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Systemic Venous Collateral Channels Causing Desaturation After Bidirectional Cavopulmonary Anastomosis: Evaluation and Management

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Objectives. **We sought to characterize the frequency, anatomic details and factors associated with the development of collateral channels between the superior and inferior vena caval systems after bidirectional cavopulmonary anastomosis.**

Background. **It is well known that systemic venous collateral channels often develop in patients who have undergone a classic Glenn shunt or bidirectional cavopulmonary anastomosis and that such collateral channels can lead to profound systemic desaturation. However, there have been few reports focusing on this problem.**

Methods. **Fifty-four patients (median age 1.4 years) who underwent bidirectional cavopulmonary anastomosis and had preoperative and postoperative angiograms available for review were studied retrospectively. Postoperative connections between the superior and inferior vena caval systems were identified and measured. Sites of collateral origin and entry from the superior and inferior venous systems, as well as the course taken in between, were recorded.**

Results. **At follow-up angiography performed 17 days to 46 months postoperatively, a total of 31 venous collateral channels were observed in 18 patients with a wide variety of primary morphologic diagnoses. The majority of these collateral channels (80%) originated from the brachiocephalic vein or its junction with the superior vena cava, and over half of them drained below the diaphragm. In patients who developed venous collateral channels, the mean transpulmonary pressure gradient early after bidirectional cavopulmonary anastomosis was higher ($p = 0.005$), and mean arterial oxygen saturation at follow-up was lower ($p = 0.009$). There were trends toward higher superior vena caval pressure early after the operation and at follow-up in patients with collateral channels and a higher likelihood of absent upper lobe pulmonary blood flow in these patients. Successful coil embolization of 10 collateral channels was performed in six patients, with a median increase in arterial oxygen saturation of 16%.**

Conclusions. **Angiographically detectable systemic venous collateral channels develop after bidirectional cavopulmonary anastomosis in a substantial number of patients (33% in the present series) with a variety of forms of a functional univentricular heart. Patients with venous collateral channels can be treated successfully with coil embolization, but the indications for embolization will depend on individual circumstances.**

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Bidirectional cavopulmonary anastomosis (BCPA) is increasingly being used as a primary palliative procedure (1) or an intermediate step in the staged repair of functional univentricular hearts (2–4). The principle of BCPA is to provide a controlled, low pressure source of effective pulmonary blood flow that has growth potential and relieves the volume load on a functionally univentricular heart (2). In BCPA physiology, the superior vena caval (SVC) and pulmonary artery systems are in direct continuity and, barring stenoses, are at equal pressures. The inferior vena cava (IVC), meanwhile, is isobaric with the right (systemic venous) and left (pulmonary venous) atria, assuming a nonrestrictive interatrial communication. Because the pressure in the pulmonary arteries must be higher than that in the pulmonary venous atrium in order to overcome pulmonary vascular resistance, SVC pressure is elevated over IVC pressure to a degree that is equivalent with the transpulmonary pressure gradient. It has been recognized since the earliest laboratory (5) and clinical (6) experience of Glenn et al. that such a physiologic arrangement can lead to the development of venous collateral connections between the higher pressure SVC system and the lower pressure IVC system. This finding has been confirmed in a number of studies of both the classic Glenn shunt (7) and the BCPA (3,8,9). In the presence of BCPA, a connection between the SVC and IVC systems allows for decompression of the higher pressure SVC system and concomitant reductions in pulmonary blood flow and arterial oxygen saturation. In more severe circum-

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Abbreviations and Acronyms

BCPA = bidirectional cavopulmonary anastomosis
IVC = inferior vena cava (caval)
SVC = superior vena cava (caval)

stances, these sequelae can result in marked hypoxia that is correctable only by occlusion of the collateral channels, either percutaneously or surgically. However, there have been few reports dedicated to this problem and none, so far as we know, that characterizes the prevalence, anatomic details and clinical correlations of systemic venous collateral channels arising after BCPA. Thus, in the present study, we describe our experience with post-BCPA venous collateral channels and discuss issues regarding their preoperative evaluation and postoperative management.

Methods

Patients and operative procedures. Fifty-five patients who underwent BCPA between January 1990 and May 1995 underwent both pre-BCPA and post-BCPA catheterization. Post-BCPA angiograms were unavailable for two patients. One of these patients was excluded from the study, but the other had a classic Glenn shunt before BCPA and was included in the study group for the purposes of characterizing systemic venous collateral channels but not for the purposes of statistical analysis. Patient diagnoses are summarized in Table 1. BCPA was the first surgical procedure performed in eight patients, whereas the remaining 46 patients had undergone one (n = 32) or more (n = 14) previous procedures, including three patients with a previously placed classic Glenn shunt.

