The fate of the unligated vertical vein after surgical correction of total anomalous pulmonary venous connection in early infancy

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The vertical vein, when present, is usually ligated or divided during surgical correction of total anomalous pulmonary venous connection (TAPVC).[1] However, some recent work has suggested that ligation of the vertical vein is not a mandatory component of successful surgical correction of this anomaly. In selected cases, nonligation of the vertical vein may be an advantage and the unligated vein could be expected to close off.[2] We investigated the fate of the vertical vein in 4 consecutive patients in whom it was left unligated for various reasons.

Clinical Summary

From 1995 to 1999, ten patients below the age of 90 days underwent surgical correction of TAPVC in our institution. Of these, 4 patients did not have vertical vein ligation for various reasons. The details of these patients are presented in Table 1 (age 5-80 days; weight 3-4.5 kg). All the patients were operated on with cardiopulmonary bypass, bicaval cannulation, and low flow when needed. The anastomosis was constructed through a superior approach with 7-0 Prolene suture (Ethicon, Inc, Somerville, NJ) between a widely opened common pulmonary vein and the roof and posterior wall of left atrium including the appendage. The foramen ovale or atrial septal defect was closed through a separate incision in the right atrium. The vertical vein was dissected and looped, and the decision to ligate or not was made while the patient was being weaned from bypass. In the 3 cases of supracardiac TAPVC, hemodynamic instability was the reason for leaving the vertical vein open. In the single case of infradiaphragmatic TAPVC, the descending vertical vein was electively left alone. The hospital stay of these patients ranged from 10 to 25 days (mean 17 days). The postoperative course was marred by pulmonary hypertensive crises and recurrent supraventricular tachycardia in patient 1 but was smooth in the others. All patients received sodium nitroprusside infusion while receiving ventilatory support and oral captopril after extubation.

Two-dimensional echocardiography with color flow imaging was performed daily on all patients during their stay in the intensive care unit, with special attention to flow in the vertical vein. After discharge from the hospital, echocardiography was repeated at 1, 3, and 6 months during clinic reviews. In patient 1, minimal flow was demonstrable in the vertical vein at the time of discharge but none was present at 1 month. An anastomotic stricture later developed in this child. In patient 2, the vertical vein remained widely patent in all examinations despite an adequate anastomosis between the common pulmonary vein and the left atrium (no Doppler gradient, smooth flow). In patient 3, no flow was demonstrable in the descending vertical vein 1 week after the operation, but there was persistent enlargement of the right atrium and right ventricle. Patient 4 had a widely patent vertical vein with profuse flow in all examinations. In this patient the anastomosis was noticed to be narrow from the beginning.

All the patients underwent cardiac catheterization 6 months to 2 years after the initial operation. Patient 1 had evidence of severe pulmonary arterial hypertension and a gradient of 17 mm Hg between the pulmonary capillary wedge pressure (PCWP) and left ventricular end-diastolic pressure (LVEDP). Selective pulmonary angiograms in levophase demonstrated anastomotic stricture. The vertical vein was not patent. Patient 2 had normal pulmonary artery pressures. The widely patent vertical vein was easily entered with a 6F balloon wedge catheter. The pulmonary venous pressure was 10 mm Hg and did not rise further on balloon occlusion. There was no gradient between the PCWP and LVEDP. Pulmonary artery angiograms showed unobstructed flow from pulmonary veins to the left atrium, but there was equally brisk flow across the vertical vein outlining a figure-of-8. The flow from the left pulmonary veins seemed to favor the vertical vein, whereas that from the right-sided veins favored the left atrium (Figure 1). Patient 3 had normal pulmonary artery pressures, no significant gradient between the PCWP and LVEDP, and no detectable shunt on oximetry. A pulmonary artery angiogram showed smooth and brisk levophase flow into the left atrium but also opacified a descending vertical vein draining into the portal vein (Figure 2). Patient 4 had

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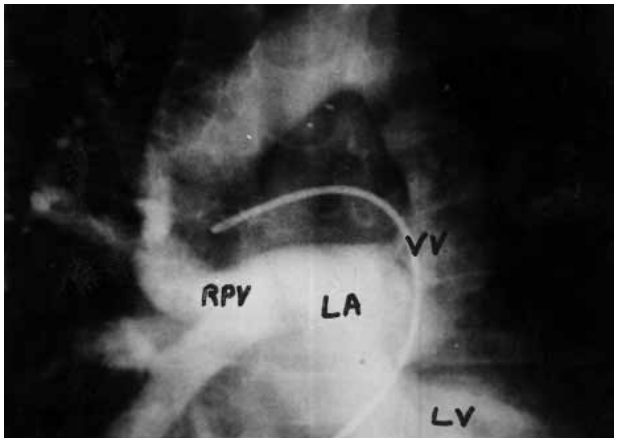

Figure 1. Postoperative pulmonary arteriogram (levophase) in patient 2. *VV,* Vertical vein; *RPV,* right pulmonary vein; *LA,* left atrium; *LV,* left ventricle.

