



ELECTRICAL STORM

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WHAT IS IT?

- ▶ Current Arbitrary Definition

- ▶ 2/3 or more separate VF/
VT episodes leading to
DCCV or ICD therapies
(including ATP) in 24 hours

WHY DO WE CARE?

- ▶ Increased incidence since AICD have become commonplace
- ▶ 10-30% of pts with ICD for secondary causes get it
- ▶ 4% of those with ICD for primary prevention (MADIT-II)

WHY DO WE CARE 2?

- ▶ Independent adverse prognostic factor (AVID, MADIT-II, Gatzoulis)
- ▶ At least 3 fold risk death/transplant/hospital in next 12 months
- ▶ Unclear if its the storm or the underlying pathology

WHY DO WE CARE 3?

- ▶ Pathophys and management from the storm EP literature can help inform ED management of refractory arrhythmias

WHAT CAUSES IT?

- ▶ **STRUCTURAL HD**
- ▶ **ABNORMAL ELECTRICAL
SUBSTRATE**

STRUCTURAL HD

- ▶ IHD
- ▶ Non Ischaemic Cardiomyopathy (DCM, HCM, ARVC)
- ▶ Valvular
- ▶ Infiltrative: Sarcoidosis, Chagas, myocarditis)
- ▶ Congenital

ABNORMAL ELECTRICAL SUBSTRATE

- ▶ Primary
 - ▶ Idiopathic
 - ▶ Brugada
 - ▶ ER
 - ▶ LQTS, SQTS
 - ▶ CPVT
- ▶ Secondary
 - ▶ Electrolytes
 - ▶ Toxins
 - ▶ Endocrine eg TFT
 - ▶ Periop CSx
 - ▶ R on T pacing

MANAGEMENT

- ▶ Issue with standard ACLS is Storm Rx can be vastly different depending on cause
 - ▶ eg High dose B Blocker vs chemical pacing
 - ▶ eg Amiodarone in LQTS
 - ▶ eg Verapamil in WPW

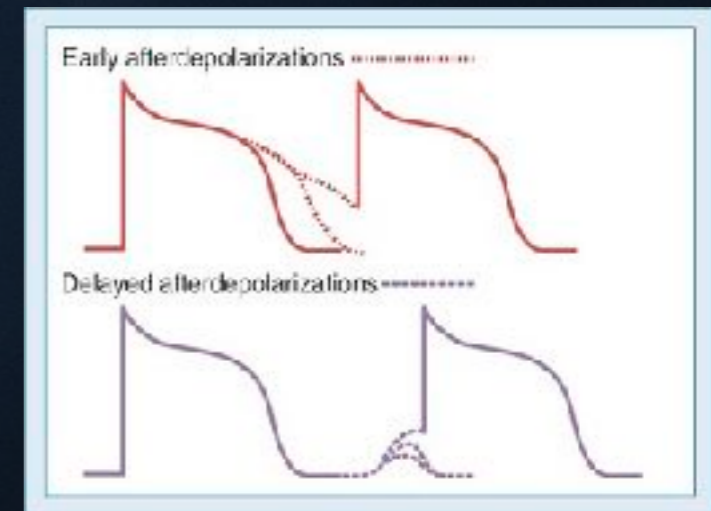
BUT HERE'S YOUR PROBLEM

- ▶ Working out which one it is can be very difficult during a recurrent arrest situation
- ▶ Use a management strategy based on likely cause from rhythm, POCUS, ECG

THUS

- ▶ Check known PHx
- ▶ Do a bedside echo ASAP
 - ▶ best in sinus but obvious abnormality seen even in arrhythmia
- ▶ Do a blood gas
- ▶ Check an ECG ASAP (hopefully sinus)

TINY BIT OF ELECTROPHYSIOLOGY NERDY STUFF



- ▶ EADs (Early After Depolarisations): secondary depol occurring before full repolarisation. Happen when AP prolonged (eg loss of Repol K⁺ currents)
- ▶ DADs (Delayed) when elevated Ca²⁺ load increases Protein Kinase II activity eg tachycardia or Beta Adrenergic stim. If big enough, I_{Na} is activated and a new AP occurs
- ▶ Automaticity: spontaneous AP generation
- ▶ Re-entry: Needs vulnerable substrate and a trigger

