

Impact of prosthesis-patient mismatch in the mitral position on left atrial and pulmonary arterial pressures: a numerical study

David Tanné, Lyes Kadem, Régis Rieu, Jean Gilles Dumesnil, Philippe

Pibarot

▶ To cite this version:

David Tanné, Lyes Kadem, Régis Rieu, Jean Gilles Dumesnil, Philippe Pibarot. Impact of prosthesis-patient mismatch in the mitral position on left atrial and pulmonary arterial pressures: a numerical study. 2005, pp.265-266, 10.1080/10255840512331389271. hal-00092134

HAL Id: hal-00092134 https://hal.science/hal-00092134

Submitted on 8 Sep 2006

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers. L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés. Impact of prosthesis-patient mismatch in the mitral position on left atrial and pulmonary arterial pressures : a numerical study

Tanné $D^{1,3}$, Kadem L^2 , Rieu R^1 , Dumesnil JG^3 , Pibarot P^3

- ¹ IRPHE, Université de la Méditerranée, Equipe Biomécanique Cardiovasculaire, Marseille, France.
- ² IRCM, Laboratoire de Génie Biomédical, Montréal, Canada.
- ³ Quebec Heart Institute, Laval Hospital, Laval University, Quebec, Canada.

Introduction

Prosthesis-patient mismatch (PPM) occurs when the effective orifice area (EOA) of the prosthesis is too small in relation to the body size and thus to the cardiac output requirement of the patient. A recent retrospective study from our group suggests that mitral PPM defined as an indexed EOA $\leq 1.2 \text{ cm}^2/\text{m}^2$ is associated with lesser regression of pulmonary hypertension after mitral valve replacement.

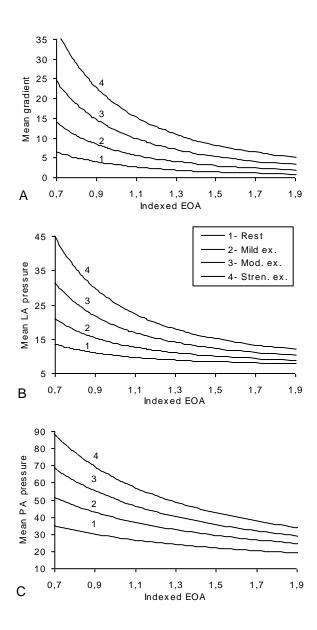
Materials and methods

We used a mathematical model of closed-loop blood circulation previously developed and validated by Thomas and al. (Thomas, 1997). The equation governing blood flow through all cardiac valves was a simplified Bernouilli equation with both resistive and inertial terms. Normalized elastance functions of ventricles and atria were used to generate realistic pressure-volume curves. The mitral valve EOA indexed for body surface area was varied between 0.7 (severe PPM) and 1.9 (no PPM) cm²/m², assuming a normal cardiac index of 3.3 L/min/m² at rest, normal LV diastolic pressures, and a normal pulmonary vascular resistance of 250 dyne.s/cm⁵. Heart rate (HR) was varied between 70 and 130 b/min and stroke volume (SV) was varied between 70 and 120 mL to simulate different flow levels as generally observed in humans at rest and during mild, moderate, and strenuous exercise.

Results and discussion

The figure shows the results for mean gradient (Panel A), mean left atrial (LA) pressure (Panel B), and mean pulmonary arterial (PA) pressure (Panel C) at rest (HR=70 b/min, SV=70 ml), mild (HR=90 b/min, SV=95 ml), moderate (HR=110 b/min, SV=110 ml), and strenuous (HR=130 b/min, SV=120 ml) exercise. An indexed EOA < $1.1-1.2 \text{ cm}^2/\text{m}^2$ is generally associated with abnormally high transmitral gradient (>5 mmHg), mean LA pressure (>12 mmHg), and mean PA pressure (>30 mmHg) at rest and/or mild exercise. At moderate and intense levels of exercise, the threshold value below

which LA and PA pressures become abnormally high is rather close to $1.5-1.6 \text{ cm}^2/\text{m}^2$.



Conclusion

This numerical study corroborates the results of our previous in vivo study suggesting that the threshold value of indexed EOA that should be used to define PPM in the mitral position at rest is $< 1.2 \text{ cm}^2/\text{m}^2$. This study also suggests that in athlete patients, the objective should rather be to provide an indexed EOA of at least $1.5 \text{ cm}^2/\text{m}^2$.

References

Thomas JD (1997): Physical and physiological determinants of pulmonary venous flow: numerical analysis. *Am. J. Physiol.* 272: H2453-2465.