The mechanism for this right ventricular filling pattern has not been clearly studied. The intraatrial baffle used in physiologic surgical repair of transposition of the great arteries limits the invasive and noninvasive study of right ventricular diastolic function. Catheterization of the neopulmonary venous atrium may be difficult in the absence of a baffle leak and requires arterial catheterization. Furthermore, the alternative represented by the echo-Doppler is confronted with the absence of a specific control group because the right ventricle receives pulmonary venous blood from a surgically elongated atrium. The right ventricular filling pattern that we documented could be a result of abnormal ventricular relaxation and decreased operative compliance due to an increased modulus of chamber stiffness. Because we did not obtain simultaneous invasive hemodynamic data, we could not establish which factors were the most important. However, studies of left ventricular diastolic Doppler flow profiles[3] indicate that, in our patients, reduction in right ventricular chamber compliance with elevated filling pressure and the larger size of the tricuspid valve orifice seem to be the dominant factors that partially overwhelm the effect of a reduced rate of ventricular relaxation. The consequence of high systolic and low systolic pressures in the right and left ventricles, respectively, is an abnormal ventricular geometry with a dilated and hypertrophied right ventricle.[7] This abnormal ventricular geometry with an increased right ventricular mass is certainly responsible for an increased ventricular stiffness.[7] Preoperative hypoxia and permanent systolic pressure overload can cause abnormal intrinsic muscular stiffness. Other potential factors influencing the right ventricular filling pattern include the postoperative adhesion of the free wall to the anterior chest wall, age at surgery, respiration, and the angle between the Doppler beam and blood flow direction.

Our study shows that asymptomatic patients undergoing a Senning procedure for simple transposition of the great arteries have a peculiar right ventricular filling pattern. The data suggest significant abnormalities on right ventricular relaxation and operative compliance. The presence of tricuspid regurgitation can influence some of the right ventricular filling parameters, even when the patient maintains a satisfying systolic function. Furthermore, Doppler indexes of diastolic function provide a noninvasive alternative for the serial assessment of right ventricular function in this group of patients.

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Incidence and Significance of a "Step-Down" in Oxygen Saturation from Superior Vena Cava to Pulmonary Artery

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Several studies[1–4] have assessed the normal variability of oxygen saturations in blood from the right-sided heart chambers. An increase in saturation from the peripheral to the central chambers that exceeds this variability suggests left-to-right intracardiac shunting. In some subjects, a decrease in saturation from the superior vena cava (SVC) to the central right-sided chambers occurs. In previous studies,[5,6] such a "step-down" was often observed in patients with shock. This study was performed to assess the incidence of a step-down in oxygen saturation of ≥5% from the SVC to the pulmonary artery (PA) and to determine if such a step-down is associated with cardiac or renal dysfunction.

We reviewed all combined right- and left-sided cardiac catheterizations performed at Parkland Memorial Hospital, Dallas, Texas, from 1978 to 1990. Of 3,296 patients, the oxygen saturation of blood from the SVC exceeded that from the PA by ≥5% in 177 (5.4%) (57 men, 120 women, age range 18 to 88 years). Each subject was matched with the next patient having combined right- and left-sided cardiac catheterization who had neither a ≥5% SVC-PA step-

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TABLE I Comparison of Patients With and Without a ≥5% SVC-PA Step-Down in Oxygen Saturation

	Step-Down Present (n = 177)	Control Subjects (n = 177)	p Value
Age (years)	53 ± 14	51 ± 14	NS
Cardiac output (L/min/m^2)	2.7 ± 0.8	2.6 ± 0.7	NS
Mean PA wedge pressure (mm Hg)	19 ± 10	13 ± 8	<0.001
Mean PA pressure (mm Hg)	30 ± 13	22 ± 10	<0.001
Mean RA pressure (mm Hg)	8 ± 6	6 ± 4	<0.001
Left ventricular ejection fraction	0.52 ± 0.19	0.54 ± 0.16	NS
Serum creatinine (mg/dl)	2.0 ± 2.7	1.2 ± 0.4	<0.001

All data are mean ± 1 standard deviation.
NS = not significant; PA = pulmonary artery; RA = right atrial; SVC = superior vena cava.

TABLE II Incidence of Abnormal Hemodynamic and Angiographic Variables in Subjects With and Without a ≥5% SVC-PA Step-Down in Oxygen Saturation

	Step-Down Present (n = 177)	Control Subjects (n = 177)	p Value
Cardiac output <2.2 L/min/m^2	47/175 (27%)	45/175 (26%)	NS
Mean PA wedge pressure >18 mm Hg	88/176 (50%)	37/174 (21%)	<0.001
Mean PA pressure >30 mm Hg	76/176 (43%)	31/176 (18%)	<0.001
Mean RA pressure >8 mm Hg	62/175 (35%)	33/175 (19%)	0.01
Left ventricular ejection fraction <0.40	36/157 (23%)	30/166 (18%)	NS

All data are mean ± 1 standard deviation.
Abbreviations as in Table I.

down nor evidence of left-to-right intracardiac shunting. This control group contained 97 men and 80 women (age range 15 to 79 years).

In each patient, a catheter was advanced sequentially to the SVC, the right atrium and the main PA. Single, 2-ml blood samples were obtained within 5 minutes from the SVC and the PA, and their oxygen saturations were determined with a reflectance oximeter (American Optical) known to be accurate when saturation is 45 to 98%.[7] Within 15 minutes, cardiac output was determined by the Fick principle; pulmonary capillary wedge, PA and right atrial pressures were recorded, and left ventriculography was performed, from which ejection fraction was calculated. Finally, serum creatinine at the time of catheterization was recorded.

