Hemodynamic Effect and Safety of Intermittent Sequential Pneumatic Compression Leg Sleeves in Patients With Congestive Heart Failure

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ABSTRACT

Background: Pneumatic leg sleeves are widely used after prolonged operations for prevention of venous stasis. In healthy volunteers they increase cardiac function. We evaluated the hemodynamic effects and safety of intermittent sequential pneumatic compression (ISPC) leg sleeves in patients with chronic congestive heart failure (CHF).

Methods and Results: We studied 19 patients with systolic left ventricular dysfunction and CHF. ISPC leg sleeves, each with 10 air cells, were operated by a computerized compressor, exerting 2 cycles/min. Hemodynamic and echocardiographic parameters were measured before, during, and after ISPC activation. The baseline mean left ventricular ejection fraction was 29 ± 9.2%, median 32%, range 10% −40%. Cardiac output (from 4.26 to 4.83 L/min; $P = .008$) and stroke volume (from 56.1 to 63.5 mL; $P = .029$) increased significantly after ISPC activation, without a reciprocal increase in heart rate, and declined after sleeve deactivation. Systemic vascular resistance (SVR) decreased significantly (from 1,520 to 1,216 dyne-s/cm^5; $P = .0005$), and remained lower than the baseline level throughout the study. There was no detrimental effect on diastolic function and no adverse clinical events, despite increased pulmonary venous return.

Conclusions: ISPC leg sleeves in patients with chronic CHF do not exacerbate symptoms and transiently improve cardiac output through an increase in stroke volume and a reduction in SVR. (*J Cardiac Fail 2014;20:739−746*)

Key Words: Laparoscopy, surgery, transthoracic echocardiography, pneumatic sleeves.

The application of pneumatic sleeves on the lower extremities improves venous circulation and prevents venous stasis in postoperative patients. The intermittent sequential pneumatic compression (ISPC) sleeves (made of 10 air cells) were originally designed for treating severe limb edema (elephantiasis), and were recently shown to improve cardiovascular hemodynamics during positive pressure pneumoperitoneum (PP) that is required during laparoscopic operations.[1] Hemodynamic derangements (such as reduced venous return, stroke volume, and cardiac output and increased systemic vascular resistance [SVR]) may follow PP and prohibit its use in patients suffering from cardiovascular disease.[2−11] ISPC and pneumatic sleeves that were activated to create pressure equilibration were shown to be effective in elimination of undesired systemic and visceral hemodynamic changes associated with PP.[1,12−14] Decreased sympathetic autonomic activity during laparoscopic operations may be an additional mechanism explaining the reduced SVR caused by the 10-cell ISPC sleeve.[15]

A recent publication claimed that the use of 3-cell sequential pneumatic sleeves may decrease cardiac output and increase SVR in healthy volunteers.[16] Because of insufficient

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information regarding the cardiovascular effects of ISPC during PP, we recently conducted a study measuring echocardiographic indices during ISPC application in healthy subjects.[17] We demonstrated an improvement in cardiac activity as expressed mainly by increased cardiac output and decreased SVR without an accompanying increase in heart rate.[17]

However, owing to increased venous return and a possible increase in pulmonary blood flow that may be associated with activation of sequential pneumatic devices, application of ISPC leg sleeves might be deleterious and unsafe in the growing population of elderly postoperative patients, particularly patients with congestive heart failure (CHF). We therefore decided to evaluate the safety and cardiovascular effects of activation of 10-cell ISPC sleeves in patients with systolic CHF.

