OBJECTIVES
We sought to compare bubble contrast echocardiography and pulmonary angiography in detecting pulmonary arteriovenous malformation (PAVM) in children with cavopulmonary anastomosis (CPA), and to examine anatomic and physiologic variables associated with the development of PAVM.

BACKGROUND
Development of PAVM in patients with CPA may cause profound cyanosis. Pulmonary arteriovenous malformation has been traditionally diagnosed by pulmonary angiography with reported incidence of 20% to 25% in patients with CPA.

METHODS
Fourteen patients (age 1.1 to 12.6 years) with any forms of CPA and normal pulmonary venous drainage formed the study population. All patients underwent cardiac catheterization and pulmonary angiography. Bubble contrast echocardiographic studies were performed with injection of 10 ml of agitated saline solution into branch pulmonary arteries. Transthoracic echocardiograms using an apical view were performed to assess the appearance of bubble contrast in the systemic ventricles. We compared the results of pulmonary angiograms and contrast echocardiograms, and findings of contrast echocardiograms between lungs with hepatic venous blood flow and lungs without hepatic venous blood.

RESULTS
Ten of the 14 patients (71%) had positive contrast echocardiographic studies, compared with three (21%) detected by pulmonary angiograms (p = 0.01). No difference was found in pulmonary artery pressure, transpulmonary gradient or presence of heterotaxy syndrome between patients with positive contrast echocardiographic studies and patients with negative studies. However, patients with positive contrast echocardiograms tended to have lower oxygen saturation (81%) and higher hemoglobin (16.4 g/dl) compared with patients with negative studies (88% and 14.7 g/dl, p = 0.10 and p = 0.18 respectively). Patients with Glenn shunt or unidirectional Fontan had higher incidence of PAVM (10/11) compared with patients with classic or lateral tunnel Fontan (0/3, p = 0.01). All 12 lungs with no perfusion of hepatic venous blood had positive contrast echocardiographic studies. Lungs with no hepatic venous blood flow were more likely to develop PAVM compared with lungs with hepatic venous blood flow (12/12 and 3/16 respectively, p < 0.01).

CONCLUSIONS
Bubble contrast echocardiography is more sensitive in detecting PAVM compared with pulmonary angiography. The prevalence of PAVM in patients with CPA may be much higher than what had been reported previously. Lungs with no hepatic venous blood flow are more likely to develop PAVM than lungs with hepatic venous blood flow. (J Am Coll Cardiol 1999;33:2052–8) © 1999 by the American College of Cardiology
a three-way stopcock. One syringe contained 10 ml of 0.9% air-filled microbubbles. Two 10-ml syringes were joined by was produced by agitating solutions manually to form size) to obtain an apical view of the heart. Contrast agent 4-Hz or 5-Hz transducer was used (depending on patient’s Model (Acuson, Mountain View, California). A 3-Hz, grams were performed using an Acuson 128XP/10SP Contrast echocardiography.

Transthoracic echocardio-

patients with CPA by contrast echocardiography. Specifically, the objectives are as follows: 1) to compare contrast echocardiography and angiography in diagnosing PAVM; 2) to investigate the prevalence of PAVM in patients with various forms of CPA by contrast echocardiography, and 3) to examine the role of hepatic venous blood in the development of PAVM in patients with CPA by contrast echocardiography.

METHODS

Patients. The study protocol was approved by the Human Subject Protection Committee of the University of California, Los Angeles. Fourteen patients with any forms of CPA were recruited in the study. Although previous studies have documented that normal individuals should have no bubble contrast pass through pulmonary capillary bed and return to the heart in contrast echocardiography (15,16), we recruited three patients with normal pulmonary venous return and without CPA as the internal control group. No patients had mucocutaneous telangiectasia or other stigmata of Osler-Weber-Rendu syndrome.

