Paediatric Intensive Care Unit

Junctional Ectopic Tachycardia (JET) UHL Children's Intensive Care Guideline

Staff relevant to:	Healthcare professionals caring for cardiac children within the Paediatric Intensive Care Unit
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1. Introduction and Who Guideline applies to

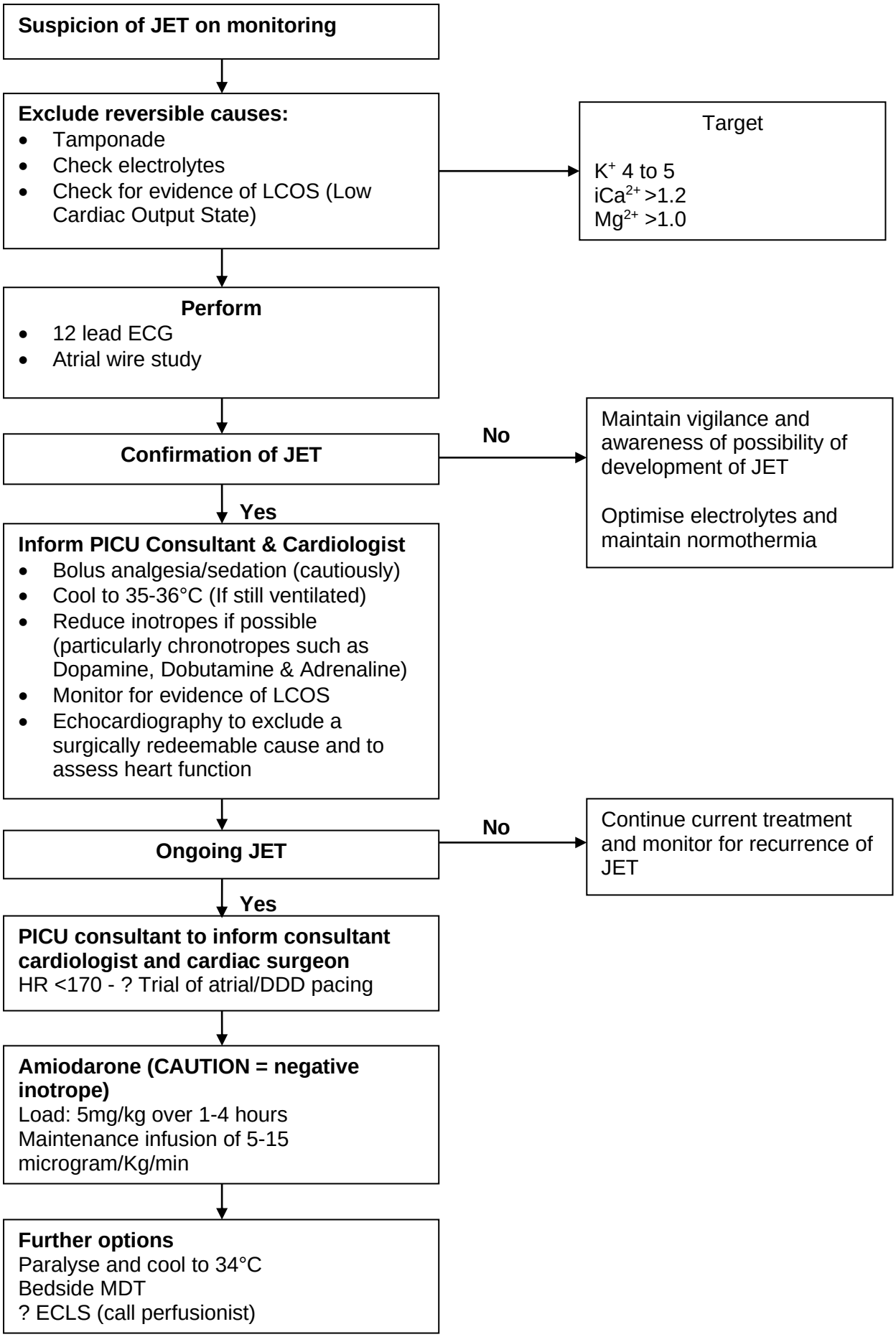
Junctional ectopic tachycardia (JET) is the most common troublesome postoperative tachyarrhythmia in children, occurring in 5-10% of post-operative cardiac patients, and can be a cause of significant morbidity and mortality in the PICU. It has a higher incidence in infants < 6 months. It is thought to be caused by trauma or oedema around the bundle of His, but is not necessarily related to surgery near the His bundle. It has been documented after all types of cardiac surgery, including extracardiac procedures such as Extra cardiac Fontan and Blalock Taussig shunt.

