Updated Approaches to Cardiac Electrical Stimulation and Pacing in Pediatrics

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Abstract: Cardiac electrical stimulation is a rarely used but required skill for pediatric emergency physicians. Children who are in cardiac arrest or who demonstrate evidence of hypoperfusion because of cardiac reasons require rapid diagnosis and intervention to minimize patient morbidity and mortality. Both hospital- and community-based personnel must have sufficient access to, and knowledge of, appropriate equipment to provide potentially lifesaving defibrillation, cardioversion, or cardiac pacing. In this review, we will discuss the primary clinical indications for cardioelectrical stimulation in pediatric patients, including the use of automated external defibrillators, internal defibrillators, and pacemakers. We discuss the types of devices that are currently available, emergency management of internal defibrillation and pacemaker devices, and the role of advocacy in improving delivery of emergency cardiovascular care of pediatric patients in the community.

Key Words: cardiac arrest, cardioversion, defibrillation, pacemaker

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TARGET AUDIENCE

This continuing medical education activity is intended for physicians, physician assistants, nurse practitioners, and emergency medical services providers who care for pediatric patients.

LEARNING OBJECTIVES

After participating in this activity, the reader will be better able to:

1. Identify the indications for defibrillation, cardioversion, and cardiac pacing in children.
2. Summarize the types of devices available for both external and internal defibrillation and pacing.
3. Describe the role of advocacy in improving delivery of emergency cardiovascular care of pediatric patients in the community setting.

Cardiac arrest is a leading cause of death worldwide, with an estimated 550,000 combined out-of-hospital and in-hospital arrests occurring annually in the United States alone.[1] Pediatric cardiac arrests occur 12,000 times per year in the United States, with approximately equal distribution of in-hospital cardiac arrests (IHCA) and out-of-hospital cardiac arrests (OHCA).[2,3] In-hospital cardiac arrest survival has improved substantially from 2001 to 2013, with rates of return of spontaneous circulation (ROSC) improving from 39% to 77%, and survival to hospital discharge improving from 24% to 43%.[4,5] In contrast, survival from OHCA remains poor, estimated between 8.3% and 15.8%.[4,6] Many factors contribute to the higher rate of survival from IHCA, including the emphasis on early recognition, high-quality cardiopulmonary resuscitation (CPR), early defibrillation when appropriate, and advances in postcardiac arrest care. In contrast, the majority of patients with OHCA present initially with asystole or pulseless electrical activity, commonly precipitated by noncardiac causes including respiratory arrest, drowning, and asphyxia.[7–9] Only 10% to 15% of pediatric patients with OHCA initially present with a "shockable" rhythm, such as pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF).[10,11] Though survival to discharge after pediatric cardiac arrest has been noted to be poor overall, survival after VF or VT arrest has been reported to be as high as 89%.[12–14] Ventricular tachycardia/ventricular fibrillation is a more common initial presenting rhythm in child and adolescent patients than infants and is a contributing factor to improved survival in noninfant pediatric patients with IHCA and OHCA.[9,15]

In this review, we will discuss the primary clinical indications for cardioelectrical stimulation in pediatric patients, including the use of defibrillation, cardioversion, and pacemakers. We will discuss the types of devices that are currently available, the emergency management of internal defibrillation and pacemaker devices, and the role of advocacy in improving delivery of emergency cardiovascular care of pediatric patients in the community setting.

Case Scenario 1

A 15-year-old, 45-kg boy was brought to the emergency department (ED) with altered mental status. His parents found him alone in his room approximately 30 minutes after he returned home from school. He was noted to be confused, and multiple empty pill bottles previously containing diphenhydramine, fluoxetine, and ibuprofen were found near his bed. Upon emergency medical services arrival, he was noted to have a temperature of 39.2°C, blood pressure of 145/90 mm Hg, heart rate 130/min, respiratory rate of 20/min, and oxygen saturation of 96% in room air. He was agitated and appeared to be having auditory and visual hallucinations. Gag reflex was intact, and no respiratory distress was present. Intravenous (IV) access was obtained, and lorazepam 4 mg was administered en-route to the ED. Upon arrival to the ED, he immediately lost consciousness. The patient was noted to be pulseless, and chest compressions were started immediately while cardiopulmonary monitors and defibrillation pads were applied. An electrocardiogram (ECG) demonstrated a wide complex tachycardia. Defibrillation was performed at 2 J/kg with immediate conversion to sinus rhythm and ROSC.

