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Guidelines on the management of patients presenting with palpitations

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These guidelines are based predominantly on:

2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC): Developed in collaboration with the Association for European Paediatric and Congenital Cardiology (AEPC). European Heart Journal (2020) 41, 655-720.

2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death European Heart Journal (2015)36, 2793–2867.

1.1 Abbreviations:

AF	Atrial fibrillation
AT	Atrial Tachycardia
AVN	Atrioventricular node
AVNRT	Atrioventricular nodal re-entrant tachycardia
AVRT	Atrioventricular re-entrant tachycardia
BBB	Bundle branch block
BHSCT	Belfast Health and Social Care Trust
BNP	Brain natriuretic peptide
BrS	Brugada Syndrome
CAD	Coronary artery disease
DVLA	Driver and Vehicle licencing agency
ECG	Electrocardiogram
EPS	Electrophysiology Study
EST	Exercise Stress Test
FBP	Full blood picture
HF	Heart failure
HFrEF	Heart failure with reduced ejection fraction
HTN	Hypertension
ICD	Implantable cardiac defibrillator
IHD	Ischaemic heart disease
IST	Inappropriate sinus tachycardia
LQTS	Long QT syndrome
PND	Paroxysmal nocturnal dyspnoea
PPM	Permanent pacemaker
PVT	Polymorphic VT
RVOT	Right ventricular outflow tract
SCD	Sudden cardiac death
SHSCT	Southern Health and Social Care Trust
ST	Sinus tachycardia
SVT	Supraventricular Tachycardia
TFT	Thyroid function tests
TTE	Transthoracic echocardiogram
U+E	Urea and Electrolytes
VA	Ventricular Arrhythmia
VHD	Valvular heart disease
VF	Ventricular Fibrillation

These guidelines are a summary of the current guidelines and consensus documents of the ESC / EACVI and are intended for internal use within the SHSC only.

1.2 Key messages:

- ✓ It is important to establish key points when taking a history from a patient presenting with palpitations.
- ✓ Establish the frequency and duration of the palpitations and if there are any exacerbating or alleviating factors i.e. stress, caffeine or alcohol.
- ✓ Enquire family history of sudden cardiac death in <45-year olds
- ✓ Assess for abnormal clinical examination
- ✓ Assess ECG for abnormalities of cardiac structure or electrophysiology
- ✓ Attempt to capture symptoms with ambulatory monitoring
- ✓ Request echocardiography / cardiac MRI only in specific cases
- ✓ Atrial and ventricular ectopy can often be diagnosed by history alone and usual respond to lifestyle changes and / or beta blocker therapy
- ✓ This document discusses other causes of palpitations in specific sections

2. Introduction

Palpitations are a common presenting complaint in primary and secondary care. Distinguishing between benign and serious causes of palpitations can often prove challenging to the physician. These guidelines aim to support and direct the initial investigation and management of patients presenting with palpitations.

3. Purpose of this policy

This policy aims to assist the attending health care professionals in the diagnosis and management of patients presenting with palpitations in both the acute and chronic setting.

4. Scope

This document provides guidance for healthcare professionals involved in the clinical management of patients presenting to primary or secondary care with palpitations. This will include:

- Consultants
- SAS doctors
- SpRs
- Junior Doctors
- Specialist Nurses
- Nursing Staff
- General Practitioners

5. Approach to Patient Presenting with Palpitations

5.1 History

- It is important to establish key points when taking a history from a patient presenting with palpitations, with particular attention paid to the high risk features described below:
 - ✓ Palpitations occurring on exertion
 - ✓ Associated with presyncope or syncope
 - ✓ Family history of cardiomyopathy or sudden cardiac death
 - ✓ Associated chest pain
 - ✓ Features of decompensated HF (orthopnoea, PND, peripheral oedema)
 - ✓ Past medical history of cardiac disease including documented arrhythmia, CAD, VHD, HF, congenital heart disease and structural heart disease

- Establish the frequency and duration of the palpitations and if there are any exacerbating or alleviating factors i.e. stress, caffeine or alcohol.

- Certain features in the history may be lower risk and point towards ectopic beats being the cause of the palpitations. These features include:
 - ✓ Increased awareness of palpitations at rest, especially in bed
 - ✓ Very short-lasting palpitations described as “skipped beats” or “dropped beats”

5.2 Examination

- **General Observation**
 - ✓ Does the patient have features of an underlying genetic condition? Or features of hyperthyroidism such as sweaty palms, exophthalmos or pretibial myxoedema?

➤ **Auscultation**

- ✓ Listen carefully for systolic or diastolic murmurs. Listen for possible third heart sound (gallop rhythm) heard in heart failure
- ✓ Auscultate the lungs for features of pulmonary oedema/pleural effusion and examine the JVP, abdomen and legs for peripheral oedema

5.3 Investigations

➤ **Bloods**

FBP, U+E and TFTs (HbA1C and BNP if indicated)

➤ **ECG**

(See below for examples of high-risk features on ECG)

➤ **TTE**

Request an TTE only if patient has:

- ✓ high risk features,
- ✓ an audible murmur, or
- ✓ BNP >400

➤ **Exercise Stress Test**

Request EST if patient has:

- ✓ Evidence of pre-excitation on ECG
- ✓ Angina or significant risk factors for CAD

➤ **Gain an ECG to coincide with presence of symptoms**

1. Cardiac holter monitor

- ✓ Usually 24 hours (up to 72 hours)

2. Event recorder

- ✓ Patient activated to record at time of symptoms

- ✓ Also records 'important events'
- ✓ Used for 1-4 weeks

3. Implantable loop recorder

- ✓ Surgically implanted device (see figure 1)
- ✓ Patient activated to record at time of symptoms
- ✓ Also records 'important events'
- ✓ Used for up to 3 years

4. 'AliveCor' device

- ✓ These devices can be purchased by patients and used to record a rhythm strip equating to lead I on an ECG which can then be automatically analysed by the Alive Cor software and / or sent by PDF to a patients cardiologist (see appendix 2).

