ADAPTATIONS TO CORONARY PHYSIOLOGY IN A PATIENT WITH SEVERE AORTIC STENOSIS AND COMPLETE HEART BLOCK UNDERGOING TRANSCATHETER AORTIC VALVE REPLACEMENT

Michael Michail MBBS\textsuperscript{ab}

Kaleab N. Asress PhD\textsuperscript{c}

James D. Cameron MD\textsuperscript{a}

Robert Gooley PhD\textsuperscript{a}

Liam M. McCormick MD\textsuperscript{a}

Alun D. Hughes PhD\textsuperscript{b}

Adam J. Brown PhD\textsuperscript{a}

\textsuperscript{a}Monash Cardiovascular Research Centre and Monash Heart, Monash University and Monash Health, Melbourne, Australia;

\textsuperscript{b}Institute of Cardiovascular Science, University College London, London, United Kingdom;

\textsuperscript{c}Department of Cardiology, Bankstown-Lidcombe Hospital, Bankstown, Australia.

Dr. Brown is supported through a Monash University Early Career Practitioner Fellowship. Drs. Gooley and McCormick are supported through a Robertson Family Research Cardiologist Fellowship. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

**Key Words:** aortic stenosis, coronary flow, coronary physiology, microcirculation, TAVR
An 82-year-old man underwent elective transcatheter aortic valve replacement (TAVR) for severe aortic stenosis. He was a participant in a study involving intracoronary Doppler and pressure assessment using a ComboWire (Volcano, San Diego, California) before and after valve deployment (Australian New Zealand Clinical Trials Registry number ACTRN12618000403235). The ComboWire was positioned in the left main coronary artery, and recordings were acquired at baseline and during hyperemia, both before and after the deployment of a 29-mm SAPIEN 3 (Edwards Lifesciences, Irvine, California) prosthesis. On both occasions, adenosine infusion precipitated complete atrioventricular block (CHB) followed by transvenous right ventricular pacing.

Analyses were performed using custom-designed software (CardiacWaves, King’s College London, London, United Kingdom) with ensembled-averaged signals. Adenosine-induced CHB decreased the ventricular rate from 60 to 39 beats/min pre-TAVR and from 72 to 37 beats/min post-TAVR (Figure 1A). Mean aortic pressure (MAP) concomitantly decreased from 81 to 65 mm Hg pre-TAVR and from 71 to 49 mm Hg post-TAVR (Figure 1B). Despite this, coronary averaged peak velocity was maintained during CHB (Figure 1C), mediated by 16.9% and 26.6% decreases in microvascular resistance pre- and post-TAVR, respectively (Figure 1D). Wave intensity analysis demonstrated preservation of the dominant systolic forward compression wave (Figure 1E) and diastolic backward expansion wave (Figure 1F) intensities during CHB. Right ventricular pacing resulted in less favorable hemodynamic status than during CHB, possibly because of accentuated ventricular desynchrony, with 15.8% and 9.9% lower MAP (pre- and post-TAVR, respectively), 24.3% and 15.5% lower coronary averaged peak velocity, 11.2% and 6.6% greater microvascular resistance, and reduced systolic forward compression wave and diastolic backward expansion wave intensities.

MAP was lower following TAVR, possibly because of myocardial stunning following rapid ventricular pacing. Despite lower MAP, however, coronary averaged peak velocity post-TAVR was consistently greater in the different heart rhythms. Higher systolic forward compression wave intensity in the presence of lower MAP is suggestive of improved flow into the coronary artery from the initial ventricular contraction, potentially because of alterations in sinotubular blood flow distribution following valve implantation (1). Conversely, a greater diastolic backward expansion wave intensity is likely attributable to improved ventricular relaxation and microcirculatory decompression (2). Although frequently used, right ventricular pacing may not improve coronary hemodynamic status, particularly at similar ventricular rates. Episodes of transient CHB are common during TAVR and may be well tolerated, because of rapid compensatory changes in the microcirculation preserving myocardial blood flow.

References

Figure 1. Hemodynamic Indexes During the Various Intraprocedural Events.

(A) Heart rate. (B) Mean aortic blood pressure. (C) Intracoronary averaged peak velocity. (D) Derived microvascular resistance. (E) Systolic forward compression wave (sFCW) intensity. (F) Diastolic backward expansion wave (dBEW) intensity. (G) Ensembled-averaged coronary pressure (blue) and flow velocity (red) during complete heart block (pre–transcatheter aortic valve replacement [TAVR]) and, beneath, the corresponding wave intensity analysis profile; positive deflections represent waves originating proximally, and negative deflections represent those originating distally. APV = averaged peak velocity; AV = atroventricular; MR = microvascular resistance; RV = right ventricular; WI = wave intensity.