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Use of a Venous Assist Device After Repair of Complex Lesions of the Right Heart

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Sixteen patients underwent hemodynamic evaluation of a venous assist device after complex operations on the right side of the heart. The device consists of an inflatable abdominal binder attached to a Jobst extremity pump causing intermittent external compression of the abdomen. In addition, six of these patients were evaluated using total lower body compression for comparison. Modifications of the Fontan procedure were performed in 14 patients, mitral valve anuloplasty and tricuspid valve replacement in 1 patient and reconstruction of the right ventricular outflow tract for treatment of pulmonary atresia with intact septum in 1 patient. The patients' ages ranged from 23 months to 31 years (mean 10.7 ± 1.8 years). Systemic blood pressure, right and left atrial pressures, heart rate and arterial-mixed venous oxygen saturation difference were recorded in each patient with and without the device in place.

With the venous assist device, mean systolic pressure increased from 95 ± 4 to 122 ± 3 mm Hg ($p < 0.05$) and diastolic pressure rose from 57 ± 3 to 70 ± 3 mm Hg ($p < 0.05$). Left atrial pressure increased from 7 ± 1 to 15 ± 1 mm Hg and right atrial pressure from 15 ± 1 to 23 ± 1 mm Hg (both $p < 0.05$). In addition, arterial-mixed venous oxygen saturation difference decreased from 29% without the device to 23% with the device in place ($p < 0.05$). Total lower body compression gave similar results to intermittent abdominal compression alone.

Use of the venous assist device improves postoperative circulatory performance in patients after complex procedures on the right side of the heart. Cardiac filling pressures and systemic blood pressure rose while peripheral perfusion was improved. The ease and effectiveness of application of abdominal compression alone offer some advantage over the previously described technique of total lower body compression.

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In 1958, Glenn (1) reported on the first successful superior vena cava to right pulmonary artery shunt. This confirmed their experimental work which showed that a lung with normal pulmonary vascular resistance can be perfused without the benefit of a pumping chamber. In 1971, Fontan and Baudet (2) went a step further and reported the successful diversion of both superior and inferior vena cava blood to the lungs by a right atrial to left pulmonary artery connection combined with a Glenn shunt. It was soon recognized that although the procedure successfully separated the pulmonary and systemic circulations, it was not a physiologic connection because of the venous hypertension that inevitably accompanies this procedure (3). Although this is well tolerated in carefully selected patients (4–6), it frequently results in fluid accumulation in the pleural, peritoneal or pericardial cavities in the early postoperative period. Some patients may also develop a low output state and require large infusions of crystalloid or colloid solutions to achieve the venous pressure necessary to maintain the cardiac output.

Heck and Doty (7) showed experimentally that external intermittent lower body compression after the Fontan procedure increased cardiac output by improving venous return from blood pooled in the abdomen and legs, and by preventing sequestration of extravascular fluid in the peritoneal cavity and lower body. We have modified the technique by using abdominal compression only and studied its effects on 16 patients, including those undergoing the Fontan procedure and those with initially impaired right ventricular function.

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Methods

Study patients (Table 1). Between June 1983 and June 1984, sixteen patients underwent an operation known to alter right-sided hemodynamics. The patients' ages ranged from

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Table 1. Hemodynamic Data in 16 Patients With Abdominal Compression

	Systolic BP		Diastolic BP		LA Pressure		RA Pressure		A-VO_2 Diff	
Case	Off	On	Off	On	Off	On	Off	On	Off	On
1	135	152	80	90	8	14	12	20	25	21
2	82	100	45	50	3	10	3	11	16	12
3	100	126	43	55	8	15	20	26	60	40
4	66	110	50	70	10	21	21	33	27	20
5	80	110	50	65	4	11	10	18	39	24
6	88	98	46	47	10	12	18	22	22	25
7	67	108	52	60	10	17	17	23	23	18
8	102	122	63	80	7	12	14	21	18	14
9	100	120	67	78	0	9	11	18	18	23
10	80	109	50	65	6	18	16	30	40	31
11	100	131	55	77	11	21	16	24	34	33
12	118	136	60	66	3	11	12	19	24	21
13	73	97	41	57	17	29	20	32	35	27
14	83	122	47	62	9	15	19	24	59	29
15	77	120	49	80	—	—	17	28	19	14
16	105	130	71	80	5	9	14	17	31	22
Mean	91.0	118.2	54.3	67.6	7.4	14.9	15.0	22.9	30.6	23.3
SEM	4.6	3.6	2.7	3.0	1.0	1.3	1.1	1.4	3.3	1.8

A-VO_2 Diff = arterial-venous oxygen saturation difference (%); BP = blood pressure; LA = left atrial; RA = right atrial. All values except last column in mm Hg.