Figure 1 – Implantable loop recorder



6. Supraventricular Tachycardia (SVT)

- An umbrella term usually used to describe all tachycardias arising from above the ventricles excluding AF (please see guidelines on AF) and ventricular tachycardia
- SVT is usually regular and can be narrow (QRS duration <120ms) or broad complex if associated with aberrant conduction (QRS duration >120ms)
- Arrhythmias usually referred to as SVT include:
 - ✓ Sinus tachycardia
 - ✓ Focal atrial tachycardia
 - ✓ Multifocal atrial tachycardia
 - ✓ Atrial Flutter (see AF guidelines)
 - ✓ AVNRT
 - ✓ AVRT

6.1 Sinus Tachycardia (ST)

- Sinus rate >100 b.p.m
- P wave is positive in leads I, II, and aVF, and biphasic/negative in lead V1 on 12 lead ECG

Inappropriate sinus tachycardia (IST)

- Fast sinus rhythm (>100 b.p.m.) at rest or minimal activity that is out of proportion with the level of physical, emotional, pathological, or pharmacological stress
- Characteristically 24 h Holter monitoring demonstrates a mean heart rate >90 b.p.m. with an exaggerated heart rate response >100 b.p.m. during waking hours

Treatment

- Evaluation and treatment of reversible causes is recommended
- IST is usually not treated with medication, however if required then Ivabradine alone or in combination with a beta-blocker should be considered in symptomatic patients

6.2 Focal Atrial Tachycardia (AT)

- Organised atrial rhythm >100bpm
- Originates within the atrial but outside the sinus node
- Ventricular rate is dependant on AV nodal conduction
- P wave morphology will depend on the particular anatomic location and can be used to localise the tachycardia (p wave algorithms are beyond the scope of this document)

Treatment

Acute Management

- Haemodynamically unstable patients (chest pain, dyspnoea, BP <90/60mmHg)
 - ✓ Synchronized DC cardioversion
- Haemodynamically stable patients
 - ✓ Adenosine I.V. (6mg followed by 12mg 1 min following the last dose, and further 12mg if necessary) should be considered. Each dose must be followed by large fluid boluses of between 20-50mls of 0.9% saline.
 - ✓ Simultaneous ECG should be recorded
 - ✓ Beta-blockers (metoprolol) should be considered in the absence of decompensated HF if adenosine fails
 - ✓ Verapamil [0.075-0.15 mg/kg I.V. (average 5-10 mg) over 2 min] or I.V. diltiazem [0.25 mg/kg (average 20 mg) over 2 min should be considered for haemodynamically stable patients in the absence of hypotension or HFrEF if adenosine fails
 - ✓ Synchronized DC cardioversion is recommended if drug therapy fails to convert

Drug cautions:

- I.V. beta-blockers are contraindicated in the presence of decompensated HF.

- I.V. verapamil and diltiazem are contraindicated in the presence of hypotension or HFrEF.
- Adenosine is contraindicated in asthma / COPD / respiratory disease / dipyridamol due to the risk of bronchospasm.
- Aminophylline should be immediately available if using adenosine

Chronic Management

- Catheter ablation may be recommended for recurrent focal AT
- Beta-blockers or verapamil/diltiazem (in the absence of HFrEF), or propafenone or flecainide (in the absence of structural or IHD), should be considered if ablation is not desirable or feasible
- Ivabradine with a beta-blocker may be considered if the above measures fail
- Amiodarone may be considered if the above measures fail

6.3 Multifocal Atrial Tachycardia (MAT)

- Rapid, irregular rhythm
- At least 3 distinct morphologies of P-waves on ECG
- Commonly associated with an underlying condition, including
 - ✓ Pulmonary disease
 - ✓ Pulmonary HTN
 - ✓ CAD
 - ✓ VHD
- Distinct isoelectric period between visible P-waves (unlike AF)

Treatment

Acute Management

- Treatment of an underlying condition is recommended as a first step, if feasible
- I.V. beta-blockers, or I.V. non-dihydropyridine calcium channel blockers (verapamil or diltiazem) should be considered

Chronic Management

- Oral verapamil or diltiazem should be considered for patients with recurrent symptomatic multifocal AT in the absence of HFrEF
- A cardioselective beta-blocker should be considered for patients with recurrent symptomatic multifocal AT
- AV nodal ablation followed by pacing (preferable biventricular pacing) may be considered for patients with LV dysfunction due to recurrent multifocal AT refractory to drug therapy

6.4 Atrioventricular Nodal Re-entrant Tachycardia (AVNRT)

- Re-entry in the area of the AVN
- Narrow complex tachycardiac (QRS <120ms)
- Rate related ST segment depression may be seen during or after tachycardia
- Retrograde P-waves may be seen following every QRS, or
- P-waves may be seen before each QRS (negative in leads II, III and aVF and V6 but positive in V1)

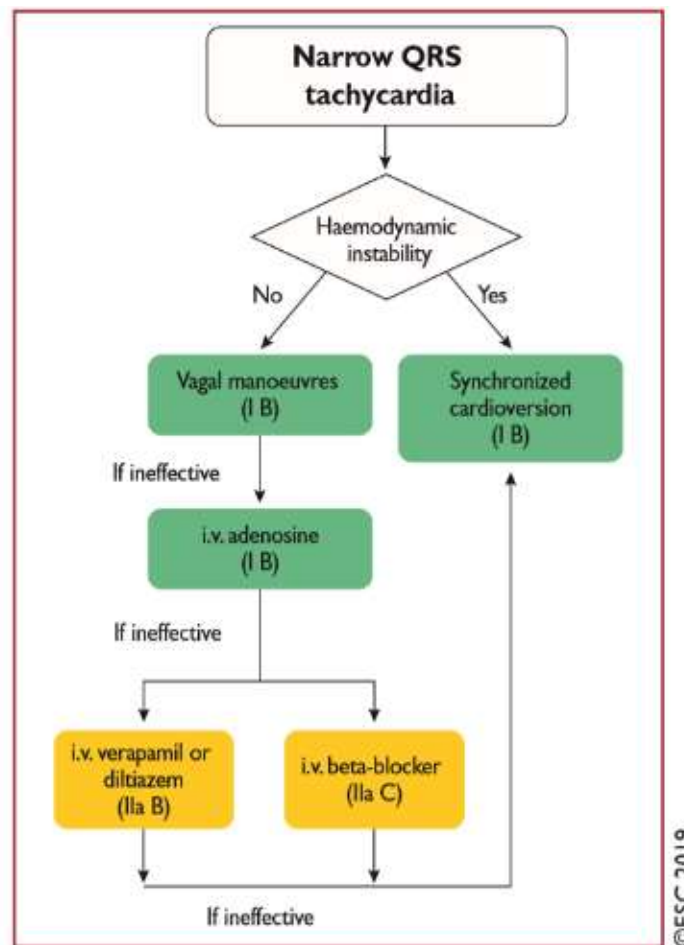
Treatment

Acute Management (See figure 2)

- Haemodynamically unstable patients
 - ✓ Synchronized DC cardioversion
- Haemodynamically stable patients
 - ✓ 12 lead ECG during tachycardia
 - ✓ Vagal manoeuvres, preferably in the supine position with leg elevation

- ✓ Adenosine I.V. (6mg followed by 12mg 1 min following the last dose, and further 12mg if necessary) if vagal manoeuvres fail. Each dose must be followed by large fluid boluses of between 20-50mls of 0.9% saline.
- ✓ Simultaneous ECG should be recorded
- ✓ Verapamil [0.075-0.15 mg/kg i.v. (average 5-10 mg) over 2 min] or i.v. diltiazem [0.25 mg/kg (average 20 mg) over 2 min (i.v.) should be considered, if vagal manoeuvres and adenosine fail or adenosine is contra-indicated
- ✓ Beta-blockers (metoprolol 2.5-15 mg given i.v. in 2.5mg boluses) should be considered if vagal manoeuvres and adenosine fail or adenosine is contra-indicated
- ✓ Synchronized direct-current cardioversion is recommended when drug therapy fails to convert or control the tachycardia

Figure 2- Acute Management of Narrow QRS Tachycardia



2019 ESC Guidelines for the management of patients with supraventricular tachycardia. The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC): Developed in collaboration with the Association for European Paediatric and Congenital Cardiology (AEPC). European Heart Journal (2020) 41, 655-720.