Contrast echocardiography. Transthoracic echocardiograms were performed using an Acuson 128XP/10SP Model (Acuson, Mountain View, California). A 3-Hz, 4-Hz or 5-Hz transducer was used (depending on patient’s size) to obtain an apical view of the heart. Contrast agent was produced by agitating solutions manually to form air-filled microbubbles. Two 10-ml syringes were joined by a three-way stopcock. One syringe contained 10 ml of 0.9% saline solution, and the other syringe contained 1 ml of air. The saline solution was agitated by rapidly flushing the solution back and forth between the two syringes through the three-way stopcock 10 times or more. A mixture of air and liquid was thereby generated. A side-hole or end-hole balloon catheter was placed with the tip of the catheter in the left or right pulmonary artery without wedging in small branches. Then a rapid hand injection of the agitated saline was performed. Simultaneous recording of the apical view echocardiogram of the injection was performed to visualize the presence of bubble contrast in the pulmonary venous atrium and systemic ventricle. In a normal individual, after injection of saline bubble contrast in the pulmonary arteries, the echo contrast quality of the bubbles is lost in the passage through the pulmonary capillary bed and therefore no bubbles should be observed in the left heart (17,18). The presence of bubble contrast in the pulmonary venous atrium and systemic ventricle indicates lack of passage of agitated saline solution through the pulmonary capillary bed, and is therefore indicative of the presence of intrapulmonary arteriovenous shunting (Fig. 1).

Pulmonary angiography. All 14 patients underwent right heart catheterization and pulmonary angiography as clinically indicated. In patients with Fontan operation, the pulmonary arteries were entered in an antegrade manner through the femoral veins. In patients with Glenn shunt or interrupted inferior vena cava withazygous continuation, the right internal jugular veins were used for access to the pulmonary arteries. The pulmonary artery pressures and pulmonary capillary wedge pressures were recorded in all patients before angiograms. Selective pulmonary angiograms were performed with a side-hole balloon catheter, or end-hole balloon catheter with proximal balloon inflation. Tips of the catheters were positioned in the branch pulmonary arteries to ensure adequate filling of the contrast in the selected arteries and to avoid wedging of the catheter in small branches. Half to 1 ml/kg body weight of Omnipaque 350 (Nycomed, New Jersey) contrast solution was injected to the selected branch pulmonary artery. The fluoroscopic cameras were angled at straight posterior–anterior and lateral positions to ensure adequate demonstration of the entire hemithorax, especially the bases of the lungs. Cineangiograms were recorded for review. Angiographic findings were examined by two board-certified pediatric cardiologists to determine the presence of PAVM.

Statistical analysis. The 14 patients were divided into groups according to types of CPA and sources of pulmonary blood flow. We first compared the contrast echocardiographic findings with angiographic findings of PAVM in all patients. Measurements of cardiac catheterization, including pulmonary artery pressure, transpulmonary gradient, aortic saturation and hemoglobin were compared between patients with positive contrast echocardiographic studies and patients with negative studies. We then examined the sources of pulmonary blood flow to the left and right lungs
for each patient. In the second analysis, we used the "lung" (right lung or left lung) as the unit of analysis. There were 28 lungs from 14 patients. Each lung was examined to determine if its blood supply included hepatic venous blood. Two groups of lungs were formed: a group with hepatic venous blood to pulmonary circulation and a group without hepatic venous blood to pulmonary circulation. Comparison of contrast echocardiograms was made between these two groups of lungs. Continuous variables were compared by Student t test, and categorical variables were compared by chi-square test or Fisher exact test. A p value of less than 0.05 was considered statistically significant. Statistical analysis was conducted using software package SPSS 7.0 for Windows (SPSS, Chicago, Illinois).

RESULTS

Patient characteristics. Clinical characteristics of 14 patients are summarized in Table 1. The mean age of patients was 7.5 years (range 1.1 to 12.6 years, median 9 years). There were eight boys and six girls. Heterotaxy syndrome was present in five patients. The interval between CPA and time of study ranged from 1 month to 8.6 years, with a mean of 4.2 years and median of 4.8 years. These 14 patients were categorized into five groups according to their initial diagnoses and types of CPA. Group 1 consisted of three patients (Patients 1 to 3) who had bidirectional Glenn shunt and no additional source of pulmonary flow from the ventricle(s) or from an aortopulmonary shunt. Group 2 consisted of two patients (Patients 4 and 5) who had interrupted inferior vena cava withazygous continuation to the superior vena cava. These two patients were status post Kawashima modification of Fontan operation with hepatic veins left to drain into the common atria (19). Group 3 consisted of two patients (Patients 6 and 7) who had unidirectional Fontan operation (connecting superior vena cava to left pulmonary artery and inferior vena cava to right pulmonary artery) (20). Group 4 consisted of four patients (Patients 8 to 11) who had bidirectional Glenn shunt and had additional sources of pulmonary flow from the ventricle(s) or an aortopulmonary shunt. Group 5 consisted of three patients (Patients 12 to 14) who had classic or lateral tunnel Fontan operation.