Methods

Every participant gave informed consent to be included in the study, which was approved by the local Ethics Committee. All patients had clinical symptoms of chronic CHF. Inclusion criteria included New York Heart Association (NYHA) functional class II–III and left ventricular ejection fraction (LVEF) ≤40% as assessed by transthoracic echocardiography (TTE). We excluded patients who could not sign an informed consent, had unstable angina, were <30 days after myocardial infarction or therapeutic coronary intervention, had NYHA functional class I or IV, had oxygen saturation (as measured by pulse oxymetry) <90% on room air, or had chronic lung disease. We also excluded postoperative patients to avoid their unnecessary mobilization for study purposes. The study was conducted in the cardiology outpatient clinic, and each patient was accompanied throughout the process by both a senior cardiologist and an anesthesiologist. Heart rate, blood pressure, and pulse oxymetry were measured every 5 minutes throughout the study. Each subject was connected to the ISPC device (Lympha-press, Mego-Afek AC, Afek, Israel) soon after arriving in the procedure room. Each leg was wrapped in a pneumatic sleeve from the tip of the toes to the proximal thigh below the inguinal region. Each sleeve was composed of 10 air cells, separately connected by an inflation tube to a computerized compressor, aimed to inflate the sleeves sequentially to a maximal pressure of 50 mm Hg, at a rate of 2 cycles/min (separated by a short interval), to enable maximal venous refilling before any successive pneumatic squeeze. Inflation pressure was not adjusted to account for varying body size, and we used the same inflation pressure that was used in our previous studies,[13–15,17] in which we have noted the cardiovascular advantages of the pneumatic sleeves.

As presented in Figure 1, the activation of ISPC lasted 40 minutes. After TTE measurements without ISPC activation, we activated the pneumatic sleeves, and after 5 minutes we started cardiac assessment which lasted 10 minutes. After an additional 15 minutes (with continuing ISPC activation), we performed a second TTE assessment which lasted another 10 minutes (total ISPC activation time 40 min). Each participant served as his or her own control. Echocardiographic measurements were conducted by experienced echocardiography specialists with the use of ultrasound with a 1–5-MHz transducer (iE33, Phillips Medical Systems, Andover, Massachusetts). During assessments, the patients were in the left lateral decubitus position. Echocardiographic parameters were measured in the parasternal long-axis view and by the apical 2- and 4- chamber view and included the velocity time integral (VTI) of the left ventricular outflow tract (LVOT), ejection fraction, cardiac output, stroke volume, peak velocity of early diastolic atrioventricular flow through the mitral valve (E), peak velocity (flow) during atrial diastolic contraction (A), and deceleration of the E-wave. Cardiac output was calculated by multiplying VTI of the LVOT by heart rate. Fractional shortening of the left ventricle was measured by M-mode still frame, and the E/A ratio was calculated. We also measured heart rate, the area of the right and left atria in the apical 4-chamber view, and the systolic and diastolic dimensions of the left ventricle. The dimensions of the left ventricle were estimated by measuring the distance between the interventricular septum and the posterior wall of the left ventricle in the parasternal long-axis view. Moderate and severe pulmonary hypertension (PHT) was defined according to European Society of Cardiology (ESC) guidelines.[18] Moderate PHT was defined as tricuspid regurgitation (TR) velocity of 2.9–3.4 m/s and systolic pulmonary artery pressure (sPAP) of 37–50 mm Hg with or without additional signs of PHT. Severe PHT was defined as TR velocity >3.4 m/s and sPAP >50 mm Hg with or without additional signs of PHT. Severe mitral regurgitation was defined according to recent ESC guidelines.[19] Color tissue Doppler imaging (TDI) in the apical 4-chamber view sampled the septal region of the mitral annulus. TDI analysis (to assess diastolic dysfunction) included peak early diastolic velocity (TDI velocity and Med E′) and the E/Med E′ ratio [(the ratio of early diastolic mitral inflow (E) to early diastolic mitral annular tissue velocity (E′)]. SVR was calculated according to the relationship between mean arterial pressure (MAP), cardiac output, and central venous pressure (CVP; directly reflected by measuring cubital vein pressure through a 17-gauge intravenous cannula). This method has been previously validated in surgical patients.[20]

Statistical Analysis

Statistical analysis was performed by with the use of computerized SPSS version 19 (SPSS, Chicago, Illinois). Quantitative data were expressed by means, medians, and standard deviations. Qualitative data was presented as frequencies and percentages. Paired-sample *t* test or Wilcoxon signed rank test were used to compare measures between time points when appropriate (evaluating the significance of the mean change in echocardiographic parameters before and after activation of the ISPC device). It was assumed that the differences, calculated for each pair, had an approximately normal distribution. Repeated-measures model was used to evaluate changes over time and was appropriate for MAP and CVP. Multiple comparisons were performed (Bonferroni test) for those measures, and trend over time period was presented for the CVP measure. *P* values of <.05 were considered to be statistically significant.