Chronic Management

- Catheter ablation is recommended for symptomatic, recurrent SVT
- Diltiazem or verapamil, in patients without HFrEF, or beta-blockers should be considered if ablation is not desirable or feasible
- Conservative management may be considered for minimally symptomatic patients with very infrequent, short-lived episodes of tachycardia

6.5 Atrioventricular Re-entrant Tachycardia (AVRT)

- Re-entrant circuit involves two limbs:
 1. AVN and his-purkinje system
 2. Accessory pathway (AP)

- Wolfe-Parkinson-White syndrome (WPW) is a condition whereby there is an accessory pathway leading to pre-excitation in combination with recurrent tachyarrhythmias (see delta wave figure 3)

- See figure 4 for treatment of asymptomatic pre-excitatio

Figure 3: Pre-excitation / Delta wave in WPW

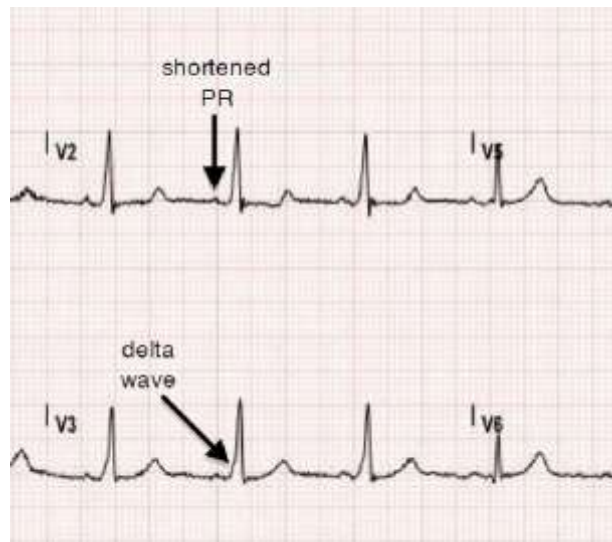
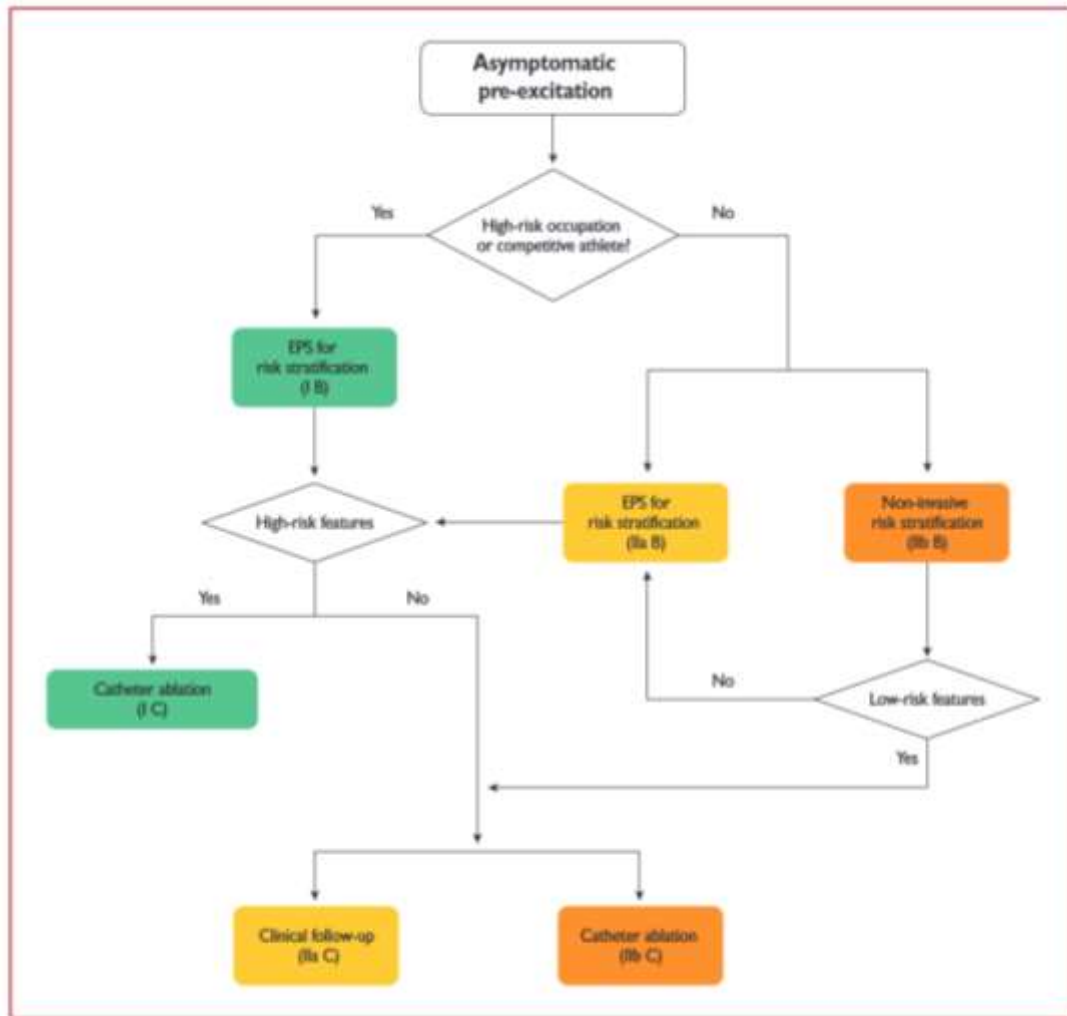


Figure 4- Management of Asymptomatic Pre-excitation



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6.51 Wolfe-Parkinson-White Syndrome

- AT leading to pre-excitation on ECG (figure 3) + recurrent tachyarrhythmias
- Typical pattern on resting ECG:
 - ✓ Short PR interval (<120ms)
 - ✓ Slurred upstroke of the QRS complex ('delta wave')
 - ✓ Wide QRS complex (>120ms), RBBB type appearance
- Usually seen in structurally normal hearts

Treatment

Acute therapy

- Haemodynamically unstable patients
 - ✓ Synchronized DC cardioversion
- Haemodynamically stable patients
 - ✓ Vagal manoeuvres, preferably in the supine position with leg elevation
 - ✓ Avoid adenosine as can precipitate VF
 - ✓ Synchronized DC cardioversion if unable to control the tachycardia

