ONE MINUTE CARDIOLOGY

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EVALUATION AND MANAGEMENT OF PVCs

PVCs are a common cause of palpitations and are also often detected incidentally. Although nearly everyone has some PVCs from time to time, people vary widely in frequency of PVCs and their sensitivity to them. Most PVCs are benign. They are more likely to be worrisome if present in the setting of underlying structural heart disease, a family history of sudden cardiac death, or prolonged QT interval.

Evaluation typically includes a 12 lead ECG and ambulatory rhythm monitoring. Holter monitoring for at least 24 hours is a critical step in assessing any patient with known or suspected PVCs as it can both quantify the total burden of ventricular ectopy and identify the presence of any related ventricular tachycardia. PVC burden is an important measurement; it is expressed as the % of heart beats that were ventricular extrasystoles during the monitoring period. Symptomatic patients with >10% PVC burden may be candidates for EP ablation. PVC burdens in the 15-20% range pose a higher risk of tachycardia-induced cardiomyopathy and heart failure, even if asymptomatic. Patients with >10% PVC burdens should be referred for cardiology consultation.

Other noninvasive testing should include a transthoracic echocardiogram. The finding of significant structural heart disease in conjunction with PVCs has prognostic implications and should prompt cardiology referral. Stress testing (combined with either echo or nuclear) is appropriate for patients who experience PVCs with exercise or for whom an evaluation for CAD is indicated. Cardiac CT, MRI and PET are not routine evaluations and are reserved for specific clinical situations including congenital heart disease, suspected cardiac sarcoidosis, infiltrative heart diseases, and cardiomyopathies such as hypertrophic CM and arrhythmogenic right ventricular dysplasia (ARVD).

PVCs without structural heart are frequently encountered in clinical practice. They tend to be catecholamine sensitive and are commonly felt during/immediately after exercise, in settings of emotional stress or fatigue, or in response to external stimulants such as caffeine. If symptomatic, they can be managed with beta blockers, verapamil or diltiazem. They are also highly curable by catheter ablation with >90% success rates.

Peri-infarct zones can be sources of PVCs but suppression of ectopy has not been associated with reduced mortality. Therefore treatment in the chronic CAD patient population is usually symptom-driven (unless very high PVC burden is present). When required, treatment usually involves beta blockers or class III antiarrhythmics such as sotalol or amiodarone. Patients with CAD and reduced EF, especially below 40%, may be candidates for ICD implantation and should be referred to cardiology for PVC management.

Most patients can be managed with medications. Beta blockers are the mainstay of medical therapy and are most strongly indicated in patients who have concomitant CAD, prior MI, or other cardiomyopathy. Calcium channel blockers may be especially appropriate in young patients without underlying structural heart disease. Catheter ablation can be curative but is typically reserved for drug-intolerant or medically refractory patients with a high PVC burden.

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