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In vitro correlation between the effective and geometric orifice area in aortic stenosis

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ABSTRACT

Background: Planimetry of aortic stenosis can be performed when Doppler measurements are unavailable. We sought to evaluate if, as advised in guidelines, the geometric orifice area (GOA) threshold value of 1 cm^2 was concordant with the threshold of 1 cm^2 of the effective orifice area (EOA), and the factors influencing the contraction coefficient (EOA/GOA ratio).
Methods: In an *in vitro* mock circulatory system, we tested 6 degrees of AS severity (3 severe and 3 non-severe), and 3 levels of flow (<150 ml/s, 150−200 ml/s, >250 ml/s). The EOA was calculated by Doppler-echocardiography, and the GOA was measured with dedicated software after camera acquisition.
Results: In all but the very low flow condition, an EOA of 1 cm^2 corresponded to a GOA of 1.2 cm^2. The contraction coefficient increased with both the flow and the stenosis severity. For very severe stenoses, the EOA and the GOA were interchangeable.
Conclusion: As observed in clinical studies, the GOA was larger than the EOA, and a GOA between 1 and 1.2 cm^2 should not discard the possibility of severe aortic stenosis.

Introduction

According to the European and US guidelines, severe aortic stenosis is defined by an effective orifice area (EOA) <1 cm^2, a peak velocity ≥4 m/s, or a mean gradient ≥40 mmHg [1,2]. The EOA represents the smallest cross-section of flow across the aortic stenosis and can be evaluated with the continuity equation. Moreover, in joint guidelines on the assessment of aortic stenosis, it is pointed out that planimetry could also be used, especially when the left ventricular outflow tract cannot be accurately measured or when Doppler data are unavailable [3]. The measure of the geometric orifice area (GOA) can be performed with transesophageal echocardiography, or more recently and accurately with imaging techniques such as computed tomography scan or cardiac magnetic resonance imaging [4–7]. However, because of the flow contraction phenomenon, the EOA is expected to be significantly smaller than the GOA [8]. Therefore, the contraction coefficient (CC), that is the EOA/GOA ratio, is expected to be <1. The use of the same cut-off values for both EOA and GOA as recommended in the guidelines may be inaccurate.

The aim of the present study was to evaluate *in vitro* the relationship between the EOA and the GOA in different stenosis settings, and the factors influencing the CC.

Methods

Experimental model

The *in vitro* circulatory system was previously described [9,10]. Briefly, the simulation system included anatomically shaped silicone-made left heart cavities and aorta, and simulation of the pulmonary and systemic circulations. Contraction of the left ventricle was achieved by a piston pump (Vivitro Inc., Victoria, Canada). Both pump activation and signal acquisition were controlled with LabVIEW8.2 (National Instruments, Austin, TX, USA). The diameter of the aorta was 39 mm at the sinuses level, and 27 mm for the tubular part.

The circulatory fluid was a saline mixture of water (53%) and glycerol (47%) mimicking blood viscosity (4 ± 0.2 cP) and maintained at 37 °C.

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Doppler echocardiographic measurements

Doppler echocardiographic measurements were performed using a General Electric Vivid 7 (GE Health Medical, Horten, Norway), with a 3.5 MHz probe. The transvalvular flow velocities, mean gradient, and aortic velocity-time integral were measured five times per condition by continuous-wave Doppler. The EOA was determined by the continuity equation, by dividing the stroke volume measured with the electromagnetic flowmeter by the echocardiographic aortic velocity-time integral [11]. Mean flow rate (Q, ml/s) was calculated by dividing this very stroke volume by the ventricle ejection time, measured on the Doppler acquisitions.

Reproduction of the aortic stenosis

To reproduce an aortic stenosis, three clamp rings were inserted through the stent frame of an Edwards SAPIEN transcatheter valve 23 mm (Edwards Lifesciences, Irvine, CA, USA). Depending on the length of the clamp rings, different degrees of valve restriction could be achieved. An example of a restricted valve before insertion in the simulator is shown in Fig. 1A.