Chronic therapy

- ✓ EP studies with catheter ablation of AP(s) is recommended in patients with symptomatic, recurrent AVRT
- ✓ Beta-blockers or non-dihydropyridine calcium-channel blockers (verapamil or diltiazem in the absence of HFrEF) should be considered if no signs of pre-excitation are present on resting ECG, if ablation is not desirable or feasible
- ✓ Propafenone or flecainide may be considered in patients with AVRT and without ischaemic or structural heart disease, if ablation is not desirable or feasible. These are usually co-prescribed with a beta-blocker and in these circumstances commencement should be considered in hospital
- ✓ Digoxin, beta-blockers, diltiazem, verapamil, and amiodarone are not recommended and are potentially harmful in patients with pre-excited AF

6.52 Pre-excited Atrial fibrillation

- Pre-excited atrial fibrillation/Paroxysmal AF has been found in 50% of patients with WPW
- May be the presenting arrhythmia in affected patients
- Patients are typically young and have no structural heart disease
- High-rate AVRT may potentially initiate AF.
- Potentially life-threatening arrhythmia in patients with WPW syndrome due to potential degeneration into VF

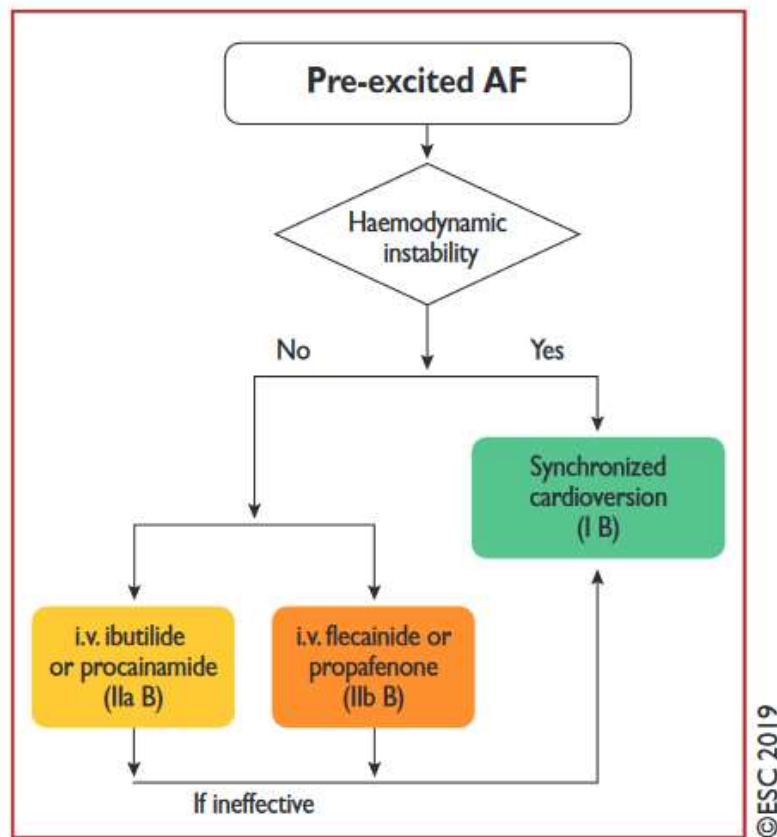
Treatment (see figure 5)

Acute

- Haemodynamically unstable patients
 - ✓ Synchronized DC cardioversion

- Haemodynamically stable patients
 - ✓ Synchronized DC cardioversion is recommended if drug therapy fails to convert or control the tachycardia
 - ✓ Adenosine is NOT recommended due to the potential induction of VF
 - ✓ Amiodarone is not recommended

Figure 5: Management of Pre-excited AF



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Chronic

- Catheter ablation is the treatment of choice for patients with symptomatic and recurrent AVRT, or pre-excited AF

7. Broad Complex Regular Tachycardia

- Wide QRS (>120ms), regular tachycardia
- Differential diagnosis includes:
 - ✓ VT
 - ✓ SVT with BBB
 - May arise due to pre-existing BBB or the development of aberrancy during tachycardia
 - ✓ SVT with conduction through an AP (pre-excited SVT)
 - ✓ SVT with widening of QRS interval induced by drugs or electrolyte disturbances
 - ✓ Pacemaker-related endless loop tachycardia and artefacts

- Correct diagnosis of VT is critical to management
- Misdiagnosis and administration of drugs usually utilised for SVT can be harmful for patients in VT
- Therefore, the default diagnosis should be VT until proven otherwise

7.1 Treatment (See Appendix 4)

A large focus should be on treating reversible factors e.g. ischaemia, heart failure, and identifying the substrate e.g. with cardiac imaging.

7.11 Acute

- Haemodynamically unstable patients
 - ✓ Synchronised DC cardioversion
 - ✓ If unsuccessful give Amiodarone 300mg I.V over 10-20 minutes and then repeat DC cardioversion
- Haemodynamically stable patients
 - ✓ Amiodarone 300mg over 20-60mins then 900mg over 24 hours (to avoid phlebitis consideration can be given to oral loading of amiodarone (see amiodarone policy))
- Correction of electrolyte abnormalities and treatment of reversible factors

7.12 Chronic

- Depends on the pathophysiology of the broad complex tachycardia
 - ✓ In structurally normal hearts
 - Approximately 70% originate from the RVOT
 - Treat only if patients are symptomatic
 - Correct electrolyte abnormalities
 - Anti-arrhythmic drug therapy
 - Beta-blockers
 - Verapamil
 - Class 1C sodium channel blockers (propafenone or flecainide)
 - Catheter ablation is recommended if treatment failure with antiarrhythmics or in patients with LV dysfunction secondary to arrhythmia
 - ✓ If secondary to left ventricular dysfunction:
 - Guideline directed HF medication (see HF policy for more details on specific drugs)
 - Withdrawal of agents which may provoke arrhythmias
 - Correct electrolyte abnormalities
 - Urgent catheter ablation for incessant VT or electrical storm
 - Oral amiodarone should be considered in combination with beta-blockers (if ablation fails or is deemed inappropriate)
 - Consideration of ICD implantation if appropriate

8. Inherited Arrhythmia syndromes

8.1 Brugada Syndrome (BrS)

- Inherited arrhythmogenic disease
- Type I is characterised by coved-type ST-segment elevation in V-1-V3 (see figure 6)
- Increased risk of SCD due to VF

- Approximately 80% of the affected individuals are men
- Onset of symptoms typically occurs at a mean age of 40

