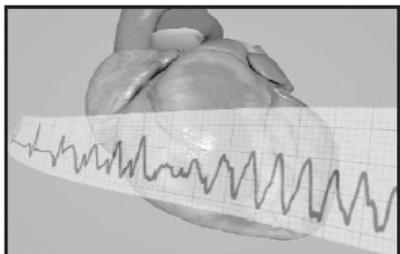
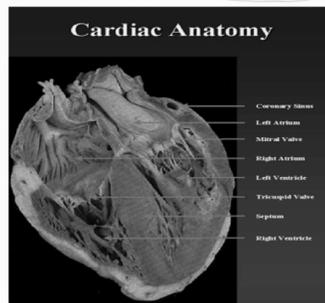


VENTRICULAR RHYTHM DISTURBANCES

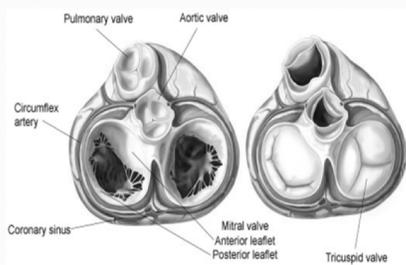


A premature ventricular beat causing ventricular fibrillation.

Cardiac Anatomy

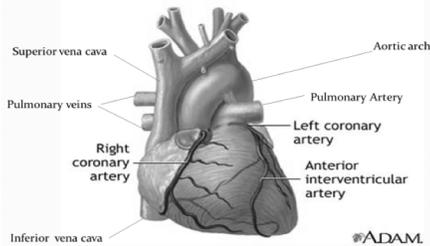


Cardiac Valves

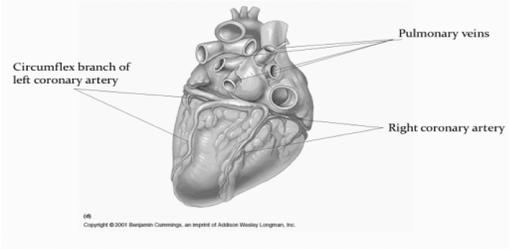


Cardiac Circulation

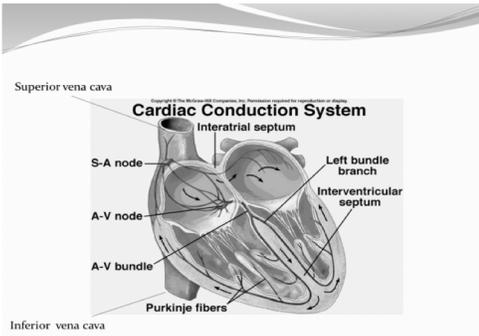
Coronary Arteries



Circumflex



Cardiac Conduction System



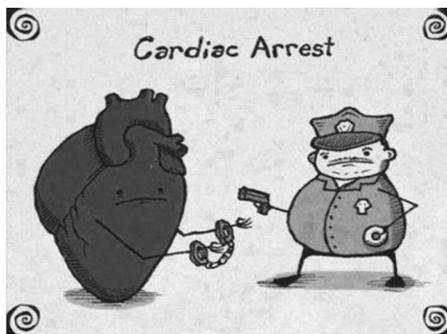
Cardiac Cycle

- ▣ <https://www.youtube.com/watch?v=CWFyxn0qDEU>
- ▣ <https://www.youtube.com/watch?v=RYZ4daFwMa8>

Normal Sinus Rhythm



Sudden Cardiac Arrest



The objectives for this session are:

- ▣ 1. Understand ventricular arrhythmias malignant vs Benign
- ▣ 2. Understand the causes of malignant ventricular arrhythmias and how to detect them
- ▣ 4. Understand pharmacological management for ventricular arrhythmias
- ▣ 5. Understand Electrophysiology options for the treatment of ventricular arrhythmias
- ▣ 6. Understand when is anticoagulation appropriate

Cardiac arrest or death

- ▣ Sudden cardiac arrest is a rarity among the young, especially athletes. According to Cleveland Clinic, sudden cardiac death kills 1 in 100,000 to 1 in 300,000, athletes under 35 years, more often males.
- ▣ Worldwide sudden cardiac death (SCD) is about 25% of the 17 million deaths every year. Cardiovascular diseases are responsible for these deaths. Men have a higher incidence of SCD than women, and it increases with age due to the prevalence of CAD in older adults.

