SUDDEN CARDIAC ARREST

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FENNVILLE, Michigan... Wes Leonard's game-winning layup in overtime brought the
Fennville High School crowd to its feet, and joyous teammates and fans quickly surrounded their star player. Then the seemingly impossible happened: Leonard collapsed and died. He was just 16. Leonard was rushed to nearby Holland Hospital, where paramedics performed CPR before he was pronounced dead. An autopsy conducted Friday by the Ottawa County medical examiner showed Leonard died of cardiac arrest due to an enlarged heart.

INTRODUCTION

SUDDEN CARDIAC DEATH is the demise of a person due to a Sudden Cardiac Arrest. Approximately 7 million worldwide and 325,000 persons in the United States die each year in the field or emergency departments due to Sudden Cardiac Death. This does not include heart attacks and typically is due to arrhythmias. Men have a higher propensity than women with blacks more susceptible than whites.

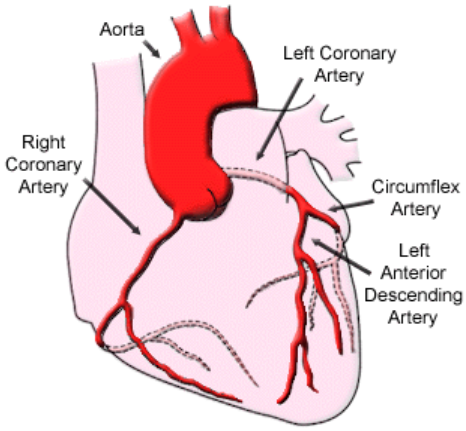
SUDDEN CARDIAC DEATH is not a phenomenon exclusive to adults. Pediatric deaths occur due to undetected cardiac abnormalities or direct traumatic insults to the heart. Bacterial and viral illnesses can weaken the heart in both adults and adolescents.

Co-morbid conditions also play a role in the evolution of Sudden Cardiac Death. Congestive heart failure has a high incidence of ventricular tachycardia and also brady-arrhythmias. Left ventricular hypertrophy has a propensity for Ventricular Premature beats that can cause sustained arrhythmias.

***"SUDDEN CARDIAC ARREST**: is the sudden cessation of cardiac activity so that the victim becomes unresponsive, with no normal breathing and no signs of circulation. If corrective measures are not taken rapidly, this condition progresses to sudden death. Cardiac arrest should be used to signify an event as described above, that is reversed, usually by CPR and/or defibrillation or cardioversion, or cardiac pacing. Sudden cardiac death should not be used to describe events that are not fatal." AHA*..... We therefore are interested in SUDDEN CARDIAC ARREST

SUDDEN CARDIAC ARREST can be classified in four groups

1. Coronary artery disease and ischemia
2. Structural abnormalities of the heart
3. Cardiac conduction defects
4. Non cardiac causes



CARDIAC ISCHEMIA

CARDIAC ISCHEMIA results in a loss or diminished blood flow to parts or all of the heart. We are all aware that the coronary arteries can become occluded and this results in an infarction to that part of the heart. **Coronary artery spasm** is seen with drug use such as cocaine and with **Takotsubo Syndrome**.
Takotsubo Syndrome is typically seen in elderly females who are suffering from some form of grief. They may present with an EKG consistent with an acute MI but the catheterization is normal

ISCHEMIA	LEADS	ARTERY	LOCATION
SEPTAL	V1	LEFT ANTERIOR DESCENDING	SEPTUM
ANTERIOR-SEPTAL	V1-V4	LEFT ANTERIOR DESCENDING	ANTERIOR WALL
ANTERIOR	V2-V4	LEFT ANTERIOR DESCENDING	ANTERIOR WALL
LATERAL	V5,V6,1,AvL	CIRCUMFLEX	LATERAL WALL
INFERIOR	II, III, AvF	RIGHT CORONARY 90% CIRCUMFLEX 10%	INFERIOR WALL
POSTERIOR	V2-V4	RIGHT CORONARY	POSTERIOR WALL

Coronary Artery Embolism results in a blockage caused by a clot which dislodges in one part of the body and plugs up an artery at a point where the clot is larger than the lumen of the artery. These may create a blockage because of the sheer size of the clot; due to a narrowing of the vessel from heart disease; or may get lodged in an area where the heart has a congenital narrowing.

Nonartherogenic coronary artery disease refers to damage to the vessels that is not due to plaque build up. Tears or **dissections** to the aorta may extend into the coronary arteries, or a coronary artery may rip on its own. **Congenital anomalies** to the coronary arteries along with **inflammation to the arteries or arteritis** can all result in lethal narrowing and blockage.

STRUCTURAL ABNORMALITIES OF THE HEART

Damage to the heart, or abnormalities of the heart can all result in sudden cardiac arrest. None of these structural causes are related to ischemia or blockage to the coronary arteries. These causes directly damage the heart itself, or secondarily impact the heart so that it cannot function as a pump. In the end, the cause of sudden cardiac arrest with this group is loss of adequate blood flow which can than result in a fatal arrhythmia. In fact, the structural abnormalities can be categorized in this way.

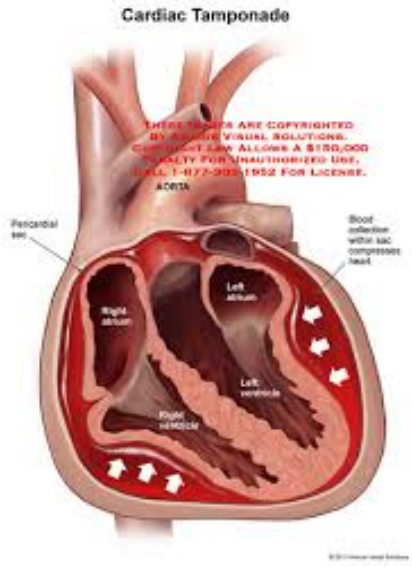
LOSS OF ADEQUATE BLOOD FLOW

- Acute pericardial tamponade
- Aortic dissection
- Acute myocardial rupture.