The median age at operation was 1.4 years (range 1 month to 43 years), and 25 patients were <1 year old. Unilateral right BCPA was performed in 40 patients; unilateral left BCPA was performed in 1 patient; and all 13 patients with bilateral SVC systems underwent bilateral BCPA. A source of pulmonary blood flow, in addition to the BCPA, was placed (as a systemic to pulmonary artery shunt, n = 3) or allowed to remain (from the native anatomy or a previous procedure) in 35 patients (65%), as either anterograde flow through a banded or stenotic main pulmonary artery (n = 12) or a systemic to pulmonary artery shunt (n = 23). In general, additional pulmonary flow was preserved in patients with anterograde flow by banding the main pulmonary artery or by tightening the band if the pulmonary artery had previously been banded. In most young patients with a previous shunt, the shunt was taken down because the BCPA was performed at the same site. Another shunt was added only if blood gases showed an arterial partial pressure of oxygen below ~30 mm Hg or if the patient was beyond toddler age, in which case an extra source of pulmonary blood flow was always added or retained. The azygous vein was routinely ligated in all patients.

Table 1. Patient Diagnoses and Frequency of Collateral Channel Development by Diagnosis*

Diagnosis	No. of Pts	No. (%) of Pts Developing Collateral Channels
Double-inlet left ventricle	12	2 (17%)
Heterotaxy single ventricle (all were asplenia variants)	8	3 (38%)
Hypoplastic left heart syndrome	7	3 (43%)
Tricuspid atresia	7	1 (14%)
Pulmonary atresia with intact ventricular septum	5	2 (40%)
Mitral atresia	3	0
Ebstein's anomaly	2	0
Other single ventricle	11	6 (55%)

*Patients (Pts) total to 55 because 1 patient with Ebstein's anomaly and pulmonary atresia with an intact ventricular septum was included under both diagnoses.

Angiographic and hemodynamic evaluation. Pre-BCPA and post-BCPA angiograms and cardiac catheterization reports were reviewed retrospectively. Pre-BCPA angiograms and catheterization reports were assessed for the presence of systemic venous anomalies on SVC or brachiocephalic vein injection and to determine whether specific methods were used to search for such anomalies. Injections into the SVC system and the pulmonary arteries on post-BCPA angiograms were reviewed for the presence of visible communications between the SVC system and the lower pressure IVC system. Venous collateral channels, when present, were characterized according to size, origin from the SVC system, entry into the IVC system and course followed from the SVC to IVC system. The diameters of all collateral channels, the SVC (both if bilateral) and the brachiocephalic vein contralateral to the SVC system (in cases of unilateral SVC) were measured and corrected for magnification by standardization to the known size of the catheter. Collateral sizes were graded relative to the diameter of the brachiocephalic vein or the smaller of the two SVC systems when bilateral SVC systems were present. Collateral channels <25% of the diameter of the brachiocephalic vein or the smaller of the two bilateral SVC systems were graded as small; collateral channels between 25% and 50% of the brachiocephalic vein diameter were graded as moderate; and collateral channels >50% of the brachiocephalic vein diameter were graded as large. In patients who underwent coil embolization of systemic venous collateral channels at the time of follow-up angiography, the collateral channels were evaluated angiographically after the embolization procedure to assess the success of collateral occlusion. In addition, significant lobar perfusion defects were recorded; systemic to pulmonary artery collateral channels were noted and measured; and pulmonary artery sizes were measured and indexed to body surface area.

Hemodynamic and oximetric data were measured at pre-BCPA and post-BCPA cardiac catheterization. Aortic, central pulmonary artery and atrial pressures and oxygen saturations were recorded.

Data analysis. Preoperative and perioperative data were collected on retrospective review of patient records. Data are reported as either mean ± SD or median (range). SPSS for Windows, version 6.01 (SPPS Inc.) was used to perform statistical calculations. The primary dependent variable analyzed was the presence of new systemic venous collateral channels on follow-up angiography. The unit of analysis was not the collateral channel but the patient, so patients with multiple collateral channels were given the same weight as those with a single collateral channel. The independent variables analyzed are listed in the Appendix. Paired two-tailed *t* test analysis was used to compare pre-BCPA and post-BCPA catheterization/angiography data. The Fisher exact test and unpaired *t* test were used to compare frequencies of dichotomous variables and means of continuous variables between patients who did and did not develop venous collateral channels during the follow-up period. Variables significant by univariate analysis were entered into forward stepwise logistic regression analysis.