All data are reported as mean ± 1 standard deviation. For each variable, patients with an SVC-PA step-down ≥5% were compared with control subjects using Student's t test. The frequency with which each variable was abnormal in the 2 groups was compared using a chi-square analysis. A p value <0.05 was considered significant.

For the 177 patients with an SVC-PA step-down, catheterization revealed no disease in 10, coronary artery disease in 71, valve disease in 52, cardiomyopathy in 30, pericardial disease in 7, pulmonary hypertension in 4 and high-output failure in 3. For these subjects, SVC saturation was 69 ± 10% and PA saturation was 62 ± 11% (p <0.001 in comparison with SVC). For the 177 control subjects, catheterization revealed no disease in 18, coronary artery disease in 98, valve disease in 37, cardiomyopathy in 21, pericardial disease in 2 and pulmonary hypertension in 1. For these subjects, SVC saturation was 68 ± 8% and PA saturation was 68 ± 9% (p = not significant in comparison with SVC). Patients with an SVC-PA step-down were similar to the control subjects in age, cardiac output and ejection fraction. Mean pulmonary capillary wedge, PA and right atrial pressures were higher (p <0.001) in subjects with a step-down, as was serum creatinine concentration (Table I). The incidence of reduced cardiac output or ejection fraction was similar in the 2 groups, but subjects with a step-down were more likely to have a mean wedge pressure >18 mm Hg, a mean PA pressure >30 mm Hg and a mean right atrial pressure >8 mm Hg (Table II).

In control subjects, oxygen saturation of inferior vena caval blood is often high because of the contribution of renal venous effluent that is highly saturated.[8] Thus, blood from the PA is usually more saturated with oxygen than that from the SVC.[1] In patients with shock, heart failure or renal disease, renal blood flow may decrease[9,10] so that renal venous effluent contributes less to the saturation of inferior vena caval blood. Because cerebral blood flow is preserved,[11] the saturation of blood obtained from the SVC may be higher than that from the inferior vena cava and the PA. In patients with shock of various causes, oxygen saturation of blood from the SVC was consistently higher than that from the PA.[5,6]

In this study, we found that the saturation of blood from the SVC exceeded that from the PA by ≥5% in only 177 of 3,296 patients (5.4%). We tried to determine if a step-down in oxygen saturation was associated with: (1) cardiac or renal dysfunction, or both, as reflected by inadequate peripheral perfusion (manifested by low cardiac output); (2) diminished left ventricular performance (manifested by a depressed ejection fraction); (3) pulmonary vascular congestion (manifested by a high wedge pressure), with a resultant increase in PA and right atrial pressures; and/or (4) reduced renal function (manifested by an elevated serum creatinine concentration). In patients with these derangements, the saturation of inferior vena caval blood may be lower than that of SVC blood,

at least in part because of reduced renal blood flow. As a result, these patients may have an oxygen saturation in PA blood lower than that of SVC blood. In our 177 subjects with an SVC-PA step-down, cardiac output and ejection fraction were similar to those of the control subjects (Table I), and the 2 groups had a similar incidence of abnormal values for these variables (Table II). However, patients with an SVC-PA step-down had higher pulmonary capillary wedge, PA and right atrial pressures than the control subjects (Table I), and the incidence with which these pressures were elevated was greater in those with a step-down (Table II). In addition, serum creatinine was higher in patients with a step-down than in control subjects. Although renal blood flow is often reduced in patients with renal disease of various etiologies, renal oxygen extraction is usually not altered. Thus, it is possible that the renal contribution to inferior vena caval blood flow is diminished in subjects with an SVC-PA step-down. Because we did not measure renal blood flow, we are uncertain if this hypothesis is correct.

In conclusion, a decrease in oxygen saturation of ≥5% from the SVC to the PA occurs infrequently. Subjects with such a step-down often have elevated pulmonary capillary wedge, PA and right atrial pressures, as well as elevated serum creatinine concentration. The mechanisms responsible for these observations are undefined, but it is possible that reduced renal blood flow (in association with renal disease or decreased renal perfusion, or both) is at least partially responsible for our findings.

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Mitral and Pulmonary Venous Flow Under Influence of Positive End-Expiratory Pressure Ventilation Analyzed by Transesophageal Pulsed Doppler Echocardiography

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It has been known since 1948[1] that positive end-expiratory pressure (PEEP) ventilation causes a decrease in cardiac output. Since then, the effects of PEEP on the heart have been studied in detail. There are mainly 2 mechanisms discussed for the decrease in cardiac output with PEEP. The first hypothesis is that cardiac output is diminished by reduction of systemic venous return. This concept[1,2] was questioned later by investigators[3,4] who studied transmural pressures and found increased filling pressures with PEEP. They concluded that left ventricular compliance decreases with PEEP owing to interventricular septal shift from right ventricular overload[4] or to compression of the heart by the expanded lungs. Subsequently, more precise measurements of filling pressure[5] provided no evidence of decreased diastolic compliance. Left ventricular function impairment could not be detected using either equilibrium-gated blood pool scintigraphy or transesophageal 2-dimensional echocardiography.[6] Most examinations have relied on hemodynamic measurements that are dependent on intricate pressure recordings that are not always reliable.[4,5] This study attempts to gain insight into changes in hemodynamics after induction of PEEP by measuring flow patterns with Doppler echocardiography. To examine the mechanism of reduction in cardiac output produced by high levels of PEEP ventilation, left atrial and left ventricular filling patterns were recorded with pulsed Doppler for 0- and 15-cm H_2O PEEP.

Fifteen patients (10 men and 5 women, mean age 58 years, range 29 to 75) were investigated. All 15 patients were undergoing prolonged mechanical ventilation for respiratory failure and were in sinus rhythm; patients undergoing mechanical ventilation for cardiac failure were excluded. Transesophageal

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