Reply to the Letter to the Editor

Reply to Aazami

Ruggero De Paulis*, Fabrizio Tomai, Luigi Chiariello
Cattedra di Cardiochirurgia, Università di Roma, Tor Vergata,
European Hospital, Via Portuense 700, 00149 Rome, Italy

Received 23 July 2004; accepted 27 July 2004; Available online 1 September 2004

Keywords: Coronary flow; Sinuses of Valsalva; Aortic root

We appreciate the interest of Dr Aazami on our work on coronary blood flow in patients with or without sinuses of Valsalva [1]. The new vascular prosthesis used in our patients was without doubt designed and promoted for its advantages in facilitating the surgical procedure and in reducing the tension on the coronary ostia anastomoses as clearly described in our previous articles [2,3] and its use has never been indicated for its possible prompting effects on coronary blood flow. Similarly, other possible implications, like the better expected longevity of stented bio-prosthesis as suggested by Dr Aazami or the better wash out at the base of the valve prosthesis, have been always considered as theoretical speculations [3]. With a similar approach we tried to speculate on the possible benefits, if present, of the sinuses of Valsalva on coronary blood flow. As a matter of fact, the use of this new Dacron graft offered us the possibility of a good experimental model to investigate if the sinuses of Valsalva might play a role in regulating the coronary blood flow or if their function is merely to regulate the opening and closing of the aortic valve. Regarding to the specific questions we would like to point out:

1. The Doppler-wire was positioned into the LAD until an optimal and stable signal was obtained. A good signal is usually recorded within the first 3–4 cm from the ostium and is referred as middle portion of the LAD. One year follow-up is usually considered a significant time for satisfactory myocardial recovery.

2. With respect to the pathophysiological settings, all patients were a mix of aortic valve stenosis and insufficiency and at the time of the experimental evaluation they were comparable for ventricular volumes, wall thickness and contractility as shown in Table 2.

3. We agree that the augmentation of the systolic fraction under maximal vasodilatation do not support, per se, a direct role of the sinuses of Valsalva on CBF, as clearly pointed out in our discussion.

4. For the sake of clarity through the manuscript we always referred to the baseline diastolic systolic integral ratio (DSIR) and its variation after adenosine infusion. However, as an example in group B patients the diastolic peak velocity integral (DPVi) and the systolic peak velocity integral (SPVi) at baseline were 14.1 ± 3.1 and 2.8 ± 0.9 cm/s, respectively, and after adenosine infusion they increased to 42.5 ± 12.3 and 10.8 ± 4 cm/s, respectively, while in group C patients they were 13.2 ± 2.8 and 3.8 ± 1.2 cm/s, respectively, and increased to 48 ± 17.8 and 16.6 ± 8.5 cm/s, respectively.

In conclusion, we want to stress our idea of using patients with different anatomical reconstructions of the aortic root simply as an experimental model to investigate the potential role of the sinuses on coronary blood flow.

References


*Corresponding author.
E-mail address: depauli@tin.it (R. De Paulis).

Letter to the Editor

Leaflet arrest in St Jude and CarboMedics valves: an experimental study

Robert L. Fortune*,1
21, 5187 Cordova Bay Road, Victoria, BC, Canada V8Y2K7

Received 17 June 2004; accepted 27 July 2004; Available online 11 September 2004

Keywords: Cardiac; Valvular; Mechanical

I read with interest “Leaflet arrest in St Jude Medical (SJM) and CarboMedics valves: an experimental study” by Grattan and associates [1] which appeared in the EJCTS June, 2004. In this paper, the authors presented two cases in which SJM Masters series aortic valves were found to be in a ‘frozen’ position following implant.