Results

Postoperative angiography was performed between 2 weeks and 46 months after BCPA (median 15.4 months). In 30 of the 54 patients, postoperative catheterization was performed as a pre-Fontan catheterization to evaluate hemodynamic data. Restriction to ventricular inflow, whether at the level of the interatrial communication or the atrioventricular valves, was not detected in any patient. A total of 31 measurable venous collateral channels were detected in 18 patients (33%). Primary morphologic diagnoses of these 18 patients are listed in Table 1. In three of these patients, collateral channels were diagnosed early after BCPA (that is, within 30 days or during the same hospital period). In six of the patients in whom collateral channels were detected, pre-BCPA evaluation of the SVC system had been performed with special techniques, including either power injection into the SVC/brachiocephalic vein or hand injection into the SVC/brachiocephalic vein during SVC balloon occlusion to increase venous pressures. No collateral channels were detected during pre-BCPA evaluation in these patients. In the other 12 patients, either special pre-BCPA evaluation was not performed (n = 10) or a pre-BCPA angiogram was not available for review (n = 2). There were no significant differences between patients who did and did not develop venous collateral channels in any demographic, morphologic or preoperative catheterization/angiographic variable.

Sites of collateral origin from the SVC system, course from the SVC to IVC system and entry into the IVC system are depicted in Figure 1. The great majority of collateral channels (81%) arose from the brachiocephalic vein or the junction between the brachiocephalic vein and the SVC (Fig. 2 and 3). Three of the collateral channels appeared to be residual left SVC systems or levoatrial cardinal veins (Fig. 3A). The course of collateral flow from the SVC to IVC system varied evenly between posterior veins (azygous/hemiazygous and lumbar; Fig. 2), middle mediastinal veins (intercostal/pericardial and left SVC; Fig. 3) and anterior veins (internal thoracic; Fig. 2 and 3). Nearly two-thirds of collateral channels drained to the IVC system below the diaphragm. Six collateral channels (19%) were small in caliber, 12 (39%) were moderate and 13 (42%) were large. Collateral size appeared to correlate with the course taken from the SVC to IVC system: 10 of 11 collateral channels draining through the left SVC or azygous/hemiazygous veins were large, whereas 8 of 9 collateral channels draining through the intercostal/pericardial veins were small or moderate in size. In one patient, significant surface venous collateral channels were visible on the anterior chest and abdominal walls, similar to the patient described by Mathur and Glenn (6).

Coil embolization of 10 venous collateral channels was performed in six patients between 2 weeks and 46 months after BCPA (median 2.2 months; Fig. 2). In all three of the patients in whom collateral channels were identified early after BCPA, the channels were successfully coiled at the time of diagnosis. Postembolization injection proximal to the occlusion revealed complete occlusion in all but one patient, in whom trace residual flow was observed (Fig. 2C), for a success rate of 100%. Arterial oxygen saturations rose from a median of 65% before embolization (range 52% to 81%) to a median of 84% after coil occlusion (range 70% to 90%). The median increase was 16% (range 9% to 20%). It should be noted that two of these patients were in the hospital for a prolonged period for multiple problems and died 2 and 8 months after coil embolization (one from a subsequent postoperative myocardial infarction and the other from acute bronchopneumonia).

Pertinent results of univariate statistical analysis for factors associated with venous collateral channels after BCPA are summarized in Table 2. Of the factors listed in this table as significant ($p < 0.05$) by univariate analysis, only early postoperative transpulmonary gradient ($p = 0.01$) was found to differ significantly between patients with and without venous collateral channels on multivariate analysis. Other than the factors listed in Table 2, there were no significant correlations between the development of venous collateral channels and any of the angiographic, hemodynamic, operative, morphologic or demographic factors analyzed (Appendix). Although saturations and transpulmonary gradient were significantly different at follow-up among patients with and without collateral channels, no threshold was detected for either measure which could serve as a reliable marker for the development of collateral channels.

The duration from BCPA to follow-up catheterization did not correlate with the presence or absence of collateral channels, but four of the seven patients who underwent catheterization within 3 months of BCPA did have venous collateral channels. In three of these patients, all with arterial oxygen saturations ≤65%, coil embolization was performed with significant improvement in saturations (at least 12% increase).