Contrast echocardiography and angiography. All three control individuals had negative contrast echocardiographic and angiographic studies for PAVM. Ten of the 14 patients with CPA (71%) had positive contrast echocardiographic studies, compared with three (21%) detected by pulmonary angiograms. The difference in prevalence of PAVM between contrast echocardiographic and angiographic diagnoses was statistically significant (p = 0.01). Of patients with and patients without PAVM detected by contrast echocardiography, there was no statistical difference in gender, presence of heterotaxy syndrome or interval between CPA and study (3.8 ± 2.7 and 5.1 ± 2.7 years respectively, p = 0.45). Patients with classic or lateral tunnel Fontan (group 5) had no PAVM found (0/3), in contrast to patients with Glenn shunt, unidirectional Fontan or Kawashima modification of Fontan (10/11, p = 0.01). Patients with PAVM detected by contrast echocardiograms tended to have lower oxygen saturation (81%) and higher hemoglobin (16.4 g/dl) compared with patients without PAVM (88% and 14.7 g/dl, p = 0.10 and p = 0.18 respectively).

The three patients who had positive angiographic findings of PAVM also had positive contrast echocardiograms. Two patients with positive angiograms at the right lung also had positive contrast echocardiograms from the right; one patient with positive angiogram at the left lung also had positive contrast echocardiogram from the left. The two patients in Group 3 (unidirectional Fontan, Patients 6 and 7) both had positive bubble contrast return in selective left pulmonary artery injections and negative studies in selective right pulmonary artery injections.

The mean pulmonary artery pressure in 14 patients was

![Figure 1. Apical view of transthoracic bubble contrast echocardiograms of a patient with bidirectional Glenn shunt and pulmonary stenosis. Preferential streaming of superior vena cava flow to the right pulmonary artery and main pulmonary artery flow to the left pulmonary artery was seen in angiograms. (A) Selective injection of bubble contrast to the left pulmonary artery showed no significant bubble contrast return to the left atrium. (B) Selective injection of bubble contrast to the right pulmonary artery showed bubble contrast opacifying the left atrium.](image-url)
The mean transpulmonary gradient was 5.1 mm Hg (median 5). No statistical
difference in pulmonary artery pressure or transpulmonary gradient
was found between patients with PAVM and patients without PAVM.

**Lungs with or without hepatic venous blood.** Patients in
Group 1 and Group 2 had no hepatic venous blood flow to
either left or right lungs. Patients in Group 3 had hepatic venous blood flow only to the right lungs but not to the left lungs. Therefore, of 28 lungs from our 14 study subjects, there was a total of 12 lungs with no hepatic venous blood perfusion and 16 lungs with hepatic venous blood (Table 2, asterisks). All 12 lungs with no hepatic venous blood perfusion had positive contrast echocardiographic studies. The difference in positive contrast echocardiograms be-

