Death beats

Sudden Cardiac Death - Primary and Secondary Prevention

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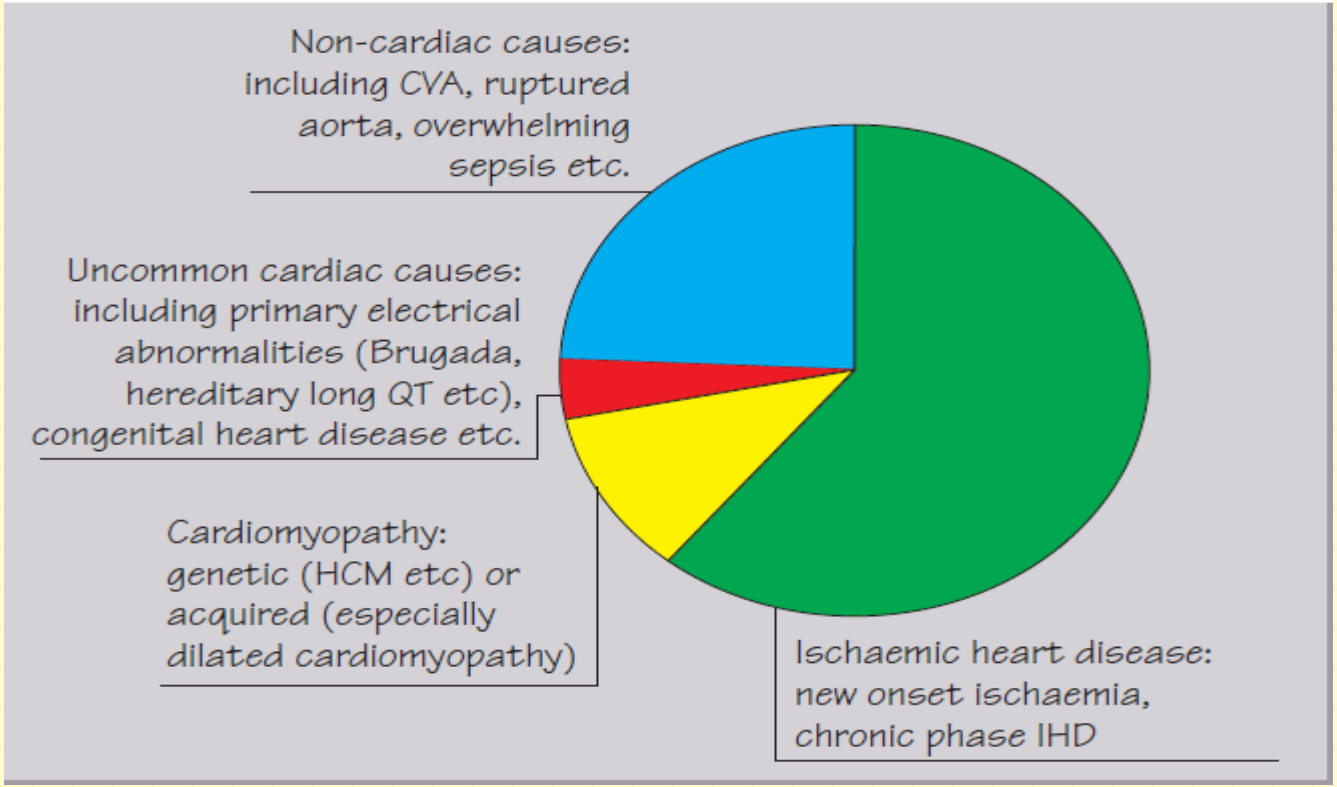
What is it?

- Sudden Cardiac Death (SCD) is:
  - an unexpected death due to cardiac causes*
  - that occurs in a short time period (generally within 1 hour of symptom onset)
  - in a person with known or unknown cardiac disease.

* Cardiac loss of function (sudden cardiac arrest) that is mostly due to abnormal heart rhythms – arrhythmias

http://emedicine.medscape.com/article/151907-overview

Causes of sudden death



Stepwise process

Cardiac disease

Malfunction in cardiac electrical propagation and signals become irregular

Tachycardia

Lowering Cardiac output and increasing sympathetic drive

Tachyarrhythmia

Ventricular fibrillation or tachycardia → Low CO → loss of consciousness

How prevalent is it?