A high-speed camera (SA3 Fastcam, Photron, Tokyo, Japan) was used to acquire 1000 images per second en-face view of the valve videos during the cardiac cycle. Determination of the GOA was performed using a program based on Matlab (Mathworks, Natick, MA, USA), after image calibration with a calibration pattern. The GOA was calculated at the maximal valve-opening frame. Fig. 1 shows the image obtained in a normal valve (Fig. 1B), a moderately stenotic valve (Fig. 1C), and an extremely stenotic valve (Fig. 1D). A video of a restricted valve obtained with the high-speed camera during a cardiac cycle is shown in the Supplemental material.

First, six degrees of stenosis based on the GOA were predefined (no stenosis, moderate stenosis, moderate-to-severe stenosis, severe stenosis, very severe stenosis, extreme stenosis). After setting up the clamp rings in the valve, the GOA was measured with the camera in normal flow rate conditions (mean flow rate >200 ml/s). Therefore, we considered it represented the true degree of stenosis of the valve, independently of any further change in stroke volume, heart rate, or mean aortic pressure. If the predefined GOA was obtained, all the other echo measurements (including the EOA) were then performed in various flow and pressure conditions. Otherwise, the valve was taken out of the simulator and the clamp rings were readjusted.

Three non-severe and three severe degrees of stenosis were obtained. The three non-severe stages included: no stenosis (control condition, maximal GOA 2.4 cm^2, maximal EOA 2.2 cm^2 in normal flow conditions), moderate stenosis (GOA 1.3 cm^2, EOA 1.1 cm^2), moderate-to-severe stenosis (GOA 1.2 cm^2, EOA 1.1 cm^2). The three severe stages included: severe stenosis (GOA 1.0 cm^2, EOA 0.9 cm^2), very severe stenosis (GOA 0.8 cm^2, EOA 0.8 cm^2), and extreme stenosis (GOA 0.5 cm^2, EOA 0.5 cm^2). For each degree of stenosis, three different values were applied for heart rate (HR: 60, 80, 100 bpm), stroke volume (SV: 30, 50, 70 ml), and mean aortic pressure (100, 120, 140 mmHg). We also defined three groups based on the mean flow rate: normal flow (Q > 200 ml/s), low flow (Q 150−200 ml/s), very low flow (Q < 150 ml/s) [12,13].