23 months to 31 years (mean 10.7 ± 1.8 years); eight patients were male and 8 were female. The preoperative diagnosis was univentricular heart in 11, tricuspid atresia in 3, pulmonary atresia with intact ventricular septum in 1 and Ebstein's anomaly in 1. A modified Fontan procedure, as described previously (4), was performed in 14 patients, including 2 who had concomitant value replacement. One patient had a mitral valve anuloplasty with tricuspid valve replacement and one had reconstruction of the right ventricular outflow tract. All patients were operated on under cardiopulmonary bypass with moderate hypothermia (28°C) and intermittent blood cardioplegic myocardial protection. Nine patients required inotropic support postoperatively.

Hemodynamic measurements. After surgery, patients were transported to the intensive care unit where stabilization was achieved over 1 to 2 hours with transfusion of fluids and blood products. Hemodynamic variables recorded during a control period included heart rate, systolic and diastolic blood pressure and both right and left atrial pressures. Simultaneous measurements of radial artery and mixed venous blood gases were obtained. The patients were then placed in a venous assist device for external compression: a MAST (Military Anti-Shock Trousers) suit in older patients, an inflatable boot that compressed both the lower limbs and the abdomen in smaller-sized patients or a modified device that consisted of an extra large blood pressure cuff placed around the abdomen just below the costal margin. Several patients underwent testing with more than one device for comparison. Each device was attached to a Jobst extremity pump and inflated to a pressure of 30 to 45 mm Hg for a period of 30 seconds and subsequently deflated for 15 seconds (Fig. 1).

After the device had been functioning for 30 minutes, all measurements were repeated. Continuous tracings of heart rate, blood pressure and right and left atrial pressures were obtained (Fig. 2). During the test period, no changes were made in respirator settings or inotropic support, and fluid administration was kept at maintenance levels. Most patients were able to discontinue use of the venous assist device at the end of the test period. However, six patients required continuous support for 6 to 78 hours despite the use of inotropic drugs.

Statistical analysis was performed by Student's t test for paired variables.

Results

Hemodynamic effects of venous assistance. Sixteen patients underwent testing with intermittent abdominal compression. In six of these patients, hemodynamic variables were also recorded during an additional period of intermittent lower body compression with either the MAST suit or the lower extremity boot. Thus, a total of 22 trials were available for analysis.

The hemodynamic data for each patient are summarized in Table 1 and those for the entire series in Table 2. All patients survived the early postoperative period and were able to be weaned from inotropic support. Systolic arterial pressure increased from 10 to 47 mm Hg (mean 26.9 ± 2.3), an average of 28.2% above preassistance control pres-

