Dilation of the Coronary Sinus on Echocardiogram: Prevalence and Significance in Patients with Chronic Pulmonary Hypertension

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Background: Although rarely seen in healthy patients, the coronary sinus (CS) is often visualized on echocardiography in patients with right-sided heart disease. However, the prevalence of this finding and its relation to right-sided heart structure and pressure remains undefined.

Methods: We examined the transthoracic echocardiograms of 43 consecutive patients referred for the evaluation of pulmonary hypertension (26 men, 17 women) with a mean age of 53 ± 15 years (range 21 to 82 years). Structural abnormalities of the tricuspid valve were absent. All patients underwent right heart catheterization within 48 hours of their echocardiogram, which revealed the following pressures: mean pulmonary artery (50 mm Hg, range 31 to 84 mm Hg) and right atrial (RA) (mean 10, range 1 to 24 mm Hg). Echocardiograms were analyzed for CS size (identified as the smallest diameter of a circular structure in the left atrioventricular groove in the parasternal long-axis view), as well as RA and right ventricular (RV) sizes. The presence and severity (grades 1 through 3) of tricuspid regurgitation (TR) were also recorded.

Results: The CS was visualized in 35 (81%) of 43 patients, and measurements ranged from 0.4 to 1.6 cm (mean 0.8 cm). No difference in RA size, RV size, TR grade, RA pressure (RAP), RV pressure (RVP), mean pulmonary artery pressure (PAP), or pulmonary vascular resistance (PVR) was observed between patients with a visualized and nonvisualized CS. Coronary sinus size correlated significantly with RA size \((r = 0.60, P < .001)\) and pressure \((r = 0.59, P < .001)\), but not with RV size, degree of TR, RVP, PAP, or PVR. Nineteen of 35 patients with a visualized CS underwent pulmonary artery thromboendarterectomy (PTE), and their CS size and RAP were unchanged \((0.8 \text{ cm and } 12 \text{ mm Hg, respectively, pre– and post–PTE; both } P = \text{NS [not significant]})\), though a decrease was observed in other measurements: RA size \((4.2 \text{ versus } 4.8 \text{ cm, } P = .02)\), RV size \((4.2 \text{ versus } 5.1 \text{ cm, } P = .0004)\), mean PAP \((37 \text{ versus } 72 \text{ mm Hg, } P < .0001)\), and PVR \((230 \text{ versus } 899 \text{ mm Hg, } P < .0001)\).

Conclusions: Coronary sinus dilation was observed in 81% of a selected group of patients with pulmonary hypertension in the absence of structural disease of the tricuspid valve. Coronary sinus dilation is related to RAP and RA size, but not to RV size, degree of TR, RVP, PA pressure, or PVR. Once dilated, CS size does not change shortly after decreases of RA size, RV size, or PA pressure produced by PTE. (J Am Soc Echocardiogr 2001;14:44-9.)

The coronary sinus (CS) is a venous structure that drains myocardial blood into the right atrium, predominantly from the left ventricle.1 In the non-pathologic state, it is a slit-like collapsed vessel that runs in the left atrioventricular groove, and is rarely visualized on transthoracic echocardiography.2 When readily visible on ultrasonographic images, the CS is often thought to represent congenital anomalous venous drainage leading to increased blood flow within the sinus with resultant dilation.3-5

In the course of interpreting clinical studies for the evaluation of right-sided heart disease, we observed that the CS was frequently dilated on transthoracic echocardiograms in these patients. However, the prevalence of this finding remains undefined, as does its relation to right-sided heart structure and...
pressure. Therefore, we undertook this study to
determine the prevalence of CS dilation in patients
with right-sided heart disease, and to define the rela-
tion between this finding and right-sided cardiac
dimensions and pressures.

METHODS

The transthoracic echocardiograms and right heart
hemodynamic data of 43 consecutive patients re-
ferred for the evaluation of pulmonary hypertension
were examined. The study group comprised 26 men
and 17 women with a mean age of 53 ± 15 years
(range 21 to 82 years). All patients had chronic long-
standing symptoms of cardiopulmonary disease and
were classified as in New York Heart Association
class III or IV. Each patient had evidence of right-
sided heart disease on physical examination, includ-
ing elevated jugular venous pressure, palpable right
ventricular (RV) heave, hepatomegaly, or a pro-
nounced P2 on auscultation. Within the population,
surgically accessible chronic thromboembolic pul-
monary hypertension was diagnosed in 25 patients
by previously described criteria, whereas 18 patients
were determined to have surgically inaccessible
primary or secondary chronic pulmonary hyper-
tension.