Known athletes with SCD



- ▣ U.S. Olympic volleyball player Flo Hyman (1986), college basketball player Hank Gathers (1990), professional basketball players Pete Maravich (1988), Reggie Lewis (1993),
- ▣ Professional soccer players; Piermario Morosini (Italy, 2012), Antunio Puerta (Seville, Spain 2007), Marc "Marco" Vivien Foe (Cameroon, 2003), and Miklos Feher (Hungary, 2004)

What is Cardiac Arrest?

BUT EXACTLY WHAT IS CARDIAC ARREST?
WHAT IS IT TRIGGERED BY AND HOW IS IT DIFFERENT FROM A HEART ATTACK?

<p>CARDIAC ARREST Occurs when the heart malfunctions and stops beating unexpectedly. It's triggered by an electrical malfunction in the heart that causes an irregular heartbeat (arrhythmia).</p> 	<p>HEART ATTACK Occurs when blood flow to the heart is blocked. It's a circulation problem that is triggered by a blocked artery that prevents oxygen-rich blood from reaching a section of the heart.</p> 
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SIGNS & SYMPTOMS TO LOOK OUT FOR

 SUDDEN COLLAPSE	 NO PULSE	 LOSS OF CONSCIOUSNESS	 NO BREATHING
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Many of us know the difference between cardiac arrest and heart attacks. As we all know, cardiac arrest is the result of the cardiac electrical system malfunction, which leads to no contractions and no perfusion, which impairs the brain, lungs, kidneys, and liver function after a few minutes and death occurs.

Cardiac arrest may occur suddenly or within an hour if symptoms are present. Symptoms can consist of shortness of breath, chest pain, chest pressure, dizziness, or fainting. Signs consist of collapse, no pulse, loss of consciousness and no breathing.

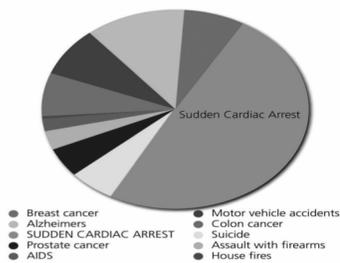
Myocardial infarction

A heart attack or myocardial infarction occurs coronary artery stenosis is more than 80% occluded which prevents oxygen-rich blood from reaching the cardiac muscle. If no intervention is done within minutes to open the vessel the cardiac muscle begins to die. During this process the person begins to experience symptoms which may be immediate and intense. Many times the symptoms begin slowly and may persist for hours, days or weeks before a myocardial infarction (MI) occurs. During an MI, most of the time the heart continues to contract and patients may remain conscious. Unlike with sudden cardiac arrest, the heart usually does not stop beating during a heart attack. The heart attack symptoms in women can be different from men.

Link

These two cardiac conditions are linked. Sudden cardiac arrest (SCA) can occur with a myocardial infarction or during recovery. The fact that a person may have an MI, will increase the risk of SCA. Most MIs do not lead to sudden cardiac arrest. However, if SCA does occur it usually is associated with an MI, especially if the patient is older (>60 years old).

CAUSES OF DEATH IN THE U.S.



SOURCE: Compiled by Sudden Cardiac Arrest Foundation, www.sca-aware.org, 2010, from Centers for Disease Control (2006) and American Heart Association (2009) data



Causes of Cardiac Arrest / Death

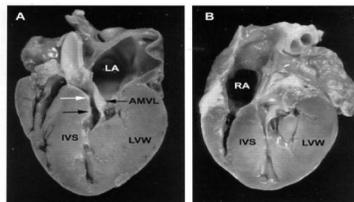
According to the European Society of Cardiology 2015 guidelines, cardiac diseases associated with Sudden cardiac death (SCD) differ in young vs. older individuals, over 40 years. In the young there is a predominance of channelopathies and cardiomyopathies, myocarditis and substance abuse, while in older populations, chronic degenerative diseases predominate (Coronary artery disease, valvular heart diseases and Heart Failure). In younger persons, the cause of SCD may be elusive even after autopsy, because conditions such as inherited channelopathies or drug-induced arrhythmias that are devoid of structural abnormalities are epidemiologically relevant in this age group.

