Determinants of Doppler Pulmonary Flow Components

Recently, CP Appelton presented an elegant animal study on the hemodynamic determinants of Doppler pulmonary venous flow velocity components (1). One of the conclusions was that the late systolic pressure increase in the pulmonary vein system was a direct result of the right ventricular stroke volume. The late systolic component of the pulmonary venous flow curve was a direct result of the right heart filling the pulmonary veins and left atrium.

Patients with univentricular hearts palliated with a Fontan or total cavopulmonary connection (TCPC) procedure do have a circulation where the systemic ventricle is the only ventricle causing pressure on the total circuit (i.e., there is no right ventricle giving a pressure head for the pulmonary circulation which is then dependant on the systemic venous pressure). We examined a group of patients with a TCPC circulation and found the same pulmonary venous flow patterns as seen in biventricular hearts with four phases: atrial reversal, early and late systolic, and diastolic flow (2). In some patients, the early and late systolic flow was difficult to separate and was not analyzed separately. Eight of 11 patients with no or minimal atrioventricular valve regurgitation did have a (late) systolic velocity within the mean value ± 2SD for normals. Our opinion is that the pulmonary vein flow patterns are controlled by left heart mechanics.

DAG E. TEIEN, MD, PhD
Department of Pediatrics, Section of Pediatric Cardiology
Umeå University Hospital
90185 Umeå, Sweden
e-mail: dag.teien@pediatri.umu.se

References

Reply

As Dr. Teien states in his letter, their study shows that patients with univentricular hearts palliated with a Fontan total cavopulmonary connection (TCPC) procedure do have a pulmonary venous (PV) flow pattern that is somewhat similar to that seen in normal biventricular hearts. Specifically, they also observe four phases of PV flow. As in normal patients, the early and late systolic flow is sometimes difficult to separate with transthoracic recordings and therefore these two flows were not analyzed separately. Because of the similarities of flow in TCPC and normal hearts, the authors conclude that pulmonary venous flow is controlled predominately by left heart mechanics. In our animal study, we had concluded that the late systolic flow was influenced by the pressure increase in the pulmonary venous system as a direct result of right ventricular stroke volume.

After reviewing the article by Dr. Teien and associates, I agree that TCPC hearts have pulmonary venous recordings with four phases. However, as shown in their Table 1 and Table 2, the TCPC patients tend to have lower systolic flow velocities and systolic flow velocity intervals. Two explanations are possible. Because pulmonary venous flow is the result of the pressure difference between pulmonary artery and left atrial pressure, a reduced systolic fraction may occur if there is a blunted rise in pulmonary venous pressure with systole, or alternatively if there is an increased pressure rise in the left atrium (usually due to left atrial noncompliance). Without direct pressure measurement, I think it is impossible in the TCPC patients to know the primary mechanism.

Overall, the conclusions made in both papers are not mutually exclusive. Our data unquestionably shows there is a late systolic pressure increase in the pulmonary venous system, which is a direct result of RV stroke volume. In TCPC patients, the increase in pressure is caused by a more constant and less pulsatile flow. Whether this latter situation results in a tendency toward a lesser increase in late systolic PV pressure and flow is speculative, but would actually support our assertion that RV output is an important and additional determinant of pulmonary venous systolic flow. On the other hand, the point is well taken that once the PV reservoir is charged with volume and pressure, alterations in “downstream” left atrial pressure due to mitral valve opening and left ventricular filling help determine the characteristic flow velocity pattern seen.

In summary, I thank Dr. Teien and associates for bringing to my attention their important article on pulmonary venous blood flow in TCPC hearts. I agree that even further study will be necessary to determine the role right ventricular stroke volume plays in determining pulmonary venous are late systolic flow that is independent from “downstream” LA events.