A modified Fontan operation has been completed in 30 of the patients in the present study, between 1 and 47 months after BCPA (median 18 months). All of these patients had an extracardiac conduit Fontan placed, except for one patient who

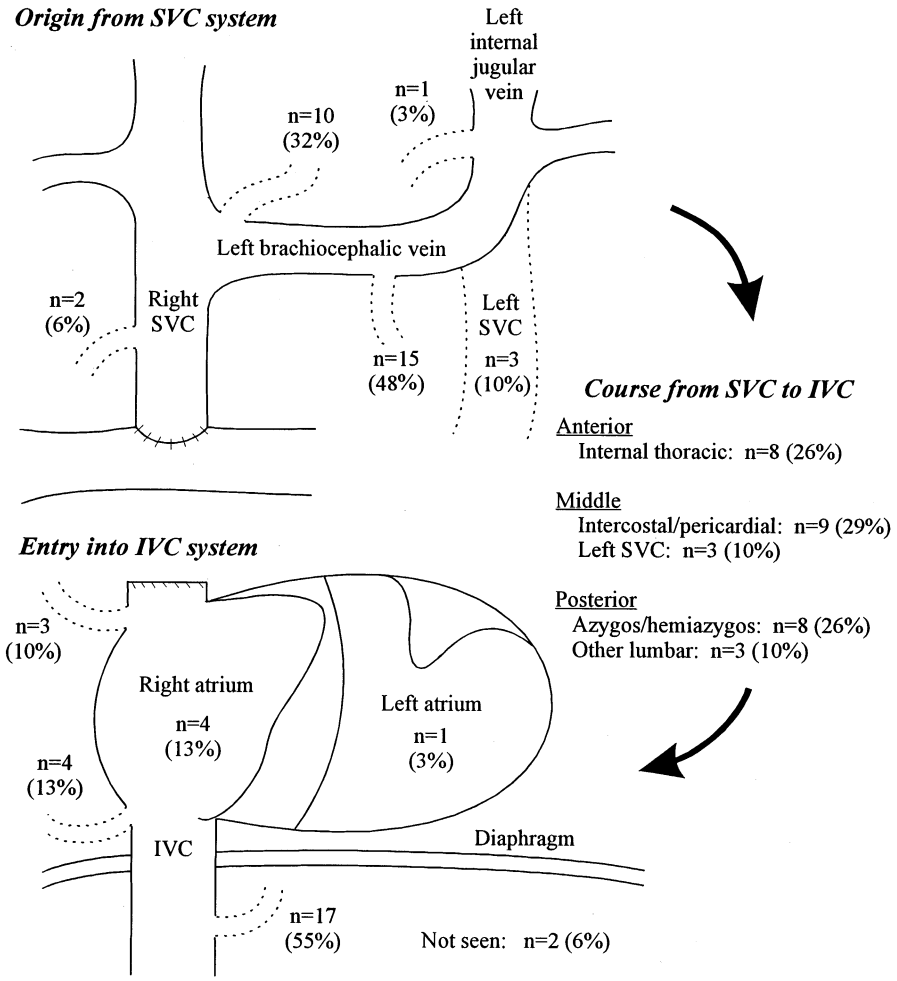

Figure 1. Sites of collateral channel origin from the SVC system **(top)** and entry into the IVC system **(bottom).** (Note: Sites of collateral channel origin and entry are not precisely as depicted; rather, they are from the indicated vessel or vascular junction.) Courses taken by the collateral channels from the SVC to IVC system are summarized at right. **Dashed lines** = collateral vessels.

underwent Fontan completion at another institution. Systemic venous collateral channels had been documented in five of these patients, two of whom had undergone embolization (one early after BCPA). There was one early death from a cerebrovascular accident after the modified Fontan operation and repair of the stenotic pulmonary venous orifice. Otherwise, there was no significant morbidity early after Fontan completion. All 29 hospital survivors are alive and have an intact Fontan circulation.

Discussion

As more and more data have become available on patients with a pulmonary circulation entirely or primarily dependent on BCPA, it has become clear that these patients frequently manifest complications related to vascular changes, including pulmonary arteriovenous fistulae (10), aortopulmonary collateral arteries (11) and venous collateral channels between the SVC and IVC. Although aortopulmonary collateral channels are of unknown physiologic significance in this setting, both arteriovenous fistulae and systemic venous collateral channels can result in dramatic and life-threatening cyanosis (9,12).

