Correspondence

Letters to the Editor must not exceed 400 words in length and may be subject to editing or abridgment. Letters must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Only some letters will be published. Authors of those selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication.

Cardiopulmonary Interactions After Fontan Operations

To the Editor:

Two fundamental observations might be drawn from the article by Shekerdemian et al1 concerning the physiological study of the Fontan circulation:

1. Stroke volume increase, as an adaptation of cardiac output, is difficult to obtain. One of the suggested explanations is that “the total afterload limits the potential for an increase in stroke volume. The total afterload of a Fontan circulation, which is equal to the total vascular resistances, or more precisely, to total impedance, may effectively lead to hemodynamic instability.” By analogy with the study on preload, afterload, and cardiac output relationship,3 stroke volume may be preserved during afterload increase, thus requiring a preload elevation. Conversely, with constant preload, stroke volume decreases when afterload increases. It is a matter of heterometric autoregulation within a constant preload, stroke volume decreases when afterload increases, thus requiring a preload elevation. Conversely, with constant preload, stroke volume decreases when afterload increases.

2. Conversely, a negative pressure ventilation may generate an important stroke volume increase. The authors1 stated that “presumably there must lie a plateau beyond which cardiac output can no longer continue to improve” when negative pressure ventilation is used. The main reported consequence of negative ventilation is a venous return variation.4 Guyton’s venous return curve5 actually admit a “plateau” effect. Reduction of intrathoracic pressure during negative pressure ventilation increases the pressure gradient between intrathoracic venous cavae and peripheral vascular beds, thus optimizing preload over Guyton’s venous return curve, accounting for an important stroke volume increase. There is a threshold beyond which venous return can no longer be increased whatever the negative pressure ventilation intensity because intrathoracic venous cavae pressure would be lower than atmospheric pressure.5

Fontan circulation studies should therefore benefit from the inclusion of Guyton’s concept6,5 on the equilibrium point between ventricular function and venous return curves. As discussed in the hemodynamic comparison between partial or total Fontan circulations,2 reported results emphasize that ventricular function and venous return curves seem to be interdependent variables of the Fontan circulation.

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3. Sagawa K. Analysis of the ventricular pumping capacity as a function of input and output pressure loads. In: Reeve EB, Guyton AC, eds. Physical


Response

We would like to thank Macé and colleagues for their additional comments related to our observations.1 A low-resistance unobstructed pulmonary circuit, sufficient preload, adequate systemic ventricular function, and physiological afterload are the key determinants of pulmonary blood flow and hence cardiac output of Fontan patients. In the acute postoperative period, these factors can be labile or simply suboptimal, and conventional methods of maintaining preload can ultimately result in fluid overload and lead to ventricular dysfunction. Manipulation of stroke volume is ideally aimed at improving pulmonary blood flow without adversely affecting ventricular function or systemic vascular resistance.

We agree that negative pressure ventilation (NPV) could theoretically compromise systemic ventricular function by increasing afterload.2 We did not directly measure intrathoracic pressure, and so in the absence of transmural pressure data, we can only assume that any effect of NPV on afterload was clinically insignificant and was exceeded by its beneficial influence on diastolic pulmonary blood flow. We have previously shown that acute transition to the Fontan circulation is associated with maintained systolic function1 and is characterized by profound changes in diastolic function consequent on the reduction in preload.3

A negative intrapleural pressure accelerates systemic venous return by increasing the pressure gradient between the intrathoracic and extrathoracic veins. We previously reported an improvement in cardiac output of ~11% in nonbypass patients receiving NPV, and in the same study, we showed an increase of 28% in postbypass patients after biventricular surgery.5 We suggested that this improvement was achieved by augmentation of venous return (along the principles of Guyton) and that this effect was more marked in postbypass patients who were likely to be more sensitive to the detrimental effects of intermittent positive pressure ventilation on venous return.