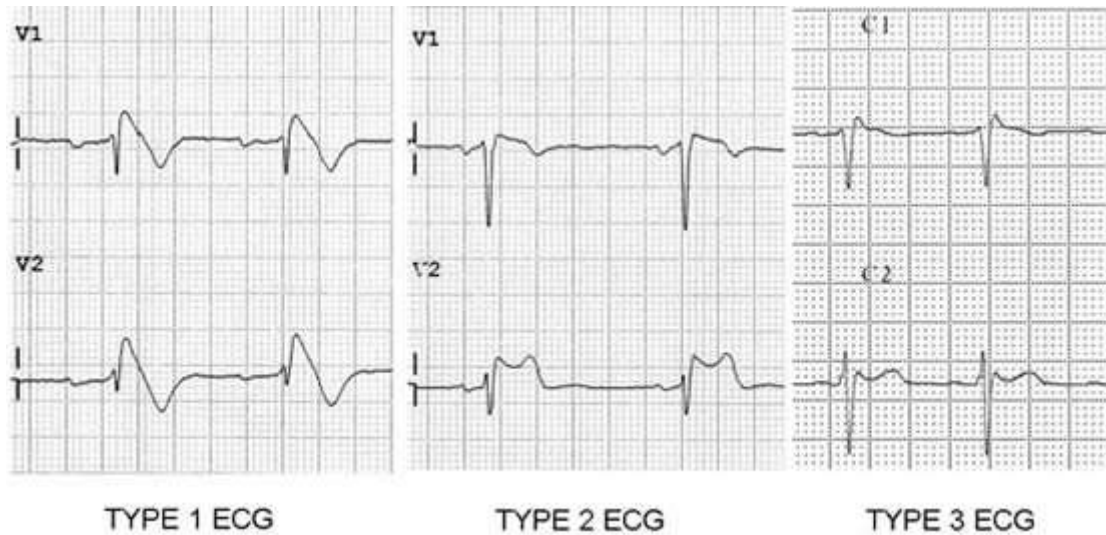
Diagnosis

- BrS is definitely diagnosed when the patient presents with:
 - ✓ A type 1 ECG (either spontaneously or elicited by a drug challenge)
and
 - ✓ At least one clinical criteria (documented VF or VT, family history SCD <45 years, coved-type ECG in family members, inducibility of VT during EP studies, syncope, nocturnal agonal respiration)
- If a type 1 ECG is observed in the absence of any clinical criteria, this should be referred to as 'idiopathic Brugada ECG pattern' and not as BrS

ECG features of Brugada Syndrome (see figure 6)

- It is recommended to place V1 and V2 leads on the second intercostal space because it may increase the sensitivity of the ECG
- There is one true diagnostic of the Brugada pattern (type 1); two others (type 2 and type 3) may suggest the disease
 - ✓ **Type 1:** Prominent coved ST-segment elevation displaying J-point amplitude or ST-segment elevation ≥ 2 mm, followed by a negative T wave (in leads V1-V3)
 - ✓ **Type 2:** ≥ 2 mm J-point elevation, ≥ 1 mm ST-segment elevation and a saddleback appearance, followed by a positive or biphasic T-wave (in leads V1-V3)
 - ✓ **Type 3:** Either a saddleback or coved appearance, but with an ST-segment elevation < 1 mm (in leads V1-V3)

Figure 6: ECG features of Brugada Syndrome



Drug challenge

- Indicated in cases in which the disease is suspected, but in which the resting ECG is normal e.g.
 - ✓ Familial screening, or
 - ✓ Suspicious, but not diagnostic (types 2 or 3)

- Flecainide, ajmaline, procainamide, disopyramide, propafenone and pilsicainide have been used to unmask

- A drug challenge is only considered positive when a conversion to the diagnostic type 1 occurs

Clinical criteria

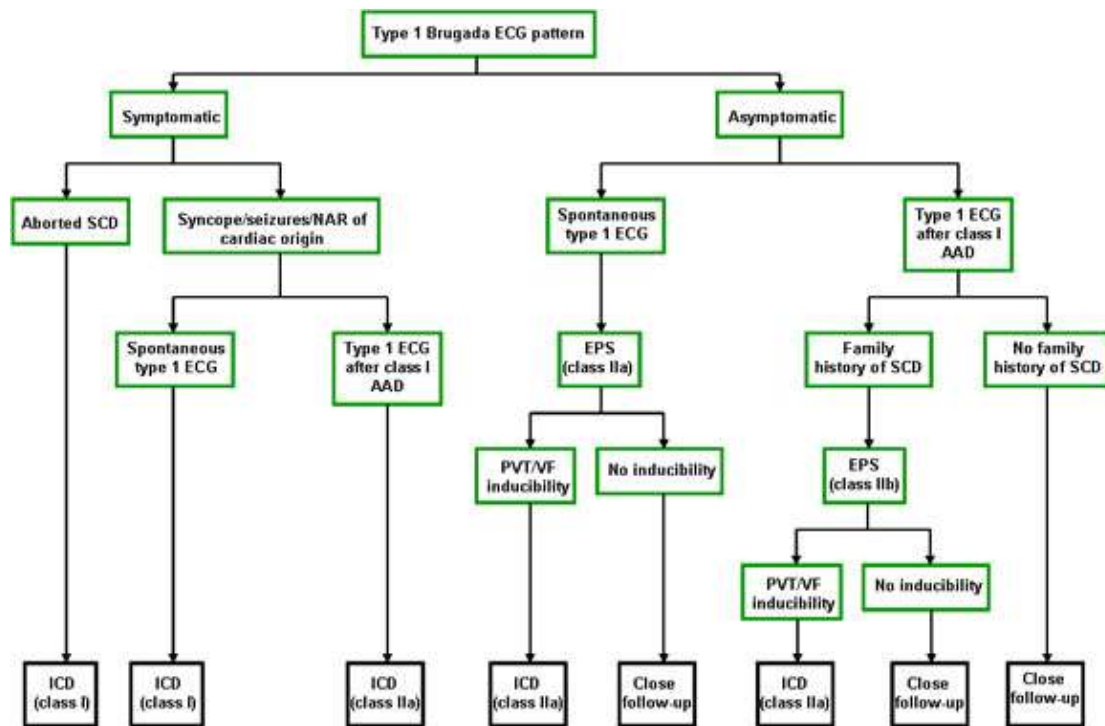
1. Data from the family history:
 - ✓ SCD in a family member younger than 45
 - ✓ ECG type 1 in family members
2. Arrhythmia-related symptoms:
 - ✓ Syncope
 - ✓ Seizures
 - ✓ Nocturnal agonal respiration
3. Documented ventricular arrhythmias:
 - ✓ Polymorphic ventricular tachycardia (PVT)
 - ✓ Ventricular fibrillation (VF)

Risk Stratification for SCD (see figure 7)

- 25% of the total population with BrS will experience SCD or VF during their lifetimes
- Only proven effective therapeutic strategy for the prevention of SCD in BrS patients is implantation of an ICD
- Circumstances in which an ICD is recommended:
 - ✓ Aborted SCD
 - ✓ Cardiac syncope + type 1 pattern on resting ECG

