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Application of Pediatric Advanced Life Support for Managing a Patient With a Defibrillator Who Develops an Intraoperative Cardiac Arrest

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Stem Case and Key Questions Content

Your case is a 7 year old female with a history of an abnormal heart rhythm scheduled for washout and debridement of suspected osteomyelitis in the right upper extremity.

The patient was seen in the Emergency Department (ED) 2 days ago with fever, right upper extremity pain, and swelling. Plain x-rays were unremarkable. The patient was started on cephalexin and acetaminophen for suspected cellulitis. The patient was subsequently discharged home.

Upon return to the ED tonight, the patient appears overall much worse than two days ago according to the mother.

The patient has the following vital signs:
Temperature (oral) 39.7 degrees Celsius
Respirations 36
Heart rate 116
Blood pressure 82/54
Room air oxygen saturation 97%
Patient weight: 29 kg

The patient had a Magnetic Resonance Imaging (MRI) study today without sedation which reported a large abscess in the right upper extremity consistent with osteomyelitis.

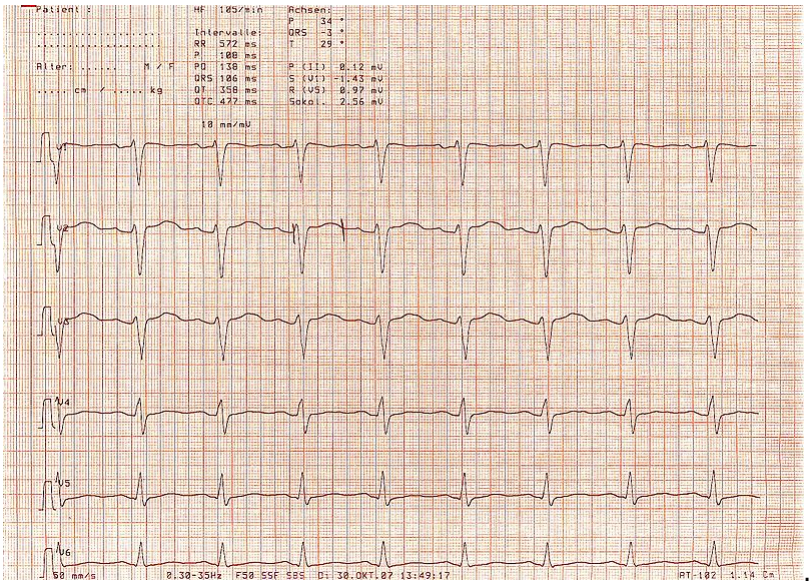
In the ED over the last three hours, the patient has received intravenous acetaminophen and two fluid boluses of lactated ringers totaling 400 milliliters (ml). The patient is currently receiving intravenous fluids at a rate of 60 ml/hour. Clindamycin has already been administered. The patient has one 22 gauge peripheral intravenous line.

The surgeon has agreed, per the admitting physician's request, to place a PICC (Peripherally Inserted Central Catheter) line during the surgical procedure due to suspected long-term intravenous antibiotic administration.

<u>Additional information from the mother:</u>
Drug allergies: penicillin- rash

Home medications: atenolol; last dose was given 3 days ago
Past Medical History: Long QT Syndrome
Past Surgical History: tonsillectomy and adenoidectomy 3 years ago; Implantable Cardioverter Defibrillator (ICD) placement 1 year ago; no reported anesthetic complications from either procedure
Last oral intake: 4 hours ago for solid food

Diagnostic tests:
Hematocrit 38%
The following EKG was obtained:



You are now evaluating the patient with the mother at the bedside.

1. The QT interval is reported in the EKG as 358; the QTc is reported as 477. What is a normal QT interval? Why is the QT interval corrected (QTc)? What is Long QT Syndrome?

You have decided to administer general anesthesia with a tracheal tube for this procedure. The patient has been brought into the operating room. Standard monitors have been applied.

