DEFINITION

- **PVC/PVE (Paroxysmal Ventricular couple/ectopic)** - single ventricular ectopic (complex) emanating from the ventricles.
- **Couplet** - 2 VE in sequence; **Triplets** - 3 VE in sequence
- **Ventricular bigemini** - sinus rhythm alternating with ventricular ectopic
Nonsustained VT (ventricular tachycardia) - 4 or more beats of VE (ventricular ectopics) terminated within 30 seconds; at a rate of greater than 100 bpm

Monomorphic - single QRS morphology

Polymorphic - changing QRS morphology at cycle length between 180 and 600 ms
- **Sustained VT** - VT greater than 30 seconds and/or require termination due to hemodynamic instability in <30 seconds

- **Ventricular Flutter** - a type of ventricular tachycardia resulting in a zig-zag pattern without clearly formed QRS complex.

- **Torsade de Pointes** - a special case of VT with wide QRS complex changing around the axis (so called twisting around the axis)
DEFINITION

- **Ventricular Fibrillation** - a totally disorganized appearance on the ECG with no discernible ventricular complexes.
- **VT storm** - 4 or more sustained VT within 24 hrs, each requiring termination by an intervention.
- **Idiopathic VT** - structural normal heart; >3 consecutive beats
AUTOMACITY:- It is a focus of cells that depolarizes faster than the SA node. It will spread out the wavefront and conduct through the whole heart. Examples such as catecholamine polymorphic ventricular tachycardia (CPVT)

RE-ENTRY:- Accounts for 75% of ventricular arrhythmia. It is caused by 2 distinct pathways that exist between an anatomical/functional block area. It usually happened to have a myocardial scar. eg. ischemia.

Triggered activity:- It is caused by triggers that provoke depolarization in EAD phase 3 or DAD phase 4. The triggers are usually premature beats. Therefore, it can be easily inducible such as torsades de pointes and digoxin toxins.
MECHANISM

- **Scar-related re-entry** – usually reentry type. Ischemic vs no ischemic eg dilated CMP; macrore-entry. Usually in *abnormal* structure heart
- **Focal** – earliest point of activation; can be automaticity, triggered or microre-entry. Usually in *normal* structure heart
PVC (Premature ventricular complex) occurred in a range of 0.5% among 20 yrs old to 2.2% in those over 50 yrs old. Mortality risk is **minimal** in a normal heart subject with PVC only during resting period. However, monomorphic NSVT (non sustained ventricular tachycardia), polymorphic VT (ventricular tachycardia) even in normal heart are indicators of risk. Many nonsustained VT are due to abnormalities of molecular level or due to electrolyte imbalance or adverse drug effects. However, some studies showed that PVC and NSVT during **exercise** and recovery phase correlated with increased risk.
Most studies cited a frequency cutoff of 10 PVCs per hour and occurrence of repetitive forms VE as increased risk. Since risk of sudden death is already quite high because of the underlying heart disease. Suppression of VE/NSVT is no longer considered a therapeutic target for the prevention of sudden cardiac death. For post MI patients, ventricular arrhythmias occurred during the 24-48 hours do not imply continuing risk over time - so called primary VT/VF. However, for non- ischemic diseased heart, there is no such primary VT/VF. Ventricular arrhythmia already carry poor prognosis.
The incidence of ventricular sudden cardiac death is low around 0.1-0.2 %. The risk of sudden cardiac death is highest in the first 6-18 months after MI or heart failure events.
CLINICAL PRESENTATION

1. Asymptomatic finding of VE/NSVT with or without ECG abnormalities
2. Symptoms: palpitations; dyspnea; syncope; chest pain; dizziness
3. VT that is hemodynamically stable—SOB; palpitations
4. VT that is hemodynamically unstable—faint; LOC
5. Cardiac arrest
CAUSES OF WIDE COMPLEX TACHYCARDIA

- Ventricular tachycardia (VT) :- 80 % of all occurrences of WCT.
- SVT with bundle branch block
- Preexcited tachycardia i.e AVRT with antidromic conduction.
- Ventricular paced rhythm.
All patients who are evaluated for ventricular arrhythmias.; Look out for congenital abnormalities such as (long QT; short QT; Brugada syndrome, etc). Identify those with electrolyte disturbance, underlying structural heart disease with bundle branch block; Q wave, ventricular hypertrophy, AV block etc. QRS duration and repolarization abnormalities are both independent predictors of sudden cardiac events too. Studies showed that a risk ratio of CVS death of 2.4 in the presence of an ischemic ECG and 4.4 for abnormal T wave axis. An QTc >440 msec predicted CVS death with relative risk 2.1
ECG HELPS LOCALIZATION

- QRS width: - Septal VT < free wall VT
- QRS axis: - Inferior- Basal area; RVOT; LVOT; Superior - from apex
- BBB - LBBB (VT arised from right ventricle); RBBB (from left ventricle)
- Concordance- Positive - posterior basal; Negative - apical
- Presence of QS or QR wave- eg QS wave in V4-V6 - apical
Class 1 indication (highly recommend) – those patients who have an intermediate or greater probability of coronary heart disease. Also, it was recommended patients with known or suspected exercise induced ventricular arrhythmias such as catecholamine VT.. to achieve a diagnosis.
- **Holter** is needed to detect arrhythmias, QT interval changes, T wave alternans, ST changes etc. It is useful when the arrhythmia appeared at least once daily.