The authors then developed an experimental model consisting of a pushrod which provided variable point pressure against the ring of the prosthesis directed at 180 degrees. Given our understanding of aortic root anatomy in both health and disease, I feel that the model in no way duplicates the forces on a prosthetic valve following aortic valve replacement.
The normal aortic root is usually 45% muscle and 55% fibrous tissue. Thus less than half of the annular area is contractile. The aortic root itself has been shown experimentally to expand during the isovolumic contraction phase, prior to ejection [2]. Also, Van Fenterghem has shown that during ejection, the basal segments adjoining the myocardium (NC-right and right-left basal lengths) shortened, whereas the aortomitral junction (left-NC basal length) lengthened [3]. Given the nature of a normal annulus then, only one pivot mechanism would be subjected to any contractile force, the other would be beside the fibrous components of the annulus and mitral valve, which expands.

Patients with long-standing aortic stenosis develop fibrosis and calcification which would negatively alter the contractility at the annular level. The authors then speculate that left ventricles generating pressures exceeding 200 mmHg could result in dynamic leaflet arrest. This surely would be of greater clinical significance, as compared to their cases which occurred in cardioplegically arrested hearts, yet there is no significant reporting of this in the literature. They report that Jaggers showed one leaflet of a SJM standard valve leaflet to arrest following mitral valve replacement. If their hypothesis is correct, how do they explain the arrest of only one leaflet of the prosthesis in this situation? The authors describe a further experiment in which prosthetic valves are placed in the mitral position of a porcine midel. The normal mitral annulus may be capable of ‘two-point’ force loading. Once again, the contractile forces of a normal porcine mitral annulus bear no relationship to the contractile forces in a normal or diseased aortic annulus.

It is impossible to reconstruct the exact intra-operative environment that resulted in the immobile leaflets as reported. However, if the proper sizing technique is utilized in which the sizer passes freely through the annulus, it would not be possible for the valve to then malfunction due to ‘compressive forces’ because that is what proper sizing is supposed to achieve. It is well recognized, however, that tissue adjacent to the valve can result in valve malfunction. These can sometimes be difficult to visualize. In such occasions, the valve can be rotated as long as a proper debridement of the annulus has been undertaken beforehand. There, one can only assume that there were technical issues involved with the initial implants that resulted in the ‘frozen leaflets.’

References


Reply to Fortune

Mark T. Grattan,*, Lars I. Thulin

*Department of Surgery, Straub Clinic and Hospital, Honolulu, HI, USA

bDepartment of Cardiothoracic Surgery, University of Lund, Lund, Sweden

Received 16 July 2004; accepted 27 July 2004; Available online 11 September 2004

Keywords: Mechanical valve; Valve replacement; Valve malfunction

The studies cited in the Letter to the Editor are indeed important contributions. However, we differ with some of the inferences Dr Fortune makes based on them. The 1988 data in the study by van Renterghem et al. were incomplete due to frequent dislodgment of the coils used to measure strain, greatly reducing the number of valid data. Their measurements of the basal segments indicated that two of three basal segments shortened during ejection. However, more recent studies of Lansac et al. used more sensitive sonometric crystals, and clearly demonstrated an expansion of all three basal segments, such that the aortic base area increased by 29.8% during systole, while the mitral annulus area contracted by −16.1% during systole due to ‘posterior displacement of the intertrigonal area corresponding to the systolic aortic root expansion’ [1]. The Lansac studies show how the aortomitral apparatus functions as an anatomic unit, and for Dr Fortune to infer from the older van Renterghem study that only one pivot of an aortic valve prosthesis (which is anchored by sutures in the aortic annulus) will be subject to that aortomitral unit’s forces is, we believe, unsupported by the current evidence. Normal myocardial forces occurring at the annulus level will be transfigured when a valve prosthesis is inserted, converting aortic annular anatomy from its normal semilunar contour to a more planar, rigid one.

The report by Jaggers cited in our study showed one leaflet of a SJM valve arrested following mitral valve replacement. In Table 1, we reported the consistent finding that, with increasing loads, first one leaflet bound up, followed by the second leaflet at greater loads. Thus, we are not surprised by the Jaggers report, and in fact, would expect that one leaflet would be affected more frequently than both