- A major cause of natural deaths worldwide >7,000,000
- M:F ratio 2-3:1
- Peak ages of onset: mid-30s to mid-40s
- Upward trend in incidence rate in developed countries – dietary and lifestyle habits

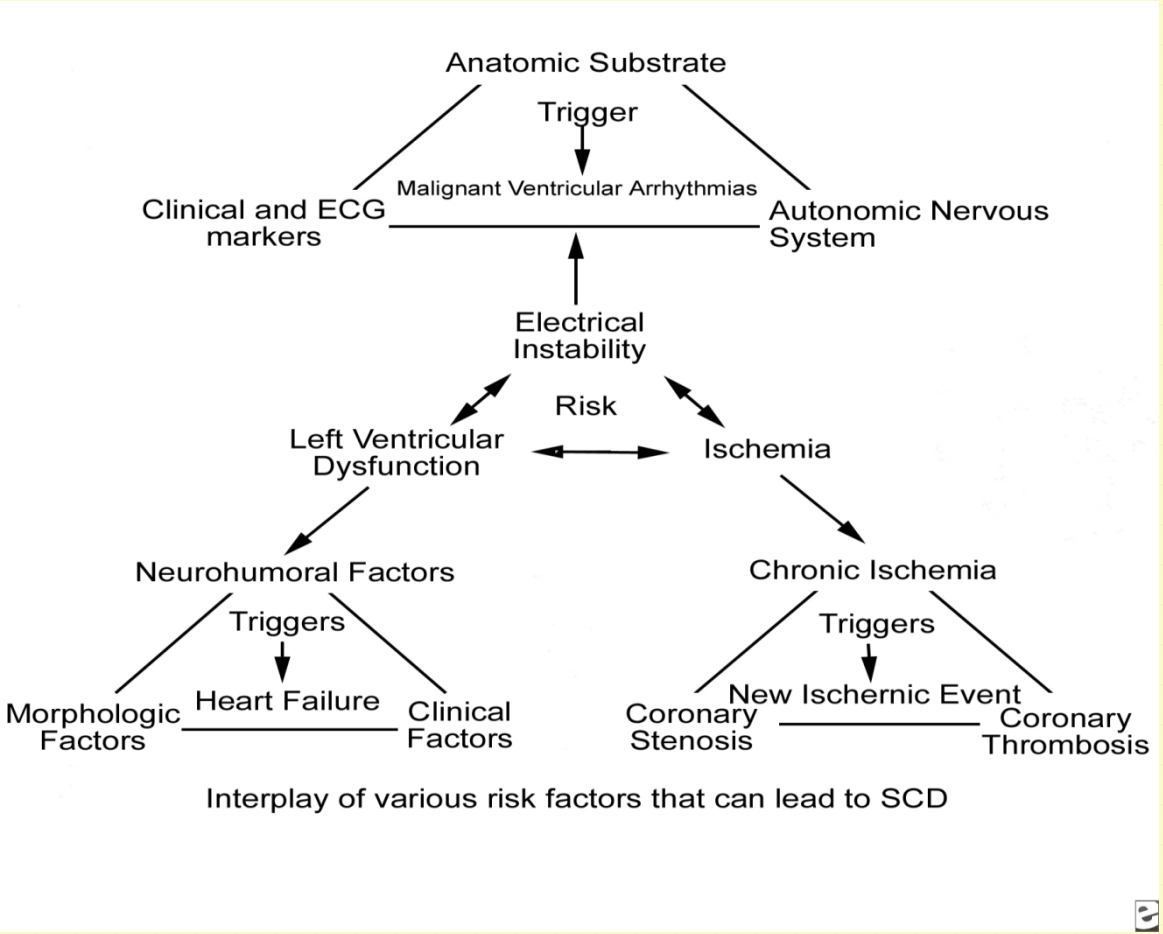
Signs and Symptoms

- prodromes of
  - chest pain
  - fatigue
  - palpitations
  - other nonspecific complaints

Table 5 Clinical presentations of patients with ventricular arrhythmias and sudden cardiac death

- Asymptomatic individuals with or without electrocardiographic abnormalities
- Persons with symptoms potentially attributable to ventricular arrhythmias
  - Palpitations
  - Dyspnea
  - Chest pain
  - Syncope and presyncope
- Ventricular tachycardia that is hemodynamically stable
- Ventricular tachycardia that is not hemodynamically stable
- Cardiac arrest
  - Asystolic (sinus arrest, atrioventricular block)
  - Ventricular tachycardia
  - Ventricular fibrillation
  - Pulseless electrical activity

What leads to SCD?