CHRISTOPHER P. APPLETON, MD, FACC
Cardiovascular Diseases, 3A
13400 East Shea Boulevard
Scottsdale, Arizona 85259

Seasonal Variations in the Incidence of Acute Myocardial Infarction

We have read with interest the recent article by Spencer et al. (1), which reported a higher incidence of acute myocardial infarction (AMI) in winter than in summer months (1). We recently analyzed a series of 9,571 computerized patient records from general practices in the UK (General Practitioner Research Database) (2). We identified 1922 cases who had a first-time AMI at age 75 or younger in the absence of recorded clinical risk factors for AMI. We matched 4 control subjects to each case on age, sex, general practice and calendar time. We conducted both a case control and a case crossover analysis to explore whether respiratory tract infections were associated with an increased risk of developing an AMI. We assessed for all cases and controls if and when they last had a chest infection recorded in the patient record before the date when the case developed the AMI (index date).

Our findings with regard to seasonal differences in the incidence of AMIs were consistent with the results of Spencer et al. (1). We have also observed the highest frequency of AMIs in January, and the lowest in summer (June). We have further observed an approximately threefold increased risk of developing an AMI in relation to an acute respiratory tract infection within the 10 days immediately preceding the index date. The risk was highest for patients who had a chest infection within 5 days of the index date (OR 3.6, 95% CI 2.2–5.7), and
gradually decreased over a period of approximately two weeks towards an OR of one (2).

The possible contribution of airway infections, which occur more frequently in cold winter months than in summer, have not been listed by Spencer et al. (1) among the possible explanations for the higher incidence of AMIs in winter time. Evidence from smaller hospital-based studies has already previously indicated that an association between acute chest infections and AMI might exist (3–5). The pathophysiologic mechanism of such an association is speculative and could involve changes in circulating clotting factors, an increased risk for acute rupture of arteriosclerotic plaque during a chest infection, or a variety of other mechanisms. The role of acute and chronic infections in the etiology of coronary heart disease and AMI needs to be better understood; it might be one of the keys towards an explanation why more AMIs occur in winter than in summer.

CHRISTOPH R. MEIER, PhD, MSc
Boston Collaborative Drug Surveillance Program
Boston University School of Medicine
11 Muzzey Street
Lexington, Massachusetts 02421

References

Anticoagulation in Dilated Cardiomyopathy

We read with interest the recent review by Koniaris et al. (1) concerning anticoagulation in dilated cardiomyopathy. Evidence from published studies does not convincingly demonstrate the benefits of anticoagulation in patients with dilated cardiomyopathy. As described by Koniaris et al., (1) a prospective, randomized clinical trial of long-term anticoagulation in patients with dilated cardiomyopathy is feasible. However, studies employing hemostatic markers indicating a biochemical imbalance between procoagulant and anticoagulant mechanisms in the blood may be useful to evaluate the appearance of thrombotic phenomena in these patients (2). By measuring plasma levels of hemostatic markers, we previously found that, in patients with dilated cardiomyopathy, plasma levels of fibrinopeptide A and thrombin-antithrombin III complex, markers of coagulation activation and thrombin generation, were significantly higher than those in normal subjects (3). Their levels showed a positive correlation with left ventricular end-diastolic volume and a negative correlation with fractional shortening of the left ventricle. Although Koniaris et al. (1) suggested that aspirin monotherapy may be beneficial for risk reduction of thromboembolism in patients with dilated cardiomyopathy, plasma levels of platelet factor-4 and beta-thromboglobulin, markers of platelet activation, were not elevated in these patients compared with normal subjects. In addition, we previously observed that aspirin monotherapy suppresses platelet function, but does not affect coagulation activity (4). These findings support the premise that anticoagulant therapy, rather than antiplatelet therapy, is more effective for the prevention of systemic embolism in patients with dilated cardiomyopathy, particularly in those with severe left ventricular enlargement and dysfunction.

UICHI IKEDA, MD
KEIJI YAMAMOTO, MD
KAZUYUKI SHIMADA, MD
Department of Cardiology
Jichi Medical School
Minamikawachi-Machi
Tochigi 329-0498, Japan

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