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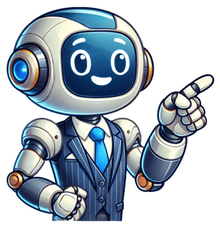

Premature atrial contractions, when isolated, are benign findings in most patients. While typically non-threatening, in association with underlying medical conditions, premature atrial contracts can foreshadow early mortality. This activity describes the evaluation and treatment of premature atrial contractions and highlights the role of the interprofessional team in improving care for patients with this condition.Objectives:Identify the etiology of premature atrial contractions medical conditions and emergencies.Review the evaluation of premature atrial contractions.Apply evidence-based management options for premature atrial contractions.Collaborate withthe interprofessional teamto improve patient care.Access free multiple choice questions on this topic.Premature atrial contractions (PACs) are contractions of the atria that are triggered by the atrial myocardium but have not originated from the sinoatrial node (SA node). PACs are also commonly referred to as atrial premature complexes (APCs), premature supraventricular complexes, premature supraventricular beat, and premature atrial beat. This phenomenon can be caused by an assortment of medical diseases, structural abnormalities, pharmaceuticals, and non-regulated compounds.Triggers forPACs can be caused by a myriad of reasons and are commonly idiopathic. Idiopathic PACs, in the absence of structural cardiac disease, frequently originate from the pulmonary veins.Identifiable causes of premature atrial contractions can be stratified into structural, chemical, or as a sequela of other conditions.Structural causes of PACs typically include coronary artery disease, hypertrophic cardiomyopathy, left atrial appendage aneurysms, left ventricular hypertrophy, valvular heart disease, septal defects, and congenital heart malformations.[1][2][3][4]At the biochemical level, sodium channel malformations and BMP2 mutations, a cause of pulmonary artery hypertension, may inadvertently predispose atrial arrhythmias.[5][6]Chemical-based causes for PACs include beta-agonists, digoxin, chemotherapeutic agents, tricyclic antidepressants, sympathomimetics amines, and monoamine oxidase inhibitors.[7][8][9]Beta-blockers have also been associated with a higher incidence of PACs.[4]A higher incidence of PACs is associated with many conditions and chronic states. Medical pathologies with associations for increased PACs include myocardial infarctions, congestive heart failure, hypertension, diabetes mellitus, a chronic obstructive pulmonary disorder, Coxsackie virus, and an overall higher CHA2DS2-VASC score.[4][10][11]Additional states such as anxiety, pregnancy, fatigue, and use of alcohol or tobacco have also predisposed for PACs.[12][13][14][15][16]Contrary to popular belief, caffeine has not been associated with an increased incidence of PACs.[17]PACsare prevalent amongst young and old patients, independent of many risk factors and previously known medical conditions.[18] Newborn infants, without associated risk factors, have been found to have variations typically considered pathologic.[19][20]Though still rare, elderly patients are more likely to present with abnormalities in their heart rhythm.[20] Nevertheless, an atypical rhythm is more likely to be found with continuous monitoring, such as a Holter monitor, than on a typical electrocardiogram strip.[21][22][23][24]This occurrence may be due in part to variations based on circadian rhythm.[25] As improvements in medical devices continue, amidst increased ubiquity of low-cost ambulatory and consumer options, the prevalence of arrhythmias may increase due to the detection of otherwise covert arrhythmias.[24][26][27][28][29]The pathophysiology for premature atrial contractions has not been well established. This is due in part to the relatively benign nature of the condition and the unnecessary invasiveness of electrophysiologic studies on humans to find a cause. Common theoretic mechanisms for this condition are based around abnormal automaticity of the cardiac myocytes, increased eliciting events from chemical or physical instigators, and reentry of a retrograde impulse. For these causes, structural heart damage or chemical use may be found during the history and physical examination.Genetic causes have also been studied, albeit in animals. Nevertheless, an LKB1 (cardiac-specific liver kinase B1) gene deletion has been associated with defects in ion channel formation of the atria. This has been shown to alter generation and conduction of action potentials, which predisposes the atria to remodeling, fibrosis, and ultimately atrial fibrillation.[30][31][32]Similar studies have also examined the formation of cytoskeletons, sarcomeres, desmosomes, and other ion channels for their role in automaticity.[33]History findings can be occult and nonspecific for the identification of PACs. Many patients are asymptomatic, and PACs are often discovered incidentally during the workup for another disease or on routine examinations. In patients who do experience symptoms, most commonly, a skipping sensation or palpitations are noted. The patient may experience shortness of breath or anxiety, as well. If the PACs are consistent, however, patients may experience signs and symptoms of heart failure.[34]Physical exam findings are also indiscriminate and lack sensitivity and specificity. The major contributor to this obscurity is the intermittent nature of PACs. When present, a clinician may palpate irregular pulses or visualize a canon a wave with jugular venous mapping. Auscultation may reveal early or additional heart sounds, as well as pauses in rhythm.The use of an electrocardiogram is standard for identifying electrical variations within the heart. PACs typically havenormal QRS complex and a normal, short, or longer PR interval than sinus rhythm. Sometimes, non conducted PACs occur in which there is no QRS complex following the PAC. PACs can be unifocal arising from one location (similar P waves in all PACs) or multifocal and arising from several locations (different P wave morphologies for PACs). The P wave of the PAC typically occurs earlier than the sinus P wave and has a different morphology and axis from the sinus P wave. It appears dissimilar from a standard sinus node generation, with variations in height, length, and shape of the P wave; furthermore, the P wave may be inverted or biphasic.Depending on the location of the generating focus, the PR interval can be shorter (