- ▣ Today's presentation will be discussing the conditions or diseases that lead to lethal ventricular arrhythmia such as Ventricular tachycardia, Ventricular Fibrillation and Torsade de Pointe.
- ▣ There are many causes and ACLS has their own list but we will be concentrating on Hypertrophic Cardiomyopathy, Arrhythmogenic right ventricular dysplasia (ARVD), Marfan syndrome, Prolong QT interval, other ventricular arrhythmia causes, and Ventricular fibrillation
- ▣ Benign conditions consist of Ventricular premature beats, ventricular tachycardia in normal structured heart

ACLS list of causes

Cardiac arrest- causes

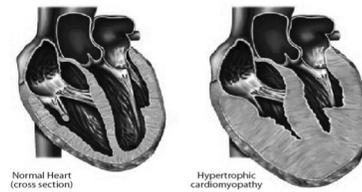
- Thrombosis- MI
- Thromboembolism- PE
- Tension pneumothorax
- Tamponade- cardiac
- Trauma
- Tablets- drugs/toxins
- Hypovolemia
- Hypoxia
- Hydrogen ions- acidosis
- Hyper/hypokalemia
- Hypothermia
- Hypo/hyperglycemia



Hypertrophic Cardiomyopathy

(HCM) is the leading cause of SCD in young people including athletes (a)

- ▣ College basketball player Hank Gathers (1990), Professional basketball player Reggie Lewis (1993), Hungarian soccer player Miklos Feher (2004)
- ▣ It is a heterogeneous disorder characterized by variable degrees of left ventricular hypertrophy, normal or small left ventricular cavity size and hyperdynamic systolic function.
- ▣ Few people with predominantly interventricular septal hypertrophy in association with anterior motion of the mitral leaflets and subvalvular left ventricular outflow tract obstruction, which leads to a condition denoted as hypertrophic obstructive cardiomyopathy. This condition affects 1 in 500 individuals in the general population.

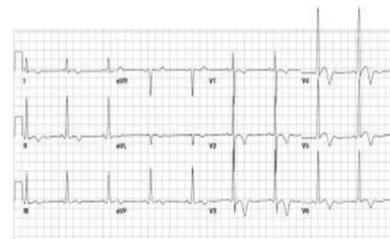


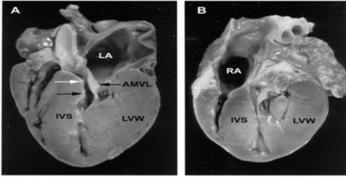
- ▣ There is a variant of HCM that is found in the elderly often-female patients with HTN, There are over 50 different mutations affecting the myosin heavy chains and myosin light chains, tropomyosin and troponin.
- ▣ HCM is characterized by the dynamic LV outflow tract obstruction (LVOFT) and diastolic dysfunction.
- ▣ The degree of LVOFT obstruction may vary below the aortic valve; this can later present itself by a narrowed septal hypertrophy.

Diagnosing HCM

- ▣ The clinical manifestations also vary. Some patients are asymptomatic for many years.
- ▣ Others present with life-threatening arrhythmias or sudden cardiac death. The most frequent symptoms is dyspnea on exertion.
- ▣ This is secondary to diastolic dysfunction and elevation in LV filling pressures. This is a progressive disease. Syncope can result from severe obstruction to the LVOFT and concomitant reduced cardiac output as well as arrhythmias. If the right ventricle is involved in the myopathy process, symptoms of right-sided heart failure may also be present.
- ▣ Patients with significant outflow tract obstruction may typically present with a harsh, crescendo-decrescendo systolic murmur heart best at left sternal border that is intensified by maneuvers that decrease left ventricular volume. An S4 is present with increased ventricular stiffness
- ▣ An echocardiogram is recommended, which will help in diagnosing patient with suspected HCM
- ▣ The EKG will demonstrate an increased QRS voltage secondary to Left Ventricular Hypertrophy (LVH). Prominent abnormal Q waves are frequently present in the inferior and lateral leads and reflect depolarization of the hypertrophied septum. Negative T waves in precordial leads and lateral leads, and deep S wave in precordial leads.

12 lead ECG: Is he having an MI ?



