

# Effect of Coronary Anatomy and Hydrostatic Pressure on Intracoronary Indices of Stenosis Severity

Tobias Härle, MD,<sup>a</sup> Mareike Luz, MD,<sup>a</sup> Sven Meyer, MD, PhD,<sup>a</sup> Kay Kronberg, MD,<sup>a</sup> Britta Nickau, MD,<sup>b</sup> Javier Escaned, MD, PhD,<sup>c</sup> Justin Davies, MD, PhD,<sup>d</sup> Albrecht Elsässer, MD<sup>a</sup>

## ABSTRACT

**OBJECTIVES** The authors sought to analyze height differences within the coronary artery tree in patients in a supine position and to quantify the impact of hydrostatic pressure on intracoronary pressure measurements in vitro.

**BACKGROUND** Although pressure equalization of the pressure sensor and the systemic pressure at the catheter tip is mandatory in intracoronary pressure measurements, subsequent measurements may be influenced by hydrostatic pressure related to the coronary anatomy in the supine position. Outlining and quantifying this phenomenon is important to interpret routine and pullback pressure measurements within the coronary tree.

**METHODS** Coronary anatomy was analyzed in computed tomography angiographies of 70 patients to calculate height differences between the catheter tip and different coronary segments in the supine position. Using a dynamic pressure simulator, the effect of the expected hydrostatic pressure resulting from such height differences on indices stenosis severity was assessed.

**RESULTS** In all patients, the left anterior and right posterior descending arteries are the highest points of the coronary tree with a mean height difference of  $-4.9$  cm (SD 1.6) and  $-3.8$  cm (SD 1.0); whereas the circumflex artery and right posterolateral branches are the lowest points, with mean height differences of  $3.9$  cm (SD 0.9) and  $2.6$  cm (SD 1.6) compared with the according ostium. In vitro measurements demonstrated a correlation of the absolute pressure differences with height differences ( $r = 0.993$ ;  $p < 0.0001$ ) and the slope was  $0.77$  mm Hg/cm. The Pd/Pa ratio and instantaneous wave-free ratio correlated also with the height difference (fractional flow reserve  $r = 0.98$ ;  $p < 0.0001$ ; instantaneous wave-free ratio  $r = 0.97$ ;  $p < 0.0001$ ), but both were influenced by the systemic pressure level.

**CONCLUSIONS** Hydrostatic pressure variations resulting from normal coronary anatomy in a supine position influence intracoronary pressure measurements and may affect their interpretation during stenosis severity assessment.

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**F**unctional assessment of coronary stenoses using intracoronary pressure guidewires constitutes a simple and clinically important diagnostic tool in the catheterization laboratory. In practice, fractional flow reserve (FFR) is calculated as the ratio of the distal trans-stenotic pressure to

the proximal coronary or aortic pressure during pharmacologically induced (usually adenosine-induced) hyperemia, assuming that the effect of boundary conditions such as central venous pressure is negligible (1). The instantaneous wave-free ratio (iFR) is a new adenosine-independent index of coronary stenosis

From the <sup>a</sup>Department of Cardiology, Klinikum Oldenburg, European Medical School Oldenburg-Groningen, Carl von Ossietzky University Oldenburg, Germany; <sup>b</sup>Department of Diagnostic and Interventional Radiology, Klinikum Oldenburg, European Medical School Oldenburg-Groningen, Carl von Ossietzky University Oldenburg, Germany; <sup>c</sup>Cardiovascular Institute, Hospital Clinico San Carlos, Madrid, Spain; and the <sup>d</sup>International Centre for Circulatory Health, National Heart and Lung Institute, Imperial College London, London, United Kingdom. The technical equipment for the dynamic pressure simulator was a loan from Volcano Corporation. Dr. Escaned has served as a consultant and a speaker at educational events for Philips-Volcano and Boston Scientific. Dr. Davies is a consultant for Volcano Corporation; and holds licensed patents pertaining to the iFR technology. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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**ABBREVIATIONS  
AND ACRONYMS****BMI** = body mass index**CT** = computed tomography**CTA** = computed tomography angiography**CX** = circumflex artery**DPS** = dynamic pressure simulator**ECG** = electrocardiogram**FFR** = fractional flow reserve**iFR** = instantaneous wave-free ratio**LAD** = left anterior descending artery**LV** = left ventricular**LVEF** = left ventricular ejection fraction**Pa** = mean systemic pressure**Pd** = mean distal pressure**RCA** = right coronary artery**RPD** = right descending posterior artery**RPL** = right posterolateral artery

severity. For iFR calculation, the aforesaid pressures are measured and indexed during a specific diastolic wave-free period, when coronary resistance is most stable and minimized over the cardiac cycle (2).