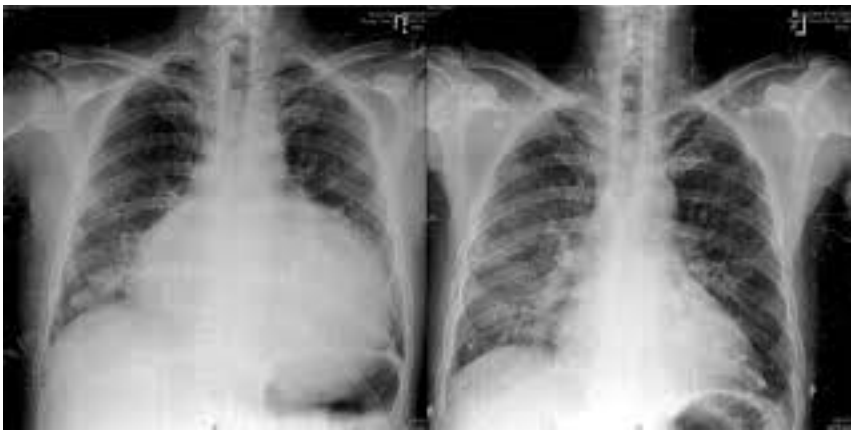
STRUCTURAL DAMAGE TO THE HEART

- Hypertrophic cardiomyopathy
- Dilated cardiomyopathy: enlarged heart typically due to coronary artery disease
- Valvular heart disease
- Congenital heart disease
- Arrhythmogenic right ventricular dysplasia: lacking or abnormal right ventricle
- Myocarditis: inflammation of the heart muscle.

ACUTE PERICARDIAL TAMPONADE results when blood or fluid fills the space between the heart and the pericardium. As the fluid increases, it creates an outward pressure that compresses the ventricles, causing a diminishing blood return into the heart. Typical finding is dyspnea and on exam one may see BECK'S TRIAD: Jugular venous distention, hypotension and muffled heart sounds due to the fluid build up in the pericardium which "muffles" the auscultation. Pulses Paradoxus or a drop of the systolic blood pressure by greater than 10 mmHg during inspiration may also be present. This is also seen in disease processes such as COPD. Echocardiogram is the gold standard for diagnosing the condition.

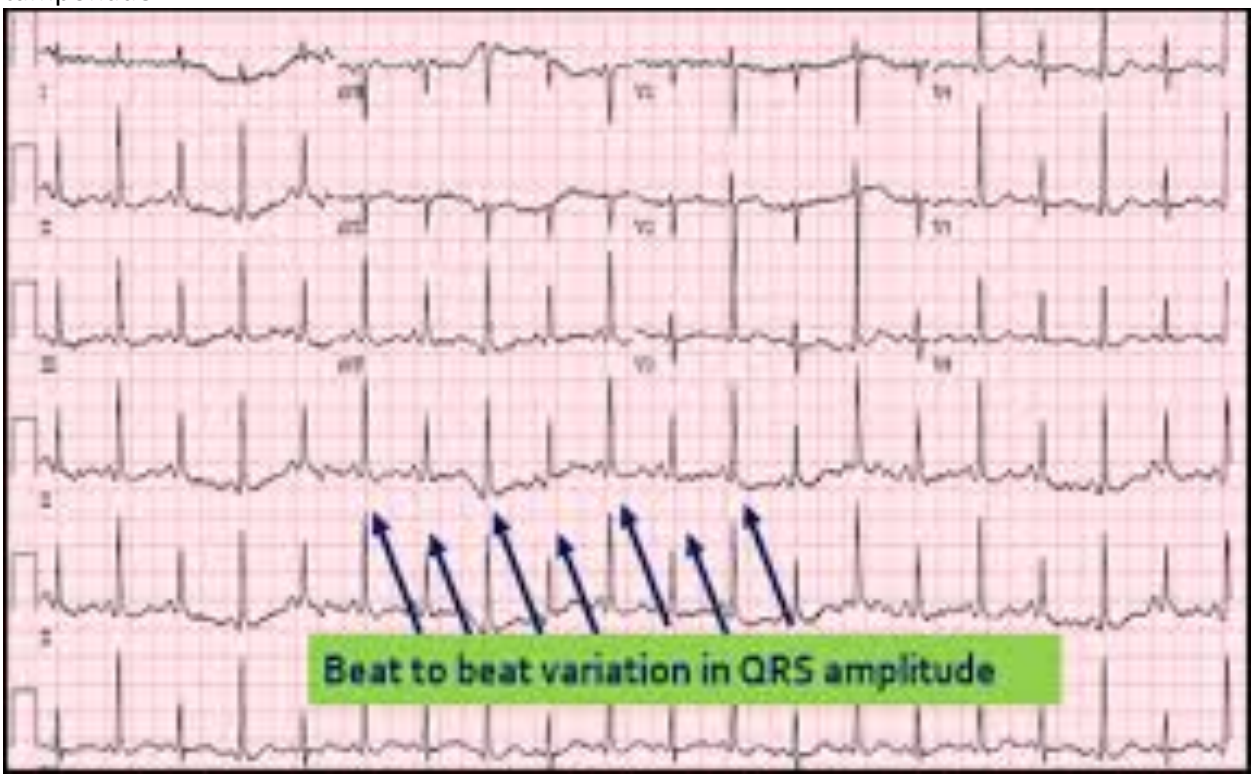


COMPRESSION IS GREATER ON THE RIGHT VENTRICLE DUE TO IT'S LOWER PRESSURES.



