required to successfully implement these operations. Te and colleagues⁴ investigated the United Network of Organ Sharing database to look at outcomes in patients undergoing CHLT.⁵ Of 47 patients, 13% (6 patients) had CHD as a presenting diagnosis. Mortality in the whole group was 13% at 6 months, although the rate in patients with CHD was not reported. We recently reported our single institution experience with CHLT. Of 14 patients undergoing CHLT since 2008, 4 had failed single ventricle/Fontan physiology; 10 patients had biventricular failure. All 4 Fontan patients had protein-losing enteropathy with central-to-portal bridging fibrosis on liver histology. The Fontan group had no mortality at 1-year follow-up, whereas the biventricular failure group had 2 deaths (20%). Outcome analysis suggested that CHLT in patients with single ventricle physiology can be performed safely, with equivocal

early outcomes to CHLT in patients with biventricular failure.

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Epicardial cryoablation resulting in precipitous left ventricular dysfunction

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Video clip is available online.

We present a case of recurrent coronary vasospasm with subsequent hemodynamic collapse that occurred during epicardial cryoablation for refractory premature ventricular contractions (PVCs). The resulting hemodynamic changes and left ventricular dysfunction were successfully treated, leading to a favorable perioperative outcome.

CLINICAL SUMMARY

A 63-year-old man with a history of coronary artery disease and remote myocardial infarction presented with 1

year of palpitations, dyspnea on exertion, and multiple syncope. Holter monitoring demonstrated frequent PVCs. Preoperative transesophageal echocardiography (TEE) showed normal valvular function, preserved ejection fraction, and left ventricular hypertrophy without wall motion abnormalities. Although 2 separate endocardial ablation procedures temporarily terminated the premature beats, PVCs persisted and medical management ultimately failed, which prompted off-pump epicardial cryoablation of the left ventricular myocardium. Both radial arterial line and internal jugular triple lumen catheters were placed followed by a standard intravenous induction and maintenance anesthesia using isoflurane, and a TEE probe was placed for intraoperative monitoring. An isoproterenol infusion was used to augment the PVCs until a regular frequency was established. Electrophysiology studies showed that the origin of the PVCs was located on the anterobasal left ventricular epicardial surface near the bifurcation of the left main coronary artery (Figure 1). The area of interest was close to the origin of the anterior cardiac vein from the great cardiac vein. A full sternotomy was performed, the pericardium was opened, and dissection to the area of interest was uneventful. Mobilization and manipulation of the coronary arteries were performed to target the areas of interest. The line of ablations of the cryoprobe (Cardioblate CryoFlex; Medtronic ATS Medical Inc, Minneapolis, Minn) was alongside

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FIGURE 1. Site of epicardial cryoablation.

and underneath the anterior cardiac vein, underneath the left main, the bifurcation, adjacent to the left anterior descending artery, and underneath the proximal circumflex artery. Cryoablations from the basal myocardium almost to the base of the left atrial appendage apically were performed with multiple 5-minute cooling periods (to -150°C). During these cooling periods, we noted the immediate development of greater than 5-mm ST-segment elevations in the anterior and lateral leads, bradycardia, hypotension, and anterior and lateral wall hypokinesis on TEE imaging (left ventricular ejection fraction from 60% to 10%), which lasted 3 to 4 minutes (Video 1). Recovery to baseline was achieved with vasopressor support (intravenous boluses of epinephrine and phenylephrine), topical normal saline and papaverine sprayed directly onto the coronary arteries, discontinuation of the cooling cycle, and limited surgical manipulation. During the cooling periods, the PVCs would stop but subsequently returned during the re-warming phase. After multiple unsuccessful cryoablation cycles, the procedure was terminated. The patient recovered without perioperative complications, although an immediate postoperative angiogram was not done. Several months after the procedure, exertional chest pain developed in this patient with known coronary artery disease and previous myocardial infarction. An angiogram revealed narrowing in the left anterior descending at the site of cryoablation that was subsequently treated with a drug-eluting stent. A follow-up angiogram showed open coronary arteries.

DISCUSSION

Although off-pump surgical epicardial cryoablation is currently a relatively uncommon approach to arrhythmia management, this procedure provides an opportunity to control refractory arrhythmias that have failed more conservative management, albeit not without risks. One theoretic advantage of cryoenergy is the reduced risk of damage to adjacent structures, such as the coronary arteries, when compared with other ablation techniques.¹

In the described case, activation of the cryoprobe was preceded temporally to each hemodynamic event. Coronary vasospasm as a direct result of cryoablation and surgical manipulation adjacent to the coronary arteries led to near hemodynamic collapse. During each episode, the patient responded favorably and rapidly to vasopressor support, topical papaverine, and discontinuation of both the cooling and surgical manipulation. Acute hemodynamic collapse occurred predictably with every subsequent cryoablation. Anticipating this left ventricular dysfunction during subsequent cryoablation cycles allowed more rapid response and shortened episodes of hypotension, bradycardia, ST changes, and left ventricular dysfunction. Unfortunately, this patient's PVCs continued both intraoperatively and postoperatively despite repeated attempts at cryoablation. Nevertheless, knowledge of the relevant anatomy, assisted by real-time TEE imaging, and vigilance and preemptive treatment during subsequent cryoablations allowed a favorable hemodynamic recovery with resolution of echocardiographic and ST-segment abnormalities.

CONCLUSIONS

On the basis of our experience in this case and consistent with previously published guidelines,² continuous TEE monitoring should be strongly considered. Furthermore, we advocate for in-line, readily available, vasopressor support before ablation or surgical manipulation near the coronaries. Last, we found that open communication among the anesthesiologist, cardiac surgeon, electrophysiologist, and other members of the operating team allowed for optimal patient outcome.

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