- Risk factors in which ICD should be considered
 - ✓ Cardiac syncope + type 1 pattern at drug challenge
 - ✓ Type 1 pattern on resting ECG + inducible arrhythmia (PVT or VF) at EPS
 - ✓ Type 1 pattern at drug challenge + family history + inducible arrhythmia (PVT or VT) at EPS

Figure 7: Risk Stratification for Sudden Cardiac Death in Brugada Syndrome



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Recommendations

- It is recommended that BrS patients should avoid all drugs that may induce a type 1 class ECG (see Table 1); <http://www.brugadadrugs.org>
- As fever may elicit the diagnostic ECG pattern and has also been recognised as a trigger of VA in BrS, patients should be encouraged to treat it aggressively with paracetamol
- Patient should avoid saunas, steam rooms and endurance athletics
- Patients should avoid sleeping after ingesting a large meal
- Patients must contact their cardiologist immediately in case of presenting syncope, seizures or nocturnal agonal respiration
- Family screening of BrS is strongly recommended in first-degree relatives (BrS is an inherited disease transmitted in an autosomal-dominant way). This is usually with a baseline ECG and an Ajmaline challenge test facilitated through the Inherited Cardiac Conditions service in the Belfast Health and Social Care Trust (BHSCT) who may also advise genetic screening for patients and their families for SCN5A analysis.
- All patients must have regular follow-ups in order to identify the development of symptoms
- Genetic testing, when available, is recommended to support clinical diagnosis, early detection of other affected family members and for research purposes. However the diagnosis should not be based on it because it still has a low diagnostic yield

Table 1- Drugs which induce Brugada like ECG patterns

Antiarrhythmic drugs	
Sodium channel blockers	Class IC drugs (Flecainide, propafenone, pilsicainide)
	Class IA drugs (Ajmaline, procainamide, dysopiramide, cibenzoline)
Calcium channel blockers	Verapamil
Beta-blockers	Propranolol, etc.
Antianginal drugs	
Calcium channel blockers	Nifedipine, diltiazem
Nitrate	Isosorbide dinitrate, nitroglycerine
Potassium channel openers	Nicorandil
Psychotropic drugs	
Tricyclic antidepressants	Amitriptyline, Nortriptyline, Desipramine, Clomipramine
Tetracyclic antidepressants	Maprotiline
Phenothiazine	Perphenazine, Cyamemazine
Selective serotonin reuptake inhibitors	Fluoxetine
Other drugs	
	Dimenhydrinate, Cocaine intoxication, Alcohol intoxication

8.2 Long QT Syndrome (LQTS)

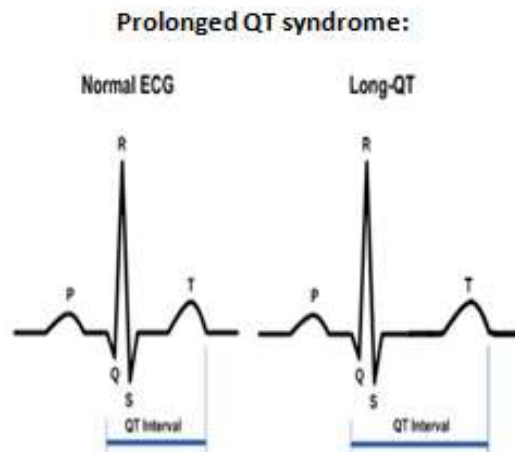
The QT interval represents the time of ventricular activity including both depolarization and repolarization. It is measured from the beginning of the QRS complex to the end of the T wave. The QT interval should be measured manually. Measurements are made in leads II and V5 because QTc (corrected QT interval) in these leads correlates best with genotype status in long QT syndrome (LQTS).

The end of the T wave (which can be difficult to define) can be most reproducibly ascertained using the tangent technique, where a line or tangent is drawn down the steepest slope of the terminal limb of the T wave. The end of the T wave is defined by the intersection of this line with the baseline.

Diagnosis

- Rule out secondary causes for QT prolongation
- $QTc \geq 480ms$ in repeated 12-lead ECGs
- LQTS is diagnosed in the presence of a confirmed pathogenic LQTS mutation, irrespective of the QT duration
- ECG diagnosis of LQTS should be considered in the presence of a $QTc \geq 460ms$ in repeated 12-lead ECGs inpatients with an unexplained syncopal episode in the absence of secondary causes for QT prolongation (see figure 8)

Figure 8: ECG showing prolonged QT interval



Risk stratification and management

- Lifestyle changes are recommended in all patients with a diagnosis of LQTS, including:
 - ✓ Avoidance of QT-prolonging drugs(<http://www.crediblemeds.org>)
 - ✓ Correction of electrolyte abnormalities (hypokalaemia, hypomagnesaemia, hypocalcaemia) that may occur during diarrhoea, vomiting or metabolic conditions
 - ✓ Avoidance of genotype-specific triggers for arrhythmias
 - Strenuous swimming, especially in LQTS1
 - Exposure to loud noises in LQTS2 patients
- Beta-blockers are recommended in patients with a clinical diagnosis of LQTS
- ICD implantation with the use of beta-blockers is recommended in LQTS patients with:
 - ✓ Previous cardiac arrest

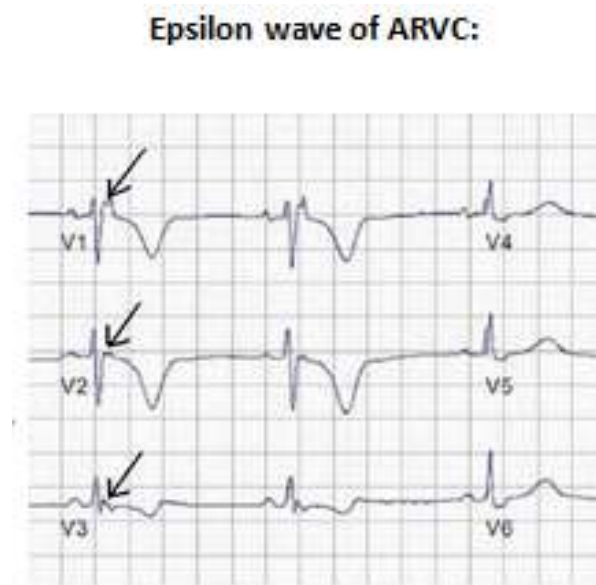
- ✓ Cardiac syncope and/or VT while receiving an adequate dose of beta-blockers
- Left cardiac sympathetic denervation should be considered in patients with symptomatic LQTS when
 - ✓ Beta-blockers are either not effective, not tolerated or contraindicated
 - ✓ ICD therapy is contraindicated or refused
 - ✓ Patients on beta-blockers with an ICD experience multiple shocks
- Sodium channel blockers (mexiletine, flecainide or ranolazine) may be considered as add-on therapy to shorten the QT interval in LQTS3 patients with aQTc >500 ms
- ICD may be considered in addition to beta-blocker therapy in asymptomatic carriers of a pathogenic mutation in KCNH2 or SCN5A when QTc is >500 ms