THE HEART ON THE LEFT IS ENLARGED DUE TO THE FLUID IN THE PERICARDIUM THAT MAKES THE HEART ROUND AND BOGGY LOOKING. NOTE THE SIDE OF THE NORMAL HEART ON THE RIGHT

However an EKG consistent with electrical alternans is nearly diagnostic of pericardial tamponade

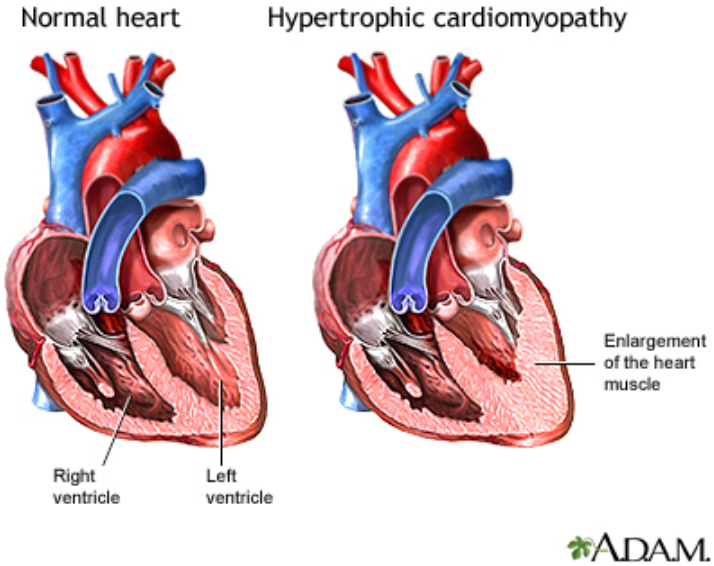


IN ELECTRICAL ALTERNANS, THE QRS COMPLEXES ALTERNATE BETWEEN A LARGE AND SMALLER ONE. THIS IS DUE TO THE WAVE LIKE FLOW BACK AND FORTH CAUSED BY THE HEART BEATING. WHEN THE FLUID IS SWISHED AWAY, THE QRS AMPLITUDE INCREASES AND WHEN IT SWISHES BACK, THE QRS IS SMALLER.

AORTIC DISSECTION is a tear to the aorta which can occur due to high blood pressure, drug use, pregnancy and congenital causes. If the tear goes all the way through the arota, a person would bleed out. In some cases, the tear extends into the coronary arteries when the arch oft he aorta is involved. The classic presentation is a complaint of pain going between the shoulder blades into the neck. Certain congential conditions such as Marfans, and connective tissue disorders have a higher propensity for dissections. Inflamation tot he aorta as well can lead to a disection.

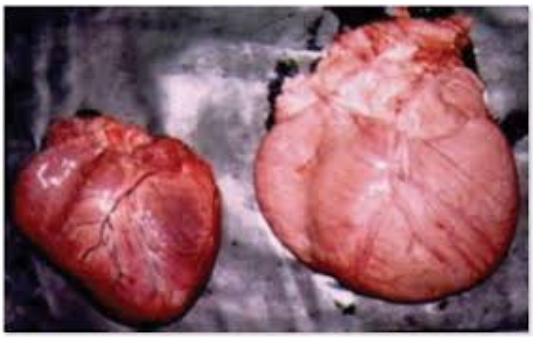
MYOCARDIAL RUPTURE is seen after acute myocardial infarctions and trauma. Mortality as would be expected is high. In some cases, the person is saved by the pericardium clotting off and compressing the bleeding.

HYPERTROPHIC CARDIOMYOPATHY is a genetic condition where the heart muscle becomes enlarged. It affects 1: 500-1000. The left ventricle and in particular, the septum, enlarges along the myocardium. Many people go undiagnosed because there is no obstruction to the blood flow. About a third however, have an enlarged septum that narrows the blood flow through the aortic valve resulting in resistance when the blood leaves the heart. As well, the Mitral Valve tilts inward pushing blood toward the juxtaposed septum instead of the hollow ventricle. This increases the amount of resistance through the aortic valve. Another name for this is **IDIOPATHIC HYPERTROPHIC SUBAORTIC STENOSIS** which more accurately describes the location as well as the condition of the heart. As stated, it is hereditary and symptoms are usually manifested during exercise. Shortness of breath, palpitations, chest pain, fatigue and syncope are all antecedent symptoms. Exertional syncope is oftentimes the first indication and in some cases, a fatal arrhythmia is the first and only noted symptom.



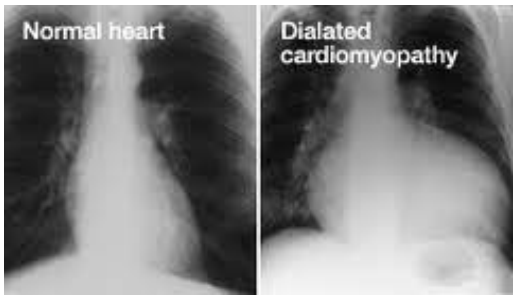
DILATED CARDIOMYOPATHY results from the heart muscle getting streched out so that the muscle fibers lose their contractile strength. Think of the heart muscle as and elastic band. Over time, an elastic band gets stretched out and loses it's ability to contract. In a cardiomyopathy, the same thing happens to the heart muscle. Since the muscle cannot contract to it's normal length, the heart dilates. The muscle also loses it's ability to forcefully contract and therefore it's ability to effeciently push blood out of heart's chambers, especially the left ventricle. Because the heart muscle cannot contract appropriately, it cannot provide it's primary function of transporting oxygenated blood. Symptoms reflect this and shortness of

breath along with fatigue are early findings. Edema and CHF are sure to follow. Now with an irritated myocardium, PVC's occur which can lead to a Ventricular Tachycardia arrhythemia.



Enlargement or cardiomegally has multiple different causes including coronary artery disease, alcoholism, drug use, diabetes, pregnancy, viral and bacterial infections and even certain minerals such as cobalt. Several years ago, cobalt was added to beer which resulted in cardiomyopathy.