Results

The patients' clinical characteristics are detailed in Table 1. As presented in Tables 1 and 2, the study population included 19 patients (16 male, 3 female), with an overall mean age of 66.8 ± 10.6 years (median 68, range 48–82). At baseline, the mean LVEF by which the patients were detected was 29 ± 9.2% (median 32%, range 10%–40%). Moderate right ventricular (RV) dysfunction was noted in 32% of the patients, and none had severe RV dysfunction. Severe TR was noted in 1 patient and moderate TR in 5 (26%). Five patients

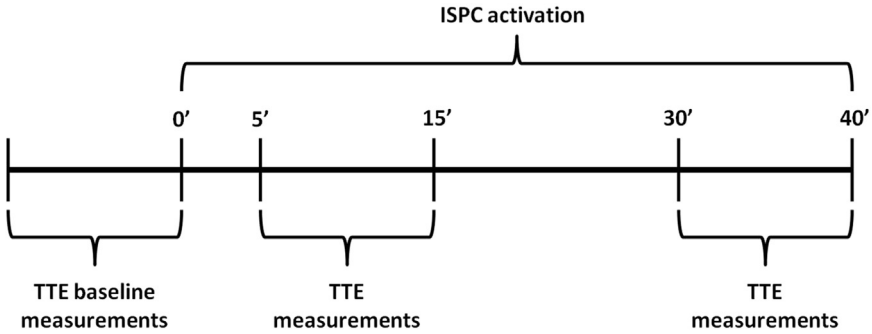

Fig. 1. Timeline of the intermittent sequential pneumatic compression (ISPC) activation study. TTE, transthoracic echocardiography.

(26%) had severe PHT and 4 (21%) had moderate PHT. The participants were patients who were admitted for acute decompensated heart failure (ADHF) after treatment with intravenous diuretics or were admitted electively for device implantation. Therefore, they did not have clinical signs of severe pulmonary congestion (ie, pulmonary rales or crackles) during the study. However, increased jugular venous pressure (JVP) was noted in 21% and lower-extremity pitting edema in 32%. None of the patients had a cardiac resynchronization therapy device. In the setting of our study (the cardiology outpatient clinic), the mean ejection fraction was low, though slightly higher than the preliminary value, and tended to increase mildly after ISPC activation (from 34.74% to 37.84%; $P = .075$; Table 3). A significant increase in cardiac output (from 4.26 to 4.83 L/min; $P = .008$) and stroke volume (from 56.11 to 63.53 mL; $P = .029$) was noted, followed by gradual decline toward baseline over time (Table 3). As presented in Table 4, these changes were not accompanied by a change in heart rate. Improved cardiac function was associated with a significantly reduced SVR (from 1,513.5 to 1,209 dyne-s/cm^5; $P < .001$), which stayed low despite relatively small gradual increment over time. The hemodynamic changes throughout the 3 phases of the study are presented in Figure 2. The mean deceleration time was low (<140 ms), and together with increased E/Med E′ (>15) and mitral valve E/A (>2), points to an additional component of severe diastolic dysfunction (mainly restrictive type). Oxygen saturation decreased slightly without clinical (albeit statistical) significance. Central venous pressure (as was reflected by cubital vein pressure) increased significantly after ISPC activation and stayed high throughout the study. The area of the right and left atrium increased after sleeve activation, reflecting increased venous return (statistically significant regarding the left atrium; Table 3). TDI during sleeve inflation revealed increased Med E′ (predicted cardiac adaptation in response to increased preload). The insignificant changes in E/Med E′ support our assumption that ISPC activation does not lead to compliance derangements or an increase in diastolic abnormalities.