Pathophysiology of Sudden Cardiac Arrest/Arrhythmia in Pediatric Patients

For pediatric patients suffering cardiac arrest secondary to a ventricular dysrhythmia, rapid defibrillation is a key factor to both improved survival and to a favorable neurologic outcome.[16,17] Recent guidelines from both the American Heart Association and the

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TABLE 1. Etiologies of Sudden Cardiac Death in Children

Congenital/ Structural	Tetralogy of Fallot
	Transposition of the Great Arteries
	Epstein's Anomaly
	Cardiac Tumors (Rhabdomyosarcoma)
	Left Heart Obstructive Lesions (Hypertrophic Obstructive Cardiomyopathy, Left Ventricle Noncompaction)
	Coronary Artery Anomalies
	Valvular Disease
	Collagen Vascular Disorders
Electrical	Long QT syndrome
	Catecholaminergic polyventricular tachycardia
	Brugada
Cardiomyopathy	Hypertrophic
	Dilated (familial or acquired)
	Arrhythmic right ventricular cardiomyopathy
	Infiltrative/restrictive (Chagas, sarcoidosis, amyloidosis, hemochromatosis, scarring)
	Myocarditis (viral, bacterial, rickettsial, fungal, toxin-mediated)
Metabolic/Toxin induced	Heavy metal exposure
	Anthracycline
	Radiation
	Carnitine deficiency
	Neuromuscular disorder
	Mitochondrial diseases
	Severe anemia
	Thyrotoxicosis
	Pompe disease

European Resuscitation Council recommend defibrillation as soon as possible, ideally within 2 minutes of recognition of cardiac arrest with a shockable rhythm.[4,18,19] However, significant barriers remain to public access defibrillation and timely recognition of cardiac arrest, both in the prehospital and in-hospital setting.

The majority of pediatric patients who suffer cardiac arrest with an initial shockable rhythm do so because of a cardiomyopathy, a congenital/structural defect, primary electrical conduction problem, or a metabolic/drug induced arrhythmia (Table 1).[20,21] Sustained VT/VF in the pediatric population is rare, estimated between 2 and 8 per 100,000 patients.[22,23] However, once a patient has suffered a cardiac arrest secondary to a dysrhythmia with shockable rhythm, defibrillation with an automatic external defibrillator (AED) or implantable cardioverter defibrillators (ICD) is often required (Fig. 1). Preventative strategies for cardiac arrests, such as use of beta blockers and implantable defibrillators, can be used to decrease the risk of sudden cardiac death in pediatric patients with known inherited arrhythmias who have not yet suffered a cardiac arrest. Cardiac ablations have also reduced the incidence of cardiac arrest in children in whom the location of the abnormal cardiac electrical signal can be located and accessed. The exact mechanism whereby an electric shock terminates VF/VT is not yet known, and there is significant debate about how cardiac tissue responds to electric shock.[24]

Methods of Delivery of Electricity

Delivery of electricity to the heart occurs using two primary mechanisms: defibrillation and cardioversion. Defibrillation is the therapeutic administration of electricity to depolarize the myocardium so that coordinated contractions can occur. It is used to terminate a nonperfusing rhythm, such as pulseless VF/VT. Cardioversion is the application of electricity to terminate a perfusing rhythm to allow normal sinus rhythm to restart. By this definition, cardioversion is a less urgent procedure compared with defibrillation, although the patient requiring cardioversion may be hypotensive or hemodynamically unstable, rather than in cardiac arrest.[25]

Defibrillation

Defibrillation in pediatric patients is indicated for pulseless VF or VT. The first documented successful resuscitation using defibrillation for VF in a human was performed in 1947 on a 14-year-old boy who was ultimately discharged home neurologically intact after the resuscitation.[26] Electricity delivered via defibrillation does not account for phase of cardiac rhythm, and it is not synchronized with the patient's intrinsic QRS complex, when present. It is not indicated for asystole, pulseless electrical activity, on in a patient with a pulse. However, when applied appropriately, defibrillation can be lifesaving. Defibrillation in patients who have a witnessed cardiac arrest with an initial shockable rhythm have consistently improved rates of ROSC with favorable neurologic outcomes.[20,27,28] Ventricular fibrillation is the initial recorded rhythm in 5% to 24% of pediatric cardiac arrests, and it is much more common in children with congenital heart defects.[9,20,28–32] The 2015 American Heart Association (AHA) guidelines suggest an initial energy dose of 2 to 4 J/kg, and in cases of refractory VF, increased energy levels may be considered up to 10 J/kg.[33] A recent systematic review revealed that ROSC is frequently achieved when the initial presenting rhythm is pulseless VF/VT with energy dosing ranging from 2 to 7 J/kg, with the higher energy levels required more frequently in infants.[29] Patients who receive defibrillation for either IHCA or OHCA do so via an AED, an ICD or a wearable cardioverter defibrillator (WCD).

