IRISH ASSOCIATION FOR EMERGENCY MEDICINE

IAEM Clinical Guideline

Treatment of Supraventricular Tachycardia in Adult Patients in the Emergency Department

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DISCLAIMER

IAEM recognises that patients, their situations, Emergency Departments and staff all vary. These guidelines cannot cover all clinical scenarios. The ultimate responsibility for the interpretation and application of these guidelines, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

Revision History

Date	Version	Section	Summary of changes	Author
August 2025	V1.0	All	Final Version	DC, JF
August 2025	V1.1	Front cover	Updated Lead Reviewer information	C. Briant

CONTENTS

GLOSSARY OF TERMS

ABCDE	Airway, Breathing, Circulation, Disability, Exposure
ACLS	Advanced Cardiac Life Support
AHA	American Heart Association
AV	Atrioventricular
AVNRT	Atrioventricular Nodal Re-entrant Tachycardia
BP	Blood Pressure
CCB	Calcium Channel Blocker
DCCV	Direct-current Cardioversion
ECG	Electrocardiogram
ED	Emergency Department
EM	Emergency Medicine
$EtCO_2$	End-tidal Carbon Dioxide
HFrEF	Heart Failure with Reduced Ejection Fraction
IV	Intravenous
mmHg	Millimetres of Mercury
PAC	Premature Atrial Contraction
RBBB	Right Bundle Branch Block
SpO_2	Oxygen Saturation
SVT	Supraventricular Tachycardia
WPW	Wolfe-Parkinson-White Syndrome

PARAMETERS

Target audience	Emergency Medicine doctors involved in the management of adult patients presenting with supraventricular tachycardia (SVT).
Patient population	Adult patients in the Emergency Department with SVT.
Exclusion criteria	Paediatric patients (under 16 years of age) Atrial tachycardia Broad complex tachycardia

AIMS

The aim of this document is to provide guidance for Emergency Medicine doctors in Ireland treating patients with SVT and to standardize care in line with current evidence.

Treatment of Supraventricular Tachycardia in adult patients in the Emergency Department

INTRODUCTION

Supraventricular tachycardia (SVT), by definition, covers a range of tachyarrhythmias originating at or proximal to the atrioventricular (AV) node. This encompasses the atrial tachycardias (e.g. atrial fibrillation, atrial flutter, multifocal atrial tachycardia) as well as the atrioventricular tachycardias. The term SVT is commonly used in the Emergency Department (ED) synonymously with atrioventricular nodal re-entrant tachycardia (AVNRT), and this guideline aims to guide treatment of AVNRT specifically and offer an approach to SVT in general.

AVNRT represents the most common regular, sustained, paroxysmal SVT. It is more common in females (approximately 70% of presentations). Its onset can occur at any age, however it is most commonly noted in the 4th and 5th decades of life. The most common symptoms reported in the literature are: palpitations (98%), dizziness (78%), dyspnoea (47%), chest pain (38%), fatigue (19%), syncope (16%).

AVNRT occurs as a result of an abnormal re-entrant circuit at the AV node. The exact anatomy and mechanism involved is not known. AVNRT is usually a narrow complex (QRS <120ms) tachycardia, unless there is a pre-existing conduction delay or a rare rate-related aberrant conduction that usually results in a right bundle branch block (RBBB) morphology.

The main consideration in the treatment of the patient with SVT is their haemodynamic status. If there is haemodynamic instability, immediate synchronised direct-current cardioversion (DCCV) is indicated.

ASSESSMENT AND DIAGNOSIS

Initial assessment should follow the standard ABCDE approach with appropriate adjuncts and monitoring applied as required (i.e. oxygen if SpO_2 <94%, $EtCO_2$ monitoring, obtain IV access, continuous ECG monitoring, BP and a 12-lead ECG.)

It is important to identify and treat reversible causes of a sinus tachycardia (hypovolaemia, electrolyte abnormalities) and recognise if there is haemodynamic instability (i.e. hypotension, altered mental status, ischaemic chest pain, acute heart failure/flash pulmonary oedema, shock) as a result of either a narrow-complex or broad-complex tachyarrhythmia since immediate synchronised DCCV is indicated.