Table 1. Baseline Clinical Characteristics

	n (%)
Age (y)	66.8 ± 10.6
Gender (male)	16/19 (84)
Dilated cardiomyopathy	5/19 (26)
Ischemic heart disease	12/19 (63)
CABG	6/19 (32)
NYHA II	4/19 (21)
NYHA III	15/19 (79)
Stable angina class III*	5/19 (26)
Right heart failure	6/19 (32)
Paroxysmal atrial fibrillation	5/19 (26)
Complete left bundle branch block	8/19 (42)
Peripheral arterial disease	5/19 (26)
Arterial hypertension	17/19 (89)
Diabetes mellitus	11/19 (58)
Hypercholesterolemia	11/19 (58)
Chronic renal failure (eGFR <60 mL/min)	4/19 (21)
Chronic anemia (hemoglobin <10 g/dL)	3/19 (16)
Morbid obesity	1/19 (5)
Chronic medications	
Beta-blockers	15/19 (79)
ACE-I/ARB	16/19 (840)
Spironolactone	5/19 (26)
2 of 3 above medications	19/19 (100)
Physical examination	
Pitting edema of lower extremities	6/19 (32)
Jugular venous distension	6/19 (21)
Dyspnea on minimal exertion	15/19 (79)
Liver enlargement	6/19 (32)

CABG, coronary artery bypass graft; NYHA, New York Heart Association functional class; eGFR, estimated glomerular filtration rate; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

*Canadian Cardiovascular Society classification.

Table 2. Baseline Echocardiographic Findings

	n (%)
Left ventricular ejection fraction ≤40%	19/19 (100)
Left ventricular ejection fraction ≤30%	8/19 (42)
Mean left ventricular ejection fraction	29 ± 9.2%
Moderate or severe RV dysfunction and enlargement	6/19 (32)
Moderate or severe tricuspid regurgitation	6/19 (32)
Moderate or severe pulmonary hypertension	11/19 (58)
Mean systolic pulmonary arterial pressure (by TTE)	49.8 ± 9.2 mm Hg
Moderate and severe mitral regurgitation	7/19 (37)
Restrictive pattern of diastolic function	7/19 (37)
Diastolic dysfunction (any grade)	19/19 (100)

RV, right ventricular; TTE, transthoracic echocardiography.

Table 3. Echocardiographic Changes Before and During Intermittent Sequential Pneumatic Compression (ISPC) Activation

	Before ISPC Activation (1)	During ISPC Activation (2)	During ISPC Activation Cont'd (3)	*P* Value (1−2) 1 Sided; 2 Sided	*P* Value (1−3) 1 Sided; 2 Sided
LVOT VTI (cm)					
Mean	31.75	31.83	—	.478*;	—
SD	17.38	16.32		.956	
Median	26.7	25.8			
EF (%)					
Mean	34.74	37.84	—	.0755*;	—
SD	8.27	13.24		.151	
Median	35	37			
FS (%)					
Mean	18.89	18.95	—	.441†;	—
SD	4.47	6.51		.882	
Median	18	18			
CO (L/min)					
Mean	4.26	4.83	4.311	.008†;	0.395*;
SD	1.45	1.46	1.4	.015	0.79
Median	4	5.2	4.2		
SV (mL)					
Mean	56.11	63.53	56.63	.029†;	0.4325*;
SD	19.96	22.83	21.49	.059	0.865
Median	52	66	52		
MV peak E (cm/s)					
Mean	87.16	88.34	—	.159*;	—
SD	22.99	25.18		.318	
Median	90.7	94.3			
MV peak A (cm/s)					
Mean	52.81	54.79	—	.159*;	—
SD	27.22	29.475		.318	
Median	50.3	50			
Decel time (ms)					
Mean	87.83	90.24	—	.13†;	—
SD	32.75	29.42		.259	
Median	87.9	92			
MV E/A					
Mean	2.13	2.18	—	.066†;	—
SD	1.19	1.24		.132	
Median	2.2	1.7			
Med E′ (cm/s)					
Mean	3.8	4.17	—	.212†;	—
SD	1.66	1.76		.424	
Median	3.51	4.09			
E/Med E′					
Mean	21.03	22.68	—	.109*;	—
SD	8.74	8.03		.218	
Median	22.4	22.3			
Area, RA					
Mean	19.46	20.27	—	.1015*;	—
SD	4.35	5.13		.203	
Median	18.9	18.2			
Area, LA					
Mean	28.03	30.9	—	.007*;	—
SD	8.24	7.99875		.014	
Median	27.5	29.7			

CO, cardiac output; Decel, deceleration; E, early diastolic mitral inflow; EF, ejection fraction; FS, fractional shortening; LA, left atrial; LVOT, left ventricular outflow tract; Med E′, early diastolic mitral annular tissue velocity; MV, mitral valve; MV peak A, flow during atrial systole; MV peak E, early diastolic atrioventricular flow; RA, right atrial; SV, stroke volume; TDI, tissue Doppler imaging; VTI, velocity time integral.