**Table 1. Patient Characteristics**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)/ Gender</th>
<th>Diagnosis</th>
<th>Heterotaxy</th>
<th>Cavopulmonary Anastomosis</th>
<th>CPA Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1—Glenn shunt with no hepatic venous blood to pulmonary arteries</td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>1</td>
<td>1.6/F</td>
<td>[S,D,D], DORV, pulmonary atresia, d-MGA, L SVC to CS, interrupted IVC with hemiazygous vein continuation to L SVC</td>
<td>Yes</td>
<td>RSVC-RPA (bidirectional)</td>
<td>0.9</td>
</tr>
<tr>
<td>2</td>
<td>7.4/M</td>
<td>[S,D,D], d-TGA, VSD, LV hypoplasia, PS and sub-PS</td>
<td>No</td>
<td>SVC-RPA (bidirectional), MPA ligation</td>
<td>2.7</td>
</tr>
<tr>
<td>3</td>
<td>1.1/M</td>
<td>[S,D,D], HLHS, s/p BAS, stenting of PDA</td>
<td>No</td>
<td>SVC-RPA (bidirectional), Norwood procedure</td>
<td>0.1</td>
</tr>
<tr>
<td>Group 2—Kawashima modification of Fontan operation</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>4</td>
<td>9.3/M</td>
<td>[A,D,D], DORV, hypoplastic LV and mitral valve, common atrium, d-MGA, bilateral SVC, interrupted IVC with aszygous continuation to L SVC, s/p right and left MBTS</td>
<td>Yes</td>
<td>RSVC-RPA, LSVC-LPA (both bidirectional)</td>
<td>5.7</td>
</tr>
<tr>
<td>5</td>
<td>8.7/M</td>
<td>[A,D,D], dextrocardia, common atrium, hypoplastic LV, interrupted IVC with aszygous continuation to RSVC, bilateral SVC, s/p modified Norwood procedure</td>
<td>Yes</td>
<td>RSVC-RPA, LSVC-LPA (both bidirectional)</td>
<td>6.3</td>
</tr>
<tr>
<td>Group 3—unidirectional Fontan</td>
<td></td>
<td></td>
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<tr>
<td>6</td>
<td>9.9/M</td>
<td>[S,D,D], DORV, TGA, PS, sub-PS, s/p PAB and Glenn shunt (SVC-RPA, bidirectional)</td>
<td>No</td>
<td>Unidirectional Fontan (IVC-RPA, SVC-LPA) and PA ligation</td>
<td>4.9</td>
</tr>
<tr>
<td>7</td>
<td>9.4/F</td>
<td>[S,L,L], DILV, 1-TGA, s/p Damus-Kaye-Stansel procedure, central shunt and Glenn shunt (SVC-RPA, bidirectional)</td>
<td>No</td>
<td>Unidirectional Fontan (IVC-RPA, SVC-LPA) and PA ligation</td>
<td>2.8</td>
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<tr>
<td>Group 4—Glenn shunt with hepatic venous blood to pulmonary arteries</td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>8</td>
<td>10/F</td>
<td>[S,D,D], dextrocardia, complete AVC, d-TGA, LV hypoplasia, PS, bilateral SVC, s/p right MBTS</td>
<td>Yes</td>
<td>LSVC-LPA (bidirectional), ligation of RSVC</td>
<td>4.6</td>
</tr>
<tr>
<td>9</td>
<td>2.4/F</td>
<td>[S,L,L], 1-TGA, DILV, ventricular inversion</td>
<td>No</td>
<td>SVC-RPA (bidirectional), PAB</td>
<td>1.4</td>
</tr>
<tr>
<td>10</td>
<td>6.2/F</td>
<td>[S,D,D], dextrocardia, common atrium, SV, d-TGA, PS, single LSVC</td>
<td>Yes</td>
<td>SVC-RPA (bidirectional)</td>
<td>5.3</td>
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<tr>
<td>11</td>
<td>9.9/M</td>
<td>[S,M,D], HLHS, s/p Norwood procedure with right MBTS, atrial septectomy</td>
<td>No</td>
<td>SVC-RPA (bidirectional), right MBTS takedown, central shunt to LPA</td>
<td>8.6</td>
</tr>
<tr>
<td>Group 5—Classic or modified Fontan</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>11.5/F</td>
<td>[S,D,D], DORV, unbalanced AVC, LV hypoplasia, PS, PAPVR</td>
<td>No</td>
<td>Lateral tunnel Fontan</td>
<td>6.1</td>
</tr>
<tr>
<td>13</td>
<td>12.6/M</td>
<td>[S,D,D], tricuspid atresia, pulmonary atresia, RV hypoplasia, s/p right MBTS</td>
<td>No</td>
<td>RA-MPA, right MBTS takedown</td>
<td>7.5</td>
</tr>
<tr>
<td>14</td>
<td>5.4/M</td>
<td>[S,L,L], SV, mitral atresia, 1-TGA, s/p PAB and Glenn shunt (SVC-RPA, bidirectional)</td>
<td>No</td>
<td>Lateral tunnel Fontan</td>
<td>1.3</td>
</tr>
</tbody>
</table>