All haemodynamically stable patients should have a thorough but focused history and examination, this can be undertaken while the patient is being connected to monitoring (BP, continuous ECG monitoring, 12-lead ECG, oxygen saturations, $EtCO_2$ monitoring). While IV access is being obtained it is recommended that a full blood count, renal function and electrolytes are requested and thyroid function bloods and other relevant tests like calcium, magnesium and phosphate be requested in clinical context.

A point of care venous blood gas will reveal any abnormal reversible electrolyte abnormalities. Patients with AVNRT will generally describe a sudden onset (and sudden termination if self-terminated) of symptoms, sometimes associated with a change in position. They will generally be aware of the fast, regular nature of their pulse. A rate of 140-280 bpm is usually demonstrated on 12 lead ECG. While rates slower than this are possible, they are rare.

There are two main subtypes of AVNRT: Slow-Fast AVNRT and Fast-Slow AVNRT. These subtypes are best understood in the context of their presumed pathophysiology. While differentiating between these subtypes has no impact on patient management, an understanding of the basic concepts of their pathophysiology helps to understand the potential variance seen in ECGs. There are two conduction pathways in the AV node, the fast and the

slow pathway. Normally, anterograde impulses travel down both the fast and slow pathways simultaneously, with the fast impulse arriving at the distal end of the slow pathway prior to the slow impulse, thus cancelling out the slow impulse.

Typical AVNRT – Slow-Fast (>90%)

This is the most common form of AVNRT. It is initiated when an impulse originating from a premature atrial contraction (PAC) arrives at the AV node during a refractory period for the fast pathway, but is conducted anterograde down the slow pathway. If this impulse arrives at the distal end of the fast pathway, and the fast pathway is no longer refractory, the impulse can continue retrograde up the fast pathway, thus initiating a cycle of tachycardia.

ECG findings with Typical AVNRT include

- P waves may be entirely buried in the QRS complex
- P waves appearing late in the QRS cycle may appear as a pseudo R' wave in V1 or V2 and as a pseudo S waves in leads II, III or aVF
- Possible rate related ST depression
- May see QRS alternans
- May see R-R interval variation

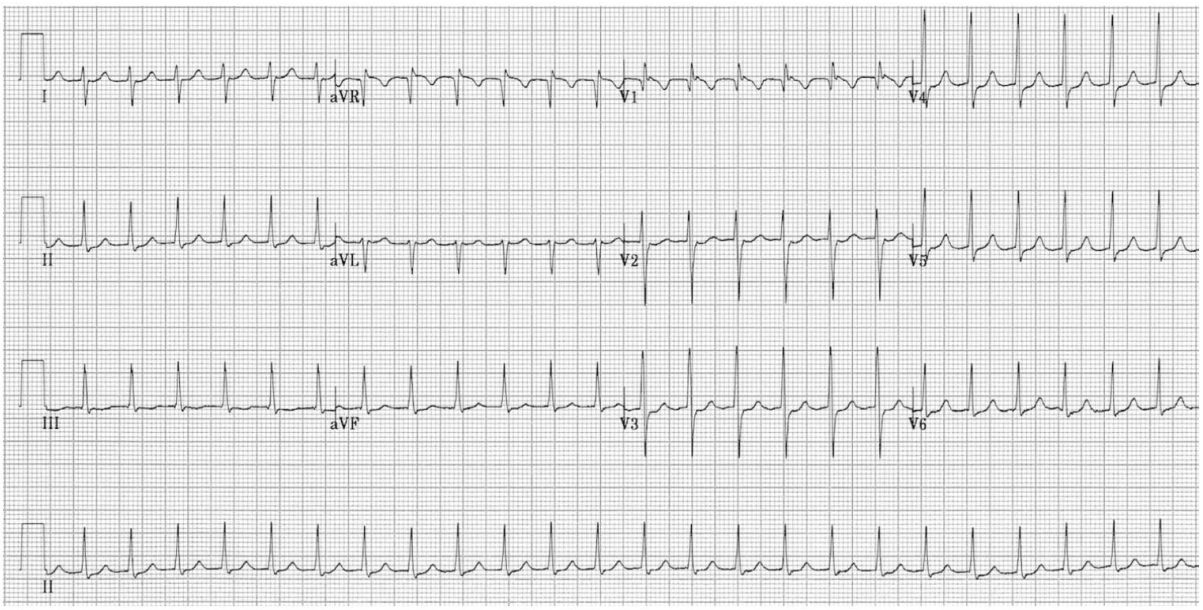

Figure 1: ECG example of a Slow-Fast AVNRT

The ECG in Figure 1 is an example of a Slow-Fast AVNRT, demonstrating the absence of clear P-waves, but the presence of pseudo R' waves in V1 and V2 as subtle positive deflections immediately following the QRS complex.