The patient has the following preoperative vital signs:
Temperature 102.1F (38.9 C)
Respirations 28
Heart rate 126, sinus tachycardia
Blood pressure 82/52
Room air oxygen saturation 98%

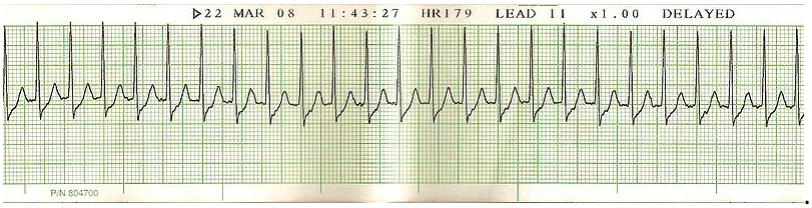
2. What are the anesthetic implications for patients with prolonged QT intervals such as Long QT Syndrome? Which medications should be avoided in a patient with a prolonged QT interval?

3. What are the anesthetic implications for a patient with an Implantable Cardioverter Defibrillator (ICD)? Is there any management required related to the ICD that should be performed prior to induction of anesthesia? Should a magnet be placed on top of the device? The surgeon requests to use a monopolar electrocautery surgical unit for this procedure; what is your response?

4. The surgeon suggests that this child may be developing septic shock. What would be your choice(s) of medications for induction of general anesthesia? Are there any medications that you should avoid?

Induction of general anesthesia was uneventful. Vital signs are stable; the airway was secured with a tracheal tube. The surgical procedure has been underway for 20 minutes.

During PICC line placement, the patient suddenly develops a blood pressure of 58/36 with the following electrocardiogram (EKG) tracing:



The PICC line guidewire is promptly removed by the surgeon. The EKG rhythm and vital signs remain unchanged.

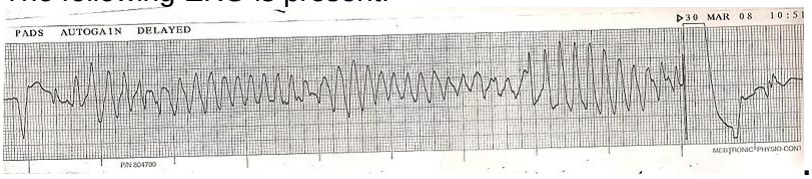
5. Suppose the code cart was not present within the operating room. What are your initial steps while the code cart is being obtained? Do you have an instinctive series of actions that you follow when any intraoperative crisis has just occurred? If so, describe your initial intraoperative crisis plan (e.g. notify surgeon, etc.)?

6. What is your interpretation of this EKG? What is your management plan? What is your next plan if the initial therapy is ineffective?

The patient is stabilized using the appropriate Pediatric Advanced Life Support (PALS) guidelines. Surgery concludes after completing a large debridement and washout to the right upper extremity. The patient received 700 milliliters of lactated ringers. The estimated blood loss was 200 milliliters.

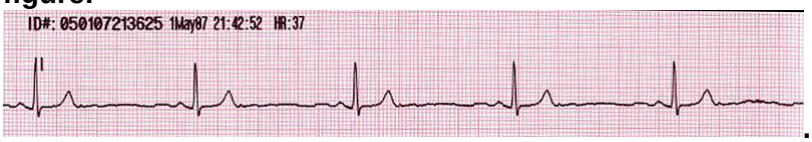
The surgical procedure has been completed. You planned to perform an awake extubation. During emergence, an alarm on the EKG monitor has been activated.

The following EKG is present:



7. What is your interpretation of this EKG? If required, what would be the order in which Pediatric Basic Life Support (BLS) should be performed? If your initial therapies are ineffective, what are your next steps in management for this rhythm? Would you administer vasopressin? If you thought the rhythm is Torsade de pointes, are there any specific therapies in addition to the standard PALS guidelines?

8. Hypothetically, suppose the rhythm developed into the one depicted in the following figure:



What is your interpretation and management of this EKG? Do you need any additional information to determine what therapies are required? If these therapies are ineffective, what are your next steps in management of this rhythm?

