 0.8$), respectively, in the control group but decreased by 4% and 10% (each $p = \text{NS}$), respectively, in the treatment group. Hence, the between-group differences in these parameters were 28% and 12%, respectively. Compared with the baseline values, less external work (LVSWI) was performed in both groups after INT, with values of -19% ($p = 0.05$) in the treatment group and -4% ($p = \text{NS}$) in the control group. However, there was no difference between the groups ($p = 0.9$).

The left ventricular efficiencies (SI/LVSWI) of the two groups diverged in opposite directions after INT until a significant difference was achieved ($\rightarrow$ **Fig. 6**). A more detailed two-dimensional plot analysis of the SI versus RPP individual traces ($\rightarrow$ **Fig. 7**) revealed that after INT, they were concentrated in the central area of the normal range in the MCP group, as opposed to the random, diffuse orientation exhibited by the control group. The protective effect of MCP against myocardial stress was clearly noted after INT in the treatment group, even though the one-shot procedure had ended approximately 20 minutes earlier.

Discussion

The use of skeletal muscles to assist circulation in cardiovascular patients has long been of interest to scientists.^{20,21} This

Table 3 Changes in the calculated hemodynamic parameters ($M \pm \delta$) at baseline, during the procedure, and 5 minutes after ceasing stimulation in the MCP group ($n = 10$)

Moment (U)	MAP (mm Hg)	MPAP (mm Hg)	CI (L/min/m ²)	SI (mL/m ²)	RPP (mm Hg/min)	LVSWI (J/m ²)	SVRI (dyn s cm ⁻⁵ /m ²)	PVRI (dyn s cm ⁻⁵ /m ²)	SI/LVSWI (mL/J)
BL	106.0 ± 10.4	19.3 ± 3.8	2.8 ± 0.6	40.2 ± 5.5	10,621 ± 2,468	8.8 ± 1.8	2964 ± 632	164 ± 68	4.7 ± 0.5
MCP on	105.0 ± 9.3	20.6 ± 4.7	2.9 ± 0.6	44.6 ± 9.9	10,542 ± 2,967	9.4 ± 2.2	2714 ± 584	170 ± 74	4.8 ± 0.5
PP				0.03			0.05		
Δ, abs	-1.0	1.30	0.2	4.4	-79	0.64	-249	5.9	0.12
Δ, %	-1%	7%	7%	10%	-1%	7%	-7%	3%	3%
MCP off	107.2 ± 12.0	21.5 ± 4.7	3.2 ± 0.5	46.5 ± 9.3	11,140 ± 3,887	9.8 ± 2.3	2444 ± 389	145 ± 71	4.8 ± 0.7
PP		0.03	0.003	0.005		0.006	0.03		
Δ, abs	1.2	2.25	0.4	6.0	520	1.14	-425	-16	0.09
Δ, %	2%	13%	15%	15%	4%	13%	-12%	-10%	3%

Abbreviations: BL, baseline; CI, cardiac index; LVSWI, left ventricular stroke work index; MAP, mean arterial pressure; MCP, muscular counterpulsation; MPAP, mean pulmonary artery pressure; PVRI, pulmonary vascular resistance index; RPP, rate pressure product; SI, stroke index; SI/LVSWI, left ventricular efficiency; SVRI, systemic vascular resistance index.

interest has led to the development of less dangerous methods of AC, including pneumomechanical enhanced external counterpulsation (EECP) in the 1970s and bio-mechanical MCP in the 1990s.^{12,22,23}

The present report of a small-cohort analysis demonstrates the beneficial effect of a one-shot MCP treatment on circulation in CAD patients with impaired systolic function ($EF \leq 45\%$). According to our findings, a 15-minute procedure led to a notable and relatively prolonged hemodynamic improvement during and after MCP. All patients completed the protocol; MCP discontinuation was not necessary in any of the cases. These patients were additionally found to become more resistant to stress from endotracheal INT. Compared with “normal values” (i.e., those usually observed during INT in patients with severe CAD), the treatment group exhibited a greater decrease in peripheral vascular resistance (SVRI and PVRI) and afterload (MAP), as well as an increase in preload (CVP). Some of the individual key hemodynamic parameters tended toward an “autonomic comfort” level (the most central areas of the normal ranges)²⁴ after MCP, which was clearly shown in the two-dimensional plots (►Figs. 5 and 7). This hemodynamic effect is often sought through complex and costly pharmacologic management in the setting of congestive heart failure.²⁵