Interplay of various risk factors that can lead to SCD

The Pathophysiology

There are multiple factors at the level of:

- **organ** (eg imbalance of autonomic tone)
- **tissue** (eg reentry, wave break, and action potential duration alternans)
- **cellular** (eg triggered activity, and automaticity)
- **subcellular** (abnormal activation or deactivation of ion channels) level involved in generation of VT or VF in different conditions

Setting the scene for Ventricle Arrhythmia

- An anatomical or a functional block in the course of impulse propagation may create a circuit with the wave front circling around it and resulting in VT.
- Other mechanisms such as wave break and collisions are involved in generating VF from VT.
- At the tissue level the reentry and wave break mechanisms are the most important known mechanisms of VT and VF,
- At the cellular level increased excitation or decreased repolarization reserve of cardiomyocytes may result in ectopic activity (eg automaticity, triggered activity), contributing to VT and VF initiation.
- At the subcellular level, altered intracellular Ca2+ currents, altered intracellular K+ currents (especially in ischemia), or mutations resulting in dysfunction of a sodium channel (Na+ channelopathy) can increase the likelihood of VT and VF.

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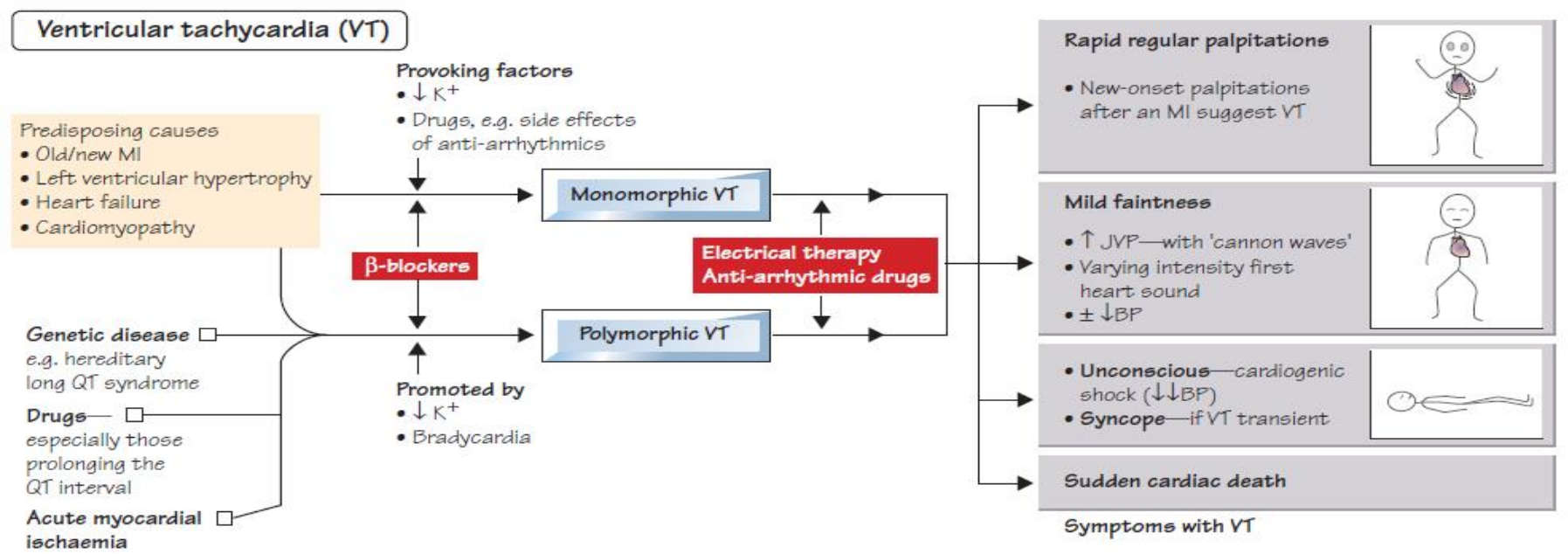
- Most cases of SCD occur in patients with **structural abnormalities** of the heart.
  - **Myocardial infarction (MI) and post-MI remodeling** of the heart is the most common structural abnormality in patients with SCD.
  - In patients who survive a myocardial infarction, the presence of premature ventricular contractions (PVCs), particularly complex forms such as multiform PVCs, short coupling intervals (R-on-T phenomenon), or VT (salvos of 3 or more ectopic beats), reflect an increased risk of sudden death.
    - However suppression of the PVCs with antiarrhythmic drugs increases mortality, owing to the proarrhythmic risk of currently available medications.
- **Hypertrophic cardiomyopathy** and **dilated cardiomyopathy** are associated with an increased risk of SCD.
- Various **valvular diseases** such as aortic stenosis are associated with increased risk of SCD.
- Acute illnesses, such as **myocarditis**, may provide both an initial and sustained risk of SCD due to inflammation and fibrosis of the myocardium.
- Inherited arrhythmia syndromes – SCD caused without apparent structural defect

