TABLE 1. Reference values for effective orifice area for prosthetic heart valves, determined from articles published in the literature, reproduced with permission from Lancellotti and colleagues³

Prosthetic valve size (mm)	19	21	23	25	27	29
Stented bioprosthetic valves						
Mosaic (Medtronic, Minneapolis, Minn)	1.1 ± 0.2	1.2 ± 0.3	1.4 ± 0.3	1.7 ± 0.4	1.8 ± 0.4	2.0 ± 0.4
Hancock II (Medtronic)	–	1.2 ± 0.2	1.3 ± 0.2	1.5 ± 0.2	1.6 ± 0.2	1.6 ± 0.2
Carpentier-Edwards Perimount (Edwards Lifesciences, Irvine, Calif)	1.1 ± 0.3	1.3 ± 0.4	1.5 ± 0.4	1.8 ± 0.4	2.1 ± 0.4	2.2 ± 0.4
Carpentier-Edwards Magna (Edwards Lifesciences)	1.3 ± 0.3	1.5 ± 0.3	1.8 ± 0.4	2.1 ± 0.5	–	–
Biocor (Epic) (St Jude Medical, St Paul, Minn)	1.0 ± 0.3	1.3 ± 0.5	1.4 ± 0.5	1.9 ± 0.7	–	–
Mitroflow (Sorin Group, Arvada, Colo)	1.1 ± 0.2	1.2 ± 0.3	1.4 ± 0.3	1.6 ± 0.3	1.8 ± 0.3	–
Trifecta (St Jude Medical)	1.4	1.6	1.8	2.0	2.2	2.4
Stentless bioprosthetic valves						
Medtronic Freestyle	1.2 ± 0.2	1.4 ± 0.2	1.5 ± 0.3	2.0 ± 0.4	2.3 ± 0.5	–
St Jude Medical Toronto SPV (St Jude Medical, St Paul, Minn)	–	1.3 ± 0.3	1.5 ± 0.5	1.7 ± 0.8	2.1 ± 0.7	2.7 ± 1.0
Prima Edwards (Edwards Lifesciences)	–	1.3 ± 0.3	1.6 ± 0.3	1.9 ± 0.4	–	–
Mechanical valves						
Medtronic-Hall	1.2 ± 0.2	1.3 ± 0.2	–	–	–	–
St Jude Medical Standard	1.0 ± 0.2	1.4 ± 0.2	1.5 ± 0.5	2.1 ± 0.4	2.7 ± 0.6	3.2 ± 0.3
St Jude Medical Regent	1.6 ± 0.4	2.0 ± 0.7	2.2 ± 0.9	2.5 ± 0.9	3.6 ± 1.3	4.4 ± 0.6
MCRI On-X (CryoLife, Kennesaw, Ga)	1.5 ± 0.2	1.7 ± 0.4	2.0 ± 0.6	2.4 ± 0.8	3.2 ± 0.6	3.2 ± 0.6
Carbomedics Standard and Top Hat (Austin, Tex)	1.0 ± 0.4	1.5 ± 0.3	1.7 ± 0.3	2.0 ± 0.4	2.5 ± 0.4	2.6 ± 0.4
ATS Medical (Minneapolis, Minn)*	1.1 ± 0.3	1.6 ± 0.4	1.8 ± 0.5	1.9 ± 0.3	2.3 ± 0.8	–

*For the ATS medical valve, the label valve sizes are 18, 20, 22, 24, and 26 mm. High velocities are common in size 19 or 21 prostheses.

REPLY FROM THE AUTHORS: ON THE VALUE OF IN VIVO EFFECTIVE ORIFICE AREAS

Reply to the Editor:

We thank Dr Doenst for discussing the role of the in vivo effective orifice area (EOA) in judging the performance of surgical heart valves (SHVs) in relation to the Position Statement of the European Association for Cardio-Thoracic Surgery Society of Thoracic Surgeons American Association for Thoracic Surgery Valve Labelling Task Force.¹

In his letter,² Dr Doenst points out the fact that in vivo EOAs are influenced by patient-related factors and claims that “in vivo EOA (...) should not allow judging the hemodynamic performance of the implant or the presence of prosthesis–patient mismatch (PPM).” We disagree with this statement and provide below further clarification.

When discussing the role of in vivo EOAs in characterizing SHV performance, a clear distinction should be made between measured (actual) and reference EOA values. In vivo reference EOAs are determined in patient populations receiving the same model of SHV and are reported for each SHV model and size.^{3,4} They provide the hemodynamic fingerprint of a given model and size of the SHV and are useful in predicting the risk of prosthesis–patient mismatch before aortic valve replacement. In fact, in vivo reference EOAs are the most

important benchmarks when predicting or assessing SHV hemodynamic performance in patients undergoing valve replacement.^{5,6}

The actual EOA of an SHV, measured in the patient after SHV implantation, is indeed dependent on the patient's hemodynamic status and may be different from the EOA predicted before valve replacement using the mean reference EOA. For example, an SHV with a nominal mean reference EOA of 1.7 cm² may have a smaller measured EOA of 1.2 cm² because the patient is in a low-flow state. In such a patient, one may erroneously conclude that the patient has prosthesis–patient mismatch, whereas in fact the reduction of EOA is pseudo-severe (ie, valve leaflet not completely opened) because of low flow. On the other hand, the reference EOA of the implanted SHV is independent of the hemodynamics of the individual patient and thus provides an accurate assessment of the presence and severity of prosthesis–patient mismatch, regardless of the hemodynamic status of the patient. Nevertheless, considering that in vivo reference EOAs are determined in a population of patients, in vivo reference EOA values should always be considered as a value distribution (described with a mean and its standard deviation), and not only as a single mean value.

Taking these into account are necessary when using in vivo EOAs for evaluating the hemodynamic performance of SHVs preoperatively or postoperatively, or when assessing the risk of prosthesis–patient mismatch. We thank Dr Doenst for raising awareness of this important issue.

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