Automated/Manual External Defibrillators

The first successful use of an alternating-current external defibrillator on a human was reported in 1956, and the first portable defibrillator in the United States was commercially available in 1968 (LifePak 33; Physio Control, Inc., Redmond, WA).[34,35] Automatic external defibrillators have the ability to independently analyze the heart rhythm to determine whether a shock should be applied. If the device determines a shock is required, the battery charges the internal capacitor to deliver the shock through the

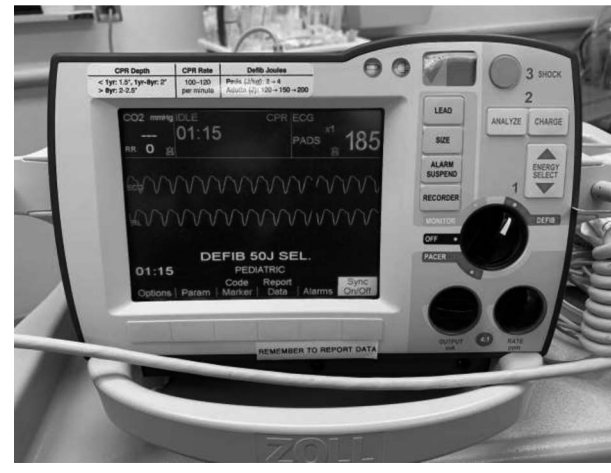

FIGURE 1. Defibrillator showing ventricular tachycardia.

chest. Electrical current flows from the cathode (negative electrode) to the anode (positive electrode), and each shock moves in an opposite polarity between the electrode pads.[35] The shock is then delivered through pads that are applied the bare chest of a patient in cardiac arrest. Modern AEDs use a biphasic waveform to deliver a shock at a lower voltage than the previously common monophasic AEDs. As a result, modern biphasic AEDs have a much lower risk for skin and soft tissue injuries, including burns, than did earlier versions of the device. Additionally, most modern AEDs provide point-of-care CPR performance feedback for compression depth, rate, and pauses in compressions. CPR feedback devices have been consistently shown to improve CPR performance; it is recommended that AEDs with the ability to provide CPR quality feedback should be universally adopted by health care facilities.[36–42] AEDs manufactured for use in the hospital setting have visible ECG tracings that hospital personnel use to identify shockable rhythms when the AED is placed in manual mode. Most AEDs have multiple utilities including defibrillation, cardioversion, pacing, and CPR feedback. Automatic external defibrillators approved for use in hospital and community settings are listed in Table 2.

Implantable Cardioverter Defibrillator

The primary indication for ICD placement is for prevention of cardiac arrest. Between 2010 and 2016, there were 3461 ICD placed in pediatric patients in the United States. Of these, 39% were placed for nonischemic dilated cardiomyopathy, 17% were placed for hypertrophic cardiomyopathy, and 13% were placed for long QT syndrome. Pediatric patients with ICDs are more likely to have structural heart disease, hypertrophic cardiomyopathy, and channelopathies when compared with adult patients.[43]

Implantable cardioverter defibrillator placement is rare in children, and indications for placement remain controversial.[44,45] Complications of ICD placement are higher in the pediatric population than in adults. This is likely secondary to the growth and increased activity level in children.[46] According to the National Cardiovascular Data Registry, pediatric patients are more likely to have an out-of-hospital complication related to ICD placement if they have a lower weight, Ebstein anomaly, worse New York Heart Association severity class, absence of beta-blocker therapy, or presence of a dual chamber ICD. However, the overall incidence of in-hospital complications is not different between adult and pediatric patients at 2.3%.[43] The most common complication is inappropriate shocks, which have a reported incidence as high as 17% to 50%, most often triggered by sinus tachycardia, supraventricular tachycardia, and lead failure.[46–48] Mechanical complications from ICD placement include lead breaks, burns, and infections, all of which have been noted to be higher in pediatric patients than in adults.[46]

Wearable Cardioverter Defibrillators

Some pediatric patients at risk for sudden cardiac death are not considered suitable candidates for ICD placement. This is usually due to contraindication of immediate placement of ICD (eg, infected device or awaiting cardiac transplantation), or in patients who are at risk but may improve over time, obviating the need for

