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Junctional Ectopic Tachycardia Management

JOHNS HOPKINS
All Children's Hospital

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Junctional Ectopic Tachycardia Clinical Pathway

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Owners: Jeremy Ramey, MD, Jamie Decker, MD, Mike Bruno, MD

Johns Hopkins All Children's Hospital – Heart Institute

Junctional Ectopic Tachycardia Management Clinical Pathway

Rationale:

This clinical pathway was developed by a consensus group of Johns Hopkins All Children's Hospital (JHACH) physicians, advanced practice providers, nurses, and pharmacists to standardize the management of junctional ectopic tachycardia (JET) primarily in patients who have recently undergone cardiac surgery with cardiopulmonary bypass.

It addresses the following clinical questions or problems:

1. Outline characteristics of JET
2. Identify patients at increased risk for developing JET
3. Clarify methods to differentiate JET from other arrhythmias
4. Outline initial management and treatment of JET

Background:

JET is an arrhythmia that may occur in the post-operative setting after cardiac surgery in pediatric patients. Specifically, JET is an automatic tachycardia that originates from the atrioventricular (AV) node or proximal bundle of His. It occurs in approximately 5 – 15% of patients after cardiac surgery and is more common in younger patients less than 1 year of age. It is also more common after surgery in patients with Tetralogy of Fallot, AV septal defect repair, arterial switch operation, and repair of anomalous pulmonary venous return. Additional risk factors include longer aortic cross-clamp times and possible genetic factors. The incidence of JET is associated with a longer duration of mechanical ventilation and longer time spent in the intensive care unit. A standardized approach to the diagnosis and management of JET in the post-operative setting will improve patient outcomes and lead to improvement in team education and collaboration.

Figure 1 – Junctional ectopic tachycardia in infants and children[2]

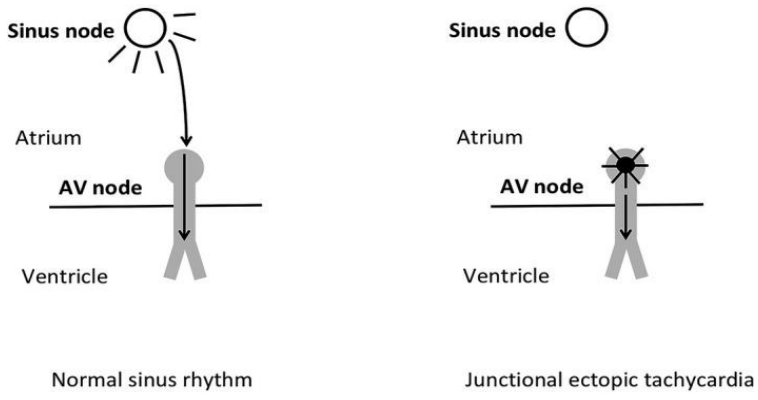

Table 1 – Definitions

JET	Automatic tachycardia that should be narrow complex (i.e., sinus QRS) The VA relationship is either 1:1 or V>A and narrow complex (sinus QRS morphology) with a "warm up" where the ventricular rate appears to increase over the atrial rate (as opposed to a reentrant mechanism like SVT)
Atrial pacing	Electrical stimulation of the atrium above the native atrial or ventricular rate Atrial pacing is usually performed using an externalized epicardial atrial pacing wire connected to a pacing source (pacing "box"), but can also be performed using an endocardial atrial pacing lead or a transesophageal pacing probe Typical pacing rate is set at 10 – 30 bpm above the native atrial or ventricular rate
Passive therapeutic hypothermia	The process of allowing core body temperature to passively drop by removing blankets and other sources of heat Typical target core temperature is 35 – 36 °C
Active therapeutic hypothermia	The process of lowering core body temperature by surface body cooling (i.e., cooling blankets) Typical target core temperature is 32 – 34 °C

Abbreviations: bpm, beats per minute; SVT, supraventricular tachycardia; VA, ventricular-atrial

Inclusion:
Verified diagnosis of JET by atrial electrocardiogram (ECG)

Exclusion:
Alternative atrial or ventricular arrhythmias

Diagnosis:

Diagnosis and initial assessment:

1. Obtain an atrial ECG per instructions on the ECG machine with interpretation by a Cardiologist and/or Cardiac Critical Care Intensivist to identify if the rhythm has any of the following features:
    a) Narrow complex tachycardia with rate > 170 bpm
    b) Retrograde p-waves
    c) Discordance between atrial and ventricular impulses
    d) Atrial rate exceeded by rapid junctional rate
2. Signs of hemodynamic compromise
    a) Hypotension
    b) Poor perfusion
    c) Rising lactate
    d) Decreased near-infrared spectroscopy (NIRS)
    e) Decreased myocardial oxygen consumption (MVO_2)