9. According to the American Heart Association, what are some common etiologies that should be considered in a child that develops cardiac arrest?

The patient has been effectively resuscitated. After discussion with the surgeon, you have decided to transfer the patient with a tracheal tube still in place to the Intensive Care Unit (ICU).

Vital signs: Temperature 37.9 degrees Celsius; Heart rate 122; Sinus tachycardia; Blood pressure 98/62; Oxygen saturation is 99% with an oxygen concentration of 100%.

10. What is your recommendation to the ICU team regarding the amount of oxygen concentration this patient should receive?

You have given report to the ICU physician and concluded transferring care to the ICU team.

Model Discussion Content

Pediatric Advanced Life Support (PALS), created by the American Heart Association, consists of nationally accepted guidelines and algorithms for cardiac resuscitation in the pediatric patient. Long QT Syndrome (LQTS) is a disorder characterized by a prolonged QT interval on the electrocardiogram (EKG)[1]. LQTS is a condition becoming recognized more frequently with important implications to the anesthesiologist. The typical age for onset of symptoms due to LQTS places pediatric patients at significant risk. Patients with LQTS are at increased risk for cardiac events including Torsade de pointes and sudden death.

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The QT interval represents the electrical depolarization and repolarization of the ventricles. The QT interval is a measure of the time interval between the start of the Q wave and the end of the T wave. The QT interval decreases with tachycardia and conversely increases with bradycardia. Therefore, the corrected QT interval (QTc) is used to compensate for the heart rate variation. The QT interval can be corrected by several formulas; the most popular method is by dividing the QT interval by the square root of the RR interval. A normal QTc interval is less than approximately 440 milliseconds (msec); a QTc interval of 450-470 msec is considered borderline prolonged. Patients with conditions such as Long QT Syndrome (LQTS) typically have a QTc interval greater than 480 msec[1].

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Causes for producing a prolonged QT interval include congenital and acquired etiologies[2]. Congenital forms of LQTS occur due to genetic mutations within the ion channels of cardiac muscle. These genetic mutations occur within the transmembrane protein mostly to the potassium channel; however, defects also occur in the sodium and calcium ion channel proteins. A prevalence as large as 1:5000 is estimated for all congenital forms of LQTS. It is proposed that there are many undiagnosed cases of sudden death due to LQTS.

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Acquired forms of LQTS commonly result from electrolyte disorders, predisposing medical conditions, and from the administration of drugs that increase the QT interval[2]. Electrolyte disorders that increase the QT interval include hypomagnesemia, hypokalemia, and hypocalcemia. Medical conditions that are associated with increasing the QT interval include hypothyroidism, hypothermia, subarachnoid hemorrhage, cocaine toxicity, organophosphate poisoning, autonomic neuropathy, and myocardial ischemia.

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Multiple congenital syndromes have been identified and associated with LQTS; a large amount of these cases have presented in the pediatric patient[2]. Currently, 8 genes have been identified and implicated in approximately 70% of the congenital cases of LQTS. Jervell Lange-Nielsen, was the first to report a case in 1957 of congenital deafness with an autosomal recessive pattern of inheritance. Romano and Ward described a similar syndrome without deafness but inherited in an autosomal dominant pattern. Andersen syndrome and Timothy Syndrome are two newly recognized syndromes also thought to be associated with mutations to the cardiac muscle ion channels.

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Patients with LQTS may report a history of ventricular arrhythmias, syncope, sudden death, and Torsade de pointes (TDP)[3]. However, many patients will be completely asymptomatic; some patients will report onset of symptoms (i.e. syncope) after physical exertion or sympathetic stimulation. Since documenting the occurrence of ventricular arrhythmias may prove to be difficult, patients with LQTS have frequently been misdiagnosed with conditions such as epilepsy and vasovagal syndrome.