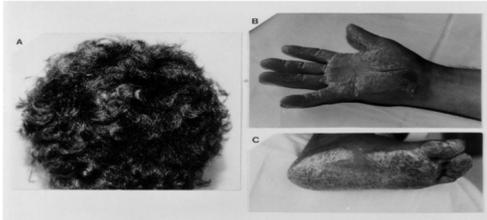


Arrhythmogenic right ventricular dysplasia

- (ARVD) is an inherited heart disease which is a progressive condition of the right ventricular cardiac muscle which leads to ventricular tachycardia, HF and SCD. The histological hallmark of the disease is replacement of cardiomyocytes by adipose and fibrous tissue.
- Clinically, arrhythmogenic right ventricular cardiomyopathy (ARVC) is defined by structural and functional abnormalities of the right ventricle, but LV involvement occurs in 50% of patients. In most cases ARVC is inherited as an autosomal dominant genetic trait caused by mutations in genes encoding for desmosomal proteins.
- ARVC has an estimated prevalence of 1 in 1000 to 1 in 5000 of the general population and is an important cause of SCD in athletes and young adults. ARVC is an important cause of ventricular arrhythmias in children and young adults. It is seen predominantly in males, and 30-50% of cases have a familial distribution

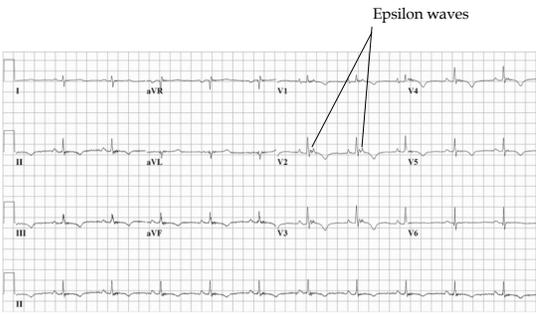
Diagnosing ARVD

- ▣ Clinical manifestations include palpitations, syncope, VT and SCD, usually develop between the second and fourth decade of life. As the disease progresses, it can result into right or biventricular heart failure.
- ▣ ARVD can be found in association with diffuse palmoplantar keratoderma, and woolly hair. It is an autosomal recessive condition called Naxos disease, because this genetic abnormality can also affect the integrity of the superficial layers of the skin most exposed to pressure stress.



- ▣ Up to two-thirds of patients have ventricular arrhythmias on ECG, holter monitoring and exercise testing, stress testing or cardiopulmonary exercise testing. These ventricular arrhythmias are usually of RV origin (i.e. show a left bundle branch morphology), but the QRS axis during VT usually differs from the QRS axis in RVOT, and many patients have multiple QRS morphologies.
- ▣ Medical treatment is not optimal, however Sotalol which is an antiarrhythmic medication has shown to reduce frequent ventricular ectopy or Non-sustained ventricular arrhythmias. However, amiodarone has shown to be superior in preventing ventricular arrhythmias vs BB therapy.
- ▣ Electrophysiology study with voltage mapping identifies regions of fibro-fatty replacement and to guide catheter ablation of ventricular arrhythmias. Acute suppression of VT is more often successful in patients presenting with a single or only a few selected dominant VT morphologies and epicardial ablation may increase success rates. As neither anti-arrhythmic drugs nor catheter ablation provides sufficient protection against SCD, ablation should be used to reduce the frequency of arrhythmia episodes rather than to improve prognosis.
- ▣ Automatic implantable cardiac defibrillators is the most appropriate therapy for sustained monomorphic ventricular tachycardia.
- ▣ Competitive exercise may exacerbate lethal ventricular arrhythmias.

ARVD EKG



ARVD CT Scan



Other Cardiomyopathies

- ▣ **Cardiac Amyloidosis** consists of light-chain amyloidosis which is caused by deposition of monoclonal light chains and hereditary transthyretin-associated amyloidosis, in which normal (wild-type) or mutant transthyretin is deposited in the myocardium.
- ▣ Cardiac amyloidosis has been associated with a very poor prognosis, usually a median survival of .1 year after the onset of heart failure symptoms. Up to half of all patients with cardiac amyloidosis die suddenly. Death is attributed to ventricular arrhythmias, however case reports reveal a successful termination of sustained ventricular arrhythmias with the aid of an implantable cardiac defibrillator (ICD).
- ▣ Holter monitoring, home monitoring and device monitoring reveals that ventricular arrhythmias are present in 25% of patients with cardiac amyloidosis, but their presence does not seem to predict SCD. ICDs should be considered in patients with light-chain amyloidosis or hereditary transthyretin-associated amyloidosis. There are insufficient data to provide recommendations on primary prophylaxis.

