

Revisiting the Historical Origins of Clinically Meaningful Coronary Artery Obstruction



To the Editor: As part of a recent informative editorial on the significance of nonobstructive coronary artery disease, Rumberger¹ reviewed the historical origins of the 50% coronary stenosis standard for the definition of myocardial ischemia and the relationship of percentage stenosis to coronary flow reserve and fractional flow reserve. I believe some corrections are warranted.

Rumberger repeated the commonly stated but incorrect assumption that the origin of the 50% stenosis threshold is attributable to the landmark 1974 article by Gould et al.² The origin of the 50% coronary stenosis standard antedates the Gould article and was adopted in 1969 as the threshold for intervention by the VA Cooperative Study, the first major randomized trial of bypass surgery.³ Even before this, in 1966, a 50% or greater diameter stenosis was identified as indicating a “severe” arteriographic narrowing by Sones’ group at the Cleveland Clinic and was subsequently used and published as the cutoff value for bypass surgery when introduced by that institution.⁴ The original Gould et al² article specifically indicated that a stenosis of “45 to 50 percent by diameter probably does not impair coronary flow reserve in man.” In the text, they were careful to indicate that coronary flow reserve begins to decline at a 30% stenosis but that a 65% to 95% stenosis is required to cause marked impairment of coronary flow reserve. Nor is the Gould et al² article the origin of the 70% stenosis standard, which was adopted by the Coronary Artery Surgical Study in 1973,⁵ before the publication of the Gould et al report. The Gould et al² article, although transformational for many fields, is not responsible for the

adoption of the 50% or 70% stenosis threshold for ischemia.

Rumberger¹ also indicated that a 70% coronary stenosis has become “the de facto stenosis measurement supported by fractional flow reserve data.” To the contrary, the major lesson of coronary flow reserve and fractional flow reserve research is that it is not possible to determine the functional significance of a coronary stenosis on the basis of percentage stenosis. Only one-third of narrowings within a stenosis range of 50% to 70% display provokable myocardial ischemia by fractional flow reserve.⁶

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In Reply—Revisiting the Historical Origins of Clinically Meaningful Coronary Artery Obstruction



To the Editor: First, I sincerely thank Rosenthal¹ for his correspondence

letter regarding my prior editorial.² We all continue to learn, and as a student of medical history I am embarrassed that I was not aware of his excellent 2015 commentary on “The 50% Stenosis.”³ In that article, Rosenthal acknowledged the commonly held physiologic studies by Gould et al⁴ as one of the original investigations on the severity of percent stenosis and coronary hemodynamics; I now stand corrected on the true origins of the clinical use of the 50% stenosis threshold. However, as also noted in my editorial, I suspect that a lot of the future issues of “percent stenosis” relied on the classic hemodynamic studies done by Katz and Linder⁵ in the 1930s.

As the use of 50% or more stenosis as possibly suggesting ischemia became popular in the 1960s and 1970s, much of this concept was fostered, legitimized, and perpetuated by studies looking at stress testing, where the sensitivity and specificity values for potential “ischemia” were found to be optimal using the angiographic 50% threshold.

I did not really state *my opinion* about the current use of a 70% stenosis as more likely being the standard for fractional flow reserve; on the contrary, I stated the current “expert consensus” using a percent stenosis threshold for “revascularization.” The latest Appropriateness Criteria for Coronary Revascularization in patients with stable ischemic heart disease uses either (1) 70% or more luminal diameter narrowing, by visual assessment, of an epicardial stenosis measured in the “worse view” angiographic projection and/or (2) a fractional flow reserve of 0.80 or less consistent with downstream inducible ischemia.⁶

I still get asked questions by patients about “percent stenosis” since this vernacular has now escaped into the public realm. We are still bound in the stenosis world in cardiology. Yet, control of microvascular