Along with the various methods of external, noninvasive CP, as well as with afterload decrease and coronary flow reinforcement, a third and more predominant component is always presented: preload increase. This component is a consequence of musculo-venous pump activation,²⁶ regardless of the method by which patients' skeletal muscles are influenced (e.g., passive [compress-release] with EECP or active [contraction-relaxation] with MCP). In contrast to EECP,^{27,28} however, it appears possible to regulate venous drainage intensity with MCP, a very important feature in heart failure patients. In this setting, MCP could provide a simple, straightforward, and—most importantly—noninvasive treatment option. Costly hospital readmissions and dangerous intensive care stays render MCP treatment using a portable device an interesting and promising option. Patients with left ventricular systolic dysfunction comprise the majority who could potentially benefit from this combined drug-device treatment.

Light muscle contractions cause skeletal muscle pump activation with enhanced venous drainage and increased preload (within the left ventricular pump tolerance), leading to an increase in SI without pulmonary circulation overload. According to theoretical physics, to reach and maintain resonance, a system requires rhythmic low-energy impulses at precisely the right times. Therefore, in an analogy to wave theory, the artificially repetitive short and slight muscle contractions used in this study created a proximally directed arterial pulse wave. Upon timing to resonate with the natural retropropagating pulse waves reflected from the periphery, this arterial pulse wave will be amplified and will augment the early diastolic pressure (►Fig. 2), resulting in an increased coronary blood supply.

Notably, accurate tuning of the muscle pulse delay and selection of the correct contraction amplitude for the

Fig. 5 Individual cardiac index (CI) against systemic vascular resistance index (SVRI) traces in MCP group patients. Dark dot with patient number—baseline, bright dot—MCP-on. Green area—autonomous comfort zone.

muscular effector are very important. The wrong settings may cause excessive preload and lead to transient tachycardia, hypertension, and other symptoms of pulmonary overload, as reported by Ostojic et al.²⁹ In our study, a low stimulation intensity was selected to induce only a light supra-threshold muscular contraction level (~10–20% of maximum tetanic tension), which was nevertheless sufficient to achieve the desired result.

The association of MCP with physical training is incorrect because physical exercise loads the body musculature, joints, and heart. In contrast, MCP gently loads only certain muscle

groups without affecting joints and off-loads the heart. Apart from the biophysics of MCP, with its purely mechanical components, more delicate biomechanisms related to neuro-reflexive control²⁴ and humoral circulatory regulation are involved, although these have not yet been studied.

Limitations

The number of patients enrolled in this pilot study was small. In addition, only 6 of the 10 patients in the treatment group had available INT data. However, this limitation was unrelated

Table 4 Measured hemodynamic parameters ($M \pm \delta$) at the baseline and after intubation in the MCP and control groups

Groups	Moment (U)	HR (beats/min)	SAP (mm Hg)	DAP (mm Hg)	CVP (mm Hg)	SPAP (mm Hg)	DPAP (mm Hg)
Main (n = 6)	BL MCP	59.2 ± 9.1	154.4 ± 26.9	82.0 ± 5.0	6.6 ± 0.8	27.3 ± 4.2	13.2 ± 6.3
	INT	62.4 ± 5.5	135.6 ± 15.9	77.2 ± 7.9	10.2 ± 2.0	26.0 ± 6.8	14.1 ± 3.2
	P _w				0.03		
	Δ, abs	3	−19	−5	4	−1.2	1.0
	Δ, %	7%	−10%	−5%	55%	−4%	6%
Control (n = 7)	BL contr	69.3 ± 4.1	152.3 ± 14.6	77.7 ± 8.5	6.1 ± 3.1	37.9 ± 16.0	17.6 ± 6.6
	INT	66.3 ± 7.5	143.0 ± 16.4	85.1 ± 16.3	5.6 ± 1.3	32.9 ± 11.9	17.3 ± 7.6
	P _w				1.0		
	Δ, abs	−4	−9	7	−0.5	−5	−0.3
	Δ, %	−5%	−5%	9%	9%	−8%	5%
P ₂					0.05		