AVC = atriovenricular canal; BAS = balloon atrial septostomy; CPA study = time interval between cavopulmonary anastomosis and study (years); CS = coronary sinus; DILV = double inlet left ventricle; DORV = double outlet right ventricle; HLHS = hypoplastic left heart syndrome; IVC = inferior vena cava; LPA = left pulmonary artery; LSVC = left superior vena cava; LV = left ventricle; MBTS = modified Blalock-Taussig shunt; MGA = malposition of great arteries; MPA = main pulmonary artery; PA = pulmonary artery; PAB = pulmonary artery band; PAPVR = partial anomalous pulmonary venous return; PDA = patent ductus arteriosus; PS = pulmonary stenosis; RA = right atrium; RPA = right pulmonary artery; RSVC = right superior vena cava; RV = right ventricle; s/p = status post; SV = single ventricle; SVC = superior vena cava; TGA = transposition of great arteries; VSD = ventricular septal defect.
between 12 lungs in the “without hepatic venous blood” group (12 of 12 or 100%) and 16 lungs in the “with hepatic venous blood” group (3 of 16 or 19%) was statistically significant (p < 0.01). Left lungs of Patients 8 and 9 and the right lung of Patient 11 were “lungs with hepatic venous blood” that had positive contrast echocardiograms. On reviewing angiograms, streaming of flow containing hepatic venous return away from the left pulmonary artery in Patient 8 and right pulmonary artery in Patient 11 was noted during injections of contrast to the Glenn shunt.

**DISCUSSION**

**Prevalence of PAVM in patients with CPA.** Although the exact prevalence of PAVM in patients with CPA remains unknown, Kopf et al. reported increasing incidence with time after CPA (3). They reported a prevalence of 31% of patients with Glenn shunts (mean 6.8 years from Glenn shunt). Other authors have described the development of PAVM in 21% to 25% of patients with Glenn shunt (5,6). These studies were based on angiographic diagnosis of PAVM. In the present study, we used contrast echocardiography and found a prevalence of 71% in patients with various forms of CPA. The prevalence of PAVM in our patients was 21% based on angiographic findings, which was consistent with previous reports. We speculate that the difference in prevalence of PAVM using contrast echocardiography in our patients might be due to the superior sensitivity of contrast echocardiography to traditional angiograms. To our knowledge, this is the first study to compare the findings of contrast echocardiography and angiography in diagnosing PAVM in patients with CPA. Further studies are needed to define the exact prevalence of PAVM by contrast echocardiography in patients with CPA.

**Contrast echocardiography versus angiography.** Hand-agitated saline solution has been proved to be safe and has been applied to many clinical settings to provide contrast during echocardiographic studies (21). Animal studies have shown that the lungs are superb filters for air bubbles (15). Although systematic evaluation of microbubbles as echocardiographic contrast is deficient, the incidence of false positive studies (i.e., air bubbles traverse the lungs and return to the left atrium) appears to be very low in lungs with intact capillary beds. It has been shown that bubbles will escape entrapment by the pulmonary capillary system only when the lungs are severely overloaded with air (20 ml of air or more) or when a balloon-tipped catheter is wedged in a pulmonary artery branch (15,22). In the present study, we injected less than 1 ml of air and avoided wedging catheters in small branches of pulmonary arteries during contrast injection. Therefore, we believe that it is unlikely that the positive contrast echocardiographic studies in our patients were falsely positive because of technical or methodological problems.

**In this study,** we demonstrated the superiority of contrast echocardiography to angiography in the sensitivity of detecting intrapulmonary arteriovenous shunting in patients with CPA. Although patients with positive contrast echocardiograms tended to have lower arterial saturation and higher hemoglobin level, this may be due to the fact that three of the four patients with negative contrast echocardiograms were in Group 5 (classic or lateral tunnel Fontan). We speculate that patients with positive contrast echocardiographic studies and negative angiograms represent a group of patients who have intrapulmonary arteriovenous shunting and are in the early stage of PAVM development. This group of patients may develop angiographically apparent PAVM over time and should be followed closely with...
periodic assessment. Contrast echocardiography can be used as a sensitive screening tool for detecting PAVM in patients with CPA. If screening contrast echocardiographic study is positive, selective angiography of both branch pulmonary arteries should be performed and examined carefully for PAVM. Because of its safety, ease of use and wide availability, bubble contrast echocardiography can also be used routinely during follow-up cardiac catheterization of patients who have initial negative angiograms for PAVM.