3. Consultation with Cardiologist and Cardiac Critical Care Intensivist to confirm diagnosis before proceeding with 'Clinical Management'

Laboratory tests and diagnostics:

- ECG
- NIRS
- Arterial blood gas (ABG) or venous blood gas (VBG)
- Complete blood count (CBC)
- Comprehensive metabolic panel (CMP)

Radiology studies:

- Consider an echocardiogram (ECHO)

Clinical Management:

Initial management stage:

1. At ALL steps in the algorithm, consider atrial pacing (AAI) at rates slightly faster (10 – 20 bpm) than the ventricular/junctional rate to improve AV synchrony
    a) Observe for any improvement in hemodynamics (e.g., improved blood pressure (BP), perfusion, decreased filling pressures)
2. Diagnosis confirmed by atrial ECG and discussion with relevant providers:
    a) Passively cool to 35 – 36 °C and minimize fevers
    b) Check and supplement electrolytes, maintaining magnesium (Mg) > 2 mg/dL, potassium (K) > 4 mmol/L, and ionized calcium (iCa) > 1.2 mmol/L
    c) Pain and sedation:
        i. Adequately control pain with acetaminophen, ketorolac (if not bleeding), and/or opioids
        ii. Avoid benzodiazepines given the risk of myocardial depression
        iii. Strongly consider the use of dexmedetomidine for sedation, as there is limited evidence that it may help to prevent and treat post-operative tachyarrhythmia
    d) Limit chronotropic agents (e.g., epinephrine, dopamine), with consideration for pure vasopressors (e.g., vasopressin, norepinephrine) if needed to maintain adequate BP
3. Rhythm refractory to initial measures with concern for worsening hemodynamic compromise:
    a) Actively cool via cooling blanket to 34 – 35 °C
    b) Increase analgesia/sedation (see Clinical Management 1c) by using infusions with strong consideration for neuromuscular blockade
    c) First-line pharmacologic therapy: (*consider either option based on the clinical condition of the patient*)
        i. amiodarone (intravenous (IV)): (refer to Table 2 for dosing)
            - Consider first-line over IV sotalol if baseline corrected QT interval (QTc) > 480 milliseconds (ms)
            - Multiple boluses may be administered if needed

- Before initiating amiodarone therapy, consider the following:
    i. Risk of hypotension with bolus dosing:
        1. Fast infusion rates of bolus have been associated with hypotension and cardiac arrest
        2. Risk factors for hypotension include:
            a. Age < 1 year
            b. Any degree of poor ventricular function
            c. Rapid infusion of amiodarone bolus over < 30 minutes
        3. For patients with risk factors for hypotension with amiodarone boluses:
            a. Consider administration of IV calcium (Ca) before administration of amiodarone bolus doses
            b. Amiodarone bolus shall be administered over a minimum of 30 minutes, with confirmation of infusion time by the prescriber with the nurse before administration
        4. Select the amiodarone bolus dose and administration method, based on the assessed ventricular function:
            a. Poor function with risk factor(s) for hypotension:
                i. “Low dose” – 2.5 mg/kg/dose (max dose: 150 mg/dose) administered over ≥ 60 minutes
            b. Adequate ventricular function:
                i. “Standard dose” – 5 mg/kg/dose (max dose: 300 mg/dose) administered over 30 – 60 minutes
    ii. Risk of QTc prolongation:
        1. Obtain daily ECGs during amiodarone loading, then at least weekly once on maintenance dosing
        2. Closer monitoring may be considered for patients with the following risk factors for QTc prolongation:
            a. Age < 9 months
            b. Doses > 10 mg/kg/DAY
            c. Persistent hypokalemia (< 3.5 mmol/mL)
            d. Adjunctive use with other QTc prolonging agents
    iii. Amiodarone therapeutic drug monitoring shall only be ordered per discussion with EP
    iv. Liver and thyroid function monitoring:
        1. If long-term therapy is anticipated, obtain baseline liver function and thyroid function tests (if not already

obtained) before or soon after initiation of amiodarone, followed by routine monitoring every 4 weeks while admitted

v. Due to the long half-life of amiodarone and potential for significant drug-drug interactions, consider discussion with pharmacy

ii. sotalol (IV): (refer to Table 2 for dosing)
- Consider first-line over amiodarone if baseline QTc is not prolonged, as it may be better tolerated
- Especially consider using in patients with left ventricular ejection fraction (LVEF) < 45% or moderate or severe right heart dysfunction
- Multiple boluses may be administered if needed
- One study demonstrated similar control to amiodarone with faster resolution of elevated heart rate (HR) within 90 minutes after administration[10]
- Obtain daily ECGs during sotalol loading, then as clinically indicated

d) Second-line pharmacologic therapy:

i. procainamide: Discuss with the Electrophysiologist (EP) before using, and the need to follow N-acetylprocainamide (NAPA) levels after administration
- Refer to Table 2 for dosing
- Consider using in patients who have poor control of JET on either amiodarone or sotalol therapy

ii. ivabradine: Used with JET, that is stable but poorly controlled in a patient taking enteral medications
- Refer to Table 2 for dosing
- Oral dosing available only; consult EP to discuss proper use and indications