NOTE THE SIZE OF THE ENLARGED HEART ON THE RIGHT OF BOTH SLIDES

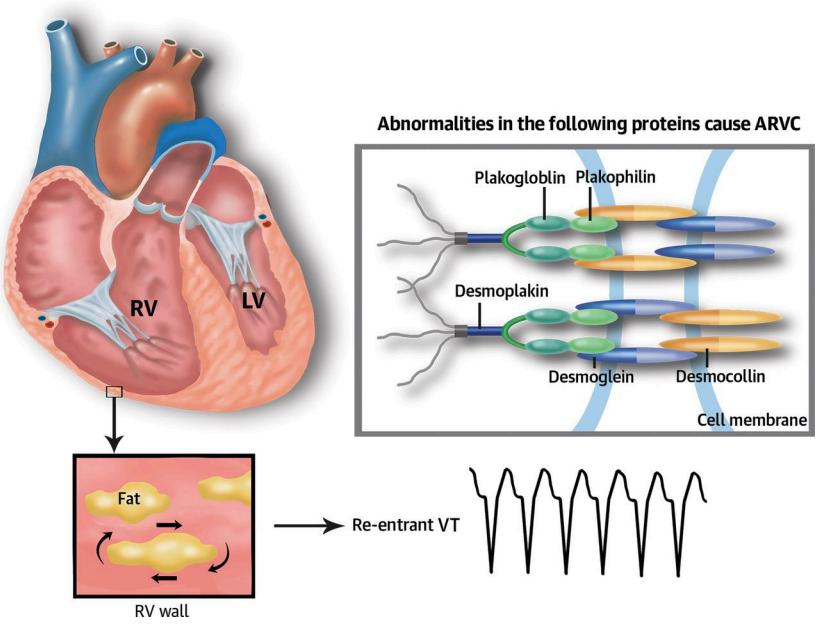


CAUSES OF CARDIOMYOPATHY

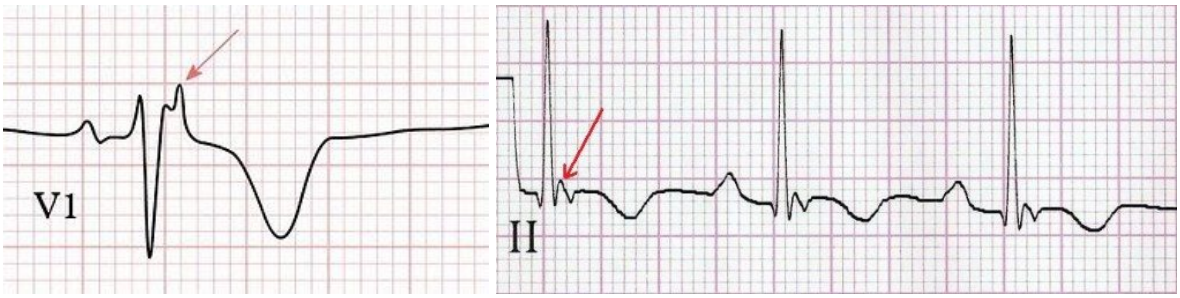
- Coronary Artery Disease, heart attack, high blood pressure, diabetes, thyroid disease, viral hepatitis and HIV
- Infections, especially viral infections that inflame the heart muscle
- Alcohol
- Complications during the last month of pregnancy or within 5 months of birth
- Certain toxins such as cobalt
- Certain drugs (such as cocaine and amphetamines)

MYOCARDITIS can result in similar symptoms as cardiomyopathy. This however is more acute and the heart is inflamed. The inflamation which could be a virus or a bacteria causes the heart to beat inefficiently potentiating a Ventricular Tachycardia Arrest.

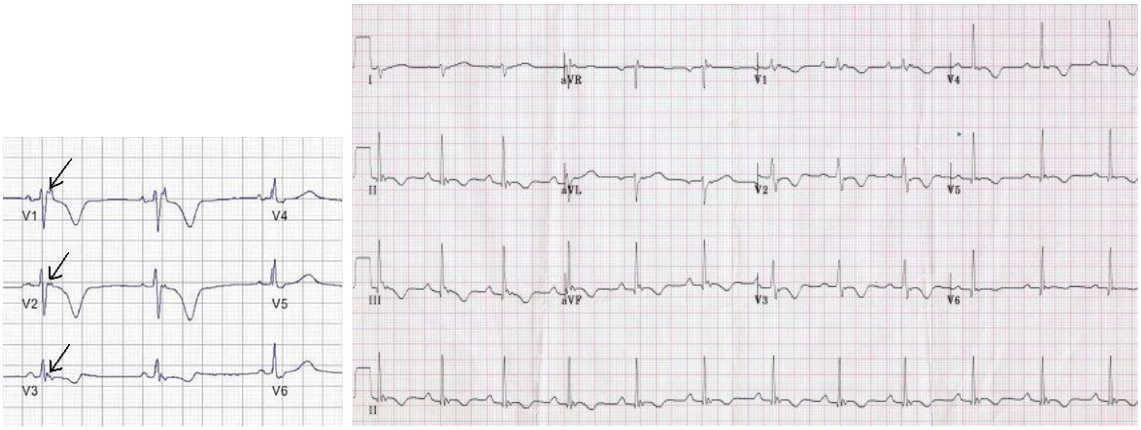
ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY (ARVC) is an inherited myocardial disease that results in a fibro-fatty replacement of the right ventricle muscle. In these cases, the muscle is transposed with fat which causes the right ventricle to initiate a ventricular arrhythmia. It is the second most common cause fo Sudden Cardiac Death for those under 35 years old and again more common in men than women.



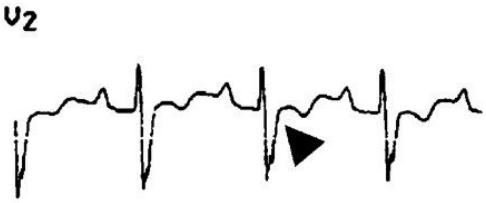
There is no pathognomonic finding that will confirm ARVC but several findings on the EKG can make the diagnosis a high probably. Epsilon wave is the most specific finding in ARVC and is on about a third of the EKG's. These patients have and upward notch at the end of the QRS

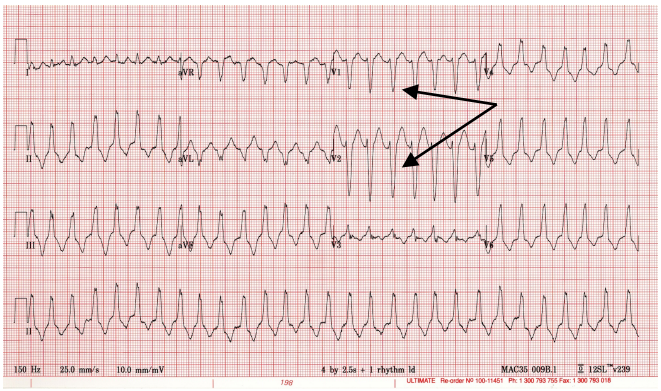


Another finding is T Wave inversion in V1-V3. This is found in about 85% of those with ARVC