TABLE 2. AEDs Approved by the United States Food and Drug Administration

Manufacturer	Device Name	AED Type	Pediatric Pads Available	Provides CPR Feedback (Rate and Depth)	Provides Visible EKG
Cardiac Science Corporation	Powerheart G3 Plus	Public Access	Yes	Yes	Yes
	Powerheart G5	Public Access	Yes	Yes	No
	Powerheart G3 Pro	Professional Use	Yes	No	Yes
Defibtech, LLC	Lifeline AED	Public Access	Yes	No	No
	Lifeline View AED	Public Access	Yes	No	No
	Lifeline Auto	Public Access	Yes	No	No
	Lifeline ECG AED	Public Access	Yes	No	Yes
HeartSine Technologies, LLC	HeartSine SAM 350P	Public Access	Yes	No	No
	HeartSine SAM 360P	Public Access	Yes	No	No
	HeartSine SAM 450P	Public Access	Yes	Yes	No
Phillips Medical Systems	HeartStart FR3	Public Access	Yes	No	No
	HeartStart OnSite	Public Access	Yes	No	No
	HeartStart FRx	Public Access	Yes	No	No
Physio-Control, Inc	LIFEPAK CR Plus Defibrillator	Public Access	Yes	No	no
	LIFEPAK CR2 Defibrillator	Public Access	Yes	No	No
	LIFEPAK 15 Monitor/ Defibrillator	Professional Use	Yes	Yes	Yes
	LIFEPAK 20E Defibrillator/ Monitor	Professional Use	Yes	Yes	Yes
	LIFEPAK 1000 Defibrillator	Professional Use	Yes	No	Yes
Zoll Medical Corporation	AED Plus and Fully Automatic AED Plus	Public Access	Yes	Yes	No
	X Series Defibrillator	Professional Use	Yes	Yes	Yes
	R Series Defibrillator	Professional Use	Yes	Yes	Yes
	AED Pro Defibrillator	Professional Use	Yes	Yes	Yes
	AED 3 BLS Defibrillator	Professional Use	Yes	Yes	No

long-term ICD therapy. For the latter, a WCD can be considered. The Food and Drug Administration has approved WCD use in children weighing at least 41 pounds and with a chest size of at least 26 in.[49] There were 455 inpatient and outpatient WCDs placed in the United States from 2009 to 2016 on pediatric patients ranging from 3 to 17 years. These patients used the WCD for a median of 33 days, and approximately 8% of the patients sustained an arrhythmia that was aborted while wearing the device.[50]

Cardioversion

The primary indication for cardioversion in pediatric patients is for supraventricular tachycardia (SVT) with cardiovascular instability and/or altered mental status.[4] Because pediatric patients often tolerate significant tachycardia without hemodynamic compromise, it is important to distinguish between sinus tachycardia, often associated with fever, dehydration, or sepsis versus supraventricular tachycardia. Supraventricular tachycardia more consistently has ventricular rates greater than 220 in infants, and greater than 180 in children older than 1 year with minimal variability. Supraventricular tachycardia frequently responds to nonelectrical treatment, including vagal maneuvers or pharmacologic therapy. Candidates for cardioversion are hemodynamically unstable with evidence of poor perfusion, such as heart failure (pulmonary edema, peripheral edema, or other indications of volume overload), cerebral hypoperfusion (altered mental status), or end-organ damage (kidney, liver, or intestinal injury). Direct-current cardioversion can promptly restore hemodynamic stability and prevent myocardial ischemia in children with cardiac tachyarrhymias.

In children with reentrant ventricular and supraventricular tachyarrhythmia with distinct R or S waves, synchronized shocks can restore sinus rhythm by depolarizing the excitable myocardium to terminate reentrant pathways. With cardioversion, the shocks are synchronized with the QRS complex to avoid the risk of triggering ventricular fibrillation if energy is delivered during the myocardial repolarization.[51] Cardioversion is painful for the patient and pretreatment sedation should be used when possible. However, electrical cardioversion in a hemodynamically unstable patient should not be delayed for sedative administration.

When performing cardioversion, the conducting pads should be placed according to the manual provided by the manufacturer of the device. Recommended electrical dosing for cardioversion in pediatric patients is 0.5 to 1.0 J/kg for both monophasic and biphasic AEDs, though exact dosages provided varies between the various devices available. If the device does not provide the desired dose of electricity, the next highest available dose should be chosen. For instance, if a 13-kg patient requires synched cardioversion, and the device only provides electricity at 10 or 15 J, then 15 J should be selected. If cardioverting patients with implanted cardiac pacemakers and/or defibrillators, it is important to ensure that the vector of delivered energy is perpendicular to the axis formed by the pulse generator, and that defibrillator pads avoid interference with, or damage to, the implanted device. All pacemakers and ICDs have shielding from the effects of cardioversion, but device interrogation after the procedure is prudent.[52]