Restrictive Cardiomyopathy

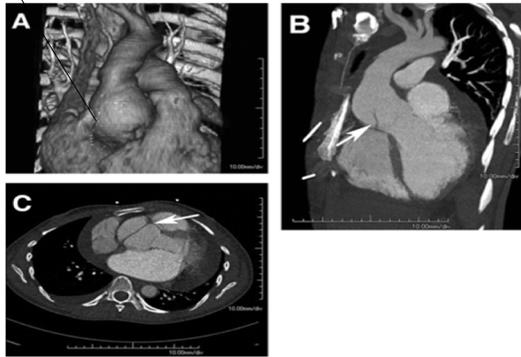
- ▣ It is the least common cardiomyopathies and is caused by number of genetic and acquired disorders. In adults the common cause is amyloidosis followed by mutations in sarcomeric protein genes and metabolic disorders. Patients with restrictive cardiomyopathy present with signs and symptoms of biventricular heart failure and diagnosed by characteristic features on non-invasive cardiac imaging and cardiac catheterization. Restrictive cardiomyopathy is associated with poor long-term prognosis.
- ▣ In children, the risk of sudden death may be higher, particularly in those with ECG evidence of myocardial ischemia. The treatment of restrictive cardiomyopathy is mostly palliative.
- ▣ HF symptoms are treated with diuretics and heart rate control to optimize LV filling. Anticoagulation should be used in all patients with atrial fibrillation. There is no prospective data on prophylactic implantation of ICDs in restrictive cardiomyopathy, so for patients with symptomatic sustained ventricular arrhythmias, indications for ICD should be similar to other cardiomyopathies, taking into account the short-term prognosis related to HF.

Marfan Syndrome

- ▣ Marfan syndrome is an autosomal dominant, connective tissue disorder which affects multiple systems. It is characterized by long bones, or tall and thin, long arms, fingers, toes, flexible joints and scoliosis. The most serious conditions deals with mitral valve prolapse and dilatation of the aortic root.
- ▣ In 1896, Antoine Bernard-Jean Marfan described the syndrome in a young patient with peculiarly long and thin digits, elongated limbs, and congenital contractures of multiple joints.
- ▣ Overtime, contracture arachnodactyly, a connective tissue disorder, was described in 1968. Fifty years later, marfan's features in other systems were described in patients with thin, elongated limbs including mitral valve disease in 1912; dislocation of the ocular lens in 1914; ruptured aortic aneurysm in 1918; aortic root dilatation and dissection in 1943.
- ▣ Marfan syndrome is clearly one of the more common, potentially lethal Mendelian conditions with an estimated prevalence of 1 case per 3000 to 5000 individuals.
- ▣ Cardiovascular conditions are the predominant feature of Marfan syndrome which includes proximal ascending aortic dilatation, dilatation of the proximal main pulmonary artery, thickening and prolapse of either or both atrioventricular valves, and mitral annular calcification.

Aortic dissection in Marfan's

Dissection lesion



Diagnosing Marfan's

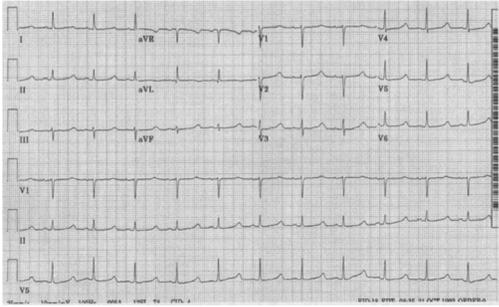
- ▣ There are EKG abnormalities in Marfan syndrome patients which include prolonged atrioventricular conduction time (first degree or second AV blocks), and ST-segment abnormalities. Ventricular repolarization abnormalities, prolong QT interval and the presence of U waves, are associated with a higher prevalence of ventricular arrhythmias.
- ▣ Ventricular arrhythmias were seen in 21% of patients and with SCD attributed to primary arrhythmias in 4%. Marfan syndrome patients with Left ventricular dilation have a higher incidence of prolong QT interval and ventricular arrhythmias.
- ▣ Women with Marfan have to worry about having a child with autosomal dominant disorder and pregnancy due to the ascending aortic dilation, which will occur during pregnancy and delivery. The Aortic size to minimize complications is <4.2 cm. These patients are referred to cardiologist who will perform a periodic echocardiogram during pregnancy.