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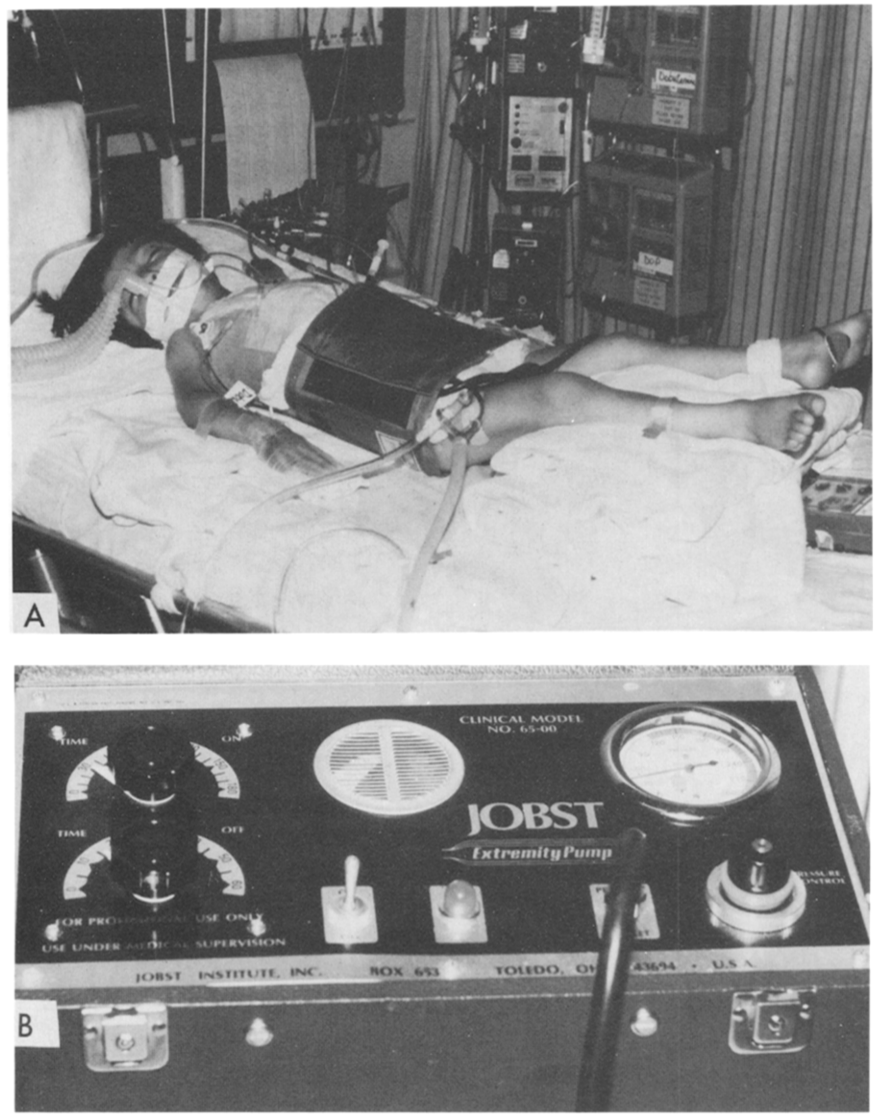

Figure 1. **A**, Modified venous assist device using abdominal compression only. A large blood pressure cuff is placed around the patient's abdomen and the legs are elevated. **B**, The Jobst extremity pump. The dials at the left allow precise timing of inflation and deflation while an internal pressure gauge monitors inflation pressure.

2.3), an average of 28.2% above preassistance control pressures ($p < 0.005$); diastolic pressure increased by 23.8%, a mean of 13.5 ± 1.4 mm Hg above the control value ($p < 0.005$). Right atrial pressure was increased from 3 to 12 mm Hg (mean 7.8 ± 0.6), an average of 52.6% over the control measurement ($p < 0.005$). Left atrial pressure increased to a similar degree, a mean of 7.6 ± 0.6 mm Hg (range 2 to 12), which represented a 111% elevation above the control value ($p < 0.005$). Most important, however, the arteriovenous oxygen saturation difference decreased with the institution of the venous assist device from 29.2 ± 2.6 to 23 ± 1.4% ($p < 0.05$). This difference is primarily related to an increase in the mixed venous oxygen saturation from 67.4 ± 2.9 to 74.0 ± 1.8% with assistance, while arterial saturation remained relatively constant. Presuming that institution of external compression did not effect a change in body oxygen consumption, this can be interpreted as evidence for an increase in cardiac output and peripheral perfusion. Although this increase in venous oxygen saturation could have resulted from arteriovenous shunting in the face of an unchanged cardiac output, this was considered unlikely without changes in clinical indicators of peripheral perfusion such as blood pH, limb skin temperature and urinary output. In addition, heart rate in the 13 patients who were not being paced in the acute postoperative period decreased from 112 ± 8 to 105 ± 7 beats/min with the venous assist device ($p < 0.001$).

All patients in the study had continuous intraarterial pressure monitoring using the radial artery. In five patients, pressure was also recorded by cuff and Doppler measurements in a lower limb during compression. In these patients, a similar increase in arterial pressure was noted at both sites, suggesting that the augmentation was not secondary to compression of the abdominal aorta and thus limited to the arms.