JET is a self-limiting tachyarrhythmia, typically occurring within 72 hours of cardiac surgery and resolving within 8 days. It is an incessant tachycardia, often with AV dyssynchrony, that results in the detrimental combination of an increased myocardial workload and oxygen consumption in the presence of a reduced myocardial oxygen supply. The global cardiac output may be reduced as a result of the impaired ventricular filling that arises due to the loss of the atrial systolic contribution and the shortened time for diastolic filling consequent upon the tachycardia. This can rapidly result in a life threatening low cardiac output state (LCOS), especially if the patient was previously haemodynamically compromised.

JET behaves like an automatic tachycardia so typically does not cardiovert in response to DC shock, adenosine or overdrive pacing. Treatment is therefore aimed at rate reduction and restoration of AV synchrony by pacing. A fall in heart rate will reduce myocardial oxygen demand while simultaneously improve myocardial oxygen delivery. Basic principles of management include adequate analgesia and sedation, correction of any electrolyte imbalance and reduction of inotropes if possible. The most effective treatment for reducing the rate of JET is a combination of modest hypothermia and IV amiodarone. Once the rate has been reduced, AV synchrony can be achieved by pacing at a rate faster than the rate of the tachyarrhythmia. ECLS is reserved for life threatening JET resistant to hypothermia and IV amiodarone.

This guideline is intended for all healthcare professionals caring for cardiac children within the Paediatric Intensive Care Unit at the Glenfield Hospital. It is provided to ensure a consistent evidence based approach to JET within the PICU

JET Treatment flow diagram



Title: Postoperative Junctional Ectopic Tachycardia
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2. Guideline Standards and Procedures

2.1 Diagnosis

The recognition and diagnosis of JET from the ECG monitor can be difficult. All patients suspected of having JET should have a 12-lead ECG and an atrial wire study if external pacing wires are in situ

Common cardiac lesions associated with the development of JET

- Tetralogy of Fallot (ToF)
- Ventricular septal defect (VSD)
- Atrioventricular septal defect (AVSD)
- Transposition of the great arteries (TGA)
- Total anomalous pulmonary venous drainage (TAPVD)

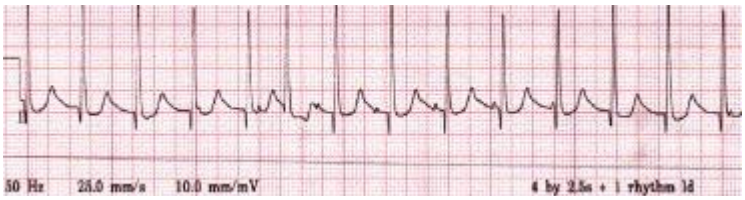
Peri-operative risk factors in the development of JET

- Infant < 6 month old
- Long cardiopulmonary bypass and cross-clamp times
- Extensive myocardial ischaemia/injury (reflected in CK-MB)
- Transient AV block immediately post cardiopulmonary bypass
- Postoperative inotropic support, particularly dopamine
- Acidosis / electrolyte abnormalities, particularly hypomagnesaemia

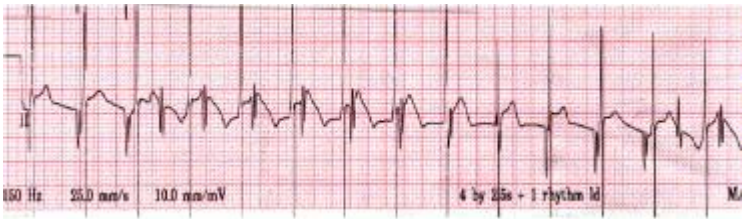
Typical features of JET on ECG monitoring

- Usual QRS complex (i.e. same QRS morphology as the preceding post-operative sinus rhythm – frequently, immediately post-operative, the QRS morphology demonstrates right bundle branch block, is therefore not narrow complex, and in the presence of tachycardia may mimic the appearance of VT). Tachycardia 170-260
- Atrial wire study can often demonstrates AV dissociation with regular ventricular rate(constant ‘R-R’ interval) faster than atrial rate, however 1:1 conduction does occur with no true A and V rate dissociation. Note also that normal conduction may still occur where a p wave is conducted to the ventricle to give the appearance of irregularity.
- If in the presence of retrograde 1:1 ventriculo-atrial conduction, the atrial activation is typically coincident with the QRS complexes.
- May have a ‘Warm-up’ or insidious onset.