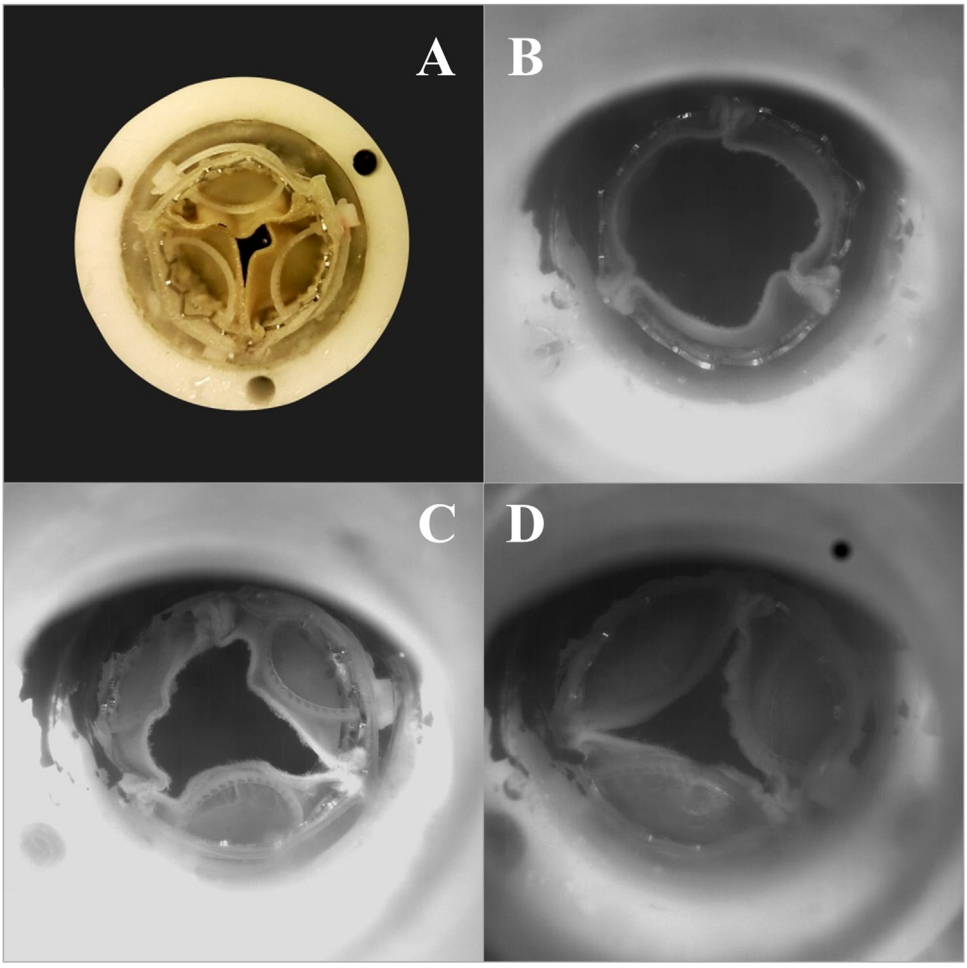

Fig. 1. (A) Example of a restricted valve before insertion in the simulator. (B) Image obtained with the high-speed camera in a normal valve; (C) in a moderately stenotic valve; (D) in an extremely stenotic valve.

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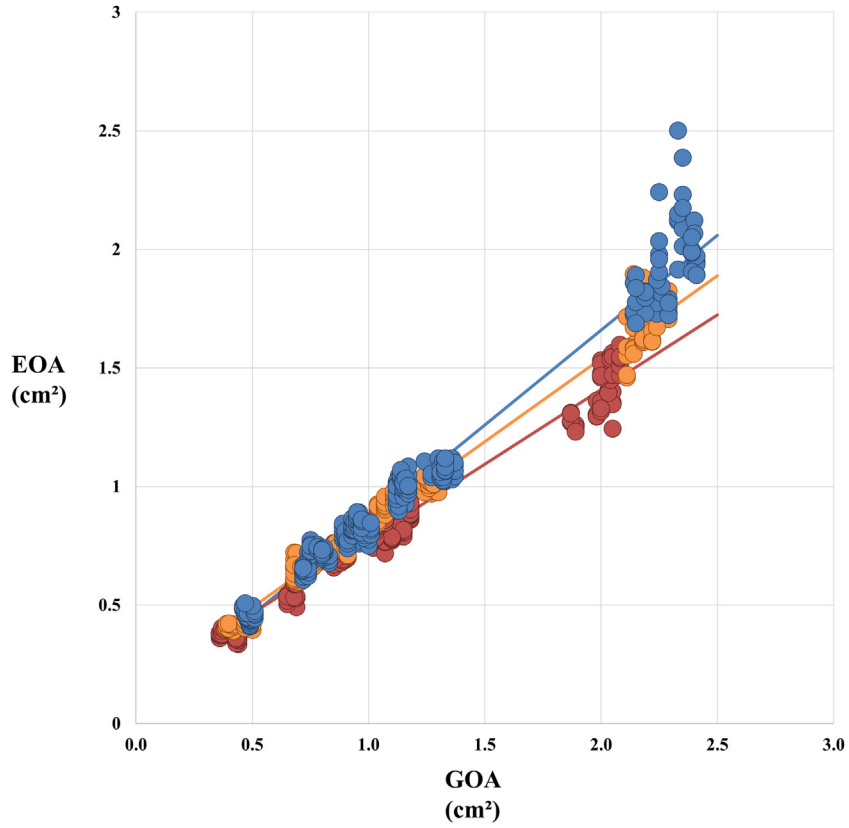

Fig. 2. Correlation between the geometric orifice area (GOA) and the effective orifice area (EOA) for normal flow (blue marks), low flow (orange marks), very low flow (red marks).

Statistical analysis

A *t*-test was performed to compare mean values of the CC between two contiguous degrees of stenosis. A paired *t*-test was performed to compare the differences between EOA and GOA, in each degree of stenosis.