Prolonged use of venous assist. Six patients in this series were dependent on the venous assist device in the

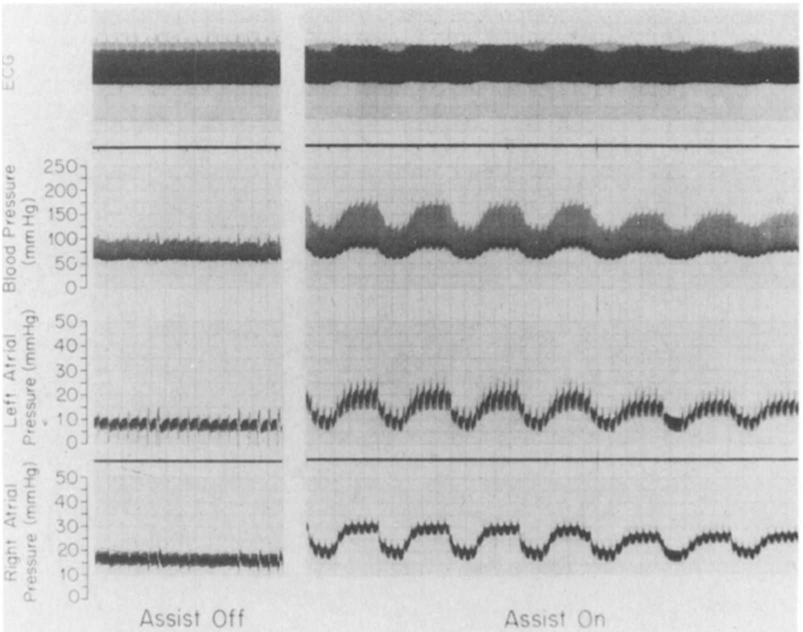

Figure 2. Typical tracing obtained in a postoperative patient before and during use of the venous assist device. Note that the device provides augmentation of the systemic and atrial pressures above control level even during periods of deflation. The device was inflated for 30 seconds and deflated for 15 seconds in each cycle.

early postoperative period and its use was continued from 6 to 78 hours. Dependence was defined as inability to maintain adequate peripheral perfusion and a systolic blood pressure above 80 mm Hg despite simultaneous infusion of dopamine and dobutamine after discontinuing the assist device. All patients were subsequently weaned from the device without complications. A single patient in this group required discontinuation of the assist device after 6 hours because of a precipitous decline in urinary output. This occurred despite appropriate improvement in overall hemodynamic profile. Urinary output increased after discontinuation of assistance, although the patient remained in a low output state over the next several hours. This represents the single example of a possible deleterious effect of intermittent external compression.

Fluid balance. In the six patients who required prolonged use of the venous assist device in the immediate postoperative period, fluid intake and output were carefully recorded. Five patients continued to have negative fluid balance while the device was in place over a period of 6 to 78 hours. This occurred even though all five patients had been hypotensive in the intensive care unit and were kept on the device to maintain a systolic pressure above 80 mm Hg, a situation that often prompts vigorous fluid resuscitation. Comparison of fluid requirements during the equivalent time period for each patient beginning at the time the device was discontinued (when a more stable state was achieved and patients no longer required assistance to maintain adequate perfusion) revealed that total fluid intake was similar between the two time periods. Thus, excessive fluid administration was avoided during the critical immediate postoperative period and the need for postoperative fluid mobilization diminished.

Table 2. Summary of Hemodynamic Changes After Application of the Venous Assist Device in 16 Patients

	Off(mm Hg)	On(mm Hg)	%Δ
Systolic BP	95 ± 4	122 ± 3	+28*
Diastolic BP	57 ± 3	70 ± 3	+24*
Pressure			
LA	6.8 ± .85	14.5 ± 1.0	+111*
RA	14.8 ± 1.0	22.7 ± 1.0	+53*
A-VO_2 Diff	29.2%	23.2%	−27%*

*$p < 0.05$. Abbreviations as in Table 1.