The 43 study patients constituted a subgroup of a
group of 70 consecutive patients referred for the
evaluation of chronic pulmonary hypertension.
Twenty-seven patients were excluded for the follow-

Figure 1 A, Transthoracic echocardiogram, parasternal
long-axis view. Note the large dilated coronary sinus (CS)
and right ventricle (RV). B, Transthoracic echocardiogram,
parasternal long-axis view in a different study
patient. Demonstrates method of CS measurement as per-
formed in a magnified view with the use of electronic
calipers. LV, Left ventricle; LA, left atrium.

ing reasons: structural abnormalities of the tricuspid
valve, suboptimal echocardiographic images (poor
endocardial border definition), right heart catheteri-
ization performed more than 48 hours after echocar-
diography, or absence of pulmonary hypertension at
right heart catheterization (mean PAP <30 mm Hg).
Twenty-five of the above 43 patients underwent pul-
monary artery thromboendarterectomy (PTE) for
surgically accessible chronic thromboembolic pul-
monary hypertension. In these patients, postopera-
tive echocardiograms and right heart catheterization
data were obtained and analyzed. Right heart cathe-
terization data were obtained in the surgical intensive
care unit within 72 hours of the postoperative
echocardiogram.

All patients underwent a standard echocardiog-
graphic examination, including 2-dimensional and M-
mode imaging and pulsed, continuous wave, and
color Doppler recordings. In addition, a contrast
echocardiogram was performed by injecting agitated
saline into the left antecubital vein. Commer-
cially available echocardiographic instruments with 2.0-
to

4.0-MHz transducers were used for 2-dimensional
imaging. Because the CS is located in the left atrio-
ventricular groove, which is best visualized in the
parasternal long-axis view, this view was used to
identify the CS in the current study. Echocardiograms
were analyzed for the presence and size of the CS,
which was defined as the smallest diameter of a cir-
cular structure in the left atrioventricular groove in
the parasternal long-axis view (Figure 1). Because the
CS was not consistently seen throughout the cardiac
cycle in every patient, we did not use a physiologic
marker such as end systole or end diastole for the
measurement of the CS. Instead the “smallest” diame-
ter in the best-visualized image of the CS was chosen
to ensure that an oblong image of the CS would not
artificially inflate the CS size measurement.

Measurements of the CS were performed with elec-
tronic calipers in a magnified display. The right atrial
(RA) and RV sizes were measured in the apical 4-
chamber view as the largest transverse dimension of each chamber in atrial and ventricular diastole, respectively (Figure 2). The presence of tricuspid regurgitation was noted and its severity graded as follows: 0 = none, 1 = mild, 2 = moderate, 3 = severe. The same criteria as applied by Helmcke et al to quantitate mitral regurgitation by color Doppler were used to quantitate tricuspid regurgitation (TR).

All echocardiograms were reviewed and interpreted by 2 observers, and no discrepancies were found. All patients underwent right heart catheterization within 24 to 48 hours of their echocardiogram. Right heart catheterization was performed in the standard manner in the cardiac catheterization laboratory with a 7.5F Swan-Ganz (Baxter Healthcare Corp, Santa Ana, Calif) catheter inserted into the right internal jugular vein. Pressure transducers were routinely balanced and positioned at the level of the right atrium. Resting phasic and electrical mean pressure measurements were recorded on paper at 50 mm/s from the right atrium, right ventricle, pulmonary artery, and pulmonary capillary wedge positions.

Cardiac output (CO) was obtained by the thermodilution method (mean of 3 injections), and pulmonary vascular resistance (PVR) was calculated by using the standard formula.

Data are expressed as mean ± SD. Linear regression analysis was used to compare CS size with RA size, RV size, RA pressure (RAP), RV pressure (RVP), pulmonary artery pressure (PAP), and PVR. Correlation coefficients with related $P$ values are reported. A Spearman rank correlation coefficient was obtained to measure the relation of CS size to TR grade. A 2-tailed Student $t$ test for unpaired populations was used to compare variables in the 2 groups (CS visualized versus nonvisualized). A 2-tailed Student $t$ test for paired populations was used to compare pre-versus post-PTE measurements in the patients who underwent this procedure. A value of $P < .05$ was considered statistically significant.