4. Continued hemodynamic compromise or issues with arrhythmia control:
   a) Discuss with EP regarding additional management
   b) Consideration of mechanical circulatory support (e.g., extracorporeal membrane oxygenation (ECMO))

Weaning/monitoring phase:

1. The underlying rhythm should be assessed twice daily (if pacing, this requires pausing the pacemaker):
   a) If the underlying rhythm is a normal sinus rhythm, de-escalation of therapy is warranted
      i. First, atrial pacing should be discontinued and normothermia established
      ii. If the patient remains in a sinus rhythm for 24 hours, anti-arrhythmic medication(s) should be gradually weaned

b) If on rhythm check, the patient is still in unstable JET, current therapy should be continued

Table 2 – Pharmacologic therapies

Medication	Mechanism of Action	Dose	Notes
amiodarone	Class III anti-arrhythmic (K channel blocker) → increases AP duration, prolongs refractory period	Bolus: Poor function with risk factor(s) for hypotension: "Low dose" – 2.5 mg/kg/dose (max dose: 150 mg/dose) administered over ≥ 60 minutes Adequate ventricular function: "Standard dose" – 5 mg/kg/dose (max dose: 300 mg/dose) administered over 30 – 60 minutes Infusion: 5 – 20 mcg/kg/min (max DAILY dose: 2,200 mg/24 hours)	• Hemodynamic instability with bolus • Consider CaCl IV 20 mg/kg/dose (max dose: 1,000 mg/dose) before amiodarone load • Discuss administration with ECMO/CVS Team if 1V physiology or ventricular dysfunction
dexmedetomidine	α2a agonist → decreases norepinephrine release and increases vagal output	0.5 – 2 mcg/kg/hour	• Infants may require higher dosing than older children • May worsen bradyarrhythmia and third-degree heart block
ivabradine	Selective inhibition of hyperpolarization-activated cyclic nucleotide gated channels (f-channels) in the SA node → slows SA and decreases HR	< 40 kg and < 18 years: 0.05 mg/kg/dose BID (max dose for patients 6 to 12 months: 0.2 mg/kg/dose BID and max dose for patients > 1 year: 0.3 mg/kg/dose (not to exceed 7.5 mg/dose) BID) ≥ 40 kg and < 18 years: 2.5 mg/dose (max dose: 7.5 mg/dose) BID ≥ 18 years: 5 mg/dose (max dose: 7.5 mg/dose) BID	• Restricted to Cardiology • Outpatient oral solution available through specialty pharmacy
procainamide	Class IA antiarrhythmic (binds to fast Na channels → inhibits recovery following repolarization)	Bolus: 2 – 6 mg/kg/dose IV (max dose: 100 mg/dose) over 10 minutes; repeat every 10 minutes up to a total cumulative dose of 10 – 15 mg/kg Infusion: 10 – 80 mcg/kg/min (max DAILY dose: 2,000 mg/24 hours)	• Consult EP before using due to lab monitoring requirements • Hemodynamic instability with bolus • Caution with decreased EF

Abbreviations: 1V, single ventricle; AP, action potential; BID, twice daily; CaCl, calcium chloride; CVS, Cardiovascular Surgery; EF, ejection fraction; Na, sodium; SA, sinoatrial

Table 2 – Pharmacologic therapies (continued)

Medication	Mechanism of Action	Dose	Notes
sotalol	Class II antiarrhythmic (β-1 and β-2 adrenergic antagonist) and class III (K channel blocker) → increases AP duration, prolongs refractory period	Bolus: 1 mg/kg/dose IV over 1 hour Maintenance doses: 120 mg/m^2/DAY divided Q8h in patients with normal renal function Dosing varies with age, BSA, and renal function (refer to *Cardiac Arrhythmia Focused Order Set* and consult with EP)	• Restricted to Cardiology • Caution if prior amiodarone administration