- ▣ Mitral valve prolapse is another condition, which can affect Marfan syndrome. The incidence can range from 50% and 80% of cases. Marfan syndrome patients with mitral valve prolapse present with variable degrees of mitral regurgitation, with up to 12% to 13% having moderate or severe mitral regurgitation.
- ▣ Although the associated left ventricular volume overload and systolic function may be associated with sudden cardiac death, this is uncommon below the age of 50 years. Surgical repair of the severely regurgitated mitral valve is possible and has been associated with a high event-free survival at 10 years.

Prolong QT interval

- ▣ The measurement of QT interval is the duration of ventricular repolarization or resting phase of ventricular myocardium. It measures the start of depolarization at the Q point of an ekg and the end of repolarization at the end of the T wave of an EKG in relation to the ventricular myocardium. When the QT is prolong there is a risk of ventricular arrhythmias which can result in early after depolarizations, provoking Torsades des Pointes, which leads to ventricular fibrillation, which ends in sudden cardiac arrest. (a)
- ▣ The 2015 European Society Cardiology panel established new guidelines on diagnosing long QT syndrome without a second cause of prolong QT. The measurement of 500 milliseconds (ms) was suggested as the threshold for asymptomatic LQTS (without a family hx of disease). This measurement is identical to the QT duration associated with high risk for arrhythmic events in SCA. The corrected QT (QTc) is >480 ms or score of three for clinical diagnosis. If unexplained syncope is present then a QTc will be >460 ms is sufficient to make diagnosis. (b)
- ▣ Long QT Syndrome (LQTS) is characterized by prolonged QT interval and ventricular arrhythmias mainly triggered by adrenergic activation. The mean age at presentation is 14 years. The annual rate of SCD in patients with untreated LQTS is estimated to be between 0.33 and 0.9%, whereas that for syncope is estimated to be 5%. Mutations in 13 genes have been associated with LQTS, most encoding for subunits of potassium, sodium or calcium voltage dependent ion channels. Genetic screening identifies a disease-causing mutation in 75% of LQTS cases and three main genes (KCNQ1, KCNH2 and SCN5A) account for 90% of positively genotyped cases. There are subtypes of LQTS. (b)

Prolong QT ekg



Diagnosing prolong QT

An electrocardiogram and genetic testing is recommended. Those patients who survive a SCA, have a high risk of recurrence, even while on Beta blocker therapy. There is a 14% risk within 5 years on therapy. Since the risk is this high an implantable cardiac defibrillation is highly recommended. These patient's risk increases for SCA if recurrence of syncope events.

Postpartum women with LQTS have an increased risk during the 9-month recovery period (especially women with the LQT2 genotype).

Silent carriers of pathogenic mutations present a modest risk of cardiac events estimated at 10% between birth and age 40 years; the use of beta-blockers should be considered in this group of patients. Prophylactic ICD therapy may be considered, on an individual basis, in high-risk patients such as women with LQT2 and QTc > 500 ms, patients with QTc > 500 ms and signs of electrical instability and patients with high-risk genetic profiles (carriers of two mutations, including Jervell and Lange-Nielsen syndrome or Timothy syndrome).