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Current therapeutic options for patients with LQTS include beta blockade, pacemaker, and implantable cardioverter-defibrillator (ICD) placement[3]. Left cardiac sympathetic denervation was previously utilized for patients who were refractory to medical management. Oral potassium supplementation and potassium sparing diuretics are currently being investigated for specific subtypes of LQTS. Beta-adrenergic blockers are the mainstay of medical therapy for patients with LQTS. Beta-blockers have been shown to reduce the number of cardiac events, including TDP, in patients with LQTS. Therapy should be continued for life; this includes the perioperative

period. In addition, many experts recommend beta-blocker therapy for asymptomatic patients with congenital forms of LQTS. Pacemakers and/or ICD's should be considered for patients with LQTS who are symptomatic despite maximal medical therapy.

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Anesthetic implications for the patient with LQTS include an increased risk of ventricular arrhythmias, Torsade de pointes (TDP), and sudden death[4]. The main goals are to avoid conditions that increase sympathetic stimulation, prevent the development of hypokalemia, hypocalcemia, hypomagnesemia, and substitute medications not associated with increasing the QT interval. However, TDP can also occur spontaneously. Conditions that increase sympathetic stimulation should be prevented by the use of sufficient preoperative anxiolysis, insuring adequate levels of anesthesia, and appropriate administration of postoperative analgesia. Conditions that increase sympathetic stimulation include hypothermia, hypertension, hypoxemia, and hypercapnia. In addition, conditions that cause bradycardia can also increase the QT interval and possibly increase the risk of TDP. Anesthetic techniques that should be considered may include deep extubation, parental presence during induction of anesthesia, and the use of regional anesthesia.

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Anesthetic management for patients with LQTS requires appropriate preparation[4]. A code cart with a defibrillator should be immediately available. Resuscitation drugs including beta-blockers, calcium, and magnesium should also be readily accessible. Continuous EKG monitoring should occur in the intraoperative and postoperative periods. Increased vigilance for the development of ventricular arrhythmias and TDP cannot be overemphasized. Preoperative beta blockade should be continued throughout the perioperative period.

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The following drugs that are commonly administered in the perioperative period have been shown to increase the QT interval (Table 1). However, the clinical significance of this is unclear; there are many reports of patients receiving these drugs without cardiac complications or the development of TDP. Ideally, medications should be selected that are not associated with prolongation of the QT interval. For example, an anesthetic plan which avoids neuromuscular blockade should be considered.

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Table 1:
Perioperative medications that may prolong the QT interval.
Anticholinergics
Neostigmine
Ondansetron
Succinylcholine
Volatile agents

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The following medications have not been shown to significantly increase the QT interval or increase the risk of TDP development (Table 2).

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Table 2:
Perioperative medications that have NOT been shown to prolong the QT interval.
Local anesthetics
Midazolam
Nitrous oxide
Nondepolarizing neuromuscular blocking agents
Opioids
Propofol

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Numerous drugs have been shown to increase the QT interval. Several drugs have been removed from clinical use or restricted due to reports of prolongation of the QT interval and the development of ventricular arrhythmias (Table 3). A comprehensive list of medications and their risk of causing TDP can be found on the Arizona Center for Education and Research on Therapeutics website http://www.azcert.org/medical-pros/drug-lists/drug-lists.cfm (accessed January 2014).

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Table 3

Medications removed from clinical use due to associated Torsade de pointes development.

Astemizole (Hismanal)
Cisapride (Propulsid)
Grepafloxacin (Raxar)
Mibefradil (Posicor)
Terfenadine (Seldane)

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Patients presenting with a pacemaker and/or ICD requires appropriate perioperative management[5]. Preoperative interrogation should occur which includes obtaining information regarding the remaining battery life, discharge history of the device, settings, and the reason for placement. The device interrogator should disable the anti-tachycardia and defibrillator functions to the ICD for the intraoperative period; this will prevent inappropriate discharges from the device. Patients with pacemakers should have all rate enhancements turned off; this will reduce the likelihood of inappropriate tachycardia from the pacemaker. Typically, the pacemaker is reprogrammed to an asynchronous mode for use during the intraoperative period. The response to the device from placement of a magnet should be determined prior to induction of anesthesia. The routine placement of a magnet on top of an ICD/pacemaker is not recommended. Postoperative interrogation is required to reactivate the defibrillator function and restore the preoperative pacemaker settings.