Abbreviations: BL, baseline; CVP, central venous pressure; DAP, diastolic arterial pressure; DPAP, diastolic pulmonary artery pressure; HR, heart rate; INT, intubation; MCP, muscular counterpulsation; SAP, systolic arterial pressure; SPAP, systolic pulmonary artery pressure.

Table 5 Calculated hemodynamic parameters ($M \pm \delta$) at the baseline and after intubation in the MCP and control groups

Groups	Moment (U)	MAP (mm Hg)	MPAP (mm Hg)	CI (L/min/m ²)	SI (mL/m ²)	RPP (mm Hg/min)	LVSWI (J/m ²)	SVRI (dyn [*] s/cm ⁵ /m ²)	PVRI (dyn [*] s/cm ⁵ /m ²)	SI/LVSWI (mL/J)
Main (n = 6)	BL MCP	107.0 ± 12.2	18.5 ± 4.5	2.4 ± 0.2	41.5 ± 6.6	9,048 ± 1,578	9.2 ± 2.3	3,351 ± 408	180 ± 84	4.6 ± 0.5
	INT	97.3 ± 9.9	18.7 ± 3.4	2.3 ± 0.2	36.0 ± 3.7	8,383 ± 553	7.1 ± 1.4	3,196 ± 448	167 ± 99	5.2 ± 0.5
	p _w				0.03		0.05			
	Δ, abs	-10	0.2	-0.4	-5.5	-665	-2.16	-154	-13	0.59
	Δ, %	-7.4%	8%	-11%	-12%	-4%	-19%	-4%	-10%	15%
Control (n = 7)	BL contr	102.6 ± 8.9	24.3 ± 9.5	2.7 ± 0.3	38.0 ± 7.9	10,964 ± 1,105	8.2 ± 2.2	2,888 ± 252	214 ± 126	4.8 ± 0.5
	INT	104.6 ± 14.4	22.6 ± 8.7	2.3 ± 0.4	30.4 ± 2.1	10,216 ± 2,529	7.1 ± 1.4	3,543 ± 507	199 ± 100	4.4 ± 0.4
	p _w			0.05	0.03			0.05		
	Δ, abs	2	-2	-0.4	-9.3	-747.4	-1.12	655	-15	-0.33
	Δ, %	2.3%	-2%	-14%	-21%	-7%	-4%	24%	2%	-7%
p ₂				0.8			0.9			0.05

Abbreviations: BL, baseline; CI, cardiac index; INT, intubation; LVSWI, left ventricular stroke work index; MAP, mean arterial pressure; MCP, muscular counterpulsation; MPAP, mean pulmonary artery pressure; PVRI, pulmonary vascular resistance index; RPP, rate pressure product; SI, stroke index; SI/LVSWI, left ventricular efficiency; SVRI, systemic vascular resistance index.

p—statistically significant.

p_p—when distribution is normal.

p_w—when distribution is not normal.

p₂—between two samples.

Fig. 6 Left ventricle efficiency (SI/LVSWI) status after intubation: point A—MCP group ($n = 6$) and point B—control group ($n = 7$) versus common baseline (BL; $n = 13$). $p = 0.05$.

to the MCP procedure. Instead, various technical measurement issues did not allow the recording of INT data in four treatment group patients. In the absence of these errors, the results observed with MCP would possibly have been more significant.

Conclusion

Some main conclusions and recommendations have emerged from this modest pilot study.

Conclusion

In patients with CAD and an impaired ejection fraction, even a 15-minute one-shot MCP application decreased the afterload while increasing cardiac output, thus achieving optimal pump efficiency of the damaged left ventricle. Preoperative MCP use facilitates hemodynamic stabilization, provides smoother anesthesiological assistance for CAD patients, and might be associated with preinterventional myocardial protection.