Not all heart problems lead to SCD

- Many patients have anatomic and functional cardiac substrates that predispose them to develop ventricular arrhythmias
  - only a small percentage develop SCD.
- Identifying the patients at risk for SCD remains a challenge.
- The strongest known predictor of SCD is **significant left ventricular dysfunction** of any cause.

Cause and effect of VT



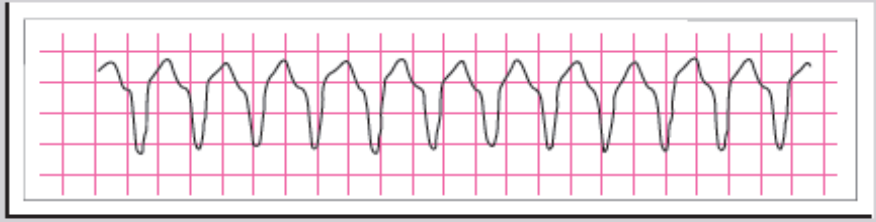
Details on VT

Pathogenesis

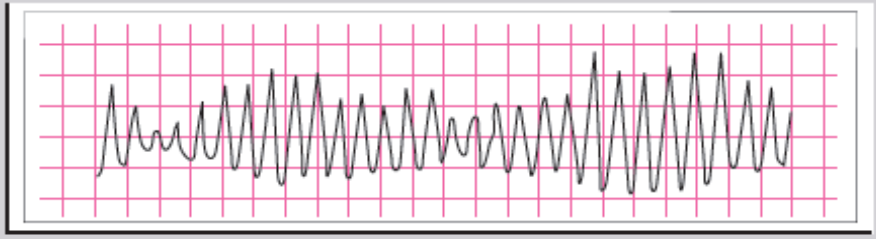
- Rapid depolarizations of the ventricular myocardium, caused by:
  - Re-entrant circuit(s) in anatomically abnormal substrates due to M infarction scar tissue
    - Lead to monomorphic VT
  - Abnormal triggering activity due to electrophysiological/metaboli c disturbances that prolong QT interval – i.e. ischemia and drugs
    - Leads to polymorphic VT

Ventricular tachycardia

Commonly due to re-entrant circuit(s) related to a scar or abnormal myocardium – results in **monomorphic VT**



Metabolic rather than anatomical substrate results in **polymorphic VT** *(Torsades de Pointes)* or VF



More on VT

- The most powerful stimulus for developing VT is myocardial damage
  - Heart failure
    - Each 10% decrease in ejection fraction, increases arrhythmic death occurring by 65%!
    - Suspect VT in anyone who has a syncope and has impaired Left ventricular dysfunction – until alternative diagnosis is found

VT ECGs

ECG diagnosis

- The QRS complexes are broad with an abnormal shape.
- VT should always be suspected when patients known to have heart disease (especially recent or remote MI) present with a regular tachycardia with broad QRS complexes:
  - In monomorphic VT, the QRS morphology is uniform; typically the rate is 120–190 beats/min. There may be evidence of independent atrial activity, i.e. dissociated P waves.
  - Polymorphic VT is less regular, more chaotic, and sometimes with a characteristic phasic variation in the QRS morphology – ‘torsades de pointes’. Polymorphic VT is inherently unstable and often degenerates early on into VF.