Abbreviations: BSA, body surface area; Q8h, every 8 hours

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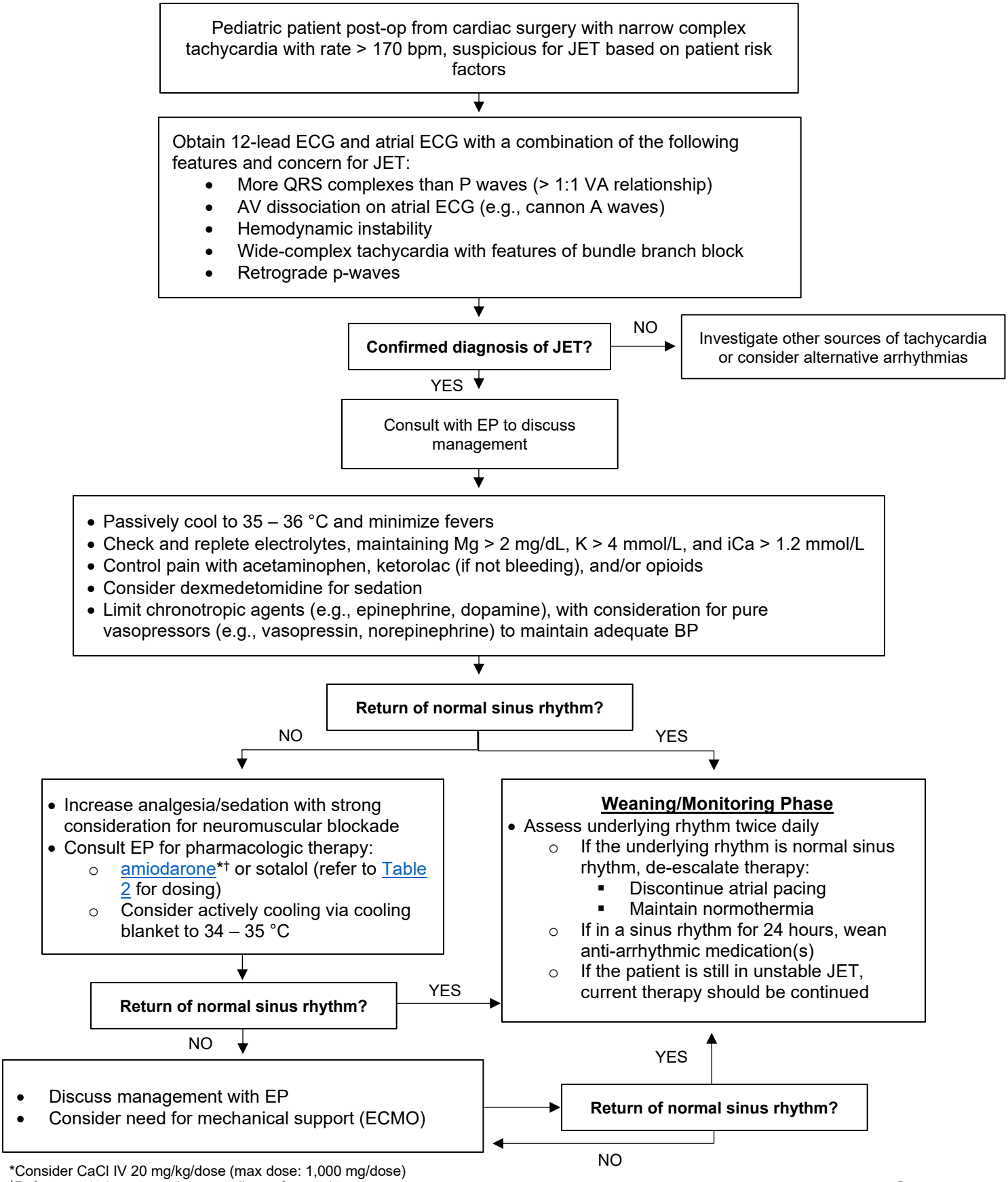

*Consider CaCl IV 20 mg/kg/dose (max dose: 1,000 mg/dose)
†Refer to amiodarone section regarding safe practices

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Outcome Measures:

- Length of stay
- Length of time in arrhythmia
- Use of pharmacologic intervention
- Mechanical support for hemodynamic compromise
- Days admission

Clinical Pathway Team
Junctional Ectopic Tachycardia Clinical Pathway
Johns Hopkins All Children's Hospital

Owner(s): Jeremy Ramey, MD, Mike Bruno, MD, Jamie Decker, MD

Also Reviewed by: Heart Institute Clinical Practice Council

Intensive Care: Reema Patel, MD
Pharmacists: Amy Kiskaddon, PharmD, Alyssa Miles, PharmD, Angela Chen, PharmD

Clinical Pathway Management Team: Courtney Titus, PA-C, Director, Kristel Lassiter, APRN, Implementation Specialist

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Disclaimer:

Clinical Pathways are intended to assist physicians, physician assistants, nurse practitioners, and other healthcare providers in clinical decision-making by describing a range of generally acceptable approaches for the diagnosis, management, or prevention of specific diseases or conditions. The physician must make the ultimate judgment regarding the care of a particular patient, considering the individual circumstances presented by the patient.

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