Recommendations in all patients with diagnosis of LQTS

- ▣ (a) Avoidance of QT-prolonging drugs
- ▣ (<http://www.crediblemeds.org>).
- ▣ (b) Correction of electrolyte abnormalities (hypokalemia, hypomagnesaemia, hypocalcaemia) that may occur during diarrhea, vomiting or metabolic conditions.
- ▣ (c) Avoidance of genotype-specific triggers for arrhythmias (strenuous swimming, especially in LQTS1, and 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. (a)
- ▣ Drug interactions play an important role in the care of patients with polypharmacy and the potential for SCA. Vandenber, Vandael, Robyns, et al., (2016), did an analysis to determine which QT correction formula to use in an automated QT-monitoring algorithm in their electronic medical record. They studied rate correction performance of different QT correction formulae and their impact on risk assessment for mortality. They took five published QT correction for heart rate and did a comparison calculation with the following formulas. Bazett9: $QTcB=QT/RR^{1/2}$; Fridericia10: $QTcFri=QT/RR^{1/3}$; Framingham11: $QTcFra=QT+0.154(1-RR)$; Hodges12: $QTcH=QT+0.00175(160/RR-60)$; Rautaharju13: $QTcR=QT-0.185(RR-1)+k(k=+0.006$ seconds for men and +0 seconds for women)

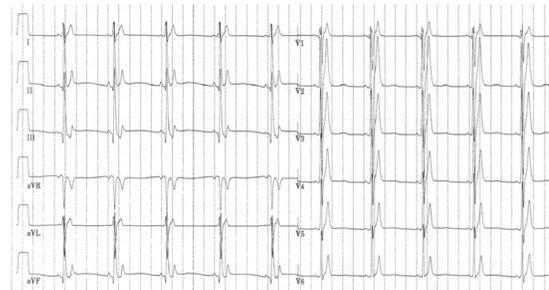
Formulas and drug interactions

- ▣ The study resulted in having two QT correction for heart rate formulas to be consistent which are Fridericia ($QTcFri= QT / RR^{1/3}$ and Framingham ($QTcFra=QT+0.154(1-RR)$), correction formula showed the best rate correction and significantly improved prediction of 30-day and 1-year mortality. (a)
- ▣ The most frequent prolong QT interval is related to adverse drug reactions.
- ▣ The following medications have been known to cause QT prolongation haloperidol, vemurafenib, ziprasidone, methadone and sertindole. Some antiarrhythmic drugs, such as amiodarone or sotalol will cause prolong QT intervals in some patients. (2015 ESC Guidelines) Other potential drugs such as second-generation antihistamines, such as astemizole, and high blood alcohol concentrations can prolong the QT interval. A possible interaction between selective serotonin reuptake inhibitors and thiazide diuretics is associated with QT prolongation. (Macrolide antibiotics are also suspected to prolong the QT interval, after it was discovered recently that azithromycin was associated with an increase in cardiovascular death [US Food and Drug Administration, 2016])

Short QT interval

- ▣ Short QT syndrome (SQTS)
- ▣ SQTS is diagnosed in the presence of a QTc \leq 340 ms. SQTS should be considered in the
- ▣ presence of a QTc \leq 360 ms and one or more of the following:
 - ▣ (a) A confirmed pathogenic mutation
 - ▣ (b) A family history of SQTS
 - ▣ (c) A family history of sudden death at 40 years
 - ▣ (d) Survival from a VT/VF episode
- ▣ Short QTs is the short interval duration of cardiac repolarization, which constitutes the substrate for the development of life threatening arrhythmias. Five genes are linked to SQTS (KCNH2, KCNQ1, KCNJ2, CACNA1C and CACNB2b), however the yield of genetic screening remains low (20% overall). The disease appears to be highly lethal in all age groups, including children in their first months of life, and the probability of a first cardiac arrest by the age of 40 years is .40%. Given the small size of the populations reported so far, the high lethality may partially reflect a reporting bias related to a reporting bias related to the under detection of SQTS in asymptomatic patients.

SQT syndrome EKG



Other causes for Ventricular arrhythmias

- ▣ There are multiple other causes for ventricular arrhythmias including Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia and early depolarization syndrome
- ▣ Congenital heart disease in children and adults, depending on the type of cardiomyopathy which will require cardiology evaluation and lifetime follow up.
 - ▣ Tetralogy of Fallot
 - ▣ Single ventricular disease (Hypoplastic left heart)