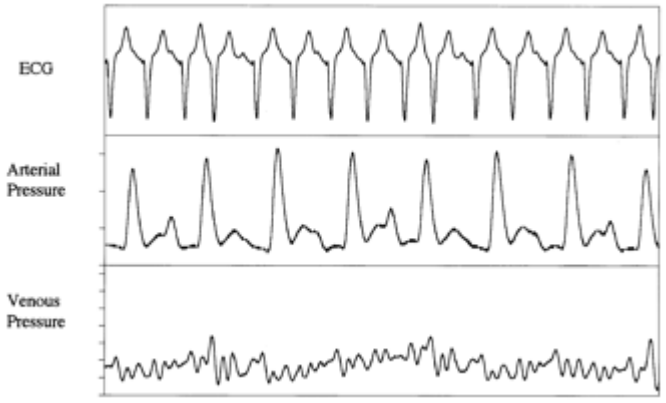
Junctional ectopic tachycardia showing AV dissociation



Atrial wire study: This rhythm strip demonstrates the faster ventricular complexes dissociated from the A 'spikes' to give the appearance of the regular A spikes 'walking through' the QRS complexes.



Loss of AV synchrony causes beat to beat variability of arterial and central venous pressure wave forms



2.2 Management options

Various treatment options exist for JET but there is no consensus on the best regimen. Treatment is aimed at slowing the 'JET driven' heart rate. However, strategies that reduce the JET rate may also reduce the rate of depolarisation of the sinus node, such that restoration of AV synchrony may not be re-established. Reduction in the tachycardia alone can be beneficial as it reduces myocardial workload, but may also allow pacing to restore AV synchrony. Markers of a low cardiac output state - a widening difference between the arterial and venous oxygen saturation, a widening of the central/peripheral temperature difference, and markers of end organ dysfunction (increasing acidosis, lactate) - should be continually monitored.

Analgesia/Sedation

Analgesia and sedation should be considered to minimise global oxygen demand, although judicial use may potentially reduce cardiac output. Despite the tachycardia being driven by a non-physiological ectopic focus, optimising analgesia/sedation will reduce endogenous catecholamine production which in turn will reduce any additional influence that the chronotropic effects may superimpose upon the JET rate. Muscle relaxation should be considered as an adjunct, especially if the desired degree of hypothermia is suspected to induce shivering (see below).

Optimisation of electrolytes and fluids

Hypomagnesaemia, hypokalaemia and hypocalcaemia have been linked with the perioperative onset of JET. (Cardiopulmonary bypass is associated with decreased magnesium levels). Magnesium should be maintained >1.5mmol/L, potassium >4.0mmol/L and ionised calcium >1.2mmol/L. Acidosis and hypovolaemia may contribute to JET although correction with bicarbonate/increased ventilation, or fluid supplementation respectively is not without side effects. Bicarbonate can cause a paradoxical intra-cellular acidosis and imparts a CO2 load; increased minute ventilation will induce complex cardiopulmonary interactions and judicious volume supplementation may not be well tolerated by a poorly functioning right ventricle operating at the top of it Starling's curve/ performance. A red cell transfusion will supplement oxygen carrying capacity.

Modest hypothermia

Modest hypothermia has been shown to reduce the rate of automatic tachycardia's such as JET. It also will minimise cellular metabolic activity and may simultaneously reduce endogenous catecholamine release. Although poorly evidenced, titrated degrees of hypothermia may sequentially reduce the JET rate, although there is no correlation between temperature reduction and rate reduction. Antipyretics should be considered, prior to using ice packs, cooling blankets or bladder/gastric irrigation with cold saline. However, hypothermia will increase the systemic vascular resistance which will increase the myocardial stroke work index and may further compromise cardiac output. Shivering will increase cellular metabolic demand. Hypothermia is associated with impaired immunity, impaired healing, deranged coagulation and increased risk of arrhythmias on re-warming. In view of these potential adverse effects, we recommend three target ranges depending upon clinical effectiveness (see flow chart)

Avoidance of inotropes

The use of inotropic drugs postoperatively is associated with an increased risk of JET due to the increase in cardiac systolic wall stress and heart rate, and may be dose dependent. Dopamine, in particular, has been shown to have a direct arrhythmogenic effect. As far as possible, inotropic support should be kept to a minimum in the immediate postoperative period. In the presence of over-vasodilation from an often co-existing 'systemic inflammatory response', the cautious introduction of vasoconstriction may permit a reduction of direct inotropic support. However, this is a potentially dangerous strategy as the dramatically detrimental effects of vasoconstriction causing an elevation in systemic vascular resistance and an increased myocardial workload cannot be overstated.