Case Scenario 2

A 10-year-old boy presented to the ED with fever and malaise. The patient had a history of coarctation of aorta repaired in infancy, with subsequent development of sick sinus syndrome. He underwent placement of transvenous Atrium Inhibit pacemaker system 3 months ago. Upon arrival, he was noted to be awake and alert with temperature of 39°C, heart rate of 120/min, blood pressure of 80/30 mm Hg and saturation of 91% on room air with respiratory rate of 32/min. Physical examination revealed a pale, diaphoretic child with nontender left infraclavicular pacemaker site without redness or purulent drainage. Intravenous access was established. Blood cultures were obtained, ECG showed atrial paced rhythm. Chest radiograph was obtained and showed intact atrial lead. Echocardiogram showed normal left ventricular function and no valvular vegetations. The patient was given IV fluids and antibiotics with improvement in blood pressure. A cardiologist was consulted, and the patient was admitted to the hospital. Subsequently, blood cultures grew methicillin susceptible *Staphylococcus aureus*.

Pacemaker Background

Pacemaker technology has improved dramatically since the first pediatric pacemaker was implanted in 1956.[53] Current pacemakers can be placed percutaneously, have longer battery lives and have diverse programming capabilities when compared with early pacemakers. Pediatric patients comprise less than 1% of the recipients of pacemakers, but the demand for implantable pacemakers in children continues to increase.[54] It is important for pediatric emergency care providers to be familiar with pacemakers and their associated implications for patient care.

Pacemaker Systems

Pacemakers consist of a pulse generator and electrode leads. A pulse generator creates the impulse, whereas the electrode leads deliver the impulse to the myocardium.[55] Typical pulse generators have a lithium iodide battery with an 8- to 12-year lifespan.[56] Leads are typically placed in the right or left ventricle (single chamber pacemaker), right atrium and right ventricle (dual chamber pacemaker) or right atrium, right ventricle, and left ventricle (biventricular pacemaker).[57] Epicardial systems are generally used in younger children and those with complex cardiac anatomy. In epicardial pacemakers, the pulse generator is placed in the abdomen and leads are attached surgically to the epicardium (Fig. 2). Endocardial (transvenous) systems are preferred in older children, though they have also been successfully implanted in infants.[58] In transvenous pacemaker systems, the pulse generator is placed in the axillary region or anterior chest wall on either left or right side (Fig. 2). Endocardial leads are placed percutaneously via axillary, subclavian, or cephalic veins.[59,60] Leadless cardiac pacing systems have been available since 2016, but these devices are still undergoing long-term safety and efficacy evaluation.[61]

Pacemaker Indications

Indications for pediatric pacemakers are complex and summarized in the 2012 American College of Cardiology Foundation, American Heart Association and the Heart Rhythm Society joint practice guideline (Fig. 3). Common indications for pacemaker placement include sinus node dysfunction, acquired or congenital heart block, long QT syndrome, and hypertrophic cardiomyopathy.[62]

Pacemaker Nomenclature

Pacemaker types are described by a 5-position code developed by the North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group (Table 3).[63] The first 3 positions are the most commonly used descriptors and are listed in Table 3. Patients with pacemakers are given identification cards which list their pacemaker type and baseline settings. Emergency care providers need to have a basic understanding of pacemaker type and nomenclature to communicate with consultants and to appropriately interpret EKGs and chest radiographs of paced patients.

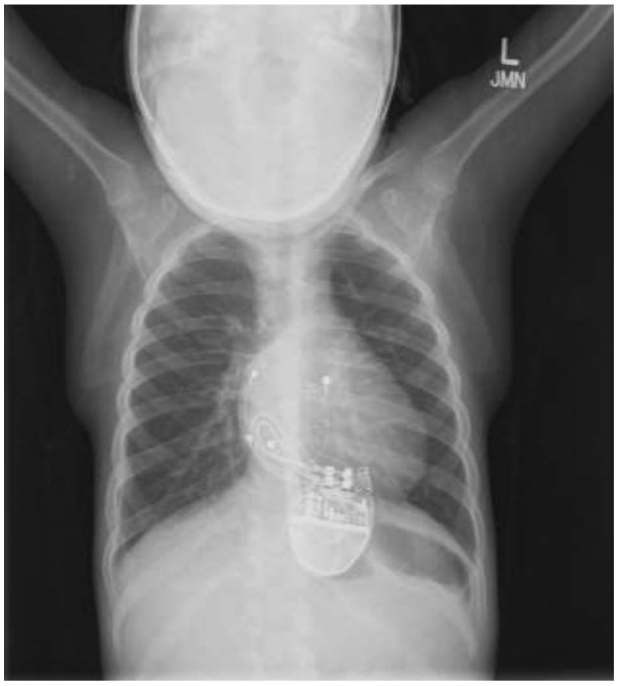

FIGURE 2. Epicardial pacemaker.

