

concomitant surgical procedure is indicated. For patients with an inaccessible epicardial space, percutaneous catheter ablation in the electrophysiology laboratory can be facilitated by a surgically created epicardial window allowing entry into the pericardial space (S9.15.4). This approach is most straightforward in patients with an apical or inferior VA substrate, because the area of the heart closest to the window is most easily accessed, and pericardial adhesions can limit extensive mapping. Surgical cryoablation can also be conducted in the operating room utilizing a lateral thoracotomy or sternotomy (S9.15.5,S9.15.6). Thoracotomy is the preferred approach to lateral and posterior substrates, whereas sternotomy is often needed for anterior substrates. Portable EAM can be utilized to help to localize the substrate, and VT can be inducible when the patient is off the cardiopulmonary bypass. When possible, preprocedural planning should include CT imaging of grafts for patients with prior bypass surgery to prevent injury to these structures. The use of surgical VT ablation has also been described in patients undergoing percutaneous LVAD implantation (S9.15.7,S9.15.8). Recently, there has been growing concern regarding the increased rates of LVAD thrombosis among patients who undergo both endocardial and epicardial surgical cryoablation at the time of LVAD implantation (S9.15.9). In highly selected patients with epicardial LVOT VAs refractory to standard techniques due to overlying epicardial fat and/or proximity to coronary arteries, open (S9.15.10) or minimally invasive (S9.15.11,S9.15.12) approaches have been used. The full characterization of the efficacy and safety of these approaches is pending.

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9.16. Sympathetic Modulation

Key Points

- Sympathetic modulation targeting the stellate ganglia by video-assisted thoracoscopy may be considered for failed VT ablation procedures or VF storms.
- A temporary effect can be obtained with the percutaneous injection or infusion of local anesthetics.

The autonomic nervous system (ANS) has a central role in regulating heart rate and cardiac function and contributes to the pathogenesis of VA in structurally normal hearts, abnormal hearts, and channelopathies (S9.16.1). The ANS includes the parasympathetic component mediated by the vagus nerve and a sympathetic component mediated by cervicothoracic paravertebral sympathetic ganglia. The ANS undergoes remodeling and becomes dysfunctional in SHD (S9.16.1). In most situations, ANS imbalances contribute to the onset and/or maintenance of VAs, especially through the activation of efferent sympathetic pathways. Interventions that decrease sympathetic tone are often beneficial.

Sympathetic activation and vagal withdrawal are associated with diminished respiratory sinus arrhythmia and heart rate variability. Iodine-123 metaiodobenzylguanidine and 11C-meta-hydroxyepinephrine imaging provide an indication of sympathetic nerve distribution and local noradrenaline reuptake that reflects sympathetic activity (S9.16.1). Although iodine-123 metaiodobenzylguanidine and 11C-meta-hydroxyepinephrine (S9.16.2) imaging findings are associated with VA in various settings, the clinical utility of using these findings for risk stratification and to guide therapy has yet to be proven (S9.16.1).

Beta blockers are a first-line therapy for VA sensitive to ANS influences. Beta blockers often decrease idiopathic RVOT PVCs in healthy individuals (S9.16.3) and reduce the rate of sudden cardiac death after MI (S9.16.4,S9.16.5). Conversely, in electrical storms due to Brugada syndrome or idiopathic VF, beta-adrenergic stimulation by isoproterenol infusion can abolish recurrent arrhythmia (S9.16.6,S9.16.7).

Transient CSD achieved by high thoracic epidural anesthesia with bupivacaine administered at the T1–T2 or T2–T3 level into the epidural space can be employed as a bridge to more definitive therapy (S9.16.8). An 80% reduction in VA episodes was observed in 6 of 8 patients undergoing thoracic epidural anesthesia, with infusion durations