Why do systemic venous collateral channels develop? It appears from our experience and other published reports that venous collateral channels can manifest at any time after BCPA and can be of varying size and significance (6–9). In the small subset of patients in whom venous collateral channels develop early after BCPA and lead to significant postoperative desaturation, this complication can be lethal (9) and must be remedied, either surgically or by means of percutaneous coil embolization. Collateral channels that are discovered later are likely to be of less acute physiologic importance but may still cause precipitous drops in arterial oxygen saturation and require embolization or ligation. Although the duration between BCPA and catheterization did not correlate with collateral channel development, four of the seven patients catheterized within 3 months of BCPA had venous collateral channels. Three of these patients had arterial oxygen saturations ≤65%. Patients who develop severe desaturation within several months of BCPA are likely to have either venous collateral channels (9) or pulmonary arteriovenous malformations (12) and should be evaluated and treated appropriately.

It remains unclear why some patients develop venous collateral channels after BCPA and others do not, and why the

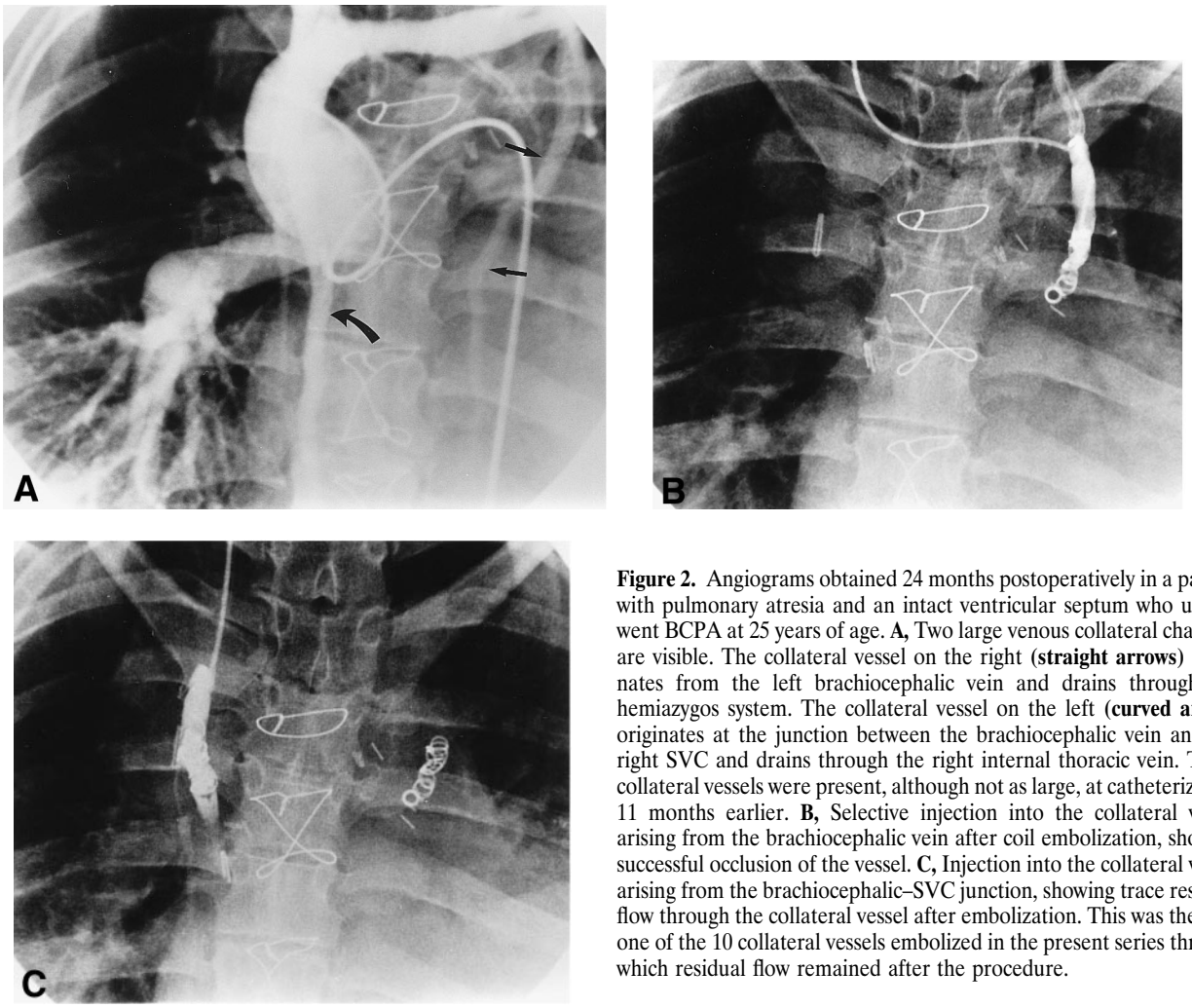

Figure 2. Angiograms obtained 24 months postoperatively in a patient with pulmonary atresia and an intact ventricular septum who underwent BCPA at 25 years of age. **A,** Two large venous collateral channels are visible. The collateral vessel on the right **(straight arrows)** originates from the left brachiocephalic vein and drains through the hemiazygos system. The collateral vessel on the left **(curved arrow)** originates at the junction between the brachiocephalic vein and the right SVC and drains through the right internal thoracic vein. These collateral vessels were present, although not as large, at catheterization 11 months earlier. **B,** Selective injection into the collateral vessel arising from the brachiocephalic vein after coil embolization, showing successful occlusion of the vessel. **C,** Injection into the collateral vessel arising from the brachiocephalic–SVC junction, showing trace residual flow through the collateral vessel after embolization. This was the only one of the 10 collateral vessels embolized in the present series through which residual flow remained after the procedure.