*Paired-sample *t* test.

†Wilcoxon signed rank test.

Discussion

In this study we demonstrated that there was no deterioration in cardiac function (transient or constant) during activation of the ISPC leg sleeves in patients with chronic CHF (systolic and diastolic). The main early hemodynamic changes included increased cardiac output, ejection fraction, and stroke volume, together with reduced SVR. However, toward the end of ISPC activation, cardiac function returned to baseline. Nevertheless, cardiac dysfunction did not worsen during ISPC activation, as was verified by insignificant changes in deceleration time, E/Med E′, and mitral valve E/A ratio as well as by TDI findings (diastolic dysfunction and cardiac compliance were not worsened). In addition,

Table 4. Hemodynamic Changes Before and During Intermittent Sequential Pneumatic Compression (ISPC) activation

	Before ISPC Activation (1); n = 19	During ISPC Activation (2); n = 19	During ISPC Activation Cont'd (3); n = 19	*P* Value (1−2) 1 Sided; 2 Sided	*P* Value (1−3) 1 Sided; 2 Sided
HR					
Mean	75.53	75.58	75.68	.277†;	.444*;
SD	13.43	12.68	11.81	.554	.888
Median	80	77	80		
SD	8.24	7.99875			
Median	27.5	29.7			
MAP (mm Hg)					
Mean	83.26	79.58	82.05	.0315*	.2325*
SD	12.64	10.83	11.66	.063	.465
Median	80	79	82	.190‡	1.00‡
PVP-CVP (cm H_2O)					
Mean	11.21	15.32	16.11	.001*;	.001*;
SD	4.77	4.93	5.91	.001‡	.001‡
Median	11	14	15		
SVR (dyne-s/cm^5)					
Mean	1520.26	1216.26	1432.21	.0005*;	.013†;
SD	599.93	557.785	836.09	.001	.027
Median	1554	1028	1291		
SD	8.74	8.03			
Median	22.4	22.3			
Sat%					
Mean	95.11	93.84	94.47	.005†;	.012*;
SD	2.38	2.69	2.34	.009	.024
Median	95	94	95		
SD	1.19	1.24			
Median	2.2	1.7			

HR, heart rate; Sat, saturation; MAP, mean arterial pressure; PVP-CVP, large peripheral vein pressure representing central venous pressure; SVR, systemic vascular resistance.

*Paired-sample *t* test.

†Wilcoxon signed rank test.

‡Bonferroni test.

the increased venous return (as demonstrated by elevated CVP and the area of both atria), was not accompanied by adverse pulmonary effects (no clinical, auscultative, or oxidative worsening was noted). The significant decrease in SVR throughout ISPC activation represents an important hemodynamic functional advantage, especially in CHF patients. Actually, the use of pneumatic leg sleeves has led to decreased afterload and to increased preload by "milking" blood from the lower venous system, thereby improving cardiac output without an accompanying increase in heart rate.

Currently, pneumatic sleeves are in very wide use in postoperative patients (to prevent venous stasis and pulmonary embolism), as well as in patients suffering from venous insufficiency and lymphatic stasis, or during prolonged laparoscopic procedures. The use of ISPC sleeves has been shown to reduce adverse cardiovascular as well as metabolic abnormalities during PP. As such, it became crucial to assess the use of pneumatic sleeves in a subpopulation of patients with CHF, as the mechanism of action of ISPC action may aggravate the clinical symptoms of CHF due to increased pulmonary blood flow and possibly increased SVR. It should be emphasized that we did not intend to offer a treatment alternative to CHF or reduce the risk of cardiac-related events in patients with CHF or other cardiac conditions. The sole purpose of our study was to investigate the cardiovascular effects following activation of 10-cell ISPC sleeves in patients with CHF and to assess its safety (while preventing venous stasis and adverse cardiovascular effects following surgery), as measured by echocardiography, with possible implications for postoperative populations.

