IMPACT OF A CONCOMITANT SUBAORTIC STENOSIS ON THE ASSESSMENT OF THE SEVERITY OF AN AORTIC VALVE STENOSIS: AN IN VITRO STUDY

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INTRODUCTION

Aortic valve stenosis (AS) is an important cardiovascular disease affecting between 2 and 7% of the elderly population in industrialized countries. AS often coexists with subaortic stenosis (SAS) [1], which is generally caused by a protrusion of the hypertrophied left ventricular outflow tract (LVOT) just below the aortic valve. The SAS may potentially interfere with the assessment of AS severity and therefore raise some uncertainties about the therapeutic management of the patient. Moreover, only a very few studies have been held on this topic to our knowledge [2]. The aim of this study was to determine the impact of SAS on the assessment of AS severity.

METHODS

For the purpose of this study, we used our validated ventriculo-aortic model [3]. The main modifications to the existing model were: 1) the addition of a homemade SAS model allowing varying the SAS severity; 2) the addition of a rigid circular orifice modelizing an AS (Figure 1).



Figure 1: Schema of the in vitro flow model.

The asymmetric SAS model was made up of a series of screws inserted in the LVOT wall section and pushing on a piece of elastic tube. This system allowed us to vary the SAS severity from 0 to a maximum of about 90% (that means that the LVOT cross-sectional area could be reduced by a maximum of 90%). In this study, four rigid circular orifices with geometric orifice areas (GOA) of 0.61 (orifice 1), 1 (orifice 2), 1.22 (orifice 3) and 1.83 (orifice 4) cm² were analyzed. For each orifice, four different severities of SAS were tested: 25% (mimicking a mild stenosis), 50% (mimicking a moderate stenosis), 75% (mimicking a severe stenosis) and 90% (mimicking a very severe stenosis).

The ventriculo-aortic flow model was adjusted to obtain typical normal hemodynamic conditions (stroke volume = 70 mL, systolic blood pressure = 120 mmHg and diastolic blood pressure = 80 mmHg) at 70 bpm. The valve effective orifice area (EOA) was measured by Doppler echocardiography using the "continuity equation" method that is used routinely in the clinical setting. The EOA represents the minimal cross-sectional area at the vena contracta of the transvalvular flow jet and determines the hemodynamic load imposed on the left ventricle.

RESULTS AND DISCUSSION

For a SAS severity less than 50%, the measured EOA did not change whatever the size of the orifice (i.e. whatever the AS severity) (Figure 2). For a SAS severity of 75%, the measured EOA decreased in the two less severe AS (orifices 3 and 4), whereas it remained unchanged in most severe AS (orifices 1 and 2). The AS severity was overestimated by about 20% for the orifice 3 and about 35% for the orifice 4. For a SAS severity of 90%, the measured EOA was underestimated by about 33%, 49% and 63% in the orifices 2, 3 and 4, respectively. For the most severe AS (orifice 1), the SAS severity had no impact on the measured EOA. On the contrary, for the less severe AS (orifice 4), beyond a SAS severity of 50%, the AS severity was progressively overestimated and reached a reduction of about 63% of EOA for a SAS severity of 90%. This corresponds to the EOA of the orifice 1, modelizing a very severe AS!



Figure 2: Relation between the subaortic stenosis (SAS) severity and the aortic valve effective orifice area (EOA) for the four orifices.

CONCLUSIONS

We have shown that the presence of a concomitant SAS may cause an overestimation of the hemodynamic severity of AS. The degree of overestimation is more important in less severe AS. Hence, the presence of SAS may lead the clinician to erroneously conclude that the AS is severe and that aortic valve replacement is indicated.

REFERENCES

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