Antiarrhythmic drugs

All antiarrhythmic drugs are myocardial suppressants. Although many classes of antiarrhythmic drugs have been used in the treatment of JET, amiodarone is widely felt to be the safest and most effective agent. Its mechanism of action is a prolongation of the duration of the action potential and refractory period of all myocardial cells, and non-competitive α- and β-adrenergic inhibition (Vaughan Williams' class III, and may be considered as having calcium channel blocking and β-blocking effects). However, Amiodarone has potentially serious side effects. It is a negative inotrope, with a slow onset of action and a very long half-life. It is also associated with arrhythmias - sinus bradycardia or atrio-ventricular block. Excessive amiodarone can lead to profound bradycardia and has resulted in the need for ECLS. Late sinus bradycardia has been documented following the cessation of amiodarone but is usually transient and easily treated with external pacing. Amiodarone may cause hypothyroidism. Recent reviews of the usage of amiodarone suggest that a loading dose of 5mg/kg over 1-4 hours, followed by an infusion of 10 micrograms/kg/min (range 5-15 micrograms/kg/min) should be considered, however in view of the potential haemodynamic consequences, the risk:benefit ratio of the need for the loading dose, the rate that this is infused, and the maintenance infusion rate should be agreed by the bedside consultant led clinical team.

Esmolol is a short acting beta blocker which reduces the rate of JET by increasing AV block. However, beta blockers may reduce systolic function and esmolol is generally only considered in refractory cases of JET. Its consideration should therefore raise concerns about the need for further support such as ECMO.

Overdrive atrial or atrio-ventricular sequential pacing to restore AV synchrony

Restoration of AV synchrony may be achieved by pacing, higher than the JET rate. Even if the JET rate is successfully reduced by following the proposed algorithm, pacing to restore AV synchrony is still recommended as the addition of atrial systole to ventricular filling will augment the cardiac output. Paradoxically, establishing synchrony may reduce myocardial oxygen consumption even though the external pacing rate will be higher than the therapeutically suppressed JET rate. More contentiously, re-establishing A-V synchrony may protect against further degeneration in cardiac rhythm. Atrial pacing alone may be associated with a long PR interval or even Wenckebach due to the effects of trauma, oedema, drugs and hypothermia on the AV node. This problem will be over-come by 'AV sequential pacing' (DDD mode) but results in ventricular pacing with ventricular dysynchrony. The best pacing mode must be determined for the individual patient depending on the presence of AV block and the haemodynamics in each mode.

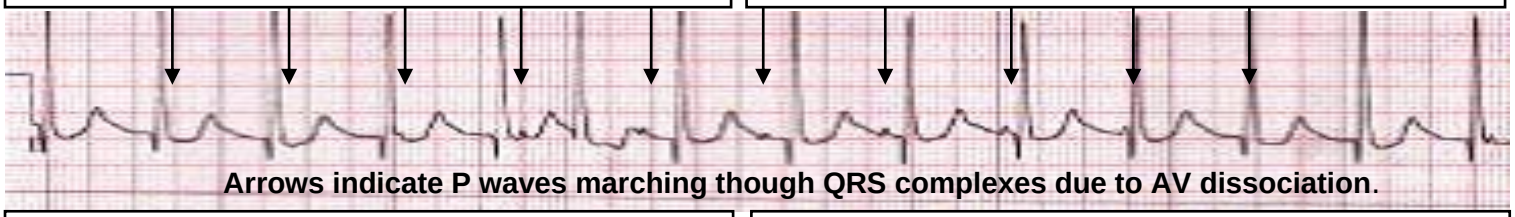
Extracorporeal life support

JET can be life threatening and all therapeutic strategies may further compromise cardiac output. ECLS should be considered in refractory cases with compromised cardiac output.

2.3 Post –operative Junctional Ectopic Tachycardia (JET)

JET is:
- the most common tachyachyarrhythmia in children post cardiac surgery;
- accounts for 5-10% of postoperative patients;
- self-limiting;
- typically occurring within 72 hours after surgery;
- significantly associated with increased morbidity;
- failure to recognize and treat can lead to rapid haemodynamic deterioration;

Be suspicious of JET if:
- persistent tachycardia, usually > 170bpm;
- neonatal or infant age groups;
- a patient is febrile and/or had a prolonged CPB time;
- a patient has had repair of such congenital defects as AV canal, VSD's, TOF or TGA. JET has also been described in extra cardiac surgeries E.g.: Fontan, BT shunts;
- ECG identified AV dissociation with faster ventricular than atrial rate, or there is retrograde 1:1 conduction;
- JET normally exhibits a 'warming up/cooling down phase';



Arrows indicate P waves marching though QRS complexes due to AV dissociation.