The current data on the hemodynamic effects of the various types of pneumatic sleeves are not conclusive, especially regarding the pathophysiologic changes in elderly and cardiac patients. For example, a nonsequential constant mode of action (persistent pressure) of pneumatic trousers (such as the medical antishock garment) did not influence cardiac output and increased SVR.[21,22] In addition, activation of nonsequential intermittent pneumatic compression on the legs of patients in the surgical intensive care unit did not significantly change the mean cardiac output as measured by the thermodilution technique.[23,24] On the other hand, application of intermittent sequential compression stockings in a cardiac patient led to augmented venous return and increased pulse pressure, suggesting increased stroke volume.[25] Comparing modes of activation, it was shown that a sequential mode of pneumatic sleeve led to a better augmentation of venous blood flow than nonsequential solitary air cuff around the legs.[26] However, a 3-cell sequential pneumatic sleeve device was reported to reduce cardiac output and to increase SVR in healthy volunteers.[16] The use of the enhanced external counterpulsation (EECP) device

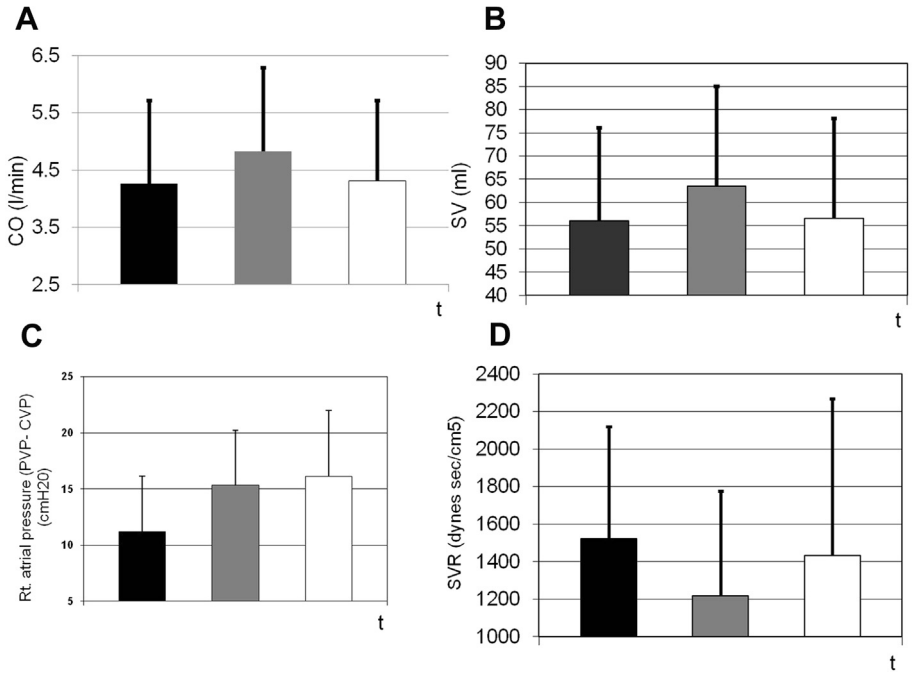

Fig. 2. The change in (A) cardiac output (CO), (B) stroke volume (SV), (C) right atrial pressure, and (D) systemic vascular resistance (SVR) at baseline (*black bar*), end of phase 1 (*gray bar*), and end of study (*white bar*). PVP-CVP, large peripheral vein pressure representing central venous pressure.

was shown to improve cardiac performance by increasing venous return and diastolic filling, decrease left ventricular end-diastolic pressure, augment cardiac output and coronary flow, and decrease SVR.[27–30] However, EECP is a complex and expensive device that requires precise synchronization with heart rate and electrocardiography for its action, and it works with much higher cuff pressures (up to 300 mm Hg). Moreover, EECP is not applicable in postoperative patients. In another study, intermittent pneumatic compression boots (in a high pressure, pulsatile, and nonsequential mode on the plantar foot aspect) in patients with CHF led to decreased SVR and pulmonary capillary wedge pressure without affecting cardiac output and stroke volume.[31] The activation of ISPC sleeves (as used in our study) improved cardiac function (increased cardiac output and stroke volume and decreased SVR), augmented visceral perfusion, and reduced oxidative stress during laparoscopic operations.[1,14,32]