### RESULTS

The echocardiographic measurements and right heart catheterization data for the group are summarized in Table 1. Pulmonary artery systolic pressure ranged from 50 to 130 mm Hg (mean 83 ± 17 mm Hg). Tricuspid regurgitation was severe in 9 patients, moderate in 24, mild in 9, and absent in 1. Neither structural abnormalities of the tricuspid valve nor anomalous venous drainage were detected in any patient.

The CS was visualized in 35 (81%) of 43 patients, and diameter measurements ranged from 0.4 to 1.6 cm (mean 0.8 cm). Regression analysis demonstrated that CS size correlated with RA size ($r = 0.60, P < .001$) and RAP ($r = 0.59, P < .001$) (Figure 3). No significant correlation was observed between CS size and RV size ($r = 0.40$), RVP ($r = 0.24$), PAP ($r = 0.28$), or PVR ($r = 0.39$).

<table>
<thead>
<tr>
<th>Table 1 Echocardiographic and catheterization data (n = 43)</th>
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<tbody>
<tr>
<td><strong>Range</strong></td>
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<tr>
<td>Echocardiographic data</td>
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<tr>
<td>RA size (cm)</td>
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<tr>
<td>RV size (cm)</td>
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<tr>
<td>Catheterization data</td>
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<tr>
<td>Mean RAP (mm Hg)</td>
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<tr>
<td>Systolic RVP (mm Hg)</td>
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<td>Mean PAP (mm Hg)</td>
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<tr>
<td>PVR (dynes/s/cm$^5$)</td>
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<td>PCWP (mm Hg)</td>
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<td>CO (L/m)</td>
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RA, Right atrium; RAP, right atrial pressure; RVP, right ventricular pressure; PAP, pulmonary artery pressure; PVR, pulmonary vascular resistance; PCWP, pulmonary capillary wedge pressure; CO, cardiac output.
We analyzed our data to determine whether patients with a visualized CS differed from patients without this finding in regard to right-sided cardiac dimensions or pressures. Though a trend existed toward a lower RA size (4.2 versus 4.8 cm, \( P = .13 \)) and RAP (8 versus 11 mm Hg, \( P = .13 \)) in patients in whom the CS was not visualized, these findings were not statistically significant. No difference was detected between the two groups with respect to RV size, or RVP, PAP, PVR, or TR severity (Table 2).

Coronary sinus size (0.8 ± 0.3 cm, pre- and post- PTE; \( P = \text{NS} \)) and RAP (12 ± 6 mm Hg, pre- and post- PTE; \( P = \text{NS} \)) were unchanged in the 19 of 35 patients with a visualized CS who underwent PTE. These structures remained constant in size despite the fact that marked reductions in RA size, RV size, PAP, and PVR were observed postoperatively in these patients (Figure 4 and Table 3).

**DISCUSSION**

The CS is the confluence of the cardiac veins that drain into the right atrium. In the nonpathologic state, it is a collapsed venous structure that is rarely visualized on transthoracic echocardiography. Although a dilated CS on echocardiogram has customarily been associated with anomalous venous drainage, it may also occur in patients with pulmonary hypertension. However, the prevalence of CS dilation in pulmonary hypertension and its relation to abnormalities of right-sided cardiac structure and pressure are undefined. The present study demonstrates that visible dilation of the CS is common in patients with elevated RV/PA pressures, occurring in 81% of patients. Nevertheless, CS size does not correlate with RV or PA pressure, but rather with RA size and pressure. Coronary sinus size is also unrelated to the extent of TR. Finally, the acute reduction of PA pressure induced by PTE is not accompanied by a
reduction of either RAP or CS size. Thus the CS behaves analogous to the venae cavae as an extension of the right atrium.

The CS is enveloped by left atrial muscular tissue as it traverses through the atrioventricular groove. Therefore it was quite possible that the CS would not dilate in response to an elevation in its drainage bed (ie, the right atrium). Our study demonstrates that the CS actually dilates in response to RA hypertension and behaves as a venous structure and not as a part of the left atrial wall.

Kronzon et al reported the ability to identify the CS in the parasternal long-axis view of the transthoracic echocardiogram in 60% of patients with a variety of cardiac diseases. None of the patients they studied had clinical signs of elevated RAP or Doppler echocardiographic evidence of severe TR. The authors designed a study to specifically identify and assess the CS. Therefore our final conclusion may have been understated. Our study is also retrospective and when the transthoracic echocardiogram was performed, special efforts had not been made to identify the CS. Therefore our final conclusion may have been understated. Our study is also limited by the fact that post-PTE echocardiograms were obtained only 1 week after surgery. Thus long-term reversibility of CS dilation is not addressed.