Atypical AVNRT – Fast-Slow (~6%)

This variant is commonly initiated by the retrograde propagation of a premature ventricular contraction (PVC) up the slow pathway while the fast pathway is refractory, with this impulse then propagating back down the fast pathway anterograde, thus completing the circuit. It is rare for this arrythmia to be sustained. This results in a relatively delayed atrial contraction as the retrograde impulse is propagating up the slow pathway, manifesting as delayed P waves visible distinct from the QRS complex. This usually results in a prolonged R-P interval and the resulting P wave often appears closer to the next QRS complex than to the QRS it originated with.

ECG Findings with Atypical AVNRT include

- Distinct P waves, often with a prolonged R-P interval
- Inverted P waves in II, III aVF, V6 and positive P waves in V1
- Possible rate related ST depression
- May see QRS alternans
- May see R-R interval variation

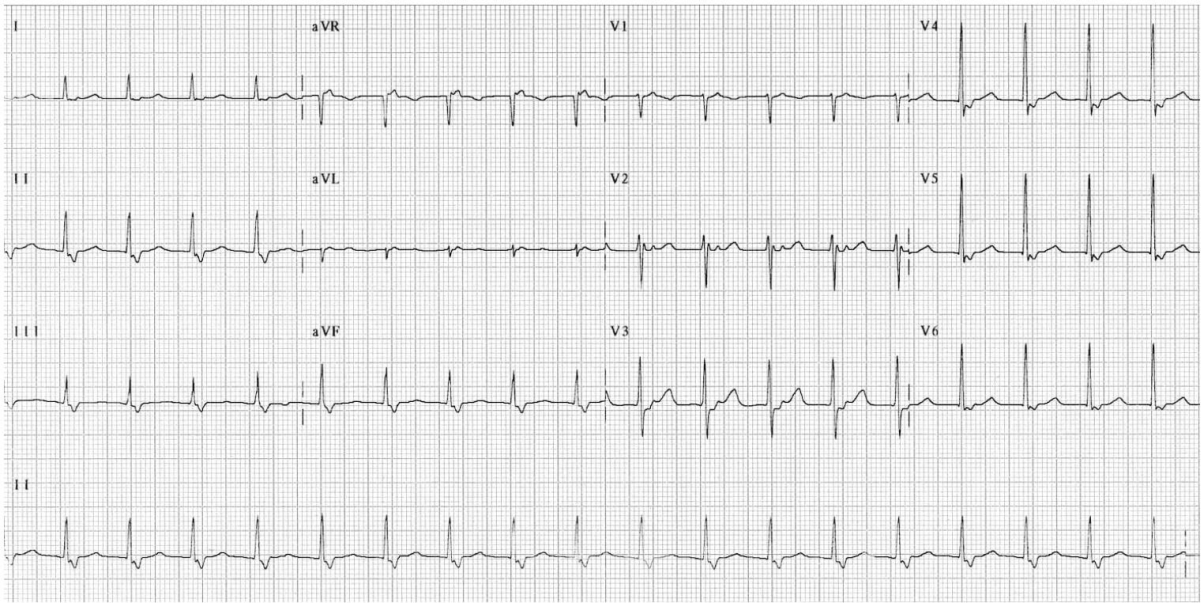

Figure 2: ECG example of Fast-Slow AVNRT

The ECG in Figure 2 is an example of a Fast-Slow AVNRT, where distinct retrograde P-waves are best seen in V2, and these P-waves are inverted in leads II, III, aVF and V6 while positive in V1.

TREATMENT

The following recommendations are all written with primary consideration given to the American Heart Association Guideline for the Management of Adults with Supraventricular Tachycardia and are also in accordance with The European Resuscitation Council Guidelines 2021: Adult advanced life support, the Resuscitation Council UK Guidelines 2021: Adult tachycardia algorithm and The European Society of Cardiology guidance.