Recommendations

MCP should not be considered as an alternative to IABP, as it has similar effects but is simple and noninvasive. MCP provides an opportunity for expanding the indications of AC to prehospital and outpatient therapy and introduces a new combined drug-device therapy niche in cardiology. Based on the presented results, a multicenter study with appropriate financial support and a sufficient number of subjects is needed. The results of such a study would hopefully substantiate the evidence obtained in this pilot study.

Acknowledgments

We would like to thank M. V. Zatevakhina, MD; Prof. V. A. Lischouk; L. V. Sazikina, PhD, for their assistance with data collection; and P. Huber for statistic analysis and Editage for English language editing.

Fig. 7 Patients' individual stroke index (SI) against rate pressure product (RPP) trace development in the control ($n = 7$) and MCP ($n = 6$) groups. Dark dot with patient number—baseline (BL), bright dot—after intubation (INT). Yellow area represents the area of normal hemodynamic values; green area indicates the autonomous comfort zone.

References

- Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. *J Clin Anesth* 1996;8(1):63–79
- Akhlagh SH, Vaziri MT, Masoumi T, Anbardan SJ. Hemodynamic response to tracheal intubation via direct laryngoscopy and intubating laryngeal mask airway (ILMA) in patients undergoing coronary artery bypass graft (CABG). *Middle East J Anaesthesiol* 2011;21(1):99–103
- Craver JM, Murrah CP. Elective intraaortic balloon counterpulsation for high-risk off-pump coronary artery bypass operations. *Ann Thorac Surg* 2001;71(4):1220–1223
- Marra C, De Santo LS, Amarelli C, et al. Coronary artery bypass grafting in patients with severe left ventricular dysfunction: a prospective randomized study on the timing of perioperative intraaortic balloon pump support. *Int J Artif Organs* 2002;25(2):141–146
- Kim KB, Lim C, Ahn H, Yang JK. Intraaortic balloon pump therapy facilitates posterior vessel off-pump coronary artery bypass grafting in high-risk patients. *Ann Thorac Surg* 2001;71(6):1964–1968
- Kang N, Edwards M, Larbalestier R. Preoperative intraaortic balloon pumps in high-risk patients undergoing open heart surgery. *Ann Thorac Surg* 2001;72(1):54–57
- Bolooki H. Clinical Application of the Intra-aortic Balloon Pump. 3rd ed. New York, NY: Futura; 1986:111
- Shumakov VI, Tolpekin VE. Vspomogatelnoe Krovoobraschenie [in Russian]. Moscow, Russia: Medicina; 1980:248. Available at: <http://urss.ru/cgi-bin/db.pl?lang=Ru&lang=ru&page=Book&id=57816>. Accessed on September 21, 2016
- Meharwal ZS, Trehan N. Vascular complications of intra-aortic balloon insertion in patients undergoing coronary revascularization: analysis of 911 cases. *Eur J Cardiothorac Surg* 2002;21(4):741–747
- Christenson JT, Cohen M. Preoperative IABP in high-risk patients reduces postoperative lactate release and subsequent mortality. *Ann Thorac Surg* 2002;73(3):1026–1027
- Santa-Cruz RA, Cohen MG, Ohman EM. Aortic counterpulsation: a review of the hemodynamic effects and indications for use. *Catheter Cardiovasc Interv* 2006;67(1):68–77
- Tozzi P, Corno AF, Lapanashvili LV, Von Segesser LK. Muscular counterpulsation: preliminary results of a non-invasive alternative to intra-aortic balloon pump. *Eur J Cardiothorac Surg* 2004;26(4):726–729
- Walpoth BH, Aregger C, Lapanashvili L, Mettler D, Carrel T, Hess OM. ECG-triggered muscular counterpulsation for treatment of low cardiac output. *Int J Artif Organs* 2007;30(5):407–414
- Lapanashvili LV, Buziashvili YI, Matskeplishvili ST, et al. Therapeutic value of muscular counterpulsation after coronary bypass grafting operation. *J Card Surg* 2009;24(2):134–140
- Barclay JK, Murrant CL, Woodley NE, Reading SA. Potential interactions among vascular and muscular functional compartments during active hyperemia. *Can J Appl Physiol* 2003;28(5):737–753
- Kots JM. Handbook "Physiology of Muscles Activity." M Fizkultura I Sport [in Russian] 1982:225. Available at: https://vk.com/doc-74650149_437132297?hash=02d0407bd111c12bfd. Accessed on September 21, 2016
- Bockeria LA, Buziashvili YI, Lapanashvili LV, et al. Original method of external muscular counter-pulsation in coronary heart disease patients at cardiosurgery clinic. [in Russian]. *Cardiovasc Ther Prophylaxes* 2005;4(5):4–11. Available at: https://www.researchgate.net/publication/268522685_Original_method_of_external_muscular_counter-pulsation_in_coronary_heart_disease_patients_at_cardiosurgery_clinic. Accessed on September 21, 2016
- Lapanashvili L, Buziashvili YI, Matskeplishvili ST, et al. ECG-triggered skeletal muscle stimulation improves hemodynamics and physical performance of heart failure patients. *Int J Artif Organs* 2008;31(3):244–251
- Babskiy EB, Zubkov AA, Kasitzkiy GI. Circulation. In: Kasitzkiy GI, ed. *Textbook of Human Physiology* [in Russian]. Moscow, Russia: Medicina; 1985:239–292
- Bokeria LA, Lapanashvili LV. [Autologous muscle tissue for reconstruction of the heart and assisted circulation] [in Russian]. *Grud Serdechnosudistaia Khir* 1990;3(3):58–63
- Lapanashvili LV. Automuscular system of assisted circulation for surgical correction of heart failure. *Il Cuore* 1992;9(1):5–27. Available at: https://www.researchgate.net/publication/263209527_Automuscular_system_of_assisted_circulation_for_surgical_correction_of_heart_failure. Accessed on September 21, 2016