Limitations

Transthoracic echocardiography and right heart catheterization were not simultaneous; up to 48 hours elapsed between them in this study. However, because the underlying medical condition in all patients was chronic pulmonary hypertension, we feel that simultaneous echocardiography and right heart catheterization would not have yielded substantially different results. Post-PTE right heart catheterization data were obtained in the intensive care unit by different operators and thus may be susceptible to variability in these measurements. However, these data were only used as confirmatory evidence of our conclusions, so they do not affect the results of the primary study. This is a retrospective study, and when the transthoracic echocardiography was performed, special efforts had not been made to identify the CS. Therefore our final conclusion may have been understated. Our study is also limited by the fact that post-PTE echocardiograms were obtained only 1 week after surgery. Thus long-term reversibility of CS dilation is not addressed.

Measurement of RA and RV sizes by echocardiography suffers from the lack of a uniformly agreed-upon method, and whether the RA and RV enlarge in

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Patient characteristics of visualized versus nonvisualized coronary sinus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiographic/catheterization data</td>
<td>CS visualized (n = 35)</td>
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<tr>
<td>RA size (cm)</td>
<td>4.8 ± 1.2</td>
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<tr>
<td>RV size (cm)</td>
<td>4.9 ± 1.0</td>
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<tr>
<td>TR (grade)</td>
<td>2.0 ± 0.7</td>
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<tr>
<td>Mean RAP (mm Hg)</td>
<td>11 ± 5</td>
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<tr>
<td>Systolic RVP (mm Hg)</td>
<td>84 ± 18</td>
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<tr>
<td>Mean PAP (mm Hg)</td>
<td>51 ± 12</td>
</tr>
<tr>
<td>PVR (dynes/s/cm⁵)</td>
<td>908 ± 397</td>
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</tbody>
</table>

CS, Coronary sinus; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; RAP, right atrial pressure; RVP, right ventricular pressure; PAP, pulmonary artery pressure; PVR, pulmonary vascular resistance.

<table>
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<tr>
<th>Table 3</th>
<th>Pre– versus post–PTE echocardiographic and hemodynamic parameters (n = 19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiographic data</td>
<td>Pre–PTE</td>
</tr>
<tr>
<td>CS size (cm)</td>
<td>0.8 ± 0.3</td>
</tr>
<tr>
<td>RA size (cm)</td>
<td>4.8 ± 1.3</td>
</tr>
<tr>
<td>RV size (cm)</td>
<td>5.1 ± 1.0</td>
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<tr>
<td>Catheterization data</td>
<td></td>
</tr>
<tr>
<td>Mean RAP (mm Hg)</td>
<td>12 ± 6</td>
</tr>
<tr>
<td>Systolic RVP (mm Hg)</td>
<td>88 ± 21</td>
</tr>
<tr>
<td>Mean PAP (mm Hg)</td>
<td>53 ± 14</td>
</tr>
<tr>
<td>PVR (dynes/s/cm⁵)</td>
<td>899 ± 436</td>
</tr>
</tbody>
</table>

PTE, Pulmonary thromboendarterectomy; CS, coronary sinus; RA, right atrium; RV, right ventricle; PAP, pulmonary artery pressure; PVR, pulmonary vascular resistance.
a symmetrical manner is unclear. With the methods in our study, minimal variability was noted in these measurements. Another limitation of our study is that the study group consisted only of patients with pulmonary hypertension. Therefore these findings cannot be extrapolated to RV volume overload states such as atrial septal defect, TR, or congestive heart failure, which could also lead to RAP elevation and an increase in RA size.

Conclusion

In the absence of structural disease of the tricuspid valve, CS dilation was observed in 81% of a selected group of patients with pulmonary hypertension. Coronary sinus dilation is directly related to RAP and RA size, but not to RV size, degree of TR, RVP, PAP, or to PVR. Once dilated, the CS diameter remains unchanged after decreases in right-sided heart dimensions and PAP induced by PTE. Therefore CS dilation is a marker of increased RAP that does not vary appreciably with other right-sided heart dimensions or hemodynamic parameters. Though we have demonstrated the presence of CS dilation in a high percentage of patients with chronic pulmonary hypertension, we were unable to relate the presence of this finding to pulmonary hypertension. However, this finding helps in understanding and defining the physiologic response of the CS to chronic RA hypertension.

REFERENCES