8.3 Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- Progressive heart muscle disorder characterized by VA, HF and SCD
- Cardiomyocytes are replaced by adipose and fibrous tissue
- Clinically, ARVC is defined by structural and functional abnormalities of the right ventricle, but LV involvement occurs in >50% of patients
- Estimated prevalence of 1 in 1000 to 1 in 5000 of the general population
- Important cause of SCD in athletes and young adults
- Clinical manifestations, including palpitations, syncope, VT and SCD, usually develop between the second and fourth decade of life
- Disease progression may result in right or biventricular HF
- Up to two-thirds of patients have VAs on resting or ambulatory ECG monitoring and exercise testing
- ECGs can show RBBB, T wave inversion in leads V1-V3 and an epsilon wave

(figure 9)

Figure 9: Epsilon wave in ARVC



Risk stratification and management

- Avoidance of competitive sports
- Beta-blockers titrated to the maximally tolerated dose are recommended as the first-line therapy to improve symptoms in patients with frequent PVC and NSVT
- ICD implantation is recommended in patients with a history of aborted SCD and haemodynamically poorly tolerated VT
- Amiodarone should be considered to improve symptoms in patients with frequent PVC or NSVT who are intolerant of or have contraindications to beta-blockers
- Catheter ablation should be considered in patients with frequent symptomatic PVC or VT unresponsive to medical therapy to improve symptoms and prevent ICD shocks

- ICD implantation should be considered in ARVC patients who have haemodynamically well-tolerated sustained VT
- ICD implantation may be considered in patients with one or more recognised risk factors for VA in adult patients with a life expectancy >1 year
- EPS may be considered for stratification of SCD risk

Appendix 3 highlights the SHSCT pathway for palpitations.

9 Driving Guidelines

This document cannot cover all driving restrictions. Ultimately the decision on whether a patient can drive or not falls with the driver and vehicle licensing agency (DVLA). All patients must be informed to self-refer to the DVLA on discharge if advised by their medical team. Further guidance on individual cases can be found on the DVLA website or on the SHSCT educational cardiology dropbox forum.

10. Update and review

- This document will be updated every 3 years.
- Revisions will be made ahead of the review date if new, relevant national guidelines are published. Where the revisions are significant and the overall policy is changed, the authors will ensure the revised document is taken through the standard consultation, approval and dissemination processes.

11. References

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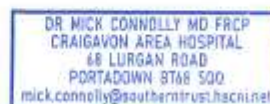
Appendix 1 – Governance information

Document title	Guidelines for the management of patients presenting with palpitations
Date issued / approved:	5th August 2020
Date valid from:	5th August 2020
Date valid to:	Initial: 5th August 2023 Extended: 1st Jan 2027 (see below)
Brief summary of contents	This document provides guidance for any professional involved in the clinical management of patients presenting to SHSCT with palpitations.
Policy objectives	To provide clear speciality agreed guidelines and pathways for the diagnosis and clinical management of patients with palpitations.
Keywords	Cardiology Palpitations Supraventricular tachycardia
Authorship	Dr David Mc Eaneyey (cardiology clinical lead, consultant cardiologist) Kay Carroll (cardiology head of service) Dr Mick Connolly (consultant cardiologist) Dr Elizabeth Banks (ST3 Cardiology)

Addendum Jan 2024:

This policy was reviewed by the consultant cardiologists and discussed at the cardiology governance meeting in Winter 2023. It was sent out for a 6 week consultation period. No amendments were suggested by the wider cardiology team. Following universal unanimous agreement by the consultant body at this governance meeting it has been approved for 3 further years until 1st Jan 2027.

Signed: Dr Mick Connolly Consultant cardiologist

Appendix 2 – AliceCor details

'AliceCor' device



AliveCor® is a family of mobile, clinical-quality electrocardiogram (ECG) recorders. This is available on Amazon for approximately £85.

Patients with known or suspected heart conditions and health conscious individuals can use Kardia Mobile or Kardia Band to record an ECG daily or whenever they are feeling symptoms and share their recordings with their cardiologist.

Users may record ECGs using Kardia™ Mobile with their smartphone or tablet, or the Kardia™ Band with their Apple Watch.

The duration of the recording is established by the Kardia phone and Kardia watch apps with a default setting of 30 seconds.

The Kardia phone app may extend recordings to a maximum time of 5 minutes.

The software application can store thousands of recordings on your smartphone or Tablet.

Medical professionals can quickly assess rate and rhythm, screen for arrhythmias, and remotely monitor and manage patients with AliveCor®.

Download the Kardia phone app

1. Using your smartphone or tablet, search for *Kardia* in the App Store or Google Play store.
2. Download and install the Kardia phone app.