rates and extent of collateral channel development vary so greatly. Intuitively, it seems logical that the likelihood of collateral channel development will correlate with the pressure differential between the SVC and IVC systems. In the present study, this logic is supported, as the transpulmonary gradient was significantly higher on the first day after BCPA in patients who went on to develop venous collateral channels than in those who did not ($p = 0.005$). In fact, the finding that this correlation was no longer significant at follow-up may reflect the decompression of the SVC system through the venous collateral channels. These findings are particularly interesting, given that the transpulmonary gradient can vary so widely after BCPA and because the magnitudes of the pressures at issue are relatively small. However, it is difficult to know whether these statistically significant differences translate into clinical differences of sufficient magnitude to affect how BCPA and Fontan repair are approached. There is some evidence from computational fluid dynamic studies of cavopulmonary connections to suggest that small variations in pressure can reflect significant differences in energy conservation at the point of

Figure 3. Angiogram from an 18-month old patient with tricuspid atresia who underwent BCPA 8 months earlier. Two venous collateral channels are shown arising from the left brachiocephalic vein and draining through a pericardial vein **(right, straight arrow)** and the left internal thoracic vein **(left, curved arrow)**. These collateral vessels were not embolized.

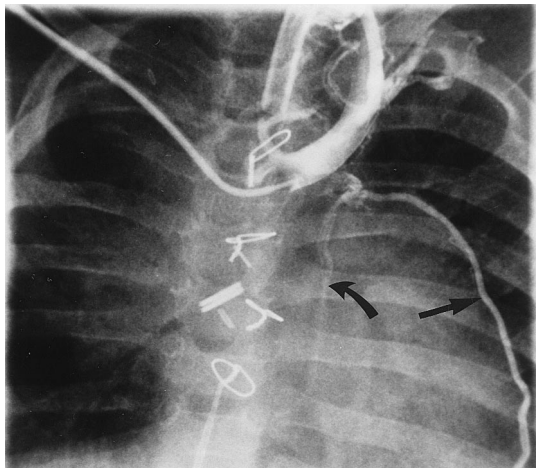

Table 2. Results of Statistical Analysis for Factors Associated With Venous Collateral Channels After Bidirectional Cavopulmonary Anastomosis

	Venous Collateral Channels		
	Present (n = 18)	Absent (n = 36)	Univariate p Value
Diagnostic and preoperative			
Heterotaxy syndrome (% of pts)	17	14	0.79
Anterograde PBF before BCPA (% of pts)	28	53	0.084
Indexed PVR (Wood units)	2.8 ± 1.0	2.4 ± 1.3	0.35
Operative: extra source of PBF	56	69	0.32
Early postoperative			
BCPA pressure (mm Hg)	16.9 ± 3.0	14.7 ± 3.8	0.056
Transpulmonary gradient (mm Hg)	10.5 ± 2.7	7.8 ± 2.6	0.005
Postextubation Sao_2 (%)	83.9 ± 7.0	86.5 ± 5.9	0.17
Follow-up catheterization			
BCPA pressure (mm Hg)	13.2 ± 4.5	10.9 ± 4.7	0.09
Transpulmonary gradient (mm Hg)	7.2 ± 3.3	5.6 ± 3.2	0.11
Sao_2 (%)	77.7 ± 8.8	82.9 ± 4.9	0.009
Aortopulmonary collateral arteries (% of pts)	46	45	0.86
Absent upper lobe flow to either lung (% of pts)	33	11	0.065