MONOMORPHIC VT IN STRUCTURAL HD

- ▶ Most common storm
- ▶ Usually re-entry (myocardial scars)
- ▶ Often focal ectopy from DAD (enhanced by Beta adrenergic activity)
- ▶ Standard Rx is Class 1 Na⁺ blockers but not as good if bad LV, so Amiodarone used
- ▶ However given sympathetic role, B1 blockade is important e.g. esmolol
- ▶ RSI/sedation can also suppress sympathetic activity

WHAT IF DRUGS DON'T WORK

- ▶ RFCA is the indicated next step
- ▶ 3D mapping to find area of substrate first
- ▶ 4% major complication rate

MONOMORPHIC VT IN ICD

- ▶ ATP (burst overdrive pacing) prior to internal defib
- ▶ ATP most appropriate to avoid unnecessary shocks particularly for fast VT

MONOMORPHIC VT IN NORMAL HEARTS

- ▶ Rare: "Idiopathic VT"
- ▶ Usually Outflow tract VT (LBBB) or Fascicular VT (narrow RBBB)
- ▶ OT-VT is usually cAMP mediated DADs. cAMP increased by sympathetic stimulation.
 - ▶ Rx thus B blockade or Ca²⁺ 1st or Class III 2nd. RFCA 3rd
- ▶ F-VT usually verapamil responsive. 2nd: RFCA

POLYMORPHIC VT/VF IN STRUCTURAL HD

- ▶ IHD most common cause (if no LQT)
- ▶ Revascularisation is a priority
- ▶ B blockade or stellate ganglion sympathetic denervation is better than Class 1 agents.
- ▶ Amiodarone still a useful agent and is first line
- ▶ In heart failure patients, Amiodarone by its effect on I(KAS) currents seems best

POLYMORPHIC VT/VF IN STRUCTURALLY NORMAL

- ▶ Rare but includes all the genetic causes
- ▶ Most important group for DDx

LONG QT SYNDROME

- ▶ Usually Torsades rhythm (T de P = polymorphic VT + long QT)
- ▶ Congenital vs Acquired
- ▶ Caused via EADs (early after depolarisations)
 - ▶ Fix electrolytes ASAP
 - ▶ Magnesium safe but ?effective
 - ▶ Beta blocker 1st line in congenital, Verapamil may be second line
 - ▶ Temporary overdrive pacing works in acquired because bradycardia prolongs QT. Beta Blockers may worsen acquired

BRUGADA

- ▶ Storm can be brought on by fever, hypoK, bradycardia and high vagal tone
- ▶ VT/VF caused by loss of AP dome in RV epicardium leading to STE and reentry
- ▶ Class 1 agents worsen this but quinidine OK
- ▶ Isoprenaline reduces STE and recovers AP dome (also used in Early Repolarisation)

OTHER

- ▶ CPVT : normal ECG
 - ▶ bidirectional VT (alternating RBBB and LBBB)
 - ▶ Rx with IV Beta Blockers (dec Ca^{2+} and DAD)
 - ▶ 2nd line verapamil. 3rd line left cardiac sympathetic denervation
- ▶ Idiopathic VF: Verapamil

BUT NOTHING IS WORKING

- ▶ ECMO:
 - ▶ Main use is as a bridge to getting RFCA done or surgical sympathetic blockade
 - ▶ May also have a role in toxic causes when storm is time dependent or as a bridge to transplant