Linear regression was performed to evaluation the correlation between the EOA and the GOA globally and then in each flow condition. It was also performed for each degree of stenosis to evaluate the correlation between the CC and the mean aortic flow or the stenosis severity. Multiple linear regression was performed to predict the effect of both EOA and flow on the CC.

All statistical analyses were performed with SPSS Statistics version 24 (IBM corp., Armonk, NY, USA). A value of $p < 0.05$ was considered significant.

Results

EOA and GOA correlation

There was a linear correlation between EOA and GOA (Fig. 2, r^2 0.95, $p < 0.0001$). Considering all flows, an EOA of 1 cm^2 corresponded to a GOA of 1.2 cm^2. This correlation was also found in each flow group (normal flow: r^2 0.97, low flow: r^2 0.98, very low flow: r^2 0.98, all $p < 0.0001$). An EOA of 1 cm^2 also corresponded to a GOA of 1.2 cm^2 in the normal and low flow groups, and 1.3 cm^2 in the very low flow group.

Contraction coefficient depending on aortic stenosis severity

The mean values of EOA and GOA are summarized in Table 1. There was a significant difference between EOA and GOA in all conditions ($p < 0.0001$ for all, Fig. 3).

Table 1
Comparison between effective orifice area (EOA) and geometric orifice area (GOA) for different stenosis degrees, including all flow rates.

	EOA (cm^2) Mean ± SD	GOA (cm^2) Mean ± SD	p
No stenosis	1.7 ± 0.3	2.2 ± 0.2	<0.0001
Moderate	1 ± 0.1	1.3 ± 0.1	<0.0001
Moderate-to-severe	0.9 ± 0.1	1.1 ± 0.1	<0.0001
Severe	0.8 ± 0.1	0.9 ± 0.1	<0.0001
Very severe	0.6 ± 0.1	0.7 ± 0.1	<0.0001
Extreme	0.4 ± 0.04	0.5 ± 0.1	<0.0001

The CC was highest for the extreme stenosis, and significantly decreased from the extreme condition to the very severe condition (0.96 ± 0.01 *vs* 0.90 ± 0.01, $p < 0.0001$), and from the very severe to the severe condition (0.90 ± 0.01 *vs* 0.85 ± 0.004, $p < 0.0001$). There was also a significant decrease from the moderate-to-severe condition to the moderate condition (0.84 ± 0.004 *vs* 0.78 ± 0.003, $p < 0.0001$), but not between the severe to the moderate-to severe conditions (0.85 ± 0.004 *vs* 0.84 ± 0.004, $p = 0.07$), nor from the moderate to the control conditions (0.78 ± 0.003 *vs* 0.77 ± 0.0007, $p = 0.14$).

Contraction coefficient depending on aortic stenosis severity and mean aortic flow

There was a significant correlation between the CC and the mean flow rate (Fig. 4, $p < 0.0001$) in all but the extreme stenosis conditions. However, the coefficient of determination r^2 decreased as the severity of the stenosis increased, from 0.75 in the control condition, to 0.11 in the very severe condition.

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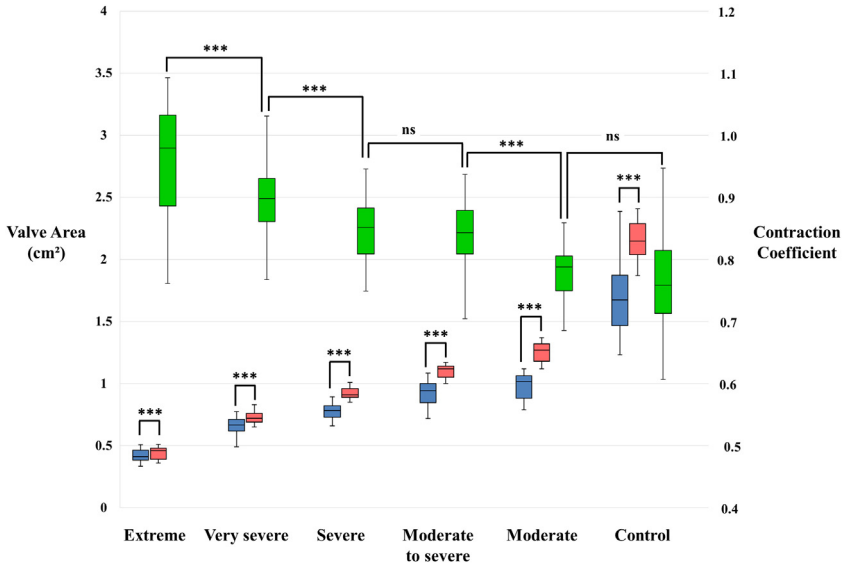