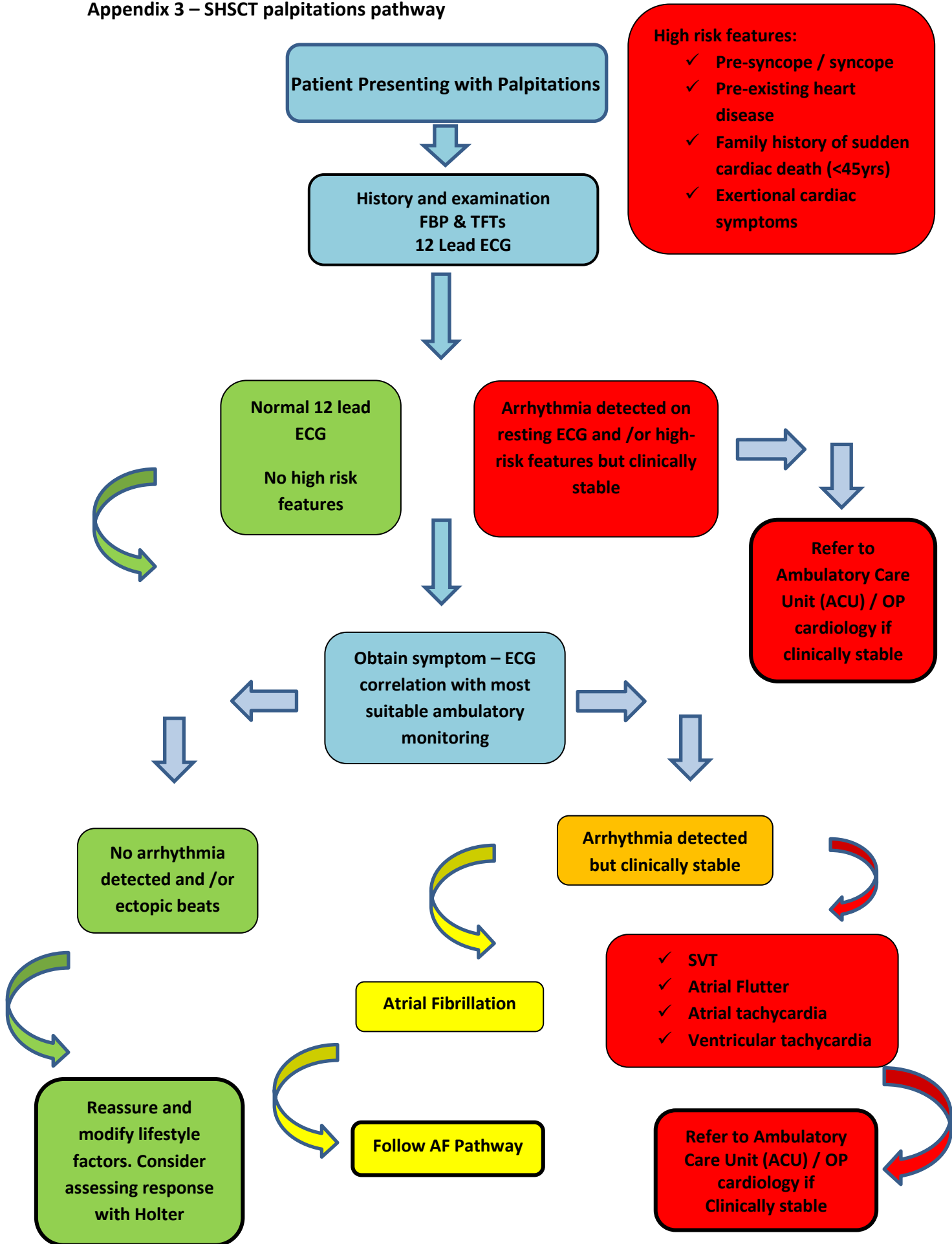
Set up an AliveCor Account

You will use your AliveCor account to access, print, and save your EKG recordings stored on the Kardia phone app and the AliveCor server. Follow the instructions presented when you open the Kardia phone app for the first time.

To take an ECG recording with Kardia Mobile, follow the instructions below.

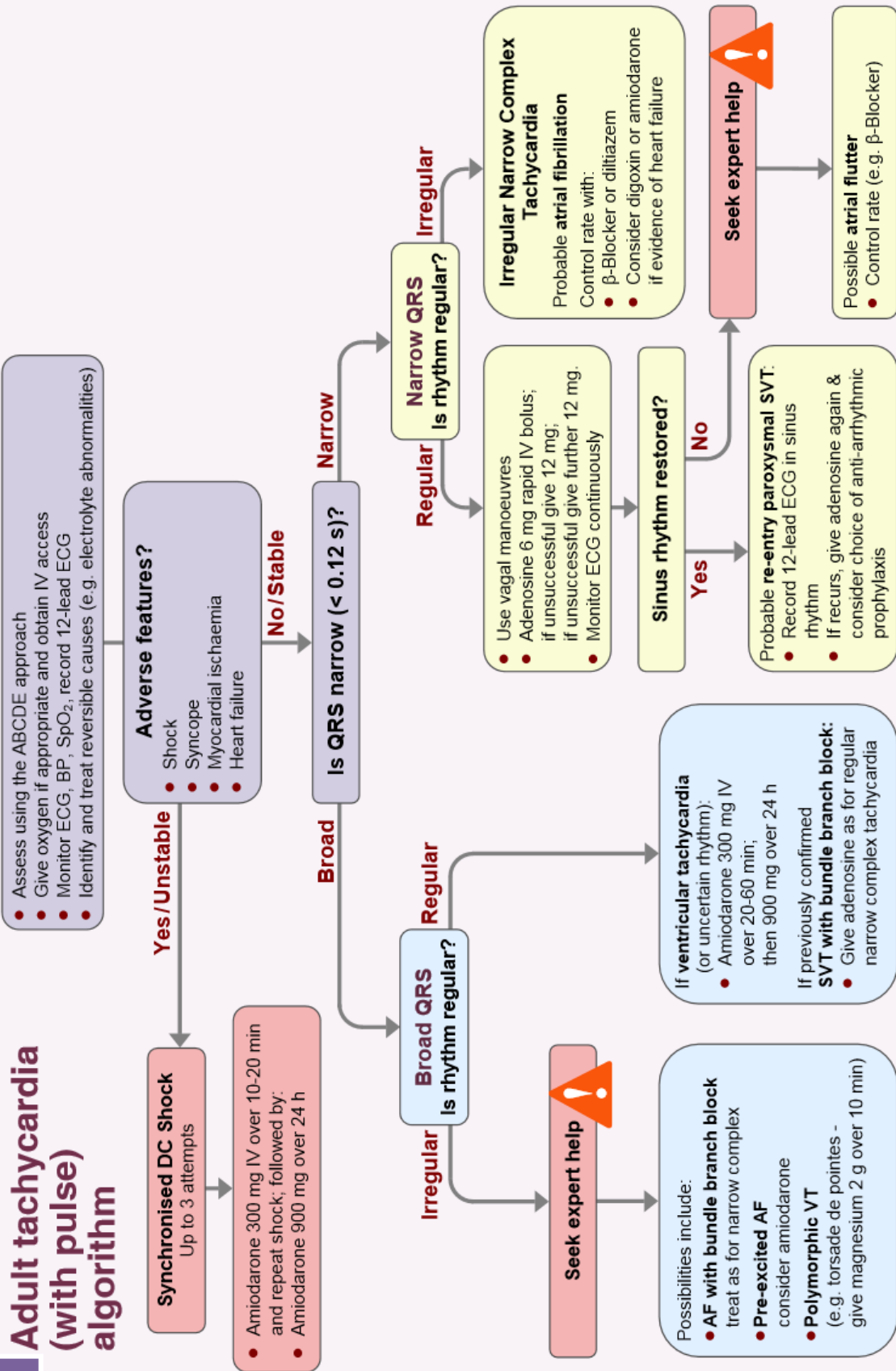
1. Select an ECG option – Standard ECG, Resting Heart Rate, or Guest EKG.
2. Rest two or more fingers on Kardia Mobile; your right hand should contact the electrode closest to the bottom of the smartphone or tablet, and your left hand should contact the electrode closest to the top of the smartphone or tablet. This is a Lead I ECG.
3. While recording your ECG, speak your symptoms (e.g. *I'm* feeling palpitations) into the smartphone. Any voice memo recorded will be transcribed to text and added to the Notes section for that ECG recording.
4. Tap the envelope icon next to the ECG you would like to email/print on the Journal screen. Tap Email. The PDF version of the ECG recording will then be attached to a new email in whatever email account you have set up on your smartphone or tablet.

Appendix 3 – SHSCT palpitations pathway



Appendix 4- Adult Tachycardia (with pulse) Algorithm

Adult tachycardia (with pulse) algorithm



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