of said complications is increasing may be true, but this again had nothing to do with the integrity of previous estimates made in a good faith effort to study results. I hope this clarifies any question in the readers' minds as to the intention of my statements regarding this issue.

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MICHAEL E. DEBAKEY

The obituary of Dr DeBakey was very interesting.[1] One of Dr DeBakey's pioneering efforts does need special emphasis. By using autologous saphenous vein(s), on November 23, 1964,[2] DeBakey performed the first human coronary artery bypass graft surgery to the left anterior descending coronary artery in an asymptomatic patient after myocardial infarction.

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PULMONARY ARTERIAL HYPERTENSION AND CONGENITAL HEART DISEASE: TARGETED THERAPIES AND OPERABILITY

To the Editor:

We read with great interest the case report of Hoetzenecker and colleagues[1] of a patient with severe pulmonary arterial hypertension associated with an atrial septal defect and the beneficial effect of bosentan, which allowed for closure of the defect. This is a very interesting topic that raises a lot of controversies in the field of pulmonary arterial hypertension associated with congenital heart disease.[2]

I would like to comment on the hemodynamic data presented in Table 1. Total pulmonary resistance at baseline is calculated at 460 dynes · s^{-1} · cm^{-5}, but pulmonary vascular resistance, taking the mean left atrial pressure in the calculation, which is commonly done in congenital heart disease, would give a value of 311 dynes · s^{-1} · cm^{-5} or 3.9 Wood units, which is very close to a value of 3 Wood units considered as totally safe for surgical repair. If we calculate the ratio of pulmonary to systemic blood flow (Qp/Qs), we have a value of 2.7, which is also considered a value that allows for repair.

Moreover, when we look at the data during the nitric oxide testing, these values reach a pulmonary vascular resistance of 1.78 Wood units or 142 dynes · s^{-1} · cm^{-5}, the Qp/Qs is 3.25, and the pulmonary arterial saturation reaches 84.5%. All these values are consistent with a reactive pulmonary vascular bed and are values that will clearly allow closure of the shunt for a congenital cardiologist.[3,4] It would be of interest also to report the ratio of pulmonary over systemic vascular resistance, as a ratio < 0.33 indicates good prognosis after closure of the shunt.[4]

Maybe the authors used the definition of reactivity used for other forms of pulmonary arterial hypertension, allowing for the use of calcium channel blockers, but this may not be applied for the evaluation of operability in patients with congenital heart disease. Why did the authors think that this particular patient required bosentan treatment based on these measurements? If we look at the data after treatment and just before repair, the hemodynamic does not look much better than with nitric oxide, as the pulmonary vascular resistance is 2.6 Wood units or 205 dynes · s^{-1} · cm^{-5} and Qp/Qs is 2.24. Why was the patient considered operable with these values but not before? If this is based only on mPAP, I would suggest the authors should discuss the fact that mLAP also had a significant decrease.

Finally, if we analyze the measurement 8 months after repair, the pulmonary vascular resistance is 4.2 and the mean pulmonary arterial pressure is 35 mm Hg—still not normal. We clearly hope that these values will remain at these levels and not rise again in the coming month, as the outcome, at least in pediatric patients with recurrent pulmonary hypertension after repair, is dismal, as shown recently by Haworth and colleagues.[5]

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Reply to the Editor:

We thank Dr Beghetti and colleagues for their thoughtful comments regarding the hemodynamic evaluation and further management of our recent case of atrial septal defect (ASD).[1] The management of patients with a degree of pulmonary vascular disease prior to shunt closure has been a matter of debate. In addition, recent studies demonstrating the efficacy of oral vasodilators in pulmonary vascular disease associated with congenital systemic-to-pulmonary shunts[2,3] have fueled an uncertainty of vasodilator pretreatments prior to shunt closure.

Despite optimized medical treatment with diuretics, antibiotics, and oral anticoagulant over 4 months since the first medical contact, our patient was severely dyspneic, with elevated atrial pressures, a pro–brain natriuretic peptide serum level of above 4000 pg/mL, and a dramatically limited 6-minute walking distance (6-MWD) of 150 m. In fact, based on the hemodynamic assessment alone, the patient was admitted to the surgical ward for ASD closure. However, surgeons refused the operation based on the patient's overall clinical profile and frailty. A 10-month treatment with bosentan on top of supportive treatment with diuretics and anticoagulation effectively decreased shunt flow and lowered pulmonary vascular resistance by 140 dynes $\cdot$ s^{-1} $\cdot$ cm^{-5} and markedly decreased atrial pressures, biomarkers, and 6-MWD in the presence of a mild arterial desaturation.

We do agree with the discussants that taking into account left atrial pressures, pulmonary arteriolar resistance was about 3 Woods. In addition, the pulmonary-to-systemic resistance ratio under oxygen and nitric oxide was <0.33 (in the patient, this ratio was 0.11), a threshold pediatric cardiologists have labeled as a criterion conveying a good prognosis after closure of the shunt.[4] Still, data in adult patients with congenital heart disease are lacking, and the criteria of a complete hemodynamic responder status in adults were not fulfilled in this case.[5] Because hemodynamic testing is a routine procedure in adult pulmonary vascular centers, we do rely on these data in the absence of firm evidence indicating their uselessness in adults with congenital heart disease. Moreover, children are usually examined under general sedation/anesthesia. For these and other reasons, it is evident that the hemodynamic response pattern in children is different from that in adults[6] and that hemodynamic criteria in children may not apply to elderly adults. Furthermore, later assessments after surgery in the patient under discussion illustrated a degree of persistent pulmonary vascular disease with a pulmonary arteriolar resistance of 530 dynes $\cdot$ s^{-1} $\cdot$ cm^{-5}, despite active treatment with bosentan.[1]

The main value of this report is to provoke discussion, because due to its single case nature, surgery in the absence of bosentan cannot be repeated.

We submit that our invasive procedure was based on an integrative clinical and hemodynamic approach and guided by numbers, rather than the reverse. Controlled data to guide a ''targeted treatment-and-repair'' strategy in adult patients with congenital heart disease are needed.

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QUALITY OF LIFE IN PATIENTS WITH PROSTHESIS–PATIENT MISMATCH

To the Editor:

I read with interest the article by Moon and colleagues[1] and thank the authors for their contribution to the continued debate on the issue of prosthesis–patient mismatch (PPM).

PPM is an important topic in current cardiac surgery, and there are several discrepancies and contrasting publications about the effect of PPM on postoperative outcome.[2,3] Some of these controversies are discussed in the accompanying counterpoint article by Dr Feindel.[4] In their work Moon and colleagues[1] provide important insight into the management of aortic valve replacement (AVR) with bioprostheses in patients older than 70