Fig. 3. Comparison between the effective orifice area (blue) and the geometric orifice area (red) for each degree of stenosis, including all flow rates. Green: contraction coefficient comparison between each degree of stenosis, including all flow rates. *** $p < 0.0001$.

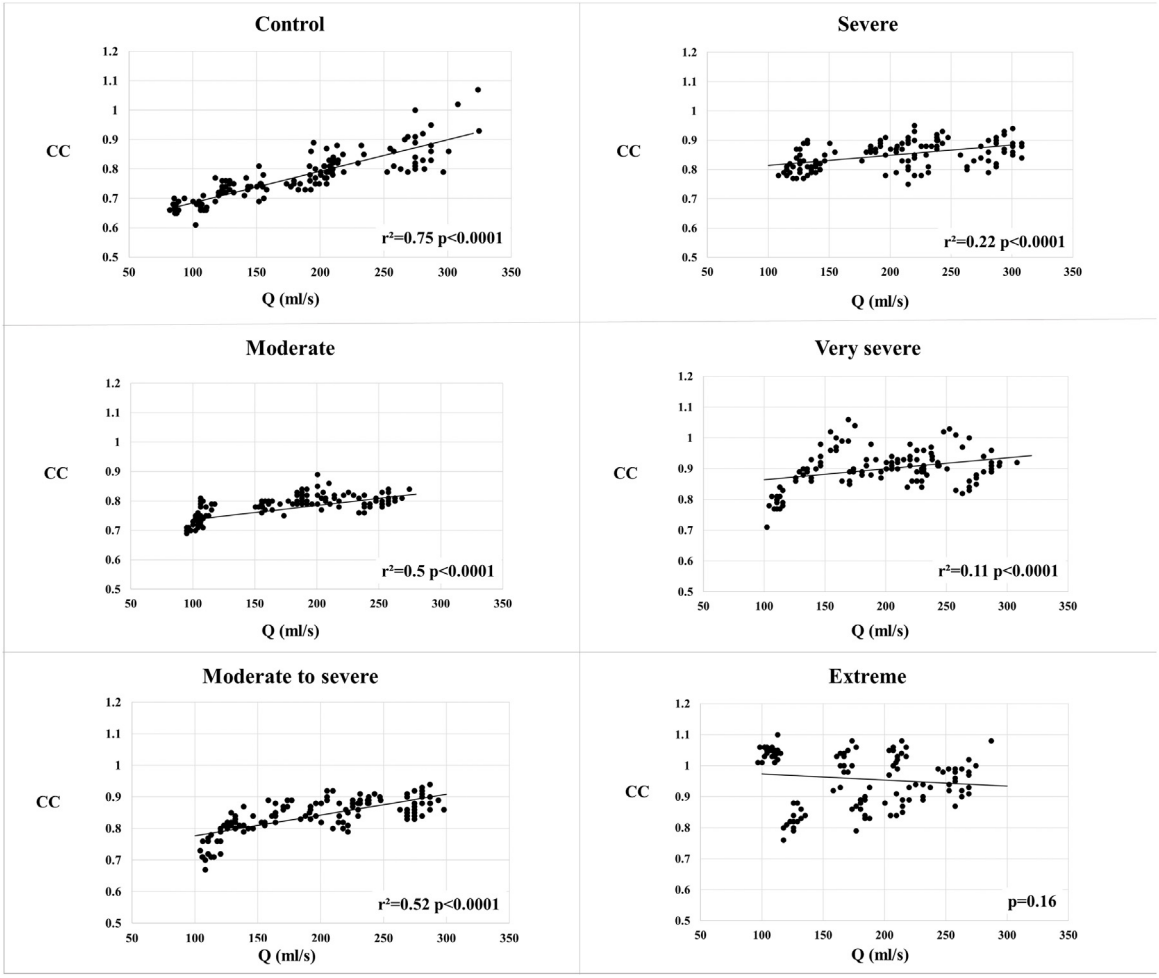

Fig. 4. Correlation between the flow (Q) and the contraction coefficient (CC), depending on the aortic stenosis severity.