Pacemaker Complications

Complications From Pacemaker Device Malfunction

Complications involving pacemaker devices include (a) problems with pulse generator function (battery failure, damage, loose connection to the leads, rotation of pulse generator (also known as Twiddler's syndrome); and (b) problems with lead function including fracture, detachment, breakdown in insulative coating, myocardial fibrosis. (Table 4) In the emergency setting, cause of the malfunction is not as important as the resulting malfunction itself. The most common pacemaker malfunctions include: failure to pace, failure to capture, and failure to sense.[64]

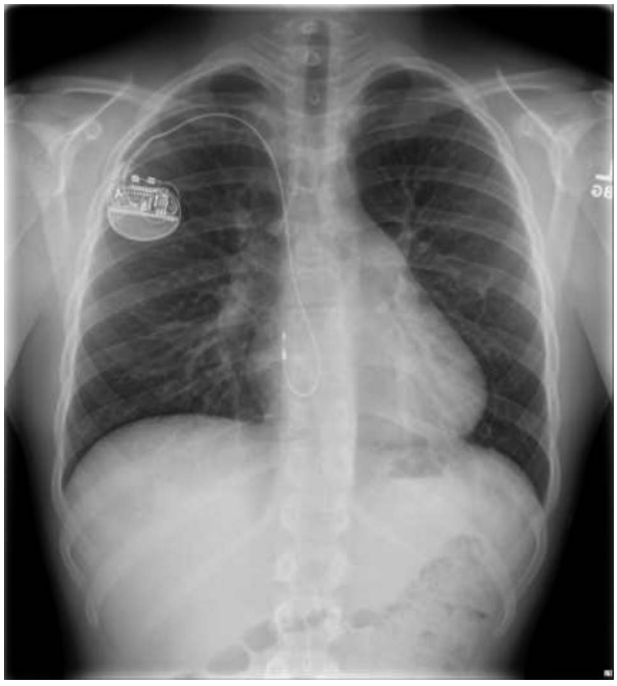

FIGURE 3. Right infraclavicular endocardial pacemaker.

TABLE 3. Nomenclature of Common Cardiac Pacemakers

Position	I	II	III
Description	Chamber paced	Chamber sensed	Response to sensing
Options	A (atrium)	A (atrium)	I (inhibit)
	V (Ventricle)	V (Ventricle)	T (trigger)
	D (A + V)	D (A + V)	D (I + T)
		O (none)	O (none)

a. *Failure to pace:* Failure to pace occurs when a pacemaker does not trigger the myocardium to depolarize. ECG tracings do not show pacer spikes, or any pacemaker-generated QRS complexes. Instead, they show the native rhythm of the patient. Causes for failure to pace include: lead malfunction, battery, and trauma to the device itself.[65]
b. *Failure to capture:* Failure to capture occurs when a pacemaker impulse is generated but is unable to depolarize myocardial tissue. Electrocardiograms demonstrate the native rhythm with noncaptured pacemaker generated spikes.[55] Causes of failure to capture include: lead malfunction, cardiac scarring, battery problems, electrolyte abnormalities (most commonly hyperkalemia), or supratherapeutic drug levels leading to changes in depolarization threshold.[65,66]
c. *Failure to sense:* Failure to sense can produce oversensing or undersensing of the paced rhythm. Oversensing occurs when the pacemaker inappropriately interprets a native cardiac rate as being too fast and pacing is inhibited. Electrocardiogram shows absent pacer spikes. If the pacemaker is inappropriately inhibited, a patient may become hemodynamically unstable due to an inadequately perfusing rhythm.[3] If pacemaker oversensing is suspected, a magnet can be placed over the pacemaker pocket, which changes the pacemaker to prefixed, asynchronous pacing mode and eliminates the need for sensing. Removal of the magnet will return the pacemaker to programmed mode.[67] In contrast, undersensing occurs when a pacemaker incorrectly interprets native cardiac activity as too slow and inappropriately sends an impulse to the myocardium to stimulate ventricular contraction. In this case, an ECG may show inappropriately placed pacer spikes with or without capture. Patients with an undersensing pacemaker may have palpitations. Causes of pacemaker sensor abnormalities include lead malfunction, cardiac fibrosis or scarring, battery failure, and electrolyte abnormalities.[65,68,69]

