Letter by Picichè Regarding Article, “Effect of Permanent Right Internal Mammary Artery Closure on Coronary Collateral Function and Myocardial Ischemia”

To the Editor:

With great interest, I read the article by Stoller and Seiler on the effect of permanent internal mammary artery (IMA) closure on coronary collateral function and myocardial ischemia. IMA occlusion is an Italian concept that dates back to 1939 and was first introduced into practice in the 50s. Though forgotten for over 50 years, I decided—to my knowledge for the first time in medical circles—to resurrect this concept because I noticed that the number of patients failing to benefit from conventional revascularization was markedly underestimated. On this topic, I earned a Canadian grant for an experimental study, completed a PhD in Paris, and started publishing from 2010 onward. Being a cardiac surgeon and spending time in operating rooms rather than catheterization laboratories, my goals were to spread word of the IMA occlusion concept among interventional cardiologists and to highlight what I believed, and still believe, is an intriguing research area in cardiology. How exactly, in man, the pericardiophrenic branch of the IMA increases blood flow to the heart and collateral function remains a puzzle in my mind, but old studies suggest that small collateral vessels arising from the pericardiophrenic branch reach the pericardial reflections surrounding the heart. Whether this regards the heart muscle microcirculation or the aorta and pulmonary artery to establish connections with local vessels and the vasa vasorum. These latter vessels, in turn, are believed linked to myocardial muscle microcirculation. From this perspective, flow from the pericardiophrenic branch might be ascribed to a network of so-called noncoronary collateral myocardial blood flow or noncoronary collateral circulation (ie, flow to the heart from thoracic arteries like the bronchial, esophageal, diahphragmatic, intercostal, and mediastinal). Despite uncertainty about the exact mechanisms and anatomic interactions between the pericardiophrenic branch and heart, it is well established that IMAs have tremendous potential to spontaneously sprout collateral branches in the presence of an ischemic stimulus. This may occur either in the heart or lower limbs. We also should not be surprised that, in humans, blood can reach the heart from extracardiac arteries. Phylogenetic differences in the arterial system between species and the fact that, in some mammals like rats, the heart receives dual blood supply from vessels corresponding to the IMAs (called cardiomedialstinal arteries) supports this conjecture. Whatever the exact mechanism by which IMA occlusion reduces myocardial ischemia, after a study I conducted with coworkers was unfruitful owing to an overly extensive protocol involving surgically treated dogs, I became convinced that the effects of IMA occlusion must be investigated via endovascular means. Reporting in leading journals, I first suggested endovascular occlusion as a potential tool to treat refractory angina, both in 2010 and 2016. In a comprehensive review, I also provided 2 angiographic images of postoperative IMA occlusion caused by surgical technique issues, to show how this could cause collaterals to develop. New branches had developed from the proximal patent segments, directed toward the heart. The article by Stoller and Sailer, albeit failing to credit my previous observations and suggestions, reinforces both my initial concept and current beliefs and should promote further research.

Disclosures

None.

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References

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