Comparison of abdominal and lower body compression (Table 3). Six patients underwent hemodynamic testing in a sequential fashion using first abdominal compression and then lower body compression with the MAST or boot device. Possible differences related to the order of use were excluded by performing a separate control measurement for each test period. Results from this fairly small number revealed a tendency toward slightly greater increases in filling pressure and blood pressure in the lower body compression group. However, comparison of the change in arteriovenous oxygen difference between groups showed a slightly larger reduction in saturation difference in the abdominal compression group (20.2% versus 22.8%, $p = 0.09$). None of the differences were statistically significant.

Discussion

Continuous external counterpressure. External counterpressure by means of a pressurized suit was first used by Crile (8) in 1903 to manage surgical patients who were

Table 3. Comparison of Hemodynamic Effects of Abdominal Compression Versus Lower Body Compression in Six Patients

	Systolic BP		Diastolic BP		LA Pressure		RA Pressure		$A-VO_2$ Diff	
Case	Off	On	Off	On	Off	On	Off	On	Off	On
					Abdominal compression					
8	102	122	63	80	7	12	14	21	18	13
12	118	136	60	66	3	11	12	19	24	21
14	83	122	47	62	9	15	19	24	59	28
15	77	120	49	80	—	—	17	28	19	14
1	135	152	80	90	8	14	12	20	25	21
9	100	120	67	78	0	9	11	18	18	23
Mean	102.5	128.7	61.0	76.0	5.4	12.2	14.2	21.7	27.2	20.2
SEM	8.1	4.8	4.5	3.8	1.5	1.0	1.2	1.4	5.8	2.1
					Lower body compression					
8	107	136	70	90	4	15	13	26	16	15
12	115	137	58	77	3	10	11	16	24	21
14	86	133	47	56	8	15	19	24	40	32
15	92	110	60	74	5	13	15	24	27	24
1	125	155	75	92	12	18	18	25	25	25
9	116	126	74	80	1	9	11	18	19	21
Mean	106.8	132.8	64.0	78.1	5.5	13.3	14.5	22.2	25.2	22.8
SEM	5.6	5.5	4.1	4.8	1.5	1.3	1.3	1.5	3.0	2.1

Abbreviations as in Table 1.

hypotensive because of excessive blood loss. This technique was little used, however, and it was not until years later that it gained popularity in the control of hemorrhagic shock during emergency resuscitation (9,10). This followed several experimental studies (11–13) indicating prolonged survival and decreased blood loss from arterial and venous injuries produced in dogs. The use of external counterpressure seemed to be well tolerated and was shown to increase mean arterial blood pressure, pulmonary artery blood volume and stroke volume (14). Augmentation of cardiac index was not reliably produced and appeared to be a transient phenomenon (15). The beneficial effects have been hypothesized to be due to a redistribution of cardiac output and an "autotransfusion" from the periphery to the central blood volume. However, this has recently become controversial (16), and it has been shown that the primary effect of continuous counterpressure may be a simple elevation in systemic vascular resistance and that deleterious effects on tissue metabolism may occur in the hypovolemic state (17). The increase in afterload would make use of prolonged counterpressure detrimental in conditions of hypotension related to depressed cardiac performance. Intermittent counterpressure, as used in this series, elevated arterial pressure in association with laboratory and clinical signs of improved tissue perfusion, indicating that the mechanism involved increased cardiac output rather than increased peripheral resistance.

Pulsed external counterpressure. In 1963, several investigators (18–20) independently reported success in achieving augmentation of cardiac output through application of pulsed counterpressure to the body in synchrony with cardiac pumping. External counterpulsation during diastole compresses the venous system in a pumping action that displaces peripheral blood rapidly to the central venous pool providing a transfusion effect. In addition, retrograde displacement of arterial blood provides a potential arterial stroke volume of 100 to 200 cc in a fashion similar to the invasive intraaortic balloon pump (21). Although these techniques are somewhat cumbersome, beneficial effects have been reported in acute myocardial infarction, with or without cardiogenic shock (22,23).