*A source of pulmonary blood flow (PBF) anterograde from the ventricle(s) was present, as opposed to that through a systemic to pulmonary artery shunt only. Data presented are mean value ± SD, unless otherwise indicated. BCPA = bidirectional cavopulmonary anastomosis; pts = patients; PVR = pulmonary vascular resistance; Sao_2 = arterial oxygen saturation.

cavopulmonary anastomosis (13,14). Moreover, the more subtle factors responsible for a Fontan circulation that is successful in the long term are poorly understood, and it may very well be the case that such subtle changes in hemodynamic factors (i.e., 1- to 2-mm Hg pressure differences) do translate into significant differences in clinical outcome. Ultimately, more sensitive and detailed studies of cardiac and vascular hemodynamic interactions and their relation to Fontan outcome will be necessary to make determinations regarding the clinical significance of such small hemodynamic differences.

In addition to the significance of the transpulmonary gradient, there are undoubtedly multiple factors that contribute to the development of venous collateral channels after BCPA. One factor that is almost certainly related to the propensity of any given individual to develop venous collateral channels after BCPA is the patient's native venous anatomy, which varies considerably even in so-called normal cardiovascular systems. Although collateral connections between the SVC and IVC systems are sometimes referred to as "abnormal" (9), it may be the case that only the patency of such channels, and not their existence, is abnormal.

Evaluation and significance of systemic venous collateral channels. Although venous anomalies are common in patients with visceral heterotaxy, and a conspicuous portion of patients who develop venous collateral channels after BCPA have heterotaxy syndromes, our experience shows that collateral channels can form in patients with any type of congenital heart disease. Thus, we believe that the SVC system should be evaluated in all patients undergoing pre-BCPA cardiac catheterization, with either a power injection in the brachiocephalic vein/SVC or a hand injection proximal to a balloon occlusion of the SVC(s). Such studies should not be considered definitive, as they will likely detect channels in only a small percentage of the patients in whom collateral channels eventually develop after BCPA. However, this evaluation will reveal anomalies in some patients and adds negligible time and risk to the catheterization procedure. Moreover, the patients in whom pre-BCPA evaluation does reveal collateral channels may be those who are most likely to suffer from decompression and desaturation in the early postoperative period, which is potentially the most serious collateral channel–related complication.

Substantially decreased or absent upper lobe pulmonary blood flow was observed on the angiogram in nearly 20% of our patients at post-BCPA and has also been observed in a significant number of patients after a classic Glenn shunt (7). Our finding that patients with venous collateral channels after BCPA are more likely than those with no collateral channels to have absent upper lobe pulmonary blood flow ($p = 0.065$) supports the report by Trusler et al. (7), who describe the same association in their cohort of patients who had undergone classic Glenn anastomosis. The nature of this relation is unclear. It is tempting to speculate that segmental loss or arterial collateral flow, or both, would increase the transpulmonary gradient, thus promoting venous collateral formation. In the present series, however, there were no significant hemodynamic or oximetric differences between patients with and without upper lobe flow, and no difference in the prevalence of pulmonary artery collateral channels in patients with and without upper lobe flow.

The most significant potential complication associated with venous collateral channels is systemic desaturation. Thus, it is not surprising that arterial oxygen saturations at follow-up

catheterization were significantly lower in patients with collateral channels than in those without ($p = 0.009$). This correlation did not hold true after extubation in the post-BCPA period, probably because collateral channels had yet to form (or become patent) in most patients. Venous collateral channels, whether they develop early or late, should always be considered as a potential cause when patients with BCPA physiology develop unexplained hypoxia or desaturation. Although saturations were significantly different at follow-up among patients with and without collateral channels, no threshold was detected which could serve as a reliable marker for the development of collateral channels. These collateral channels are readily diagnosed, and, importantly, they are fairly simple to treat.

Management of systemic venous collateral channels after BCPA. In our experience, coil embolization of venous collateral channels has been highly successful, as has also proven to be the case with other types of undesired vessels (15). We achieved complete occlusion of all but one collateral channel in which only trace residual flow was detected, with resulting increases in arterial oxygen saturation of between 9% and 20%. In most cases, embolization of venous collateral channels is technically straightforward. However, it is important to recognize that there may be circumstances in which it is difficult or impossible to navigate the catheter into the collateral channel, such as when the channel arises from a point close to the catheter access site. Such difficulties can usually be overcome with more proximal or contralateral access, although the technical options available in challenging circumstances are limited by the discontinuity between the SVC and IVC systems.