Normal structured and VA

- ▣ Ventricular arrhythmias in a structurally normal heart are called idiopathic Ventricular tachycardia or premature ventricular contractions. These occur about 70% and it originates from the right ventricular outflow tract. Other origins include the aortic sinuses of Valsalva, LVOT, great cardiac veins, and epicardial myocardium, aorta-mitral continuity and rarely the pulmonary artery.
- ▣ This idiopathic arrhythmia may occur without structural heart disease; however, subtle wall abnormalities have been demonstrated on Cardiac magnetic resonance imaging in some patients. They have a focal mechanism secondary to automaticity, micro-re-entry or triggered activity.
- ▣ Idiopathic RVOT-VT typically presents between the ages of 20 and 50 years and more frequently in women. There are two typical forms: exercise/stress-induced VT and repetitive monomorphic VT occurring at rest. Repetitive NSVT occurs in 60-92% of cases while incessant VT occurs only occasionally. Paroxysmal sustained VT separated by long periods of infrequent PVCs is less common. Episodes increase in frequency and duration during exercise and/or emotional stress; exercise tests may provoke focal OT-VT during the exercise or recovery phases.
- ▣ Typical QRS morphology is an inferior axis with dominant LBBB morphology. VT is monomorphic, however, the QRS morphology may vary slightly. Multiple distinct VT morphologies are very rare and raise the suspicion for scar-related VT, such as in ARVC. Although idiopathic OT-VT follows a benign course, malignant VT may occasionally occur. ECG during sinus rhythm is usually normal, however, 10% have complete or incomplete RBBB. Exercise testing and cardiac imaging should be performed to exclude the presence of underlying structural heart disease, and cardiac catheterization may be warranted in some cases.

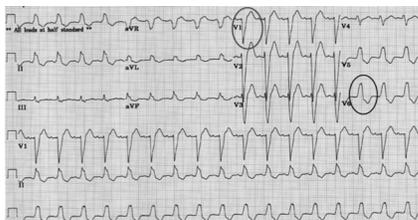
Normal Sinus Rhythm



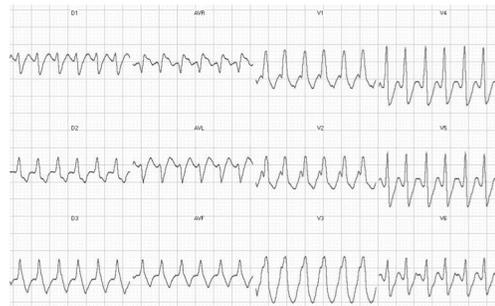
PVCs



Left Bundle Branch Block



Right Bundle Branch Block



Treatment

- ▣ Treatment is only warranted if patients are symptomatic. It is worth noting that symptoms may be related to LV dysfunction, considering that idiopathic VT may be a cause of tachycardia-induced Cardiomyopathy.
- ▣ In such patients, treatment with sodium channel blockers (class IC agents) or catheter ablation should be considered. In patients with RVOT-VT/PVCs, primary catheter ablation is recommended, whereas in patients with LVOT-VT/PVCs, catheter ablation should only be considered after failed anti-arrhythmic therapy. The close anatomical proximity of the RVOT, LVOT and great cardiac veins limits precise localization of the VT origin based on QRS morphology except for classic RVOT tachycardia.

Are you screening?

- ▣ Patients coming in for a physical exam if over 40 years old a 12 lead EKG is recommended. An EKG is recommended on anyone presenting with chest discomfort, fainting, or near syncope
- ▣ An ekg should be considered on anyone with a cardiac murmur, family hx of sudden cardiac death
- ▣ Patients who present for the first time with high blood pressure >160/ 90 in the office should have an EKG done for evaluation.
- ▣ Patients with hx of CAD, HTN, CHF, obese and over 65 years old should have an EKG screening. Any abnormal ekg should be further evaluated by Cardiology consideration of an echocardiogram. Any premature beats of 2 or more should undergo a 24 hour holter monitor to evaluate for PVC or PAC burden (normal burden is 1-5%) anything above 6% or higher an echocardiogram and a two week or 30 day event monitor is recommended for symptom correlation
- ▣ Any abnormal echocardiogram will require a stress test or cardiac CT scan with contrast vs Cardiac MRI with contrast especially if looking for ARVD vs Coronary artery disease or CHF scarring. Cardiology may recommend a cardiac catheterization of the left side for coronary angiography, if COPD is present a right sided cardiac catheterization is recommended, if echocardiogram PAP is >40 mm HG

Questions



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