Use of external counterpressure to augment venous return. In 1977, Hellberg et al. (24) introduced a new indication for synchronous external counterpulsion. This group used the technique primarily as a right-sided assist to reverse low output after physiologic repair of tricuspid atresia. They recorded pressure waves of 25 to 50 mm Hg in the inferior vena cava and suggested that the assist device acted as a "peripheral pump ventricle" to augment pulmonary blood flow and increase left ventricular end-diastolic pressure. Fearing that these pressure waves act to impede venous return from the head and splanchnic circulation, Heck and Doty (7) conducted experiments using phasic external compression. Mongrel dogs underwent tricuspid valve closure with creation of a right atrial to pulmonary artery anastomosis. Lower limb and abdominal compression to a pressure of 60 mm Hg was instituted immediately postoperatively in cycles of 6 to 8 compressions/min. They were able to demonstrate increases in cardiac output, mean arterial pressure and right and left atrial pressures when compared with values in a control group of animals.

Heck and Doty (7) subsequently applied a similar technique in nine patients after they had undergone the Fontan procedure. Phasic external compression was applied from the ankles to the costal margin to a pressure of 45 to 50 mm Hg for 45 seconds followed by release for 15 seconds. Compression was initiated in the operating room as cardiopulmonary bypass was terminated. Increases in hemodynamic variables in the patient group were similar to those seen in the experimental animal group. Two patients had cardiac output determinations during compression and showed increases of 15 and 21%, respectively. These investigators hypothesized that the beneficial effects of the venous assist device were attributable to relocation of peripherally sequestered fluid to the central circulation. Our results support this concept and indicate that total lower body compression may not be necessary to achieve similar hemodynamic improvement.

Effects of intermittent abdominal compression. Because hepatic enlargement and ascites secondary to passive venous congestion appear earlier than peripheral edema with postoperative right heart failure, we evaluated the effect of compressing the abdomen alone in addition to that of the more cumbersome approach of compressing the whole lower body. Our data indicate that an inflatable abdominal binder with leg elevation produces hemodynamic improvement equivalent to that seen with pneumatic trousers. Although there was a suggestion of slightly greater increases in filling pressures and systemic pressure in the group with lower body compression, this was not reflected by a greater change in arteriovenous oxygen saturation difference when compared with values in those patients undergoing intermittent abdominal compression only. Presumably, compression of the entire lower body produced a greater increase in peripheral vascular resistance that may have offset the increase in cardiac output in terms of oxygen delivery. Thus we have a simplified technique that does not require a specially manufactured suit for the pediatric patient and that can be applied quickly and easily in the intensive care unit so as to avoid additional cumbersome equipment in transport.

In a similar study, Guyton et al. (25) used a ventilator reservoir bag connected to a volume ventilator that compressed the abdomen (from xiphoid to knees) up to a pressure of 50 cm water 10 to 20 times per minute. Fourteen postoperative pediatric patients underwent compression for 2 to 43 hours. As in the present series, these investigators demonstrated a consistent increase in right atrial filling pressure in association with an elevation in mean arterial pressure similar to that observed in patients having total lower body compression. In addition, their study demonstrated a net negative fluid balance in survivors during the first 12 hours of compression, similar to that seen in our patients requiring abdominal compression for prolonged periods.

Physiologic mechanism of a venous assistance. Hemodynamic studies of patients undergoing right heart bypass procedures of the Fontan type have shown that few patients demonstrate an effective pumping mechanism for passage of blood into the pulmonary circulation (4,26). Most, instead, depend on elevated right-sided filling pressures to provide for passive flow into the pulmonary artery. The situation is somewhat analogous to that seen in isolated right heart failure in which low cardiac output results from poor performance of the right ventricle after right ventricular infarction. Goldstein et al. (27) in an experimental model have shown improvement in aortic pressure and cardiac output after right ventricular infarction by simple volume loading to raise right-sided filling pressures and augment left ventricular preload. Clinically, volume expansion is usually required in the early postoperative period in patients with compromised right-sided function after the modified Fontan procedure. Transient elevation of intraabdominal pressure by external compression would theoretically empty the congested liver and splanchnic bed into the central circulation in anterograde fashion, because venous valves prevent retrograde flow.

