A Review of Cardiac Arrhythmias with Hospital-Based Observations on Presentation, Diagnosis, and Treatment

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ABSTRACT

Adult patients with brady arrhythmias and atrioventricular conduction abnormalities are increasingly being treated with conduction system pacing, which includes His bundle and left bundle branch pacing. This method aids in preventing the development of heart failure brought on by the mechanical and electrical dyssynchrony linked to long-term ventricular pacing. These results confirm that this method is feasible for younger pediatric patients in similar clinical settings. Children who have had congenital heart defect correction and need permanent cardiac pacing fall into a separate category. Due to non-physiologic ventricular contraction, these patients are more likely to develop pacing-induced cardiomyopathy, and there are currently few preventative and treatment alternatives. When complete atrioventricular block is present, techniques for minimizing ventricular pacing cannot be used, and biventricular pacing in pediatric patients is technically difficult. We describe a case of left bundle branch pacing in a child who had a permanent pacemaker implanted for postoperative full atrioventricular block and tetralogy of Fallot surgically repaired.

Keywords: conduction system pacing; His pack pacing; left pack branch pacing; pediatric pacing; natural heart complaint; postoperative atrioventricular block; complete heart block; endless trendsetter; pacing- convinced cardiomyopathy; tetralogy of Fallot; ventricular dyssynchrony; physiologic pacing.

INTRODUCTION

Heart arrhythmias are characterized by abnormalities in the pace, regularity, or activation sequence of the heart that differ from the typical sinus rhythm. [12,18] They may show no symptoms at all or exhibit palpitations, syncope, disorientation, hemodynamic compromise, or abrupt cardiac death. [15,16,18] Current reviews highlight that arrhythmias are not a single disease but rather a wide range of conditions, with

significant overlap between structural and electrical heart disease. [12,2,1]

ARRHYTHMIA CLASSES

Several complimentary frameworks that are helpful for clinical description and ECG-based diagnosis can be used to classify arrhythmias. [2,11]

1. Heart rate classification

- Bradyarrhythmias: rhythms marked by an abnormally low heart rate (usually less than 60 beats per minute in adults). [13,9]
- Tachyarrhythmias: rhythms linked to an abnormally high heart rate (usually greater than 100 beats per minute in individuals at rest). [14,12]

2. Anatomical location of origin classification

- Rhythms associated with sinus nodes, such as sinus tachycardia and bradycardia. [12]
- Atrial arrhythmias, including tachycardia, flutter, and fibrillation. [6,7]
- Atrioventricular nodal re-entrant tachycardia (AVNRT) and atrioventricular re-entrant tachycardia (AVRT) are examples of atrioventricular junctional and re-entrant supraventricular tachycardias (SVTs), which may be associated with accessory pathways (e.g., Wolff–Parkinson–White physiology). [11]
- Ventricular arrhythmias, which include malignant rhythms including ventricular tachycardia (VT) and ventricular fibrillation (VF) as well as premature ventricular complexes (PVCs). [1,22,23]

3. ECG feature classification (especially for tachyarrhythmias)

A useful ECG-based strategy prioritizes rhythm regularity and QRS duration: [11]

- The breadth of QRS

Wide-complex tachycardia: QRS ≥ 120 ms, usually of ventricular origin, however it can also be a sign of SVT with pre-excitation or aberrant conduction. [19]

- Consistency

Tachyarrhythmias are typically classified using a combined narrow/wide and regular/irregular paradigm, which contrasts regular and irregular rhythms. [11]

4. Electrophysiologic mechanism classification

Three classical processes are responsible for the majority of arrhythmias from a mechanistic

perspective: [2]

- Unusual automaticity [2]
- Activities that are triggered [2]
- Re-entry [2]

5. Categorization based on duration and temporal pattern

Episode structure can also be used to characterize rhythmic burden: [22,23]

- Isolated ectopy, such as PVCs or PACs [21,22]
- Short runs or couplets [23]
- Sustained versus nonsustained episodes (a crucial distinction in VT) [23,1]

PATHOPHYSIOLOGY

Cardiac arrhythmias arise when impulse formation is abnormal, impulse propagation is impaired, or both. [2] Broadly, the underlying mechanisms can be organized into disturbances of automaticity, afterdepolarization-mediated activity, and re-entrant conduction, with bradyarrhythmias often reflecting pacemaker failure or conduction block. [2,9,24]