TABLE 4. Types of Pacemaker Device Malfunction

Location of Malfunction	Type of Malfunction
Pulse generator	Battery failure
	Damage
	Loose connection to the leads
	Rotation (Twiddler syndrome)
Leads	Fracture
	Detachment
	Insulation breakdown
	Myocardial fibrosis

Complications Resulting From Pacemaker Placement

a. *Pneumothorax:* Pneumothorax or hemothorax is an early complication of lead placement and occurs in about 2.2% of pediatric patients.[54] Patients typically present within the first 48 hours after lead placement and may have respiratory distress, chest pain, hypoxia, and subcutaneous emphysema. A chest radiograph usually reveals the diagnosis, and in severe cases, chest tube placement maybe required.[70,71]
b. *Infection:* Generator pocket infections may occur early or late after pacemaker implantation and are often associated with lead or battery replacement, dental care or with other medical procedures. Patients may have purulent drainage, induration or fluctuance at the pacemaker site as well as fever or other signs of sepsis. Common bacterial etiologies of pacemaker infection include commensal skin flora such as *Staphylococcus aureus* and *Staphylococcus epidermidis.* Treatment of pacemaker infection includes IV antibiotics; in many cases surgical removal of the pacemaker is indicated.[70] Pacemaker lead infections tend to occur later after implantation. Patients may present with signs of sepsis, endocarditis and septic pulmonary emboli. Staphylococcal species are often the causative bacterial. Appropriate IV antibiotics should be initiated rapidly. Definitive treatment includes lead removal.[70,72,73]
c. *Pericardial Effusion:* Pericardial effusion from perforation of the myocardium is a rare complication of pacemaker placement, occurring in 0.06% of patients.[74] While most patients present in the first 24 hours after implantation, delayed presentations have been reported.[75] Patients may be asymptomatic or present with signs of cardiac tamponade including chest pain, dyspnea and hypotension.[76] Chest radiography should be obtained to visualize pacemaker leads. Echocardiography is useful to visualize pericardial effusion.[76] Emergency pericardiocentesis should be considered in those patients presenting in extremis.[70]
d. *Thrombosis:* Venous thrombosis and occlusion, sometimes leading to superior vena cava (SVC) syndrome and pulmonary embolism, can be an early or late complication of pacemaker placement. Patients with endocardial pacemaker systems are at particularly high risk of development of thromboses. Vascular occlusion is reported in about 7%–13% of pediatric patients with transvenous leads.[77,78] Typical symptoms of acute thrombosis include extremity, facial and neck swelling as well as dyspnea and hypotension. Ultrasonography maybe helpful in diagnosis. Treatment of pacemaker related thrombosis most commonly includes anticoagulation.[79]
e. *Pacemaker syndrome:* Pacemaker syndrome is a constellation of symptoms including fatigue, confusion, headache, shortness of breath, chest pain and palpitations. It is thought to be related to loss of atrioventricular synchrony. This is a diagnosis of exclusion in patients with pacemakers; other causes of these symptoms should be fully evaluated before attribution to pacemaker systems. Supportive care is indicated in the acute care setting.[22,70]

Emergency Evaluation and Management of Patients With Pacemakers

Pacemaker malfunction may lead to hemodynamic instability. A thorough history and physical examination, as well as a complete set of vital signs are indicated in all patients. Common symptoms of pacemaker malfunction include fatigue, presyncope/syncope, dizziness, palpitations, and shortness of breath.[67] The pacemaker site location should be examined for signs of infection, trauma and migration. Additional physical examination signs such as jugular vein distention and new heart murmurs or rubs should be noted.[80] Pacemaker type and setting information should be reviewed, when available. Pacemaker manufacturer symbol maybe visible on a chest radiograph.[81] Diagnostic evaluation should include an ECG to evaluate the cardiac rhythm and pacer activity (Fig. 4). Chest radiography should be obtained and compared to previous films, if available, to evaluate for lead fracture, displacement, or rotation of pulse generator.[82] Patients also should be assessed for metabolic abnormalities such acidosis, hypokalemia, hypoxia, and hypothyroidism because these may alter pacemaker function by changing depolarization threshold[83] Transcutaneous pacing pads should be placed on the patient if pacemaker malfunction is suspected. In rare cases, transvenous pacing might be necessary. Urgent cardiology consultation is required in all cases.[81]