- **Event recorder** is used for sporadic cases with symptoms such as palpitations; dizziness; syncope that are caused by transient arrhythmia.
extremely useful in diagnosing serious tachyarrhythmias and bradyarrhythmias in patients with life-threatening symptoms such as syncope.
To identify the structural abnormalities of heart. LV systolic function and regional wall motion can be assessed. Stress echo may also be needed who are suspected to have ischemia causing ventricular arrhythmia.
We use 8 ventricular stimuli at cycle lengths of 400 ms and 600 ms at RV apex then deliver 1-3 extra ventricular stimuli. EP testing is recommended for patients with ischemic heart history who had symptoms of ventricular tachyarrhythmias such as palpitations, presyncope, or syncope. It is also useful to differentiate wide complex tachycardia. For patients with asymptomatic NSVT and EF <40 %, inducibility was around 20-40 %. Positive predictive value in ischemic heart patient is around 70 %. However, for non-ischemic etiology, EP testing is of very low value in predicting cardiovascular events.
For simple VE or NSVT, we can treat the underlying pathology. If the heart is structurally normal. We can just reassure the patient or just give B blocker or Ca blocker to relieve symptoms. We should try to avoid anti-arrhythmic drugs for ectopic treatment.
SPECIFIC TYPES OF VT

RVOT VT/ LVOT VT

- Structural Normal Heart
- LBBB and inferior axis for RVOT
- Caused by Ca-dependent triggered activity.
- Sensitive to Ca blocker Isoptin (Verapamil) and ATP.
- Common in female; exercise/stress related
FASCICULAR VT

- Usually occurred at left posterior fasicle of left ventricular septum.
- Common in men
- Structural normal heart
- RBBB and left superior axis
- Exercised related
- Micorre-entry of the Purkinjie fasicles.
CATECHOLAMINERGIC POLYMORPHIC VT (CPVT)

- Genetic determined adrenergic sensitive VT
- Chromosome 1 – RyR2 cardiac ryanodine receptor gene – some families had autosomal dominant.; responsible for Ca release.
- DAD – induced triggered activity
- Occurred during exercise or stress. No resing ECG hallmark could be identified.
- > 60% - 1st episode of syncopy or cardiac arrest by 20 yrs old.
- Can be bidirectional, monomorphic; polymorphic.
- Tx - B blocker and ICD
BRUGADA CHANNELOPATHY

- Usually blocked in Na channel SCN5A mutations.
- ECG – ST segment elevation in right precordial leads with coved shape or saddle shaped
- Structural normal heart
- Presented as polymorphic VT/VF
- QTC < 360 ms
- Polymorphic VT
- High incidence in infants; children and young age
- Usually gain in function in K channel KCNH2
LONG QT CHANNELOPATHY

- QTc > 460 ms
- 75% are caused by mutations in 3 genes: KCNQ1; KCNH2, SCN5A
- Some associated with deafness
- LQT1 – sympathetic stimulation prolongs QT causing Torsades
- B blocker maybe useful in treatment
- ICD - definitive
ISCHEMIC VT

- Scar related macrore- entry VT actually accounted for 70% VT/VF.
- Catheter ablatable but easily recurred in different forms.
- ICD – definitive treatment
- Use drug/ RF ablation to ameliorate VT attack frequency only
BB RE-ENTRY VT

- Usually occurred in dilated CMP
- Typical LBBB with bear the same shape in sinus rhythm
- VE goes thru the Right Bundle and back to left bundle.
- Macrone-entry; His preceds the V signal
- Treatment – ablated the Right bundle but still needs ICD.
ARVD
ARRHYTHMOCGENIC RIGHT VENTRICULAR DYSPLASIA

- Can be autosomal dominant
- 1 in 5000 in general population.
- Linked to mutations in proteins of cardiac desmosome; essential for mechanical coupling between cells.
- Right heart replaced with fibrofatty tissue
- ECG – QRS duration in lead V1-V3 >110 ms; so called the Epsilon wave – delayed activation in R ventricle.
VT

- IV Ca blocker or B blocker for structural normal heart VT especially RVOT; Fasciular
- For incessant or recurrent VT, IV amiodarone especially in abnormal heart patient
- Cardiovert in BP unstable patient.
- Anti-arrhythmic medications in structural abnormal heart had fair efficacy and side effects
For sustained monomorphic VT with a normal structure heart such as RVOT VT, we can try radiofrequency ablation.

For scar related VT, we can try to ablate to stop the VT storm. That would ameliorate the VT attack frequencies.

Recurrent VT – ideal treatment for catheter ablation.
- In polymorphic VT or in structurally abnormal heart
- Had cardiac arrest before (class 1 indication).
- For MADIT trial, ICD improved survival by 20% in 1 year. For EF <35%, prophylactic ICD advocated.