Macé and colleagues have rightly pointed out that the augmentation of venous return is ultimately limited by collapse of intrathoracic veins as the right atrial pressure approaches zero. Although the pulmonary artery pressure and hence, right atrial pressure fell in our patients, at no point in the respiratory cycle did these pressures fall to atmospheric or below. The Fontan circulation in particular is likely to be relatively resistant to this phenomenon because of the high baseline preload that exists in these patients. Indeed, the potential improvement that could be achieved by this adjustment of cardiopulmonary interaction in the Fontan circulation may be even greater than that which we demonstrated in our study.

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Spatial and Temporal Periodicity During Atrial Fibrillation

To the Editor:

The article by Skanes et al1 presented interesting evidence of spatial and temporal periodicity during atrial fibrillation, contrary to the traditional concept of totally disorganized random atrial activity in atrial fibrillation. Skanes et al referred to recent work by others2,3 in accord with their own.

With former colleagues in Chicago, Ill, I called attention 20 years ago to the fact that some patients with ECG features of atrial fibrillation have M-mode echocardiographic findings indicating regular atrial contractions. Regular deflections on the mitral valve echogram were demonstrated in Figures 14 and 15 of that paper,4 although the ECG was typical of atrial fibrillation. At the time, we speculated that one explanation for this apparent discrepancy was one dominant circus-motion wave (producing regular atrial contractions), with smaller subsidiary irregular “eddies” of electrical activity responsible for the irregular contour of ECG atrial activity.4 Over the last 2 decades, we have noted that this phenomenon (regular diastolic mitral deflections on echocardiography concomitant with irregular atrial ECG deflections) is not very rare in clinical M-mode recordings, yet few if any similar observations have been published.

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Subclinical Aneurysm Is Not A False Aneurysm

To the Editor:

I enjoyed reading the beautifully illustrated case report of subclinical aneurysm by Kontozis et al.1 However, I questioned the appropriateness of the term “false aneurysm” used by the authors to describe the submral left ventricular aneurysm.

False aneurysm is a healed myocardial free-wall rupture of a myocardial infarct.2 Because the authors stated clearly that submral left ventricular aneurysms are caused by a congenital defect in the posterior portion of the mitral anulus, they are not true ‘false aneurysms.’

Submral left ventricular aneurysm is a peculiar form of left ventricular aneurysm, occurring almost exclusively in Negroid people and being noncoronary in etiology, that is, situated immediately beneath the posterior leaflet of the mitral valve.3,4 Submral left ventricular aneurysms have a variety of clinical presentations. Distortion of the mitral anulus produces mitral regurgitation.5 The larger submral aneurysm may accommodate a large regurgitant volume during systole that is returned to the left ventricle in diastole, thus placing an additional hemodynamic burden on the left ventricle.6 Rupture of the aneurysm may cause cardiac tamponade or death.7,8 In rare situations, submral left ventricular aneurysm may, via several mechanisms, cause coronary artery obstruction.


Submitral Aneurysm Is Not A False Aneurysm

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latter, in turn, may result in acute myocardial infarct, which may rupture, thus possibly causing a false aneurysm of the left ventricle if the patient survives.

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Response

We appreciate the comments of Dr. Cheng. The origin of submitral aneurysms is unknown. A congenital origin has been postulated, although an inflammatory origin has also been proposed. The congenital origin is supported by the fact that submitral aneurysms are almost exclusively observed in black African patients. Chesler et al. suggested that a defect in the muscular fibrous junction below the intermediate portion of the posterior mitral leaflet leads to a hematoma contained by the epicardium. Organization of this hematoma results in a fibrous walled epicardial aneurysm that may expand under the influence of the high pressure in the left ventricle. Thus, submitral aneurysms are essentially false aneurysms. These aneurysms can also be situated between the aorta and the heart, and in this case, a similar etiology has been postulated that results in the formation of a false aneurysm.