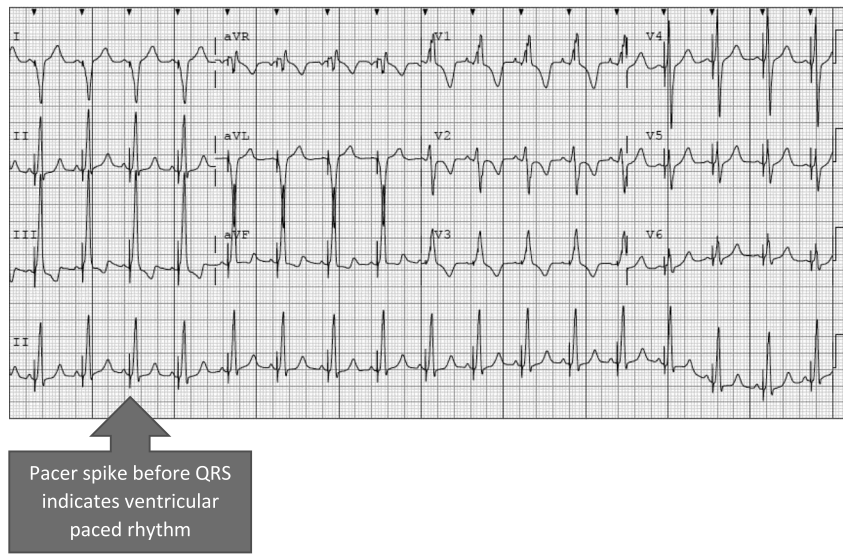

FIGURE 4. Atrial sensed, ventricular paced rhythm.

Advocacy/Future Research

Improved survival from pediatric IHCA and OHCA has been associated with multiple factors. These include presence of witnessed arrest, immediate provision of high quality chest compressions, and early defibrillation for shockable rhythm.[11,84,85] IHCA are more likely to occur in the intensive care unit, to be witnessed, and to be monitored, and this results in improved survival of patients who suffer IHCA.[14,86–88] ED and ICU teams who undergo regular performance and simulation training are more likely to provide optimal care of pediatric patients in cardiac arrest.[38,89,90] In contrast, the majority of children who suffer OHCA have an unwitnessed arrest, do not receive CPR, and do not receive defibrillation.[91] Barriers to public access defibrillation and provision of bystander CPR have been identified in prior studies. The main barriers noted include acquisition and maintenance of the defibrillation device itself, public knowledge and awareness of the device location, training, medicolegal issues, and layperson willingness to use the device.[92–96]

Multiple solutions have been recommended to overcome these barriers, with varying levels of success. Telephone-CPR (TCPR) is one method of increasing early need for CPR by lay bystanders. Using TCPR, emergency dispatchers recommend CPR when a patient is either not responsive or not breathing normally. While TCPR has been shown to improve recognition of cardiac arrest, it does not necessarily improve the rate at which bystanders actually provide CPR.[17,97] One known barrier to bystander conventional CPR is the need for rescue breaths; for this reason hands-only CPR is recommended in adult patients with cardiac arrest. While pediatric patients have better outcomes from OHCA with conventional CPR than they do with hands-only CPR, children who receive any form of CPR have better outcomes than those who do not.[98,99]

For those patients with OHCA who require defibrillation, mobile strategies have been used throughout the world to improve access to AEDs. These include mobile phone positioning systems that enable trained members of the general public to be notified if they are within 500 meters of a person suffering a cardiac arrest, as well as mobile applications that connect bystanders emergency dispatch centers that give the exact location of a person with OHCA and the location of the nearest AED.[17,100,101] To facilitate the most cost-effective placement of AEDs in the community, public spaces with large numbers or circulating people have been targeted. These include placement in transportation hubs (airports, train stations, metro stations), casinos, stadiums, convention centers, post offices, and schools.[102–104] The American Heart Association recommends that every school athletic program should have access to an AED within 5 minutes of collapse.[105,106] Where this is not feasible, drone technology has been discussed as a method for rapid delivery of AEDs to the location of a person in cardiac arrest. In 2016, Google patented drone technology to deliver medical equipment, including AEDs, in the case of an emergency (US Patent 9,307,383 B1). Mathematical models suggest that in rural areas in particular, drone technology could reduce the delivery time of an AED by 6 to 10 minutes, thus potentially improving the time from cardiac arrest to defibrillation.[17,107]

In both the in-hospital and out-of-hospital settings, clinical providers have an obligation to provide competent and timely care to pediatric patients who require cardiac resuscitation. In the ED, resuscitation teams must be prepared to provide optimal CPR and appropriate cardioelectrical stimulation to any patient, at any time of day or night, regardless of underlying physiology or cause of cardiopulmonary instability. In the community setting, resources must be allocated to improve both access to AEDs and to education/training on the critical importance of bystander CPR and AED use.

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