This simple approach facilitates physiological assessment of coronary stenosis severity in the catheterization laboratory. However, it is potentially fraught by the boundary conditions and assumptions of the model. Thus, although stenosis location should, from a theoretical perspective, not influence FFR or iFR measurements, significant differences of both iFR and FFR values between anterior and posterior coronary territories have been observed in clinical practice (3,4), with higher values of both indices in the circumflex (CX), obtuse marginal, and right coronary arteries (RCA), when compared with the left anterior descending coronary artery (LAD) and its diagonal branches. This difference was independent of the effects of potential clinical and anatomic confounders (4).

We hypothesized that variations in hydrostatic pressure, related to coronary anatomy, might be the cause of these findings. This might be important, not only for the interpretation of conventional FFR or iFR measurements, but also for intracoronary pressure mapping, an increasingly used

application of both indices, and for measurements of microcirculatory resistance.

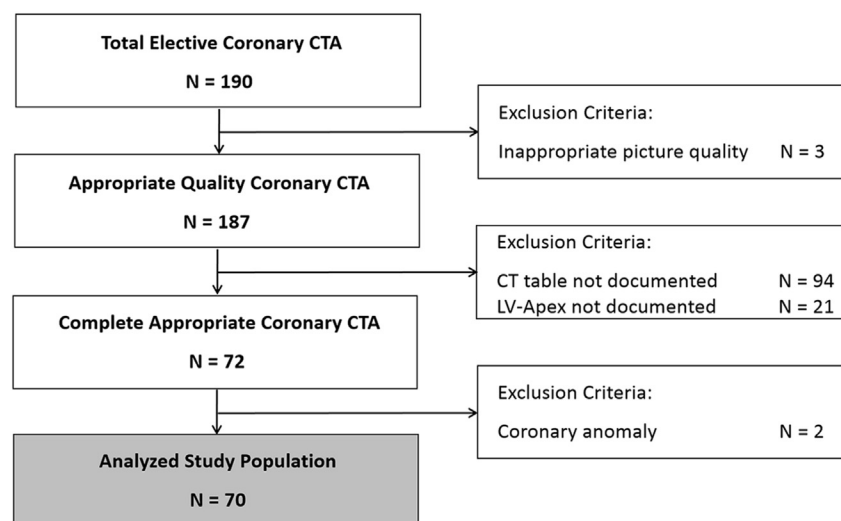
In the study presented, we sought to analyze and quantify the impact of hydrostatic pressure on the results of intracoronary pressure measurements using an *in vitro* model. Furthermore, we aimed to quantify the height differences between the distal coronary vessels and the corresponding coronary ostia in real patients in a supine position in order to quantify a theoretical impact of hydrostatic pressure *in vivo*.

**METHODS**

**STUDY POPULATION.** From May 2015 to June 2016 a total of 190 elective coronary computed tomography angiographies (CTAs) were performed in our hospital. Inclusion criteria for this analysis was any elective coronary CTA with capture of the complete heart as well as the computed tomography (CT) table. Patients with incomplete opacification of the coronary vessels ( $n = 2$ ), extensive artefacts ( $n = 1$ ), and coronary anomalies ( $n = 2$ ) were excluded from analysis. Overall, a number of 70 CTA was identified as appropriate for the intended analysis (Figure 1).

**ANALYSIS OF CORONARY ARTERY HEIGHT DIFFERENCES.**

Coronary CTA was performed according to common standards using a CT scanner with 64 detector rows. Beta-blockers were administered intravenously if necessary, targeting a heart rate below 90 beats per

**FIGURE 1** Flow Chart of Patient Selection

CTA = computed tomography angiography; LV = left ventricular.

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