Confirmed AVNRT can be managed with the same principles and treatment algorithm as the management of acute narrow-complex tachycardia without an established diagnosis (Appendix 1). Broad complex tachyarrhythmias follow a different treatment algorithm which is also summarised for reference (Appendix 2).

Unstable Patient

The presence of haemodynamic instability (i.e., hypotension, altered mental status, ischaemic chest pain, acute heart failure/flash pulmonary oedema, shock) as a result of either a narrow-complex or broad-complex tachyarrhythmia, is an indication for immediate synchronised DCCV.

Expert help must be sought as soon as possible.

Initial energy dosing recommendations vary, the American Heart Association Advanced Cardiac Life Support recommends referring to your specific devices' recommended energy level and the European Resuscitation Council recommends 70-120 Joules. This is delivered for up to 3 attempts with increasing energy levels. Analgesia and sedation are recommended prior to the DCCV of a conscious patient.

If DCCV is unsuccessful and haemodynamic instability persists, amiodarone 150 - 300mg IV over 10-20 minutes is given as the next line of treatment, followed by either an initial infusion

of amiodarone 1mg/minute for the first 6 hours or amiodarone 900mg IV infusion over 24 hours. Further attempts at DCCV may be made.

Stable Patient

1. Vagal Manoeuvres

The first line treatment of a stable AVNRT or any narrow complex tachyarrhythmia should be the modified Valsalva manoeuvre. The modified manoeuvre was described in the REVERT trial which demonstrated an increased rate of return to normal sinus rhythm after one minute in 47% of their modified group vs 17% in the standard treatment group.

The patient is positioned in semi-recumbent position and asked to blow into a 20ml syringe with enough force to move the plunger for 15 seconds. Some studies have calculated this to be approximately 40 mmHg. After this straining, the head of the bed can be lowered to move the patient supine, and the legs passively elevated to above 45 degrees and held for up to 45 seconds (Figure 3).