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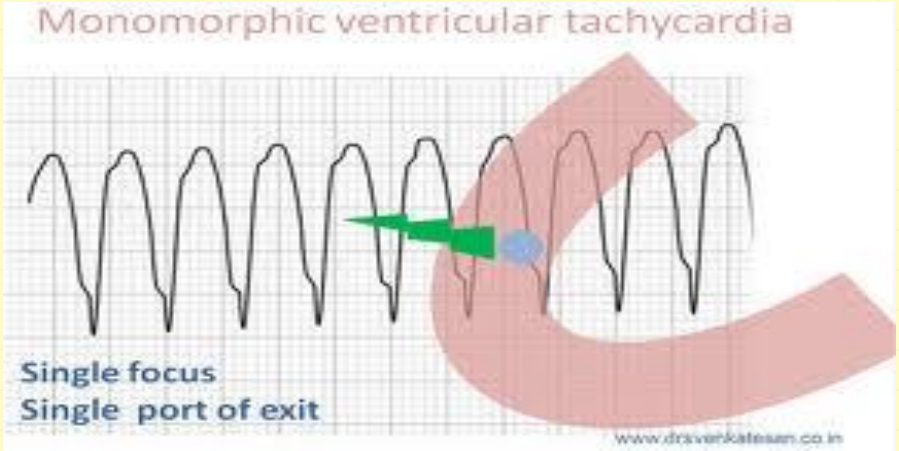

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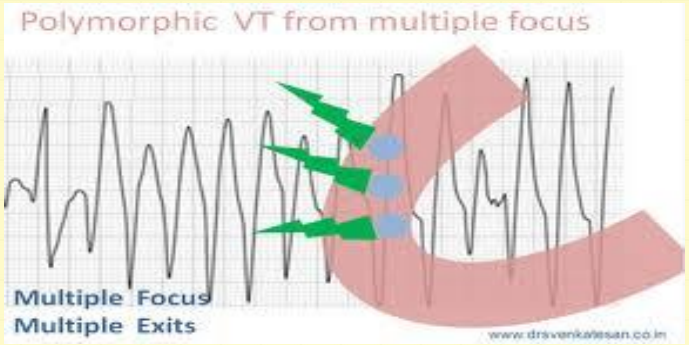

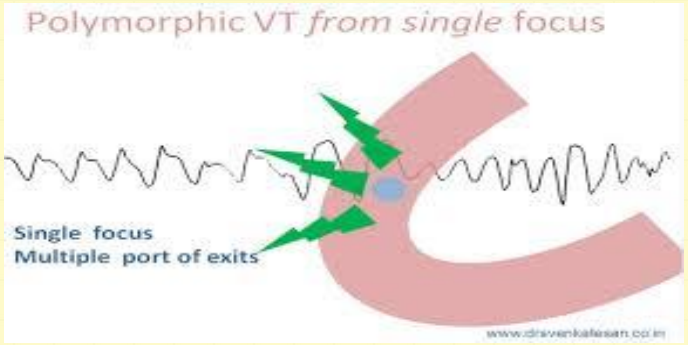

drsvenkatesan.wordpress.com

Causes of polymorphic VTs

Table 92.1 Causes of polymorphic ventricular tachycardia.

- Acute ischaemia
- Drugs (cause prolongation of the QT interval): quinidine, sotalol, amiodarone, tricyclic antidepressants, antihistamines
- Hypokalaemia
- Hypomagnesaemia
- Hypocalcaemia
- Bradycardias (any cause)
- Congenital QT prolongation syndromes (ion channel mutations): Jervell–Lange–Nielsen syndrome, Romano–Ward syndrome

Treating VTs – during an episode

Pulseless VT, or impending cardiovascular collapse:

- DC cardioversion, either immediate or after urgent anaesthesia/sedation.

Haemodynamically stable VT:

- intravenous lidocaine (lignocaine) or drugs such as amiodarone. Never use multiple drug combinations.
- If drugs are unsuccessful, use DC cardioversion.

Polymorphic VT:

- DC cardioversion, intravenous magnesium, correction of the underlying metabolic or electrophysiological abnormality.
- Slow heart rates prolong the QT interval and may worsen polymorphic VT.

Treating VTs - prevention

Long-term drug therapy:

- b-blockers, amiodarone, angiotensinconverting enzyme inhibitors and spironolactone to improve LV function and maintain K+.