Other findings would include a localized widening of the QRS along with a Left Ventricular Tachycardia which is charaterized by a downward slope in V1 and V2





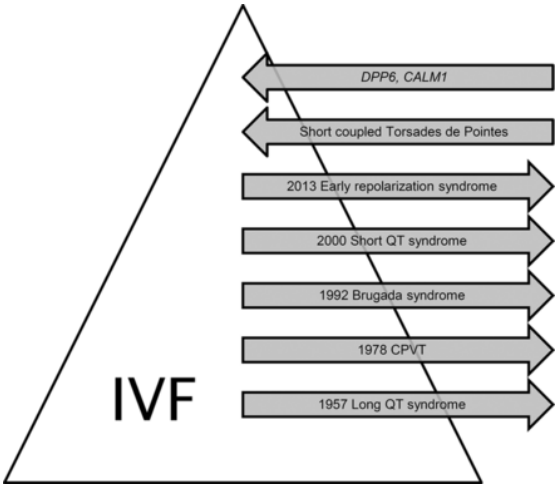
DOWNWARD SLOPE V1& V2 (ARROWS)

NON STRUCTURAL CAUSES OF SUDDEN CARDIAC ARREST

NON STRUCTURAL CAUSES OF SUDDEN CARDIAC ARREST can be considered conduction disturbances of the heart. They are conditions which result in a predisposition to an arrhythmia or heart block. Oftentimes they go unnoticed until a life-threatening event occurs, and in other cases, they are picked up incidentally on an EKG or from the family history due to their hereditary nature.

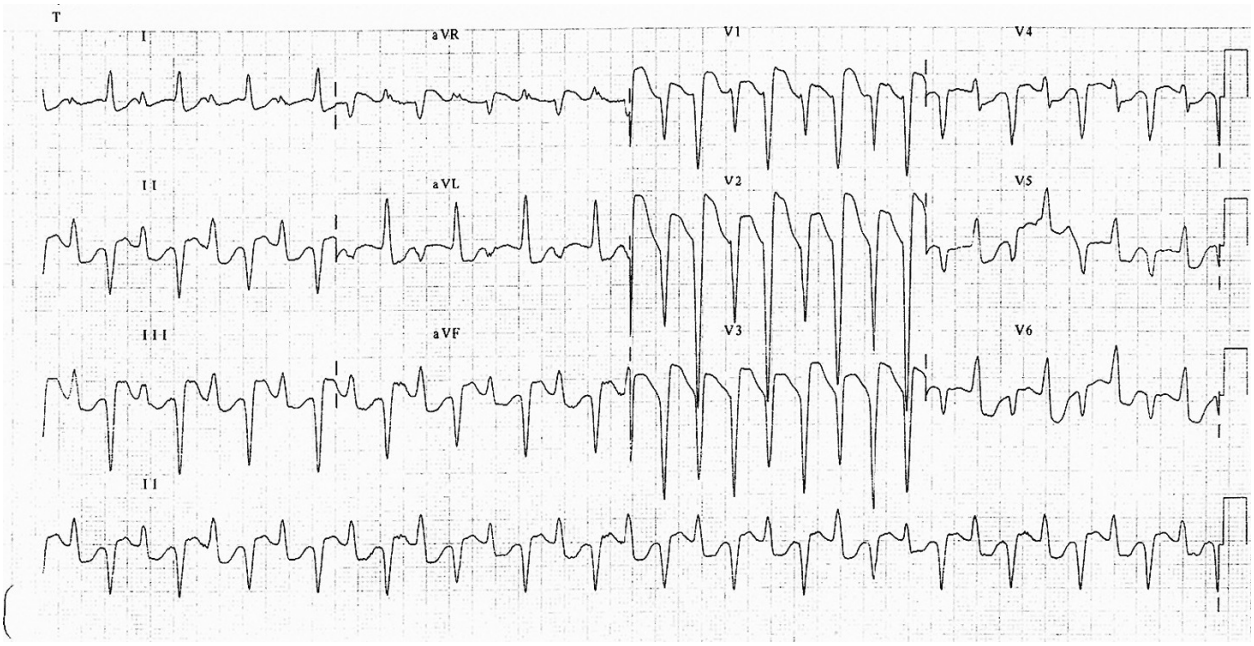
The list would include:

- Primary Electrical Disease: idiopathic ventricular fibrillation
- Brugada Syndrome
- Long QT Syndrome
- Pre-excitation Syndrome
- Complete Heart Block
- Familial Sudden Cardiac Death
- Commotio Cordis: chest wall trauma