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Handling Complexity in Oxygen Delivery in the Univentricular Circulation

To the Editor:

Barnea et al present an important analysis of pulmonary-to-systemic flow balance (Qp:Qs) and total systemic oxygen delivery (DO2) in patients with complete mixing of blood. We feel that they are too modest about the analytical utility of their equations. A central theme of their work is the relationship between DO2 and its determinants: metabolic rate (VO2), pulmonary venous oxygen content (CpO2), pulmonary blood flow (Qp), systemic blood flow (Qs), and cardiac output (CO)=Qp+Qs. They present the following equation relating these parameters, which appeared too complex to proceed to algebraic maximization of DO2 in the general case, and hence the authors were obliged to perform computer modeling for a few individual cases:

\[
DO2 = \frac{1}{1 + \frac{Qp}{Qs}} \times CO \times CpO2 - \frac{1}{Qp/Qs} \times VO2
\]

However, we would like to add the observation that this relationship may be expressed more simply, with each relevant variable appearing only once:

\[
DO2 = Qs \times \left( \frac{CpO2 - VO2}{Qp} \right)
\]

From this, one can readily deduce that for maximization of DO2, the chosen value of pulmonary blood flow should be: Qp for max DO2=\(\sqrt{(CO \times VO2)/CpO2}\), and the fraction of blood sent to the lung should be: \(Qp/CO = \sqrt{VOCO/(CO \times CpO2)}\).

Applying values of VO2 (0.009 L \cdot min^{-1} \cdot kg^{-1}), CO (0.30 L \cdot min^{-1} \cdot kg^{-1}), and CpO2 (0.184 L O2/L blood), we obtain a target Qp of 0.12 L \cdot min^{-1} \cdot kg^{-1}, or a Qp/CO of 40%.

The clinical significance of this for a clinician aiming to maximize DO2 is that if cardiac output rises in proportion to metabolism (VO2/CO ratio conserved) in the recovery period after surgery, then the target Qp/CO balance remains constant, whereas if the rise in cardiac output fails to match that in metabolism, then one would have to divert progressively larger fractions of that cardiac output to the lungs.

The final comment we have is that it is far from clear that total oxygen delivery is the most important quantity to maximize, because it disregards the fact that oxygen of different saturations (and hence partial pressures) is of different metabolic utility in protecting tissue from hypoxic damage. Nevertheless, we support this analytical approach to developing a theoretical basis for our clinical choices in these difficult and unstable patients. We contend that these relationships are not as complex and unfathomable as they may seem at first, and further analyses may give yet more useful additions to the clinician’s armory of rules of thumb.

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Response

We thank Drs. Francis, Davies, and Coats for their thoughtful comments. The perioperative management of children with hypoplastic left heart syndrome (HLHS) presents a challenge. Given the small patient size and precarious hemodynamics, it is
difficult to make measurements. For this reason, we developed a simple model to predict systemic oxygen delivery. In our first article, we used this model to demonstrate that maximal oxygen delivery occurs at a pulmonary-to-systemic flow ratio (Qp/Qs) ≤1.1 In our second study, we examined the efficacy of arterial and mixed venous oxygen saturations and indexes derived from these blood gases to monitor system oxygen delivery.2

We are delighted that our article stimulated you and your colleagues to reformulate the basic equations. Obviously, there are many different ways to express these relationships. You expressed the pulmonary artery blood flow at maximal systemic oxygen delivery as a function of cardiac output, whole-body oxygen consumption, and pulmonary venous oxygen capacity. We plotted this relationship: pulmonary artery blood flow versus cardiac output. Similar to our first study, for maximal systemic oxygen delivery, the Qp/Qs ratio was <1. As cardiac output increases, pulmonary blood flow increases, but not in a linear fashion. Thus, Qp/Qs continues to decrease. There is no one value of Qp/Qs for maximal oxygen delivery.

Your assumption that cardiac output and whole-body oxygen consumption would increase proportionally is too restrictive. Clearly, changes in oxygen extraction occur in these patients. Also, most HLHS patients suffer from depressed ventricular function and a high Qp/Qs after surgery. Thus, in most patients, the problem is too much, and not too little, pulmonary blood flow.

Again, we are glad of your interest. For our last article,2 we solved the basic equations and plotted the results in Excel. This spreadsheet (in Excel for PC) is available free of charge. Just send an e-mail message to Dr Barnea (barneao@eng.tau.ac.il), and he will send you a copy. We hope that you, other clinicians, and investigators will try this demonstration. We are all looking for the best way to monitor these patients and save lives.1–4

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