Revascularization (coronary artery bypass graft or percutaneous coronary intervention):

- for severe coronary disease.

Implantable defibrillators (ICDs):

- monitor the cardiac rhythm and deliver antitachycardic therapy (overdrive pacing and/or intracardiac shocks) if VT or VF is detected.
- They are indicated for survivors of cardiac arrest, and in symptomatic VT with impaired LV function - Secondary prevention

Ventricular Fibrillation

- A very lethal arrhythmia – especially out of hospital setting
- Those who are resuscitated, without major neurological damage should get a full workup to find underlying cause

Vfib video youtube

Causes of VFs

Ischaemic heart disease (IHD):

- either acute MI, which is then treated in the standard fashion, or critical coronary stenosis, treated with complete revascularization.
- If there is no new ischaemia, and the problem relates to an old MI-related scar, the treatment is an ICD.

Other structural heart disease:

- especially cardiomyopathies. Many patients in these categories need an ICD.

Channelopathies:

- these are usually genetically mediated proarrhythmic conditions, and include hereditary long QT syndrome and Brugada's syndrome.

Other rare conditions:

- including WPW syndrome with complicating AF and very high heart rates, and acquired long QT syndromes (including starvation-related syndromes).

Diagnosis

- Laboratory studies
  - Cardiac enzymes (creatine kinase, myoglobin, troponin)
  - Electrolytes, calcium, and magnesium
  - Quantitative drug levels (quinidine, procainamide, tricyclic antidepressants, digoxin): High or low drug levels may have a proarrhythmic effect
  - Toxicology screen: For drugs, such as cocaine, that cause vasospasm-induced ischemia
  - Thyroid-stimulating hormone
  - Brain natriuretic peptide (BNP)

- Other tests to evaluate or predict risk of SCD
  - Imaging studies: Chest radiography, **echocardiography**, nuclear scintigraphy
  - Electrocardiography (ECG): Including, possibly, signal-averaged ECG
  - Coronary angiography
  - Electrophysiology

Primary Prevention

- Methods to avoid occurrence of disease.
- Most population-based health promotion efforts target here.

- Several studies have evaluated the use of prophylactic ICDs in patients who have not yet experienced SCD but are at high risk for future SCD:
  - Multicenter Automatic Defibrillator Implantation Trail (**MADIT**) demonstrated that patients with ischemic cardiomyopathy (LVEF ≤35%) and inducible but nonsuppressible VT on EPS had a survival advantage by implanting an ICD.
  - MADIT-2, demonstrating that post-MI patients with an LVEF ≤30% have a survival benefit with ICD implantation.
  - The Defibrillators in Nonischemic Cardiomyopathy Treatment Evaluation (**DEFINITE**) study showed that implantation of an ICD reduced the risk of sudden cardiac death in a patient population of nonischemic cardiomyopathy (LVEF < 36%) who also had PVCs or nonsustained VT.

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- Sudden Cardiac Death in Heart Failure Trial **(SCD-Heft)** demonstrated that patients with either ischemic or nonischemic cardiomyopathy on optimal medical therapy, LVEF ≤35%, and NYHA II or III treated with an ICD demonstrate greater survival as compared with either amiodarone or placebo.

- The recent Home Automated Defibrillator Trial (**HAT**) demonstrated no survival benefit for the use of a home AED in patients surviving a recent anterior MI who were not candidates for an ICD.

- The use of **microvolt T wave alternans** (MTWA) to determine which patients with depressed LV systolic function would best benefit from prophylactic ICD placement has been the subject of several recent clinical trials. To date, the results of these clinical trials has not been conclusive.

Treating SCD events

- **Advanced Cardiac Life Support** protocols
- **Bystander CPR** – in the community
  - Use of Automated Electric Defibrillators (AED)
- **Defibrillation** – for VF or VT causes
- **Pulseless Electrical activity**:
  - Drugs: Adrenaline. Atropine in bradycardia. Sodium bicarbonate in hyperkalemia.
- Asystole - ?
- **Medical Stabilisation** – avoiding the 50% re-arrest rate
- **Therapeutic Hypothermia** – limit neurologic injury due to brain ischemia during cardiac arrest and reperfusion injury with resuscitation

Secondary Prevention

- Methods to diagnose and treat existent disease in early stages before it causes significant morbidity