- Triggered activity: afterdepolarizations occurring during or after repolarization can reach threshold and produce extra beats; this phenomenon is often facilitated by metabolic or electrolyte derangements, drug effects, ischemia, or heightened adrenergic tone. [2,24]
- Re-entry: conduction slowing and heterogeneity—commonly due to scar, fibrosis, or the presence of dual pathways—permit a self-sustaining circulating wavefront, producing abrupt-onset tachycardias. [2]
- Mechanisms of slow rhythms: bradyarrhythmias typically result from reduced sinus node impulse generation or impaired conduction through the AV node/His–Purkinje system, leading to intermittent or complete failure of atrioventricular transmission. [9]

THE EPIDEMIOLOGY OF ARRHYTHMIAS

In clinical practice, cardiac arrhythmias are very common. As people age and have underlying cardiovascular disease, [7,8,10] their frequency and overall burden increase significantly. The most common persistent arrhythmia, atrial fibrillation (AF), has a significant age-dependent rise in prevalence. [7,8,10] Supraventricular tachycardias (SVTs) are less prevalent than atrial fibrillation (AF) and are frequently seen in people who do not have major structural heart problems. [11] In the general population,

premature atrial and ventricular complexes (PACs/PVCs) are quite common and often benign in the absence of concomitant heart disease. [21,22] Ventricular tachycardia (VT) and ventricular fibrillation (VF), on the other hand, are less common but have significant therapeutic implications because to their correlation with sudden cardiac death, which typically occurs in individuals with severe structural or ischemic heart disease.[1,23]Arrhythmia-related morbidity is rising at the population level due to aging populations, better survival rates, and an increase in the number of people with chronic heart disease. [7,10]

RISK FACTORS AND ETIOLOGY

Cardiac substrate abnormalities and systemic or environmental stimuli often interact to cause the multifactorial development of cardiac arrhythmias. [2,7]

- Structural and ischemic heart disease: Electrical heterogeneity and scarring caused by myocardial ischemia or previous infarction might encourage atrial and ventricular arrhythmias. Similar effects include chamber dilatation, hypertrophy, fibrosis, and changed loading conditions in cardiomyopathies and valvular diseases. [1,2]
- Electrolyte imbalances: Abnormal serum levels of important ions, such as potassium, sodium, calcium, and magnesium, can change the excitability and repolarization of membranes, making them more vulnerable to tachyarrhythmias and bradyarrhythmias. [24]
- Thyroid dysfunction: Excess thyroid hormone frequently predisposes to tachyarrhythmias (particularly AF), whereas decreased thyroid function may lead to bradycardia and conduction delay. Both hyperthyroidism and hypothyroidism are linked to rhythm abnormalities. [25]
- Obstructive sleep apnea: Intrathoracic pressure fluctuations and intermittent hypoxemia might raise myocardial stress and sympathetic activation, resulting in a pro-arrhythmic environment. [26]

SYMPTOMS AND CLINICAL PRESENTATION

The kind of rhythm, ventricular rate, length of the episode, and underlying cardiac reserve all affect the symptom profile of cardiac arrhythmias. [15,18] Typical expressions consist of:

- Palpitations: the awareness of an

irregular heartbeat, which can be felt in the throat, neck, or chest and is frequently described as fluttering, fast pounding, or sporadic "skipped" beats. [15,18]

- Dizziness or lightheadedness: caused by brief decreases in cerebral perfusion, especially during severe bradycardia or fast tachyarrhythmias. [18]
- Dyspnea: shortness of breath during rest or exertion, which may be caused by higher filling pressures during arrhythmic episodes or reduced cardiac output. [18]
- Fatigue and widespread weakness: poor energy and decreased exercise tolerance, frequently accompanied with bradyarrhythmias, frequent ectopy, or persistent tachycardia. [18]
- Pain, tightness, or pressure in the chest that may accompany arrhythmias, particularly when myocardial ischemia or elevated oxygen demand is present. [18]

DIAGNOSTIC ANALYSIS

In order to determine reversible causes and underlying heart illness, [15,7] the evaluation of suspected arrhythmia starts with a thorough clinical assessment and is further refined utilizing ECG-based documentation and focused studies.