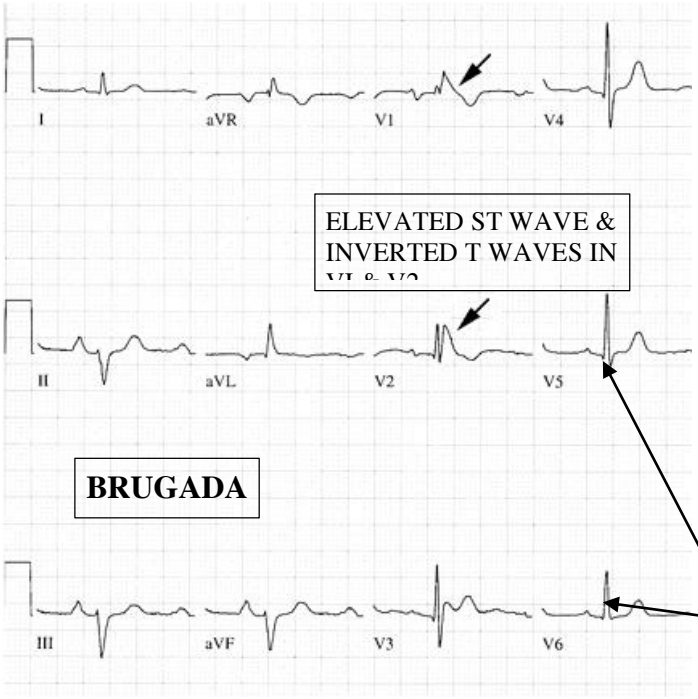


PRIMARY ELECTRICAL DISEASE or idiopathic ventricular fibrillation is a fluid term that constantly changes as we learn more about different disease states and pathology. It accounts for approximately 5% of the sudden cardiac deaths. It is more common in females and oftentimes, they have a history of previous syncopal episodes. When diagnosed pre-mortem, the treatment is an internal defibrillator. Postmortem, the autopsy reveals a normal looking heart.
For those lucky to survive, the treatment is an internal defibrillator.

CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA is a ventricular tachycardia that is triggered by emotional stress or physical exertion. It has a genetic component and the mean age of occurrence is 7-9 years of age. Early symptoms are lightheadedness, palpitations, syncope and death. The pathognomonic finding is Biventricular Tachycardia. The QRS morphologic has two distinct characteristics and alternates 180 degrees. It is only seen in Catecholaminergic Polymorphic Ventricular Tachycardia and Digoxin toxicity.

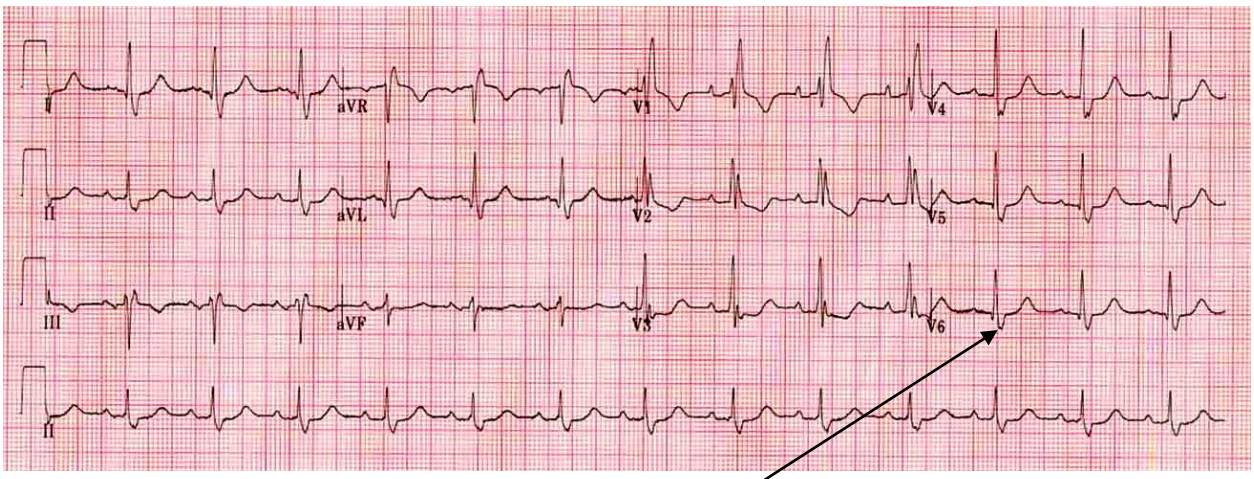


BRUGADA SYNDROME is a hereditary condition that results in disruption of the sodium channels causing ventricular arrhythmias. There is a characteristic EKG which looks like a right bundle branch with ST elevation in V1-3. Oftentimes there is a family history of a relative dying at a young age from an unknown cause.

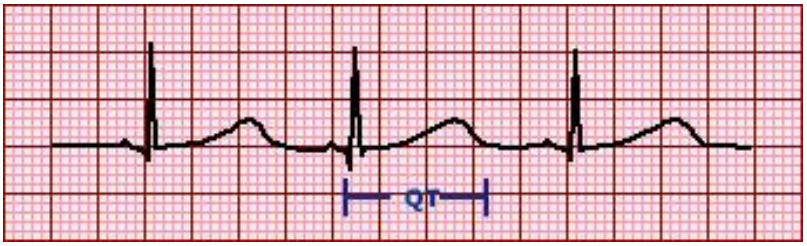


12-lead electrocardiogram from a patient with the **Brugada syndrome** shows down-sloping ST elevation ST segment elevation and T wave inversion in the right precordial leads V1 and V2 (arrows); the QRS is normal. The widened S wave in left lateral leads that is characteristic of right bundle branch block is absent. *Courtesy of Dr Rory Childers, University of Chicago.*

In contrast, a Right Bundle Branch Block……

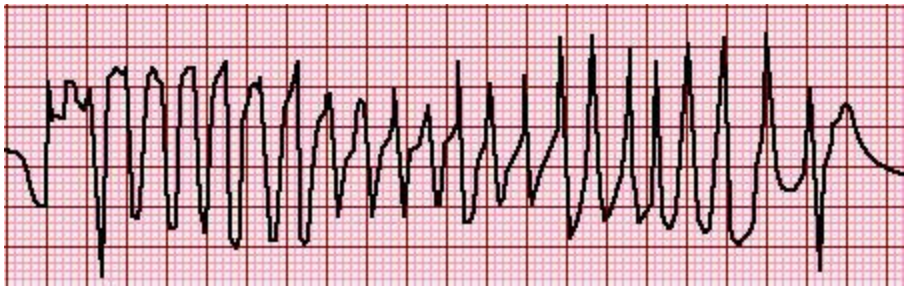


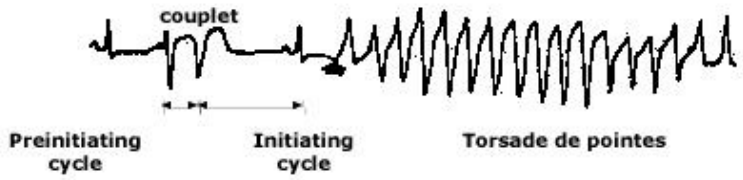
LONG QT SYNDROME is a disorder where the cardiac muscle does not repolarize appropriately causing a prolonged repolarization and potentiating a ventricular arrhythmias. Torsades de Pointe is the most prevalent arrhythmia with this condition. Like other conditions of conduction disturbances, there may have been a previous presentation of lightheadedness, palpitations and syncope.



