Is D2B the Only Metric to Determine Outcome With STEMI?

We appreciate Dr. Conti’s remarks highlighting the recent conundrum of lower door-to-balloon times not equating to reduced mortality or 30-day readmission rates in our paper (1) as well as that reported in another recent publication (2). As we disclosed in the discussion, our 4-year study began in 2007 at a time when many demographics nationwide, including symptom onset, first medical contact, and total ischemia time, also known as symptom-to-balloon (S2B) time, were not identified tracking elements. Fortunately, many regional systems of care have now incorporated these into their data acquisition through the ACTION (Acute Coronary Treatment and Intervention Outcome Network) Registry—Get With the Guidelines, and indeed we began adopting these in 2009 as we recently reported at the 2013 Transcatheter Cardiovascular Therapeutics Conference (3,4). Even so, these are not the entire universe of important metrics regarding acute ST-segment elevation myocardial infarction treatment. Oh, how we wish we could quantitate preservation of ejection fraction, lower morbidity associated with less heart failure, and quality of life measures, to name a few. It is not good enough to presume that these are improved by better door-to-balloon times, but it does seem intuitive. As has been said by the North Carolina RACE-ER (Reperfusion of Acute myocardial infarction in Carolina Emergency departments—Emergency Response) investigators Dr. Jollis, Dr. Granger, and Ms. Roettig, a door-to-balloon time <90 min has largely been solved by most well-functioning hospitals in this country and abroad (5). Only through achieving the overarching goal of reducing total ischemia time or S2B time through coordinated regional systems of care may we be able to further reduce an already low ST-segment elevation myocardial infarction mortality rate and improve outcomes. It is hoped that our paper and the 5 or so tools that we described may be useful in helping systems reach these goals. We all need to continue work on S2B time and extended outcomes measures.

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Renal Denervation of Accessory Renal Arteries

In the October issue of JACC: Cardiovascular Interventions, Id et al. (1) compared the results of renal denervation using the Symplicity catheter system (Medtronic, Mountain View, California) in patients with and without accessory renal arteries. This issue is not insignificant considering that 27% of the resistant hypertension population may have accessory renal arteries. These patients have, for the most part, been excluded from clinical trials of renal denervation devices. The drop in office-based systolic blood pressure at 6 months was significantly less in the patients with accessory renal arteries than in those with only main renal artery trunks (~6.2 mm Hg vs. ~16.6 mm Hg). The patients with accessory renal arteries were further subdivided into 2 groups; the subgroup with complete accessory renal artery denervation experienced a slightly greater drop in systolic blood pressure than did patients with incomplete treatment of their accessory arteries (~8.8 mm Hg vs. ~4.1 mm Hg).

Renal denervation targets the sympathetic nerves located in the adventitia of the renal artery wall (2). The nerves travel from the spinal cord along the artery and then to the kidney where extensive branching occurs. Early anatomic studies using electron microscopic autoradiography demonstrated that the majority of renal nerves terminate at vascular structures within the kidney (3). Based on these anatomic findings, the nerve traffic to and from the kidney corresponds at least roughly to the blood supply, traveling with both accessory and main renal arteries. So patients with accessory renal arteries may be predisposed to a lack of blood pressure response if treated with a denervation technique targeting nerves only in the main renal artery—just as was reported in the article by Id et al. (1).

Yet, why was the blood pressure-lowering response in the subgroup with “complete” renal denervation of accessory renal arteries less than that in the group with only main renal artery trunks? This difference may in part be explained by limitations of the monopolar renal denervation system used for ablation of accessory renal arteries. Cooling of monopolar catheters from blood flow is critical to avoid tissue damage from catheter overheating. In low flow conditions such as with accessory renal arteries, cooling may be inadequate. The system generator will then terminate energy delivery to prevent overheating, but it also halts the ablation, potentially compromising denervation efficacy. Other renal denervation systems that do not require cooling may be more effective for accessory renal arteries (4). It will be critical to understand the
differences in renal denervation technologies and their applicability to the subset of patients with accessory renal artery anatomy.

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Please note: Dr. Mendelsohn has received consulting fees from Medtronic and Boston Scientific/Vessix and has served on the medical advisory board of Boston Scientific.

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Reply
Renal Denervation of Accessory Renal Arteries

We appreciate the interest and comments of Dr. Mendelsohn regarding our recently published paper (1). The paper described the blood pressure–lowering effect of catheter-based renal denervation in patients with bilateral single renal arteries compared with those with accessory renal arteries. 

As outlined in our paper, systolic blood pressure reduction was less pronounced in patients with accessory renal arteries (regardless of complete or incomplete denervation). Furthermore, blood pressure reduction, even in patients with complete denervation of accessory renal arteries, was numerically less pronounced than in patients with bilateral single renal arteries; however, this difference did not reach statistical significance. We postulated that the latter observation might have been related to a true absence of a difference in blood pressure reductions or to a true difference that did not reach statistical significance due to insufficient power, a result of the limited number of patients in the respective subgroups. Studies with larger patient numbers and adequate power to clarify the importance of complete denervation including accessory renal arteries are needed. We agree with Dr. Mendelsohn that, provided these studies confirm a less pronounced blood pressure reduction in patients with accessory renal arteries, this may be the result of incomplete denervation due to technical limitations related to overheating as a consequence of insufficient cooling by altered flow dynamics in accessory renal arteries. It is also possible that limited maneuverability of the catheter used in our study in smaller caliber arteries interferes with circumferential energy application, perhaps preventing complete denervation. In this context, potential solutions are being actively pursued by a number of companies. These may include, but are not limited to, irrigated radiofrequency catheter systems, lower profile catheters with superior maneuverability that do not interfere with flow dynamics in smaller vessels, the use of a cooling balloon during energy delivery, chemical neurolysis, or noninvasive denervation. Some of these technologies have reached the clinical stage and approval for use in some countries. It will now be very important to examine the efficacy (and safety) of these technologies in smaller accessory renal arteries. Hence, studies examining devices that allow application in smaller (<4 mm) caliber arteries should not exclude patients with accessory renal arteries.

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