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Additional concerns for patients with ICD's and pacemakers include several equipment modifications[5]. The artifact filter on the electrocardiographic monitor should be disabled; this will allow the pacemaker spikes to be displayed within the electrocardiograph. Since the defibrillator function has been disabled, an external defibrillator should be immediately available during the intraoperative period. Alternative pacing equipment including transcutaneous or transvenous systems should be immediately available for patients with pacemakers. Placement of an arterial line is not mandatory; however, the pulse should be measured by a mechanical method such as pulse oximetry, palpation, or invasive pressure monitoring. Monopolar electrocautery should be avoided due to the production of electromagnetic interference. Electromagnetic interference can result in triggering of the ICD. Bipolar electrocautery is recommended for patients with an ICD or pacemaker.

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The selection of medications required for induction of anesthesia in this case should include the concerns for maintenance of hemodynamic stability, increased risk for aspiration, increased risk for cardiac arrhythmias, and the potential for septic shock. According to the revised PALS guidelines, etomidate is not recommended for routine use in patients with suspected septic shock due to the association of increased mortality even after receiving a single dose for airway management[6]. One example of an acceptable medication regimen for a rapid sequence induction of anesthesia in this patient may include a reduced dose of propofol (e.g. 1 mg/kg)

along with an increased dose of rocuronium (e.g. 1.2 mg/kg).

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In late 2010, the American Heart Association (AHA) released updates to Pediatric Basic Life Support (BLS) and Pediatric Advanced Life Support (PALS)[7]. Most of the PALS algorithms have been predominantly unchanged from the last version. However, a major change in the 2010 Pediatric BLS guidelines is regarding the order of cardiopulmonary resuscitation. The previous "ABC" order of airway, breathing, and then circulation has been replaced with "CAB"; this sequence requires chest compressions to be started before providing rescue breathing.

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The change from "ABC" to "CAB" was made by the AHA for the following reasons:
-Desire to have one algorithm for adults and children
------Adults comprise the majority of cardiac arrests
-Better outcome in adults if compressions started early
-Minimal delay in starting ventilations for children
-Less delay in ability to start chest compressions
------Head positioning/opening airway is longer process to initiate
------Unwillingness to provide rescue breaths

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Other changes made to the 2010 Pediatric BLS guidelines include[7]:
-Pulse check: take no longer than10 seconds
-If no pulse or unsure, start chest compressions
-"Look, listen, and feel" terminology eliminated
-Apply an Automatic External Defibrillator (AED) if available
-Ideally with a pediatric attenuator if <8 years old
-May use a standard AED in all patients if a pediatric attenuator is unavailable

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Other significant changes made to the 2010 PALS guidelines include[6]:
-Use of continuous pulse oximetry is strongly recommended
------Appropriate to use 100% oxygen concentration during resuscitation
------Once stabilized, titrate oxygen concentration to maintain an oxygen saturation of >94%
-No recommendation for the routine use of cricoid pressure during intubation for prevention of pulmonary aspiration
-Utilize the actual body weight for drug dose calculations
-Calcium and sodium bicarbonate are not routinely recommended
-Defibrillate first at 2 Joules/kg; the maximum dose has been increased to 10 Joules/kg
-New sections on Congenital Heart Disease which includes:
------Single ventricle: consider the use of heparin, maintain an oxygen saturation of approximately 80%, consider the use of medications to lower systemic vascular resistance, and use of extracorporeal membrane oxygenation (ECMO)
------Pulmonary Hypertension: consider the use of inhaled nitric oxide, prostacyclin, and ECMO
-Etomidate is not recommended for patients with septic shock due to an association with increased mortality
-Wide-complex tachycardia is defined as having a QRS width >0.09 seconds.
-No reliable predictors to guide termination of resuscitation efforts
-Vasopressin is not part of the PALS guidelines