Surgery

- Temporary cardiac pacing
- Radiofrequency ablation
- Cardioverter defibrillator therapy
- Coronary artery bypass grafting (CABG)
- Excision of ventricular tachycardia foci
- Excision of left ventricular aneurysms
- Aortic valve replacement
- Orthotopic heart transplantation

ACLS

ACC/AHA/ESC 2006 guidelines for managing patients with ventricular arrhythmia and prevention of SCD

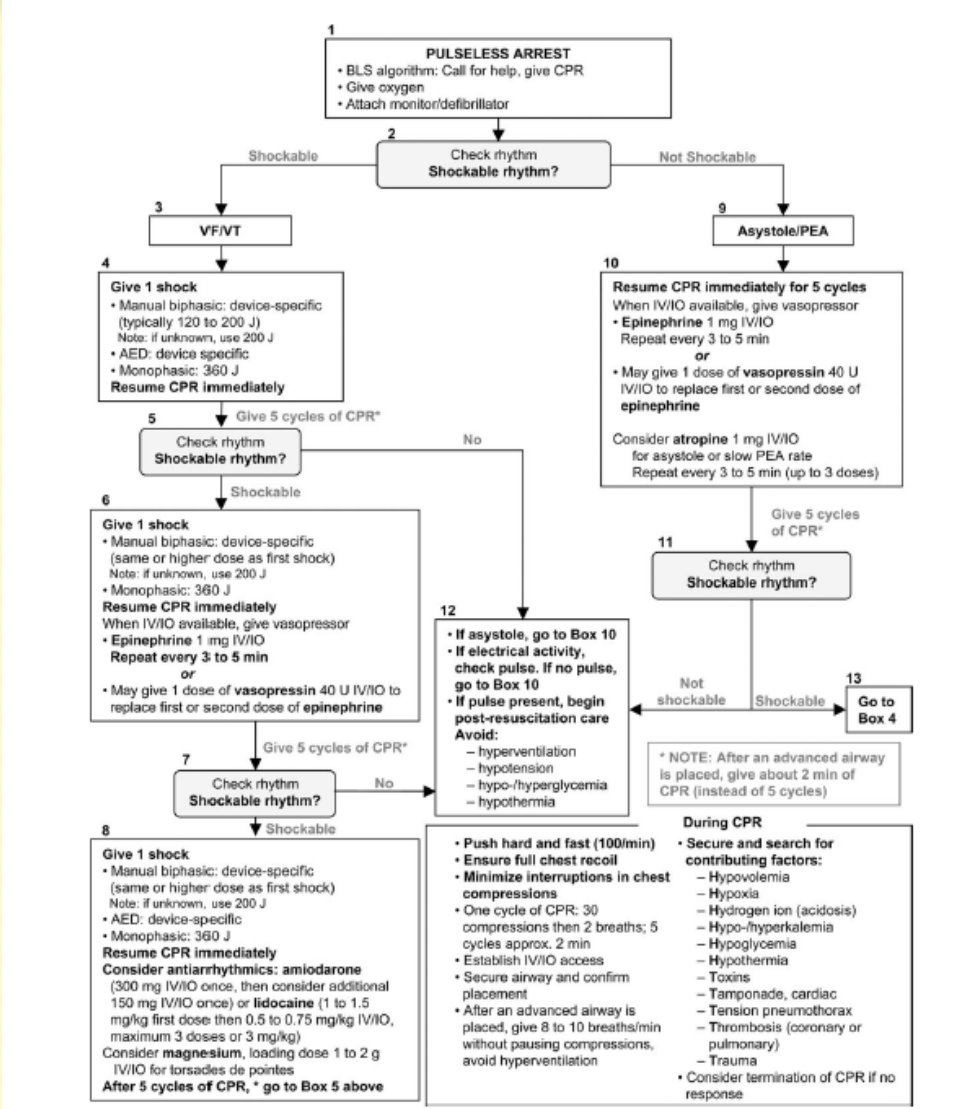

Figure 3 Advanced cardiac life support pulseless arrest algorithm. Reprinted with permission from *Circulation* 2005;112:IV57–66. AED, automated external defibrillator; BLS, basic life support; CPR, cardiopulmonary resuscitation; IV/IO, intravenous/intraosseous; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia.

Bibliography

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- Medscape – Sudden Cardiac Death
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