Prolonged QT interval The corrected QT interval (QTc) is calculated by dividing the QT interval (0.60 seconds) by the square root of the RR interval (0.84 seconds). In this case, the QTc is 0.65 seconds.

TORSADES de POINTES

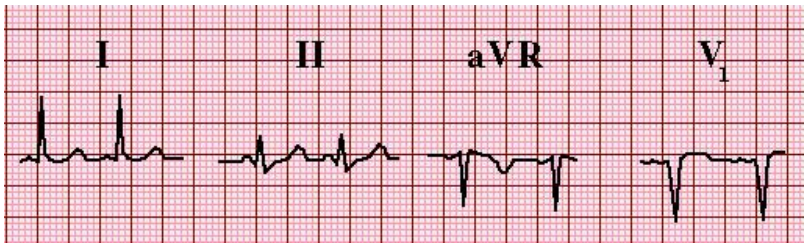




The electrocardiographic rhythm strip shows torsade de pointes, a polymorphic ventricular tachycardia associated with QT prolongation. There is a short, pre-initiating RR interval due to a ventricular couplet which is followed by a long, initiating cycle resulting from the compensatory pause after the couplet.

PRE-EXCITATION SYNDROME
(LOWN-GANONG-LEVINE SYNDROME)

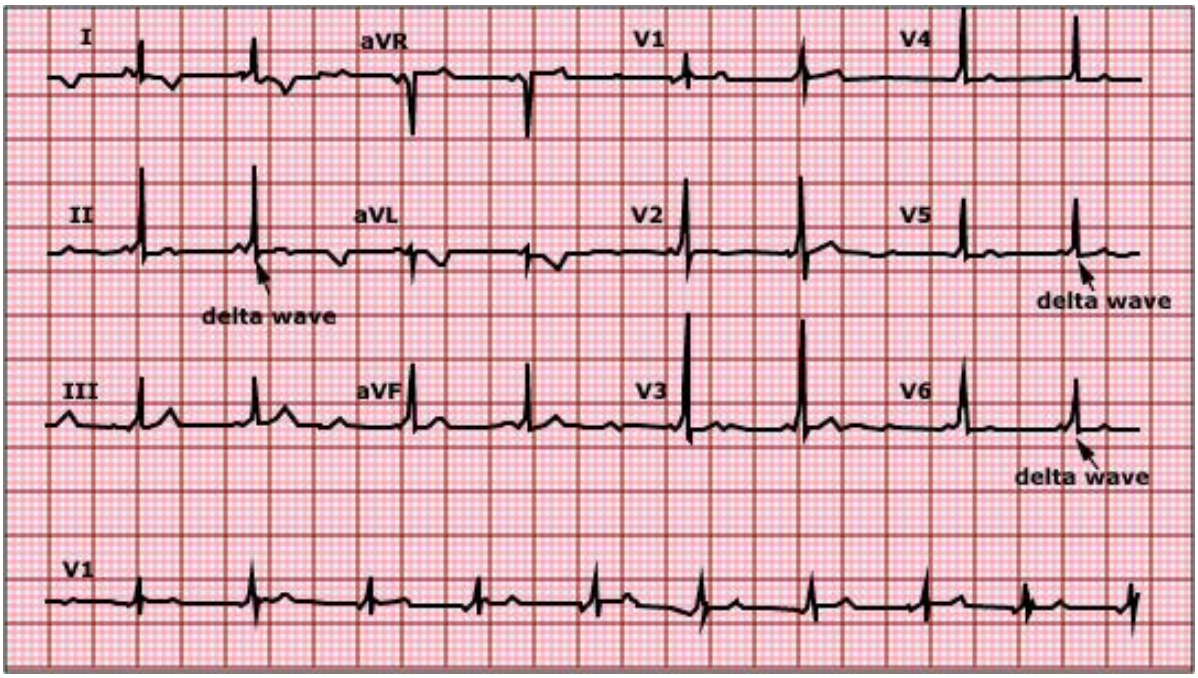
The syndrome results in a reentrant tachycardia. The ekg has a short pr interval of less than 0.12 seconds. The tachycardia passes through an accessory bundle (James Bundle). The syndrome circuits through the AV node resulting in a normal QRS complex.



This syndrome is characterized by a short PR interval (<0.12 seconds) and a normal QRS complex due to the rapid atrioventricular conduction (passage through the AV node)

PRE-EXCITATION SYNDROME
WOLFE-PARKINSON-WHITE SYNDROME

WOLFE-PARKINSON-WHITE SYNDROME or WPW is another reentry supraventricular tachycardia. Unlike Lown-Ganogn-Levine Syndrome, WPW by passes the AV node connecting the atria and ventricle through the Kent Bundle. WPW also has a short PR interval but unlike LGL syndrome, it has a distinctive delta wave juxtaposed upon the QRS complex.



Note the upward slanting at the beginning of the QRS complexes

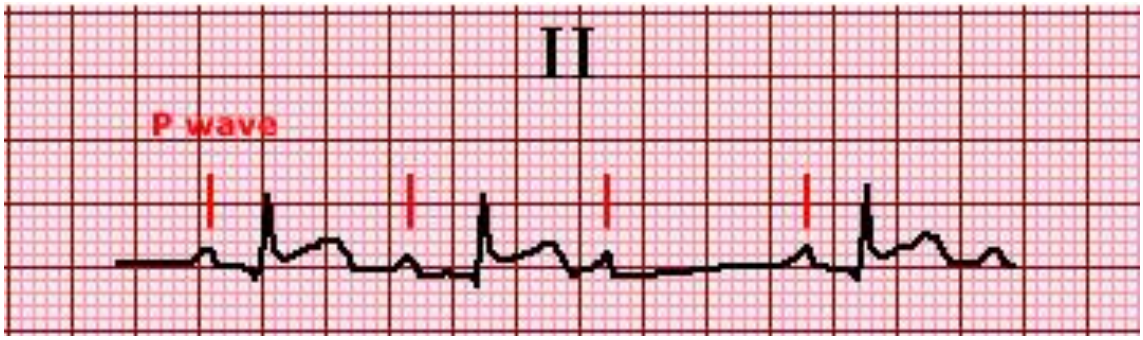
In the end, the pre-excitation syndromes can result in inefficiency of the chambers from filling. Dizziness and hypotension result. Ultimately, these syndromes degenerate from an SVT to Ventricular Tachycardia/Fibrillation.