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Many anesthesia providers report that they have developed an instinctive series of actions to

follow after a generic intraoperative crisis has occurred[8]. These series of actions would occur prior to instituting the PALS guidelines. One example of a generic initial intraoperative crisis plan (not in any particular order but in most situations can be accomplished in a short period of time) includes:

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-Call for help/Notify surgeon
-Call for additional equipment/supplies (e.g. code cart, Malignant Hyperthermia cart, blood products, difficult airway equipment, etc.)
-Turn vaporizers off/medication infusions modified
-Increase to 100% oxygen concentration
-Open intravenous fluids
-Listen to breath sounds/verify airway
-Check pulse/Assess vital signs/Start CPR if indicated

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According to the new BLS guidelines, chest compressions should be started first and without delay. The oxygen concentration should be 100% with the vaporizers turned off[9]. Emergency drugs can be delivered by endotracheal or intraosseous routes if loss of intravenous access should occur. The smaller defibrillator paddles are used for infants less than 1 year old or less than 10 kg.

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The first rhythm illustrated in this case depicts supraventricular tachycardia (SVT). The tachycardia algorithm of the 2010 PALS guidelines[6] assumes a pulse is present along with evidence of poor perfusion. First, determine if the QRS duration is narrow or wide. If the QRS duration is narrow, assume either sinus tachycardia or SVT is present. SVT is a rapid, regular rhythm with nonidentifiable P waves. SVT may be distinguished from sinus tachycardia by a rate that is usually greater than 220 in infants and greater than 180 in children. SVT with a pulse can initially be treated with adenosine if intravenous access is present or synchronized cardioversion. Vagal maneuvers may be attempted first as long as no delay occurs in providing drug or electrical therapy. Adenosine is administered at a dose of 0.1 mg/kg followed by 0.2 mg/kg if the first dose is ineffective. Adenosine must be flushed in rapidly during its administration. If adenosine is ineffective or if intravenous access is not present, synchronized cardioversion is used with an initial dose of 0.5-1 J/kg followed by 2 J/kg for further attempts. If cardioversion remains ineffective, consultation with a pediatric cardiologist is strongly encouraged. In addition, amiodarone 5 mg/kg given over 20-60 minutes or procainamide 15 mg/kg given over 30-60 minutes should be administered.

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If a wide QRS duration is present, treat as presumptive ventricular tachycardia using synchronized cardioversion at 0.5-1.0 J/kg followed by 2 J/kg if ineffective. If cardioversion is ineffective, consultation with a pediatric cardiologist is strongly encouraged. In addition, amiodarone 5 mg/kg IV given over 20-60 minutes or procainamide 15 mg/kg IV given over 30-60 minutes should be administered.

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A summary for the management of a pediatric patient with tachycardia and with poor perfusion includes[6]:

<u>-First determine the QRS interval:</u>

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-If a Normal QRS interval (Sinus Tachycardia) is present: treat the underlying cause such as