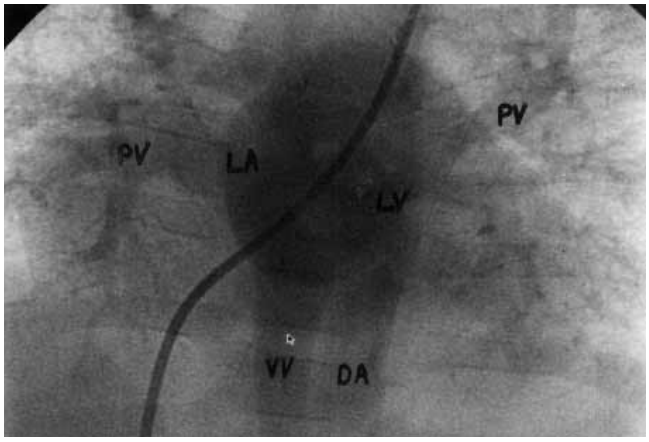

Figure 2. Postoperative pulmonary arteriogram (levophase) in patient 3. *VV,* Vertical vein; *PV,* pulmonary veins; *LA,* left atrium; *LV,* left ventricle; *DA,* descending aorta.

TABLE 1. Postoperative cardiac catheterization data

Patient No.	1	2	3	4
Time to catheterization (mo after surgery)	8	6	24	3
PAP (mm Hg)				
Systolic/diastolic	70/30	35/7	25/10	55/25
Mean	45	17	15	35
PCWP (mm Hg)	25	10	12.5	22
Vertical vein status	Closed	Patent	Patent	Patent
Innominate vein oxygen saturation (%)	75	97	56 (IVC 61)	95
Angiogram	Anastomotic stricture	Large vertical vein; wide anastomosis	Unobstructed pulmonary venous return to LA; descending vertical vein to portal vein	Large vertical vein; anastomotic stricture

PAP, Pulmonary artery pressure; *PCWP,* pulmonary capillary wedge pressure; *IVC,* inferior vena cava; *LA,* left atrium.

severe pulmonary hypertension and a gradient of 15 mm Hg between the PCWP and LVEDP. Vertical vein occlusion with a 5F balloon wedge catheter caused an increase in pulmonary venous pressure of 7 mm Hg. Selective pulmonary artery angiograms showed anastomotic obstruction and vigorous flow along the vertical vein.

Patient 1 had revision of the anastomosis. However, 6 months later she again had anastomotic stricture, necessitating balloon dilatation. Patient 2 underwent vertical vein ligation through a left anterior thoracotomy, as the vein and its connection to the left atrium were considered too big for any form of transcatheter closure. Patient 3 is awaiting surgery for ligation of the descending vertical vein. Patient 4 had anastomotic revision and vertical vein ligation. All the patients were clinically doing well at last follow-up examination.

Discussion

This report was prompted by the recent article by Cope and associates,[2] questioning the necessity and desirability of vertical vein ligation in surgical correction of TAPVC. Previous studies have shown that in TAPVC, the left atrium is small and lacking in normal compliance and reservoir function. The left ventricle also may be small and dysfunctional, especially when pulmonary venous obstruction coexists.[3] In such cases, it has been argued that elective nonligation of the vertical vein provides a "safety valve" to the small left atrium, which may not be able to handle the entire pulmonary venous return.[2] It remains speculative whether diverting part of the pulmonary venous return into a patent vertical vein would deprive the left side of the heart of its physiologic stimulus to develop.

In infradiaphragmatic TAPVC, successful surgical correction without ligation of the descending vertical vein was reported in 1967.[4] It was postulated that the high resistance of the hepatic parenchyma would discourage flow and promote closure of the vein. In their series of 9 cases, Cope and colleagues[2] argued that such an approach was also valid for the management of an ascending vertical vein in supracardiac TAPVC. They suggested that nonligation of the vertical vein might be particularly useful and life-

saving when the patient was in hemodynamically unstable condition. Importantly, they observed in their cases that the unligated vertical vein invariably closed spontaneously if the anastomosis between the common pulmonary vein and left atrium was adequate. However, our data show that such spontaneous closure does not necessarily occur, nor did we find any reliable predictors of spontaneous closure. In patient 2, who had a persistent large vertical vein, the anastomosis was adequate and pressure in the common pulmonary vein was low. Both these factors should have discouraged flow in the anomalous channel. It appeared that the streaming of flow from the left pulmonary veins favored the vertical vein, contributing to its patency. In patient 1, who had late anastomotic stricture and high pressure in the common pulmonary vein, the vertical vein remained closed. In the case of infradiaphragmatic TAPVC, echocardiography showed persistent enlargement of the right atrium and ventricle, even though Doppler color flow imaging failed to show any flow in the descending vertical vein. An angiogram revealed persistent patency of the vertical vein after 2 years, even though the anastomosis was adequate.

Our findings do not support *elective* nonligation of the vertical vein in surgical correction of TAPVC. We do wish to emphasize that spontaneous closure of an unligated vertical vein cannot be taken for granted. If the vertical vein is left unligated in a TAPVC correction, the patient should have close echocardiographic monitoring until complete cessation of flow is documented. In case the vertical vein remains patent, adequacy of the anastomosis should be verified. If the anastomosis is adequate, the vertical vein should be interrupted surgically or interventionally. Patients with anastomotic narrowing would need reoperation and vertical vein ligation.

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