An additional mechanism is suggested by the report of Pinsky and Summer (28) in which patients subjected to abdominal and chest wall binding during positive pressure ventilation showed improved cardiac output without changes in left-sided filling pressure. They postulated that phasic increases in intrathoracic pressure produced by this mechanism reduced transmural left ventricular pressure and lowered left ventricular afterload. It is not known to what extent this contributed to the results in our series. The patients in the present study had significant elevation in left-sided filling pressures and arterial blood pressure with intermittent abdominal compression alone. That this represents a true increase in forward flow is suggested by the maintenance of indicators of peripheral perfusion such as urine output and skin temperature, and by lowering of the arteriovenous oxygen saturation difference. This would be unlikely if the pressure elevation was due to a simple increase in peripheral vascular resistance as has been suggested with the pneumatic compression suits under constant inflation.

Effect of circulatory volume. Kashtan et al. (29) performed an elegant series of experiments with dogs to define the effects of increased abdominal pressure on venous return and cardiac function. They found that increasing abdominal pressure to 40 mm Hg by infusing fluid into the abdomen of anesthetized dogs increased cardiac output only in animals previously made hypervolemic. Venous return was in fact compromised in hypovolemic and normovolemic subjects, resulting in proportionate decreases in cardiac output. Although the model used continuous rather than intermittent elevation of abdominal pressure, the findings emphasize that the hypervolemic state is best suited for optimal response to the application of artificial increases in intraabdominal pressure. Our data, to a certain extent, support this concept. As can be seen in Table 1, those patients with the highest right atrial pressures without the assist device seemed to show the greatest reduction in arteriovenous oxygen saturation differences. A notable exception can be seen in Patient 6, who had a decrease in mixed venous saturation after application of the assist device. This patient had excessive mediastinal bleeding that caused progressive hypovolemia during the testing period. In fact, this patient was dependent on the assist device and eventually required reexploration for bleeding. Whether the pressures generated by the assist device contributed to the excessive bleeding is subject to speculation.

Complications. Only one patient in this study demonstrated a marked decrease in urinary output after institution of the venous assist device. This occurred despite appropriate increases in filling pressures and a significant decrease in arteriovenous oxygen saturation difference, suggesting an adequate cardiac output. Experimental (30,31) and clinical (32) reports have suggested that elevated intraabdominal pressure may have adverse affects on renal perfusion despite efforts to maintain cardiac output. Again, these studies involved continuous elevation of abdominal pressure. We have not been able to determine why oliguria occurred only in this patient, but note that the condition was reversed by discontinuation of the assist device. This suggests that renal function should be closely monitored in all patients on whom the assist device is applied. Hematuria was reported in 12 of 14 patients in Guyton's series (25) and resolved with termination of compression. None of our patients had this complication.

Indications for venous assistance. The majority of carefully selected patients who have undergone the Fontan procedure do not require the venous assist device and maintain adequate cardiac output and urine production without the need for excessive fluid administration. Some patients, however, who have an elevated right atrial pressure (usually above 17 mm Hg), lose fluid to the extravascular space. In this group the venous assist device is valuable in maintaining adequate right- and left-sided filling pressures without excessive fluid administration. Usually this phase resolves after 12 to 24 hours and the diuresis persists without the use of the assist device. This may be due to improved ventricular function and a reduction in the pulmonary vascular resistance.

Finally, two patients in our series underwent procedures other than the modified Fontan operation. The first was a patient with Ebstein's anomaly who had a tricuspid valve replacement combined with mitral valve anuloplasty, and the second was a patient with pulmonary atresia with intact ventricular septum who had undergone reconstruction of the right ventricular outflow tract. Both patients were given a trial of intermittent abdominal compression when a low output state persisted despite large volumes of intravenous fluid administration and high dose inotropic support. In each case, the venous assist device provided valuable support to the circulation and was used for a period of 7 and 26 hours, respectively. This suggests that the venous assist device need not be limited to patients undergoing the Fontan procedure, but may be beneficial to other patients having complex operations whose right ventricular function is compromised postoperatively.

Conclusions. The venous assist device is a useful adjunct in the postoperative management of patients with altered right-sided hemodynamics, particularly those undergoing the Fontan procedure. Its application in the acute postoperative period augments right-sided filling pressures and improves left-sided hemodynamics during the period of circulatory adjustment of the venous and pulmonary vascular beds after right heart bypass procedures. The primary mechanism of action appears to involve peripheral venous blood displacement to the central circulation. The effectiveness and ease of application of abdominal compression offers some advantage over the previously described technique using specially designed pneumatic trousers for lower body compression.

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