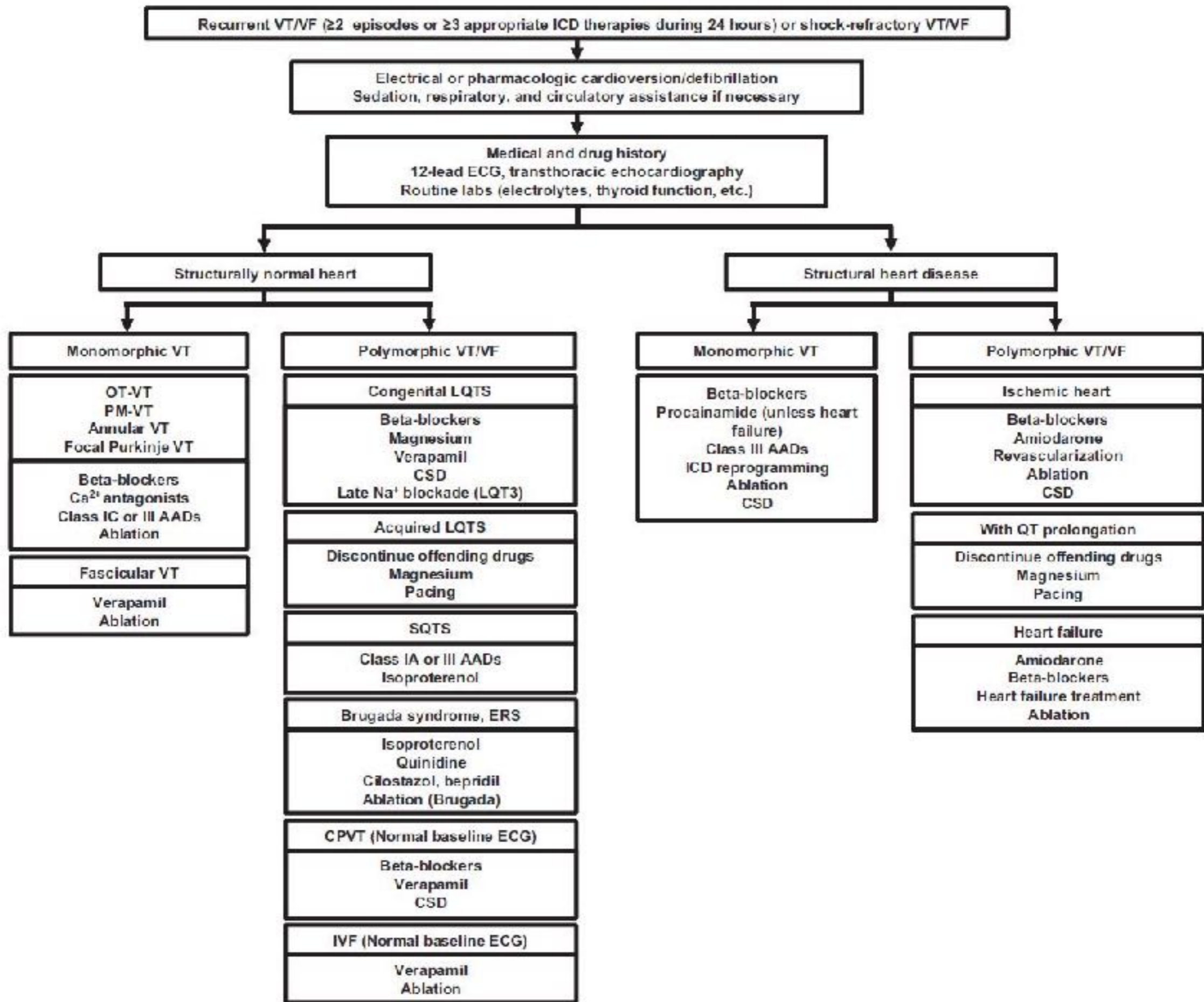


Fig. 1. Management of electrical storms. AAD= antiarrhythmic drugs; CPVT=catecholaminergic polymorphic ventricular tachycardia; CSD=cardiac sympathetic denervation; ERS=early repolarization syndrome; ICD=implantable cardioverter defibrillator; LQTS=long QT syndrome; OT-VT=outflow ventricular tachycardia; PM-VT=papillary muscle ventricular tachycardia; SQTS=short QT syndrome; VT=ventricular tachycardia; VF=ventricular fibrillation.

TAKE HOME MESSAGE

- ▶ Rx of Storm is sympathetic blockade
- ▶ Only exceptions where this is likely to be unhelpful is Brugada and Acquired LQTS
- ▶ Does Adrenaline still make sense??
- ▶ see [Should Adrenaline Use Be Arrested?](#) and [Sudden Cardiac Death](#) for more!

REFERENCES

- ▶ **Amazing summary of management**
 - ▶ Maruyama, M. Management of electrical storm: The mechanism matters. J of Arrhyth, 30 (2014) 242-249.
- ▶ **Very Detailed Pathophysiology**
 - ▶ Tsuji Y et al. Electrical storm: recent pathophysiological insights and therapeutic consequences. Basic Res Cardiol (2013) 108:336
- ▶ **Relatively simple overview**
 - ▶ Gao, Sapp. Electrical storm: definitions, clinical importance and treatment. Curr Opin Cardiol 2013, 28:72-79

THANKS

The logo features the letters 'T+CME' in a bold, red, stylized font with a thick black outline. The 'T' is tall and blocky, with a small red plus sign positioned above the 'C'. The 'CME' is written in a similar bold, rounded font. The entire logo is set against a white background.

TIME CRITICAL MEDICAL EDUCATION