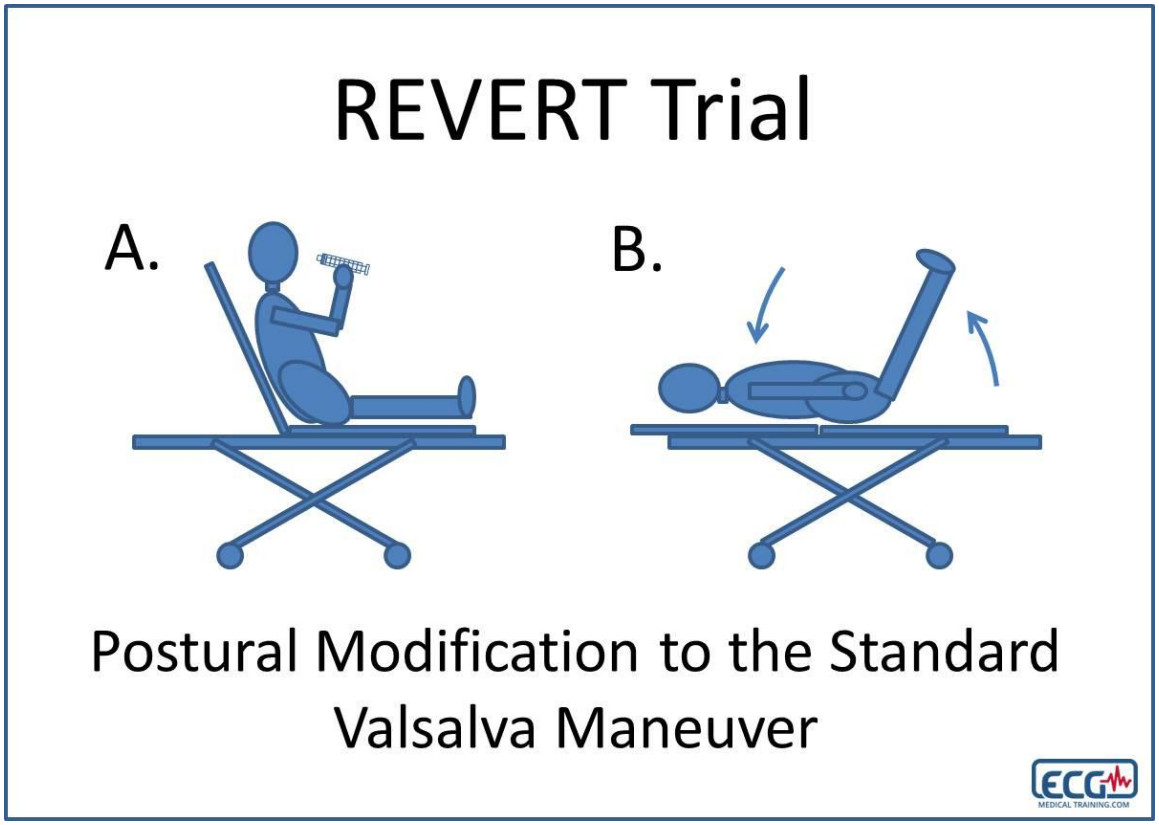

Figure 3: Image demonstrating the Modified Valsalva Manoeuvre

2. Adenosine

Adenosine is second line for the treatment of narrow complex tachycardia, provided that there is no evidence of pre-excitation (delta waves on ECG).

Pre-excitation occurs when antegrade depolarisation occurs via an accessory pathway as well as via the AV node. The accessory pathway depolarises the ventricle via a non-specialised conduction pathway, causing relatively slow depolarisation, resulting in the delta wave. Then as the other wave of depolarisation propagates beyond the AV node down the faster, specialised conducting pathways, this results in rapid depolarisation and the subsequent QRS complex succeeding the delta wave. AV node blockers risk leaving only the accessory pathways active, with potential for a poorly organised ventricular depolarisation, which can deteriorate into ventricular fibrillation.

Bronchoconstriction is another rare but recognised complication of adenosine administration. It should only be given in settings capable of managing such complications (i.e. the resuscitation room). Bronchoconstriction can occur in patients with or without respiratory disease. Whilst adenosine can be used cautiously in patients with known asthma, verapamil should be considered in patients with known severe asthma.

Adenosine has a relatively short half-life of less than 10 seconds. It should be given via a wide-bore cannula placed in the antecubital fossa, and immediately flushed with a 20ml fluid bolus, ideally via a 3-way tap.

Patients must be connected to monitoring and a defibrillator, including a continuous ECG rhythm strip. Following treatment, a review of this strip may unmask a different underlying rhythm such as atrial flutter and thus change the forward management plan.

Adenosine dosing:

- Initial dose 6mg adenosine IV.
- If unsuccessful, 12mg adenosine IV can be given a minute after first dose.
- If still unsuccessful, a third and final dose of 18mg adenosine IV can be given one minute after the previous dose.
- If administered via a central line then the initial dose should be reduced to 3mg adenosine IV.

3. Calcium Channel Blocker (CCB) OR Beta-blocker

Third line treatment is with either a calcium channel blocker (verapamil or diltiazem) or a beta-blocker (metoprolol or esmolol). However, these classes should not be used together because their combination risks potentiating severe hypotension and/or bradycardia.

Calcium Channel Blockers

- Verapamil 5mg – 10mg (0.075-0.15mg/kg) given IV over 2 minutes.

CCB have an associated risk of hypotension and are contraindicated in patients with haemodynamic instability, heart failure with a reduced ejection fraction (HFrEF) of less than 40%, suspected ventricular tachycardia (VT) or suspected or known pre-excitation (i.e. delta waves, or known Wolff-Parkinson-White).

Beta-blockers

- Metoprolol 2.5mg IV over 2 minutes or 50mg orally.

If a CCB has not been given, the use of a beta-blocker such as metoprolol can be considered given the relatively favourable safety profile in a haemodynamically stable patient. Beta-blockers are more commonly associated with a reduction in the rate of tachycardia as opposed to termination of the arrhythmia. They are contraindicated in patients with known heart failure, or evident failure in the ED.