Diagnosis:
- Echo to rule out cardiac tamponade; if in doubt Adenosine or a fluid bolus can help to distinguish other types of tachycardia/arrhythmia
- 12 lead ECG's show narrow QRS complexes without bundle branch block,
- perform atrial study: attach atrial pacing wires to V2 lead
- use 3 lead mode using lead, aVF and V2 to:
  - confirm atrial depolarisation, to define the relationship between atrial and ventricular activity and to define wide QRS complex tachycardias,
- P waves can be seen 'marching' through QRS complexes, the ventricular rate is often faster than the atrial rate and sometimes there is 1:1 VA conduction.

Rationale for treatment:
- JET causes Low Cardiac Output State (LCOS) due to
  - loss of AV synchrony;
  - increased myocardial oxygen consumption due to tachycardia
  - limited oxygen delivery due to impaired ventricular filling (loss of atrial contribution and short diastole)
- Patients with JET have increased ventilation time, ICU, hospital length of stay and higher mortality rates

What does not work (does not cardiovert):
- Digoxin, Beta-blockers, Calcium Channel Blockers, DC Cardioversion. (but these medications could help slowing down tachycardia, and allowing for pacing)

General Treatment Measures:
- Notify duty Consultant Intensivist, Cardiologist, Surgeon.
- Combination of mild hypothermia (35-36dgrC) and Amiodarone is the most effective way of slowing down the tachycardia. AV synchrony can be restored by various pacing modes.
- **Provide adequate analgesia and sedation**, this helps to control release of internal inotropes and with management of LCOS (decreases oxygen consumption). Deep sedation, and paralysis with prevent shivering when cooled,
- **Cool to 35-36 degrees** using passive therapies or extracorporeal methods, cooling ↓HR. Avoid hyperthermia (use NSAID's with caution as they may inhibit platelet function, risk of AKI with LCOS),
- **Optimise** ionised calcium >1.2, potassium >4.0 and magnesium >1.0 mmol/l; correct hypovolemia and address acidosis
- **Reduce inotropes** to as low as tolerated, careful introduction of vasoconstrictors (Noradrenaline, Vasopressin) may allow for reduction of direct inotropes - especially avoid Dopamine, minimize Adrenaline if possible
- Observe for markers of LCOS - widening arterial and venous saturations, increasing lactic acidosis.

Treatment escalation: No consensus, various options available.

Do not attempt to pace until the heart rate has lowered.
- Pace atrium at 10-20bpm above the JET rate; if AV function impaired, use AV sequential pacing,
- Use Amiodarone as 1st line pharmacology therapy (avoid infusing both Amiodarone and Dexmedetomidine as the combination could cause profound bradycardia),
- Avoid loading dose of Amiodarone: can destabilise impaired haemodynamics (Amiodarone is cardiodepressant)
- Give Amiodarone centrally if possible, a maintenance infusion will be 5-10 mcg/kg/min
- For children <20kgs prepare infusion: 15mg/kg into 50mls diluted with 5% Glucose (do not use 0.9% sodium chloride)
  For children >20kgs prepare infusion: 300mg in 50mls, rate 5-10 mcg/kg/min
- For peripheral infusion, dilute to between 600mcg/ml and 2mg/ml. **See local guidance for monitoring of Amiodarone.**
- Side effects: ↓HR, ↓BP, AV block, liver dysfunction check TFTs, LFT.
- Send baseline TFT

Other considerations: MDT to consider 2nd line adjuncts, such as Flecainide, Procainamide or Ivabradine, and ECMO. Reduction in systolic function might lead to haemodynamic deterioration. Beta blockers may an option - by increasing AV block, JET rate is reduced. If these measures fail, initiate ECMO.

3. Education and Training

None

4. Monitoring Compliance

What will be measured to monitor compliance	How will compliance be monitored	Monitoring Lead	Frequency	Reporting arrangements
Treatment algorithm followed and documented	Audit	Consultant	2 Yearly	CPM

5. Supporting References

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6. Key Words

Junctional ectopic tachycardia (JET), Postoperative, Tachyarrhythmia

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The Trust recognises the diversity of the local community it serves. Our aim therefore is to provide a safe environment free from discrimination and treat all individuals fairly with dignity and appropriately according to their needs.
As part of its development, this policy and its impact on equality have been reviewed and no detriment was identified.

CONTACT AND REVIEW DETAILS	
Guideline Lead (Name and Title) James Whitelaw	**Executive Lead** **Chief Medical Officer**
Details of Changes made during review: Added post –operative JET guidance	