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Do Ameroid Constrictors Reliably Occlude Porcine Coronary Arteries?

Jeremy L. Herrmann, M.D.¹

¹ Department of Surgery, Indiana University School of Medicine, Indianapolis, Indiana

Article Commentary

Ameroid constrictors are devices capable of inducing chronic myocardial ischemia generally through occlusion of the left circumflex artery in animal models [1]. The ameroid constrictor consists of an inner hygroscopic ring that upon fluid absorption expands against a fixed metal sheath thereby occluding the encompassed vessel. Luminal occlusion occurs over 4–5 weeks with the most rapid rate of closure occurring in the first 3–14 days, and chronic ischemia ensues with subsequent development of collateral vessels [2]. Vessel occlusion depends on vessel and constrictor size, and an important question regarding the use of this device is whether there is a predictable correlation with ameroid constrictor size and vessel size with regard to reducing coronary flow. In this study, Tuzun and colleagues evaluate the relationship between ameroid constrictor size with coronary flow to determine whether the diameter of the ameroid constrictor determines degree of coronary artery occlusion [3].

In this experiment, pigs were grouped according to the internal diameter of the ameroid constrictor. Thirty days after placement of the ameroid constrictor, angiography was performed to measure coronary flow, and endocardial electromechanical (NOGA) mapping was used to evaluate the extent of the injured myocardial territory. Coronary occlusion was observed in all animals at the end of the study period, and there was no correlation between coronary flow and device diameter in the range tested. After normalizing the device diameter to vessel diameter, no statistical differences were observed with regard to percentage of injured LV area and device diameter. These results suggest these ameroid constrictor sizes may be reliably used in to produce chronic myocardial ischemia in porcine models.

There are several important potential limitations of this study, though. NOGA mapping is a percutaneous catheter-based technique which has been proved to be useful for differentiating ischemic but viable hibernating myocardium from nonviable tissue. It has been employed in gene-based [4,5] and cell-based therapies [6] for myocardial ischemia as a method of evaluating the effects of treatment as well as for guiding injections. As with any technique, the success of NOGA mapping is highly dependent on operator skill. However, as the authors mention, those performing the NOGA mapping in this study were experienced operators. Secondly, large degrees of heterogeneity in endocardial voltages have been reported for normal and hibernating myocardium [7,8]. This likely results from the mixed composition of fibrotic and viable

Correspondence: Jeremy L. Herrmann, M.D. 545 Barnhill Drive, Emerson Hall Room 203, Indianapolis, Indiana 46202, jlherrma@iupui.edu, Phone: 317-278-8625, Fax: 317-274-6896.

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myocardial elements in hibernating myocardium and may account for the large confidence intervals observed in this and previous studies.

The authors note that one mechanism of ameroid-induced vessel occlusion involves mechanical trauma to the endothelium and subsequent thrombus formation. They further mention that this mechanism may cause arterial occlusion despite imprecise matching of the ameroid and coronary artery sizes. Further work to evaluate the frequency of this effect will be necessary to fully understand how these devices effect their results.

The present study offers no myocardial functional data to correlate the observed coronary flow rates and NOGA mapping with functional impairment. Determining whether LV functional impairment correlates with ameroid constrictor size in this setting would also be a useful outcome to fully answer this question in the setting of this model.

While we might conclude that these sizes of ameroid constrictors may reliably produce chronic myocardial ischemia in porcine models, the sizes selected for this study are not representative of the broader range of sizes available. Further work will be needed to determine what if any relationship exists for other sizes of ameroid constrictors and their target vessels. However, despite these limitations, it is reassuring to know that ameroid constrictors, when used in porcine models of chronic myocardial ischemia, reliably achieve their intended objective.

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