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EDITORIAL COMMENT

New Insights Into an Old Problem*

Charanjit S. Rihal, MD, MBA,[a] Joseph J. Maleszewski, MD[b]

In this issue of *JACC: Cardiovascular Interventions*, Hamilton et al. (1) from the Bristol Royal Infirmary make an important contribution to our understanding of the in vivo anatomy of post-myocardial infarction mechanical complications. Post-infarction ventricular septal defects (PIVSD) are an infrequent, but feared, complication with resultant high morbidity and mortality (2). The low incidence, likely <1% in the modern reperfusion era, has paradoxically hampered attempts to develop surgical and interventional therapies. As a consequence, outcomes remain poor with high mortality rates at 1 year, regardless of therapy (3).

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Hamilton et al. (1) present the first series of comprehensive tomographic imaging in 32 patients with PIVSD collected over a 10-year period. A number of important findings emerge. Firstly, just over 50% of their cohort had died by 1 year, confirming the poor outcomes (with some exceptions) and high risk of this deadly complication. From a technical standpoint, they demonstrate that computed tomography images can be acquired rapidly and safely and be of high enough quality to permit detailed morphological analysis. PIVSDs can occur secondary to infarctions involving either the left or right coronary artery territories, and often are extremely complex, with serpiginous tracks, multiple orifices, and can involve adjacent regions of the myocardium; though they may be "simple" and completely surrounded by septal tissue (**Figure 1**). Infarctions of the posterobasal septum appear to be particularly prone to complex morphology, a finding that may relate to the arrangement of the myofibers in this region of the heart. The septum can literally be pulled off adjacent ventricular walls, resulting in a left-to-right shunt.

None of the PIVSDs were round, and the size and shape of the defects varied between systole and diastole, confirming the need for detailed imaging over the entire cardiac cycle. Many of the defects were large, and had very thin tissue margins at their edges, all factors that make percutaneous closure and even operative repair difficult if not impossible particularly in light of the fact that the myocardium adjacent to the defect is often friable (4). Tissue integrity diminishes rapidly in the hours and days following infarction, with maximal softening occurring around day 5 as the acute inflammatory infiltrate begins to transition to a chronic "clean-up" phase (**Figure 2**). Only 50% of the defects were small enough for even the largest device to occlude the defect in both systole and diastole. Complicating matters, even the largest plug would have reached adequate tissue margins in about 75% of cases in both systole and diastole. Despite valiant efforts, only about one-half of cases that underwent percutaneous closure survived to 1 year.

These findings raise important questions. Is it time for us to quit trying to fit round pegs into complex anatomic defects? The construction of most commercially available closure devices is predicated upon congenital atrial and ventricular septal defects, which have more predictable sizes and border regions. These devices, in the first instance, were not designed to be implanted into the necrotic and inflammatory tissue seen in the wake of massive myocardial infarction (**Figure 1**). The same anatomic problems and poor tissue quality make surgical therapy problematic, high risk, and fraught with hazard. Although new closure devices have entered the market, none of these were designed specifically for PIVSD, and it is unlikely that

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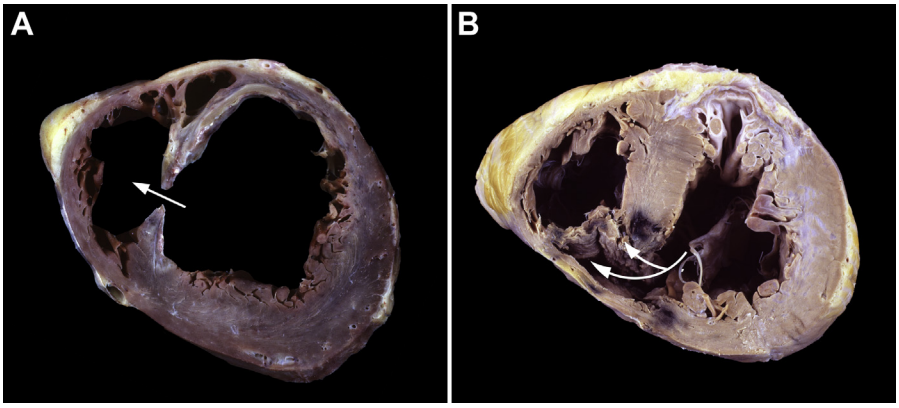
From the [a]Department of Cardiovascular Medicine, Mayo Clinic, Rochester, Minnesota; and the [b]Departments of Laboratory Medicine and Pathology and Medicine, Mayo Clinic, Rochester, Minnesota. Dr. Rihal has received research funding from Edwards Lifesciences and Abbott Vascular; and has been a consultant to Edwards Lifesciences. Dr. Maleszewski has reported that he has no relationships relevant to the contents of this paper to disclose.