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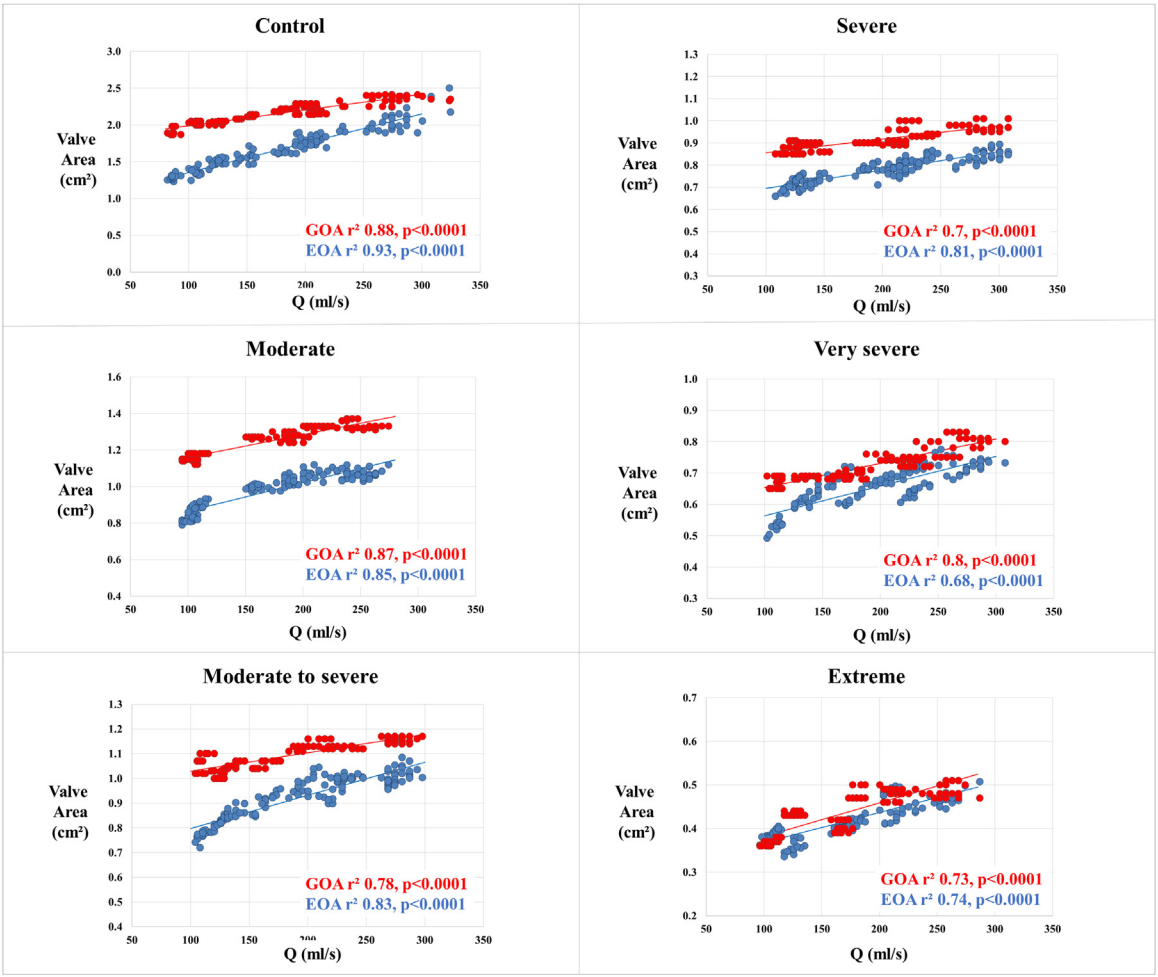

Fig. 5. Correlation between the mean flow rate (Q) and the effective orifice area (blue) or the geometric orifice area (red), for each degree of stenosis. EOA, effective orifice area; GOA, geometric orifice area.