**Hepatic factor.** Pulmonary arteriovenous malformation is a known complication of patients with liver disease (8). Srivastava et al. reported a series of 10 patients with PAVM and found a common anatomic feature of exclusion of hepatic venous blood from pulmonary circulation (7). Therefore, the authors inferred that the development of PAVM after CPA is related to the diversion of normal hepatic venous flow from the pulmonary circulation. In the present study, we found all lungs with no hepatic venous blood to pulmonary circulation had positive contrast echocardiographic studies. There was significant difference in PAVM between lungs with hepatic venous flow to pulmonary circulation and lungs without hepatic venous flow to pulmonary circulation. Interestingly, two of the three patients with hepatic venous blood to pulmonary circulation who had positive contrast echocardiograms showed preferential streaming of inferior vena cava and hepatic venous flow away from the lungs where positive studies were obtained. In addition, patients with unidirectional Fontan operation (that diverts inferior vena cava and hepatic veins to the right pulmonary artery) had bubble contrast returned only from selective left pulmonary artery injection but not in selective right pulmonary artery injections. These findings are consistent with our previous experience and also with observations by others (20,23). Whether the so-called “hepatic factor” exists or not remains an unsolved problem. Results of the present study are strongly suggestive that lack of hepatic venous blood is a major contributing factor to the development of PAVM in patients after CPA. In addition, patients with CPA who have additional sources of pulmonary flow may also develop PAVM if streaming of hepatic venous return away from one lung is present. On the basis of our findings, we recommend that patients with no hepatic venous blood to the pulmonary circulation after CPA should receive careful examination of both lungs with contrast echocardiograms performed during subsequent cardiac catheterization.

**Additional sources of pulmonary flow.** Many centers are now performing bidirectional Glenn shunt as an intermediate stage of palliation with very low mortality and morbidity for young infants having functional single ventricle (24–26). Surgical strategy regarding additional sources of pulmonary flow for patients undergoing bidirectional Glenn shunt is debatable (27,28). Advocates for additional sources of pulmonary flow argue that pulmonary vascular complications, such as development of venovenous collateral vessels and poor growth of pulmonary vasculature, may result from the lack of additional sources of pulmonary blood flow (29). In the present study, we found that all lungs with no hepatic venous blood perfusion had positive contrast echocardiograms. Therefore, we believe that additional sources of pulmonary flow that include hepatic venous blood are advantageous in reducing the late complication of PAVM development.

**Study limitations.** This study has important limitations. We have demonstrated the usefulness of bubble contrast echocardiography in detecting angiographically negative PAVM. Bubble contrast echocardiography is safe and easy to perform during cardiac catheterization. In addition, the results can be recorded and analyzed semiquantitatively by comparing the amount of contrast return to the heart in a standard echocardiographic view. Therefore, contrast echocardiography offers an excellent tool for periodic follow-up assessment of patients after CPA. In this study, we did not attempt to quantify the amount of bubble contrast, and no follow-up comparison of the same patients over time was conducted. Further studies are needed to evaluate the correlation among clinical course, development of PAVM and contrast echocardiographic findings in patients with CPA over time.

One limitation of the applicability of this study protocol in clinical use is the need for selective pulmonary artery injections which can only be performed during cardiac catheterization. If contrast echocardiography is performed by injecting agitated saline solution to a peripheral vein of the upper extremity or neck in patients with Glenn shunt, the interpretation of results may be difficult due to 1) poor localization of bubble contrast return from left or right lung; or 2) positive study from other forms of abnormal connections such as systemic venovenous collaterals.

**Conclusions.** Bubble contrast echocardiography, compared with the traditional method of pulmonary angiography, is a more sensitive tool in detecting early development of PAVM in patients with CPA. The prevalence of PAVM in patients with CPA, measured by contrast echocardiography, may be much higher than what had been reported previously by using angiography. Lungs with no perfusion of hepatic venous blood are more likely to develop PAVM than lungs with hepatic venous blood flow. We recommend that children with CPA and unexplained desaturation should have screening and periodic follow-up bubble contrast echocardiograms.

**Reprint requests and correspondence:** Dr. Ruey-Kang R. Chang, Division of Cardiology, Department of Pediatrics, UCLA Medical Center, 10833 Le Conte Avenue, Los Angeles, California 90024. E-mail: rkchang@ucla.edu.
REFERENCES