FIGURE 1 Post-Infarction Ventricular Septal Defects



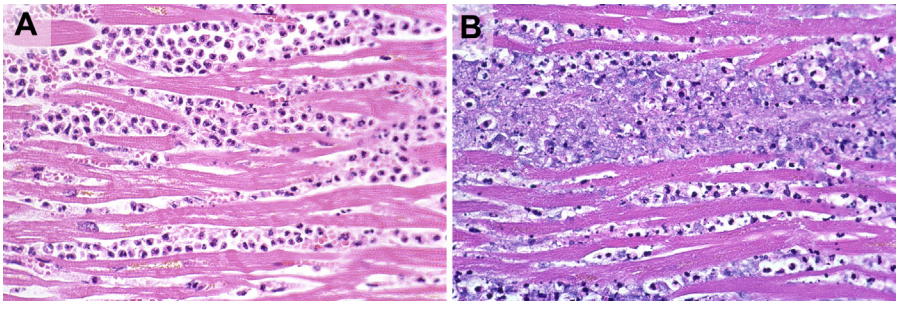
Autopsy-derived gross specimens demonstrating **(A)** a simple-type lesion **(arrows)**, occurring in the apical mid-septum, completely surrounded by septal muscle and **(B)** a complex-type lesion, occurring in the inferobasal septum with dissection along the inferior wall of the right ventricle **(lower arrow)** in addition to the septal defect **(upper arrow)**. **Arrows** indicate direction of blood flow.

they will represent a major therapeutic advance for this high-risk patient subset.

So, what can be done? The time has come for manufacturers and the interventional community to design devices that may allow for meaningful recovery either as a standalone procedure or as a bridge to definitive surgical repair. Tomographic imaging may facilitate the development of such devices, because 3-dimensional reconstruction technology, either computer-assisted design or the use of 3-dimensional printing technology could make a practical platform on which to design new devices and/or tailor such to the patient (5).

What would the characteristics of such devices be? First, they would have to be large enough, and conformable enough to anchor with stability, and provide near complete cessation of left-to-right shunting. The use of thick braid metallic devices can be problematic in post-infarction tissue, and new materials are needed. The ability to perform detailed tomographic images in vivo, and subsequently reconstruct the defects, would allow for improved procedural and surgical planning. The use of modern technologies, new materials, and personalized design characteristics should hopefully act as a springboard to innovation. To this end, manufacturers and investigators should

FIGURE 2 Histopathology of Acute Myocardial Infarction



(A) Prominent acute inflammatory (neutrophilic) infiltrate is seen in the first 24 to 48 h following the infarction. **(B)** Breakdown of the myocytes and the neutrophils around days 4 to 5 impart a grungy blue character to the infarcted tissue and correlate with substantial tissue softening both in and around the region of the post-infarction rupture.

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work with regulatory agencies to facilitate the bringing of effective new therapies to this important subset of patients. The findings by Hamilton et al. (1) represent an important advance toward this goal.

ADDRESS FOR CORRESPONDENCE: Dr. Charanjit S. Rihal, Mayo Clinic, 200 First Street SW, Rochester, Minnesota 55905. E-mail: rihal@mayo.edu.

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