EOA/GOA depending on mean aortic flow and aortic stenosis severity

In each stenosis condition, the EOA and the GOA were directly dependent on the mean aortic flow ($p < 0.0001$ for all, Fig. 5). However, the determination coefficient decreased as the severity of the stenosis increased, from 0.93 to 0.74 for the EOA, and 0.88 to 0.73 for the GOA.

EAO and mean aortic flow significantly predicted the CC in multiple linear regression analysis ($r = 0.62$ $p < 0.0001$), with the formula $CC = 0.825 + Q/1000 - EOA^{*}0.113$ (CC dimensionless, Q in ml/s, EOA in cm^2).

Discussion

Our study aimed to assess *in vitro* the relationship between EOA and GOA, and the factors influencing the CC.

Our results are as follows: (1) an EOA of 1 cm^2 corresponds to a GOA of 1.2 cm^2 and (2) the CC is influenced by both the aortic stenosis severity and the transaortic flow, gradually increasing towards 1 as the severity of the stenosis and the flow increase.

GOA threshold

The latest guidelines on the assessment of aortic stenosis consider that any of the three criteria: a valve area <1.0 cm^2, a peak velocity ≥4.0 m/s, or a mean gradient ≥40 mmHg can be considered to suggest severe AS [3]. Although it does not take into account the afterload on the ventricle, the guidelines also note that transesophageal planimetry can be used as well with the same cut-off value, especially if Doppler measurements are unavailable. Still, it is noted that the GOA can be slightly larger than the EOA [14,15]. Indeed, previous *in vitro* studies have shown that the CC, that is the ratio between the EOA and the GOA, was dependent on the transvalvular flow and the shape of the orifice [8,16]. Our data confirm that except in very severe stenoses or in high flow, the EOA is indeed smaller than the GOA. More specifically, in conditions of flow observed in patients, we showed that an EOA of 1 cm^2 corresponded to a GOA of 1.2 cm^2. The corresponding GOA was mildly higher in the very low flow condition, but the cardiac output, lower than 2 l/min, was hardly compatible with human physiology. If previous studies had already highlighted the relationship between effective and geometric areas in funnels or normal valves, this is to our knowledge the first time that an *in vitro* study defines a GOA severity threshold from the EOA. Therefore, based on this study, we would consider a GOA cut-off of 1.2 cm^2 to define severe aortic stenosis.

Moreover, these data were already observed in previous clinical studies. In a study including 27 patients, the GOA obtained with multislice computed tomography and magnetic resonance imaging was respectively 0.17 and 0.16 cm^2 larger than the EOA obtained with the continuity equation from echocardiography [5]. The optimal threshold with computed tomography scan and cardiac magnetic resonance imaging to detect severe aortic stenosis as determined by echocardiography was respectively 1.25 cm^2 and

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1.3 cm^2. Two other trials also studied the correlation between the EOA with echocardiography and the GOA from cardiac magnetic resonance imaging and computed tomography scan, and concluded that an EOA of 1 cm^2 corresponded to a GOA of 1.2 cm^2 with both methods [7,17].

In another study, Clavel et al. evaluated the measurement of the aortic valve area both by echocardiography and computed tomography scan on 269 patients [18]. With planimetry obtained by computed tomography scan, the aortic area of severe stenosis (based on an EOA obtained with echocardiography $\leq$1 cm^2) could range up to 1.4 cm^2. Another study on 169 patients showed an excellent correlation between the areas obtained with echocardiography and computed tomography scan ($r = 0.81$), but similarly, the planimetry of severe aortic stenosis could range up to 1.4 cm^2 [19]. Therefore, based on both our *in vitro* study and clinical studies, a GOA between 1 and 1.2 cm^2 should nevertheless raise the possibility of severe aortic stenosis. Although the cut-off value in the guidelines of 1 cm^2 for the GOA is very specific, it may lack sensitivity and may lead to erroneous diagnosis of moderate aortic stenosis [3].