SPECIAL CONSIDERATIONS

Special Considerations in Pregnancy

SVT occurs frequently in pregnancy (23/100,000 pregnancies) and is associated with an increased risk of death during pregnancy. This increased susceptibility to arrhythmia in pregnancy is attributed to several adaptations of the cardiovascular system that occur during pregnancy. These include increased heart rate, cardiac output, plasma catecholamine concentrations, adrenergic receptor sensitivity and increased intravascular volumes resulting in increased end-diastolic volumes and atrial stretch, along with a reduction in systemic resistance. Expert help must be sought for any pregnant patient presenting with SVT.

The initial treatment is similar to that of the general population. Unstable patients require immediate synchronised DCCV (Follow device recommendations or 70-120J). In the stable patient the first line treatment is vagal manoeuvres. Adenosine (the same dosing regimen of 6mg then 12mg then 18mg) is safe for use as a second line agent.[3] Due to paucity of evidence and potential teratogenicity of antiarrhythmic agents, any further treatment decisions will require expert opinion from appropriate specialists.

Special Considerations in Adults with Congenital Heart Disease

Treatment will be influenced by the underlying anatomy and surgical history, therefore expert specialist opinion should be sought in the treatment of any adult with congenital heart disease presenting to the ED with SVT. However, in line with previous recommendations, a haemodynamically unstable adult with congenital heart disease in SVT should be treated with immediate synchronised DCCV (following device recommendations or 70-120J initially).

Adults with congenital heart disease represent a group of patients with heterogenous anatomy and pathology. While their treatment, *in general*, is similar to those with no congenital heart

disease, it is recommended that expert specialist opinion is sought prior to chemical cardioversion of the stable patient.

Special Considerations in Adults with Wolff-Parkinson-White

As previously discussed, the initial treatment is the same as for the general population and in the event of signs of shock, a haemodynamically unstable adult with WPW and SVT should be treated with immediate synchronised DCCV (following device recommendations or 70-120J initially).[4,6]

The stable patient with WPW and SVT can be managed with the same principles as those without WPW. Orthodromic AVRT (anterograde conduction via the AV node), which accounts for approx. 95% of cases in WPW will manifest as a narrow complex (QRS <120ms) SVT and in these instances the treatment recommendations are the same as for the general population, namely vagal manoeuvres, adenosine and then beta-blockers/calcium channel blockers with expert input.[6] Orthodromic AVRT is distinguishable from the less common, antidromic AVRT, describes anterograde conduction via the accessory pathways resulting in regular, wide complex tachycardia, which can be difficult to distinguish from ventricular tachycardia and should be managed as a wide complex tachycardia, with expert input.

Although adenosine carries a small risk of precipitating atrial fibrillation in all patient, the presence of accessory pathways increases the risk of rapid ventricular conduction and potential deterioration to ventricular fibrillation. All patients receiving adenosine should be monitored with a connected defibrillator; however, this precaution is critical in those with known or suspected accessory pathways. Defibrillator pads should be applied and connected prior to adenosine administration.

IMPLICATION FOR DRIVING

SVT has implications on a driver's permission to drive a vehicle, in accordance with the National Sláinte agus Tiomáint, Medical Fitness to Drive Guidelines (https://www.ndls.ie/images/PDF_Documents/Slainte_agus_Tiomaint_Medical_Fitness_to_Drive_Guidelines.pdf).

Group 1 Entitlement drivers	**Group 2 Entitlement drivers**
Are not permitted to drive if the arrhythmia has caused or is likely to cause incapacity Are permitted to drive provided: - The condition has been effectively treated (i.e. the underlying cause has been identified and controlled for at least 4 weeks - Competent medical authority has been obtained - Where appropriate, regular medical assessment is conducted - There is no other disqualifying condition NDLS does not need to be notified unless there are distracting/disabling symptoms	Are not permitted to drive if the arrhythmia has caused or is likely to cause incapacity Are permitted to drive provided: - The condition has been effectively treated (i.e. the underlying cause has been identified and controlled for at least 3 months - The LVEF is ≥ 35% - Competent medical authority has been obtained - Where appropriate, regular medical assessment is conducted - There is no other disqualifying condition NDLS does not need to be notified unless there are distracting/disabling symptoms

Table 1

COMPANION DOCUMENTS

Appendix 1: Narrow complex tachycardia algorithm

Appendix 2: Adult tachycardia treatment algorithm

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