In an attempt to elucidate the cardiovascular effects of the activation of ISPC, we recently studied the effect of such sleeves in healthy volunteers and showed improved preload as well as afterload parameters.[17] Compared with the present study, we found similar (though temporary) increase in cardiac output, stroke volume, and ejection fraction. It should be noted that the impact of ISPC on systolic function was more pronounced in healthy subjects, as the left ventricular outflow tract velocity-time integral and fractional shortening parameters increased during ISPC.[17] However, in both studies, SVR decreased significantly during the study, which has important physiologic impact in CHF patients.

The suggested mechanism of action of ISPC is rather complex. The increase in preload may be due to the 'milking' effect and an increase in venous return as manifested by the echocardiographic parameters, and results in increased ejection fraction, stroke volume and cardiac output. Using TDI to detect myocardial strain, the Med E′ and E/Med E′ parameters suggest adequate cardiac adaptation in response to the increase in preload, without worsening diastolic dysfunction.[33–35]

We found a significant reduction in afterload (SVR) during ISPC activation. Whether the mechanism resembles that of EECP remains speculative, as ISPC activation does not require ECG synchronization.[27–29] The effect of SVR reduction with ISPC was persistent in all our previous studies, with and without PP.[1,15,17] Animal studies suggest that the activation of ISPC leg sleeves increased blood flow, possibly owing to an increased secretion of nitric oxide from the vascular endothelium, causing vasodilatation and subsequently decreased SVR. Nitric oxide inhibitors were shown to block such hemodynamic improvement, thus confirming this proposed mechanism of action.[36–38] An additional explanation for afterload reduction leading to improved contractility is suggested from our previous study, in which ISPC decreased sympathetic autonomic activity during laparoscopic operations.[15] The mechanism underlying the late decline in the hemodynamic effect of ISPC is unclear, and neurohormonal activation is one plausible mechanism. We believe that the early effect of ISPC is a result of an increase in cardiac preload, reaching a "steady state" in our next measurement. We therefore assume that continuing ISPC activation for a longer duration would not have increased CO or other hemodynamic measurements.

Study Limitations

The present study is somewhat limited by the small number of patients and the relatively short duration of our examinations. The patients did not undergo the anesthetic and physiologic insult associated with a surgical procedure, so the data may not be extrapolated to ''real surgical'' patients. As stated in the Methods section, we did not study postoperative patients owing to possible inconvenience during mobilization for examinations, yet we think that our study population is representative of these patients as well. In addition, the optimal pneumatic pressure to be used in ISPC is as yet undetermined and remains arbitrary, and our results may not be applicable to other pneumatic devices. We did not use invasive hemodynamic monitoring, although in the heterogeneous population of patients with heart failure it is pivotal to have a better understanding of the hemodynamic situation of each patient before, during, and after ISPC treatment, especially regarding the relationship between filling pressures and pulmonary wedge pressure. Yet the use of invasive monitoring would have made patient recruitment to a research protocol nearly impossible. A longer duration ($>$40 min) could have resulted in a more stable hemodynamic effect of ISPC activation. However, we think that our last measurements show that there was no deleterious effect of ISPC in such a severely ill group of patients. This technique should probably be used with caution in patients with high right atrial pressures, severe TR, or severe RV systolic dysfunction, because abruptly increasing venous return does not translate to improvement in LV stroke volume and cardiac output, despite the decrease in systemic vascular resistance.

Conclusion

The extensive use of pneumatic sleeves in medical procedures, together with the increasing number of elderly patients, as well as patients with CHF who undergo surgery, makes the selection of the pneumatic device highly important for improved clinical outcome. We have demonstrated that 10-cell ISPC leg sleeves may be safely used in patients with CHF (systolic as well as diastolic), and may also improve cardiac function through changes in preload and afterload parameters, without causing reactive tachycardia.

Disclosures

None.

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