- 22 Cohen J, Grossman W, Michaels AD. Portable enhanced external counterpulsation for acute coronary syndrome and cardiogenic shock: a pilot study. *Clin Cardiol* 2007;30(5):223–228
- 23 Okrasov MU, Ivanov GG, Dvornikov VE. External muscular counter pulsation benefits in acute heart failure patients. *Diagnostic and Treatment Disorders of Regulations Cardio-vascular System*. Moscow, Russia: Materials of the 13th conference; 2011:356–361:RU
- 24 Zamostian VP, Lapanashvili LV. Muscular counterpulsation impact in the circulation regulation [in Russian]. *Clin Physiol Circulation* 2005;4:39–48. Available at: https://www.researchgate.net/publication/263213052_Muscular_Counterpulsation_Regulation_of_Circulation_RU. Accessed on September 21, 2016
- 25 Iakobishvili Z, Behar S, Boyko V, Battler A, Hasdai D. Does current treatment of cardiogenic shock complicating the acute coronary syndromes comply with guidelines? *Am Heart J* 2005;149(1):98–103
- 26 Arinchin NI, Feketa VP, Kamishenko VG. [Effect of passive movements in the talocrural joints on central hemodynamics and heart functional state]. *Fiziol Cheloveka* 1992;18(3):83–86
- 27 Soran O, Fleishman B, Demarco T, et al. Enhanced external counterpulsation in patients with heart failure: a multicenter feasibility study. *Congest Heart Fail* 2002;8(4):204–208, 227
- 28 Feldman AM, Silver MA, Francis GS, et al; PEECH Investigators. Enhanced external counterpulsation improves exercise tolerance in patients with chronic heart failure. *J Am Coll Cardiol* 2006;48(6):1198–1205
- 29 Ostojic D, Seifert I, Pütz V, et al. Acute effects of muscular counterpulsation therapy on cardiac output and safety in patients with chronic heart failure. *Artif Organs* 2012;36(6):559–564