fever, pain, or anxiety
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-If a Narrow QRS interval (Supraventricular Tachycardia) is present: the key components include vagal maneuvers, adenosine (0.1 then 0.2 mg/kg), and synchronized cardioversion (0.5-1 J/kg then 2 J/kg)
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-If a Wide QRS interval is present (Ventricular Tachycardia): the key components include synchronized cardioversion (0.5-1 J/kg then 2 J/kg), consideration for amiodarone 5 mg/kg over 20-60 min or procainamide 15 mg/kg over 30-60 min, expert consultation is recommended, remember to not give both amiodarone and procanamide, and to avoid adenosine if Wolff-Parkinson-White syndrome is present
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The most recent 2010 PALS Pulseless Arrest Algorithm[6] incorporates 4 cardiac rhythms that are divided into shockable and not shockable categories. Pulseless electrical activity (PEA) and asystole comprise the not shockable rhythms. Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) include the shockable rhythms. The first decision to be made is if the rhythm is shockable (VF/VT) or not shockable (Asystole/PEA).
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The second rhythm illustrated in this case depicts a special variant of ventricular tachycardia (VT) termed Torsade de pointes (TDP). For all shockable rhythms such as VF or VT, defibrillate one time at 2 J/kg and resume cardiopulmonary resuscitation (CPR) immediately. Five cycles of CPR follow (which typically takes 2 minutes). If the rhythm is still shockable, defibrillate once at 4 J/kg and resume CPR. After defibrillation, epinephrine is given every 3-5 minutes. The dose for epinephrine is 10 mcg/kg (IV or IO) and 100 mcg/kg if given via the endotracheal tube. Five cycles of CPR then occur followed by evaluation of the rhythm. If the rhythm is shockable, defibrillate at 4 J/kg followed by resuming CPR. One then will administer a bolus of amiodarone 5 mg/kg IV. This cycle of 1 shock followed by 5 cycles of CPR is repeated until a decision is made to terminate efforts or the rhythm is not shockable..Torsade de pointes, a French term meaning "twisting of the points", can be classified as a less common form of polymorphic, ventricular tachycardia. TDP can spontaneously resolve or progress into other arrhythmias such as ventricular fibrillation and ultimately asystole. The ventricular rate in TDP can vary from 150-250 beats per minute. In TDP, the QRS complexes will be seen rotating around the horizontal axis. Patients with TDP may present pulseless and in cardiac arrest. However, many patients may present with ventricular tachycardia with a pulse. Management of TDP centers on magnesium sulfate administration and the implementation of emergency cardiac care guidelines such as PALS[6].
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A summary for the management of a pediatric patient without a pulse and with a shockable rhythm includes[6]:
-Defibrillate 2 J/kg, 5 cycles CPR, Reassess
<u>If still shockable</u>:
-Defibrillate at 4 J/kg
-Administer epinephrine bolus10 mcg/kg every 3-5 minutes
-Provide 5 cycles of CPR
-Reassess and repeat if applicable
<u>If patient remains in a shockable rhythm despite the previous therapies:</u>
-Defibrillate 4 J/kg then bolus amiodarone 5 mg/kg

-Magnesium only if suspected Torsade de pointes (25-50 mg/kg)
-Provide 5 cycles of CPR then Reassess

If the rhythm is not or becomes shockable, follow the asystole/PEA algorithm[6]. Resume CPR and administer epinephrine every 3-5 minutes in the same doses as for VF/VT. After 5 cycles of CPR, reevaluate the rhythm to determine if it is shockable or not shockable. After this has been determined, follow the appropriate algorithm.

The third rhythm illustrated in this case depicts bradycardia. The bradycardia algorithm of PALS[6] assumes that cardiorespiratory compromise is present and the heart rate is < 100. If the EKG displays bradycardia but no pulse is present, use the PEA section of the PALS pulseless arrest algorithm. Initiate chest compressions if the heart rate is <60 and poor perfusion is present. According to the PALS guidelines, if symptomatic bradycardia is still present administer epinephrine 10 mcg/kg IV (intravenous)/IO (intraosseous) every 3-5 minutes. If increased vagal tone or primary AV (atrioventricular) block is present, administer atropine 0.02 mg/kg; minimum dose 0.1 mg, maximum total dose 1 mg. Cardiac pacing should be strongly considered for patients refractory to medical therapy or if pacemaker dependent.

The AHA PALS guidelines[6] recommend searching for reversible causes of cardiac compromise. For simplicity, many of these common etiologies for cardiac compromise can be classified as the “H’s and T’s” and should be strongly considered during resuscitation of a child:

H’s: hypovolemia, hypoxia, hydrogen ion (acidosis), hypoglycemia, hypo/hyperkalemia, hypothermia
T’s: tension pneumothorax, tamponade (cardiac), toxins, thrombosis (pulmonary, cardiac)

According to the PALS guidelines[6], it is appropriate to use 100% oxygen concentration during resuscitation and if clinically indicated. These guidelines also recommend to titrate the oxygen concentration to maintain an oxygen saturation of >94% once the patient has been stabilized. A prolonged duration of exposure to high oxygen concentrations has been associated with increased oxidative injury after ischemia reperfusion.

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