Of course, methods employed to measure the GOA in clinical practice, such as multislice computed tomography and magnetic resonance imaging, cannot compete with the availability, innocuity, and low cost of transthoracic echocardiography, and therefore cannot be used as first-line imaging techniques. However, in the era of multimodality imaging, they can be of use in anechogenic patients or in discrepant situations.

Contraction coefficient determinants

Previous *in vitro* studies have shown that the CC was dependent, among other parameters, on the shape of the orifice inflow and on the transvalvular flow [8,16]. Depending on the shape of the orifice (rigid dome or funnel), the theorical CC could range respectively from 0.6 to 1. However, these experiments used plates, and as opposed to what is observed in valves, the CC did not vary with flow, making these results hardly applicable in clinical practice [20]. This is one of the reasons why our approach, based on trileaflet aortic valves with different degrees of restriction, is innovative. In stenotic valves, we showed that both the valve area and the mean flow rate had an influence on the CC, ranging from 0.6 to 1 in non-stenotic orifices, and from 0.8 to 1 in extreme stenosis. This dependence on flow could also be predicted from the Gorlin equation: $GOA = Q/(CC \times 44.3 \times (\text{mean gradient})^{1/2})$, also expressed as $CC = Q/[GOA \times 44.3 \times (\text{mean gradient})^{1/2}]$ [21]. In our experiment, for a set GOA, an increase of the mean flow rate led to a lesser increase of the mean gradient, and therefore an increase of the CC. For example, for the same GOA of 0.9 cm^2, an increase of the mean flow rate from 132 to 300 ml/s led to an increase of the mean gradient from 14 to 48 mmHg, and of the CC from 0.8 to 0.91. These CC values are slightly different from the ones obtained with the Gorlin equation, as we used the continuity equation to calculate an EOA/GOA ratio, but they varied in the same direction.

From the equation obtained with multiple linear regression, we could calculate that at normal flow ($Q > 200$ ml/s), the CC would always be >0.9 for an EOA < 1 cm^2, making in these specific conditions the EOA and GOA interchangeable. Although the EOA and GOA were statistically different, even in very severe stenosis, a difference <0.1 cm^2 did not seem relevant in clinical practice.

Limitations

The main limitation of this study is the design of the stenotic valves, based on the restriction of the leaflets by clamp rings. In degenerative disease, a very stenotic orifice would be heavily calcified, which is difficult to obtain *in vitro*. This may lead to differences in the CC variation compared to clinical practice. However, this model, based on the restriction of a bioprothesis leaflets, is more physiological than a plain orifice obtained with funnel and plates observed in most *in-vivo* studies, and has never been used before.

Another limitation may be the clinical applicability of the measurement of the GOA with cross-sectional imaging techniques of very severe and heavily calcified aortic stenosis. In our study, the GOA was measured with a high-speed camera, on non-calcified valves, making the delineation quite simple. With computed tomography, valve calcifications may cause reverberations, hampering the contouring of the orifice. A signal void may also be observed in cardiovascular magnetic resonance imaging because of the calcifications and turbulent flow, complicating the delineation of the leaflets. However, this measurement was shown to be possible even in calcified aortic stenosis [22].

Lastly, we observed the effect of the valve area and the flow on the CC, but other influencing factors, such as the shape of the orifice or the aorta diameter, could not be studied here. However, the shape of the orifice was an uneven triangle without commissural fusion (as shown in Fig. 1), which corresponds to what can be observed in degenerative aortic stenosis.

Conclusion

This study highlights the relationship between the EOA and the GOA in a controlled physiological environment. Specifically, we found out, for the first time in an *in vitro* study using stenotic bioprosthetic valves, that an EOA of 1 cm^2 corresponds to a GOA of 1.2 cm^2. Therefore, a GOA measured with planimetry between 1 and 1.2 cm^2, either by cardiovascular magnetic resonance imaging or computed tomography, should not discard the possibility of severe aortic stenosis. We also showed that the relation between the EOA and the GOA is dependent both on flow and stenosis severity, and that for a very severe stenosis at high flow both could be used interchangeably.

Declarations of interest

The authors declare that there is no conflict of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jjcc.2020.08.003.

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