In the majority of patients, BCPA is an intermediate procedure en route to a modified Fontan operation. Once the Fontan procedure is completed, venous collateral channels draining below the heart are of no physiologic significance. Thus, the importance of treating venous collateral channels will vary according to the circumstances. There is no need to embolize collateral channels draining below the diaphragm in an asymptomatic or mildly cyanotic patient undergoing BCPA if a Fontan completion is planned. In contrast, collateral channels to the pulmonary venous atrium in such patients should be embolized at the time of diagnosis, unless there is absolute certainty that the collateral vessels will be identifiable and easily accessible during the Fontan operation, in which case either embolization or surgical ligation may be elected. However, even in such circumstances, we believe that the option of surgical ligation should be reserved for situations in which coil embolization is technically difficult or unsuccessful. In all other patients, collateral channels draining to the heart itself, to a coronary sinus or to either cavoatrial junction should be embolized whenever they are encountered, as they are an obligatory right to left shunt. An exception are patients in whom the BCPA is used as part of a one-and-a-half ventricle repair, in which case only collateral channels draining to the pulmonary venous atrium need to be occluded. If collateral channels are suspected in patients in whom BCPA is planned as an intermediate or long-term palliation, treatment should be undertaken on the basis of symptoms. Catheterization specifically for the purpose of evaluating venous collateral channels in such patients is generally not indicated, unless the patients are symptomatic or significantly desaturated. However, if collateral channels are detected in an asymptomatic patient who undergoes catheterization for some other reason, it is reasonable to embolize the collateral channels. Another option for patients on a Fontan track who are found to have collateral channels is to proceed to the Fontan operation instead of embolization. However, this may be inadvisable for several reasons. The factors leading to systemic venous collateral channels may be indicative of a patient with suboptimal hemodynamic data for a Fontan circulation, and experience has shown that proper selection is probably one of the most important determinants of Fontan outcome. Also, timing may be a reason not to proceed to the Fontan operation earlier than planned. For example, because we perform an extracardiac conduit Fontan operation in almost all patients with a single-ventricle, we prefer to wait until the patient is ~15 kg before completing the Fontan operation, at which time the patient should be large enough to accommodate an adult-sized conduit (20 to 22 mm).

Conclusions. Venous collateral channels develop in up to one-third of patients after BCPA and can cause significant desaturation, even in the early postoperative period. Evaluation for venous collateral channels at catheterization is simple and low risk and should be performed routinely in all patients before BCPA and at follow-up catheterizations. If present, venous collateral channels can be successfully closed using coil embolization techniques.

Appendix

*Variables Analyzed for Association/Correlation With Development of Systemic Venous Collateral Channels**

Demographic and morphologic variables
- Age at operation
  - Continuous
  - <6 months (yes/no)
- Previous palliative procedure (yes/no)
- Previous systemic–pulmonary artery shunt (yes/no)
- Anterograde pulmonary blood flow before BCPA
- Diagnosis
  - Heterotaxy single ventricle (yes/no)

Catheterization and angiographic variables†
- Arterial oxygen saturation
- Mean pulmonary artery blood pressure
- Ventricular (dominant/single) end-diastolic pressure
- Indexed pulmonary blood flow

*Only factors in Table 2 were found to correlate with the development of systemic venous collateral channels.

†Pre-BCPA and post-BCPA absolute values, as well as absolute and percent changes from pre-BCPA to post-BCPA, were considered separate variables.

Pulmonary/systemic blood flow ratio
Continuous
Indexed pulmonary vascular resistance
Pulmonary artery index
Total (bifurcation, lower lobe)
Right (bifurcation, lower lobe)
Left (bifurcation, lower lobe)
Systemic–pulmonary collateral arteries (yes/no)
Absent upper lobe blood flow (yes/no)

Operative variables

Bilateral BCPA (yes/no)
Pulmonary artery augmentation (yes/no)
Intracardiac procedures performed (yes/no)
Extra source of pulmonary blood flow (yes/no)
Type (shunt/anterograde)
Cardiopulmonary bypass (yes/no)
Time

Early postoperative variables

Arterial oxygen saturation
Mean pulmonary artery blood pressure
Ventricular (dominant/single) end-diastolic pressure
Pulmonary blood flow-related reoperation (yes/no)
Effusions (chest tube drainage) lasting >7 days (yes/no)

Late postoperative variables

Pulmonary blood flow-related reoperation before Fontan (yes/no)
Time between pre-BCPA and post-BCPA catheterization
Time between BCPA and post-BCPA catheterization

Variables relating to Fontan completion

Time between BCPA and Fontan completion
Early post-Fontan death (yes/no)
Duration of post-Fontan effusions (chest tube drainage)
Continuous; >7 days (yes/no)

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