- Clinical background and evaluation at the bedside: Characterization of symptoms (e.g., palpitations, presyncope/syncope, chest discomfort), frequency and duration of episodes, triggering circumstances, and related disorders are important components. Together with a review of drugs, stimulants, and other possible triggers, a targeted examination—especially a pulse assessment for rate and irregularity—can offer quick hints. [15]
- 12-lead electrocardiography: The resting ECG, which offers details on rhythm mechanism, QRS shape, conduction intervals, and indications of structural or ischemic disease, is still the primary diagnostic test for rhythm and conduction disorders. [15]
- Ambulatory rhythm monitoring: Holter monitoring (usually 24–48

hours) or longer-term event/patch monitoring (days to weeks) are used to connect symptoms with rhythm in cases with intermittent symptoms or paroxysmal arrhythmias that are not recorded on a conventional ECG. [17,27,28]

TREATMENT AND MANAGEMENT

Treatment for cardiac arrhythmias is tailored based on the severity of symptoms, the influence on hemodynamics, the mechanism of the arrhythmia, and the underlying cardiac substrate. The focus is on treating reversible contributors and avoiding recurrence or unfavorable consequences. [7,11,1,9]

- Take care of reversible triggers and causes: Correcting triggering variables such electrolyte abnormalities, treating thyroid dysfunction, optimizing ischemic or structural heart disease, and avoiding pro-arrhythmic exposures (such as excessive alcohol or caffeine consumption, nicotine use, and stimulant usage) are frequently the first steps in management. To find substances that could cause or exacerbate arrhythmias, a thorough examination of both prescription and over-the-counter drugs is crucial. [7,24,25,26]
- Pharmacologic therapy: Depending on the type of arrhythmia, comorbidities, and safety profile, antiarrhythmic management may entail medications targeted at rate control (lowering ventricular response) and/or rhythm control (restoring and maintaining sinus rhythm). [7,11]
- Electrical cardioversion: For certain tachyarrhythmias, synchronized direct-current cardioversion is employed, especially when symptoms, hemodynamic compromise, or persistently irregular rhythms call for a quick restoration of sinus rhythm. [7,6,11]
- Catheter ablation: By removing or isolating the arrhythmogenic center or re-entrant circuit, catheter-based

ablation provides a definitive treatment for a number of supraventricular tachycardias and certain atrial or ventricular arrhythmias. [11,7]

OBSERVATIONS FROM HOSPITAL-BASED SURVEYS

The majority of episodic, nonspecific symptoms, most frequently palpitations, dizziness or lightheadedness, dyspnea, chest tightness, and sporadic syncope, are regularly reported in hospital-based surveys of patients assessed for cardiac arrhythmias. While paroxysmal episodes are more commonly recorded with ambulatory monitoring (Holter or event/loop recorders), arrhythmias are often discovered by accident on a routine 12-lead ECG. While ventricular tachyarrhythmias typically concentrate in higher-acuity settings like emergency rooms and intensive/coronary care units, atrial fibrillation and supraventricular tachycardias are frequently seen in outpatient services. [1]

Pharmacologic rate and/or rhythm control (e.g., beta-blockers, non-dihydropyridine calcium channel blockers, and specific antiarrhythmic medicines) is a common therapeutic strategy documented in these surveys. Anticoagulation is commonly used to lower thromboembolic risk in eligible patients, especially those with atrial fibrillation. A portion of patients need to be escalated to electrical cardioversion, catheter ablation, or device-based therapy, such as implanted cardioverter-defibrillators for people at increased risk of malignant ventricular arrhythmias or permanent pacemakers for clinically significant bradyarrhythmias or atrioventricular block. [7,6,11]

The practical significance of early detection of red-flag symptoms, adherence to recommended therapy, avoidance of frequent triggers (such as excessive caffeine or alcohol and pro-arrhythmic medications/substances), and organized follow-up is highlighted by patient-facing findings from these settings. These actions are frequently highlighted as essential elements of preventative methods meant to lessen consequences like stroke, heart failure progression, and abrupt cardiac events. [7,1]

CONCLUSION

A common and clinically varied colle ction of rhythm abnormalities, cardia c arrhythmias range from benign, self

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limited episodes to malignant disorde rs linked to significant morbidity and mortality.

In order to reduce unfavorable outco mes like thromboembolism and strok e, heart failure progression, and sudd en cardiac death, prompt recognition —
based on meticulous clinical assessm ent and confirmation with ECG-based testing, including ambulatory monitoring where appropriate— remains crucial.

Individualized and all-encompassing management that inclu des lifestyle and trigger adjustment, r eversible contributor repair, and judic ious use of pharmacologic therapy fo r rhythm and/or rate control is most e ffective. Procedural and device-based therapies, including as electric al cardioversion, catheter ablation, pe rmanent pacing, and implanted defibr illator therapy, offer significant choic es for risk reduction and symptom co ntrol in certain individuals. Long-term improvements in the safety and durability of arrhythmia treatment ar e anticipated as a result of ongoing a dvancements in mechanistic understa nding, monitoring techniques, and fo cused therapeutic approaches. [7,6,11,1,9]

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