ATRIOVENTRICULAR CARDIAC CONDUCTION DEFECTS

ATRIOVENTRICULAR CARDIAC CONDUCTION DEFECTS result in an interruption of the conduction impulse through the AV Node. A normal impulse ignites at the SA node, but the P wave cannot effectively traverse from the atria to the ventricle due to the AV node damage. Typically, the AV node gets scarred over causing second and third degree blocks. This is most commonly due to aging. However, infection, drugs and ischemia can all cause temporary or permanent damage to the AV node.

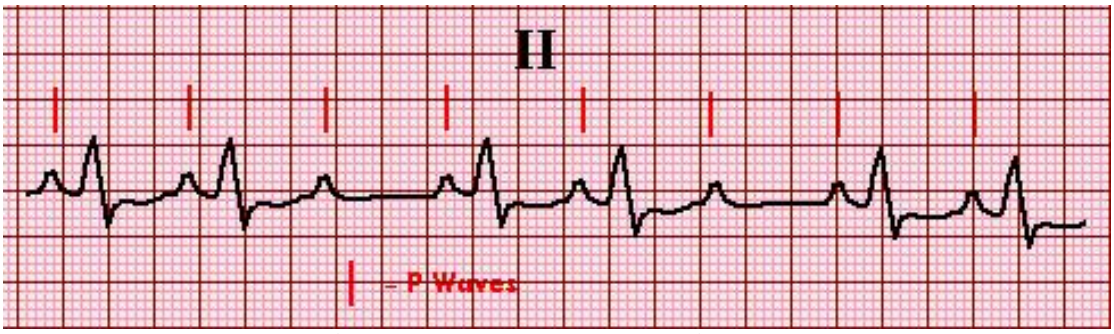
- Infection: Lyme Disease
- Drugs: Beta blockers, calcium channel blockers, digoxin
- Ischemia: Septal infarct can result in permanent "burn out" of the node. Inferior infarcts result in temporary disruption due to swelling around the AV node.

SECOND DEGREE AV CONDUCTION BLOCKS cause a prolongation through the AV node as well as episodic complete blockage of impulses. In **MOBITZ 1 OR WENCHEBACH**, a pattern forms where over the course of several beats, the resistance in the AV node continually increases causing a prolongation of the passage of the impulse. This is reflected in the PR interval getting longer or increasing. It then hits a complete refractory period where the impulse cannot get through. This is seen as a free-standing P wave without any associated QRS complex. The cycle than repeats itself.



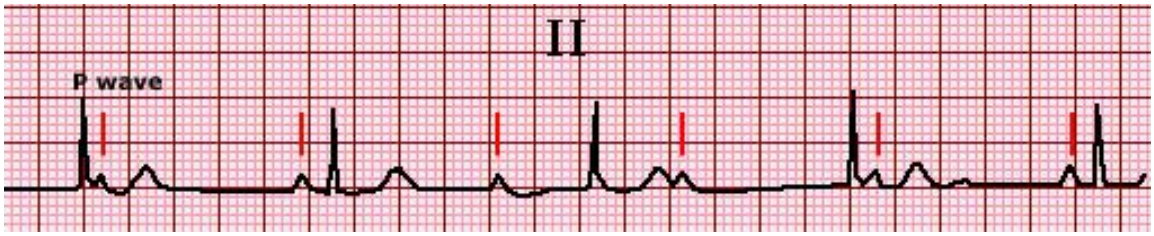
PR interval ***Longer PR interval*** ***P wave No QRS*** ***Repeat cycle***

In **MOBITZ II,** the AV node will allow some impulses to pass through normally and then slam shut resulting in no conduction to the ventricle. This appears on the EKG as normal and similar PR intervals associated QRS complexes. When the AV Node shuts on the P wave there will be a free standing P waves without any associated QRS complexes.



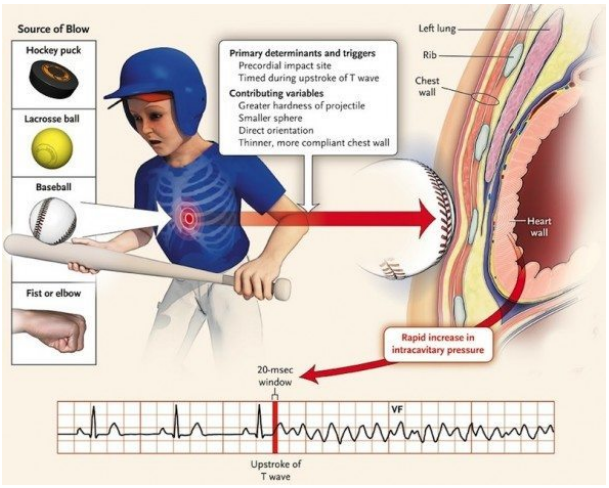
PR intervals ***P wave/ No QRS*** ***PR intervals*** ***P wave/ No QRS*** ***PR intervals***

In **THIRD DEGREE AV BLOCK**, the AV node is completely blocked or obstructed. P waves will continue to beat but cannot traverse through the AV node and ignite the ventricle. Because there is no impulse from the atria, the ventricle initiates a backup mechanism that allows the ventricle to create its own impulse and contract the heart. The impulse ignites high up in the ventricle and therefore the QRS complexes are typically narrow. The rate however is alway slower than the atrial rate and typically the ventricular rate is in the 30's to 40's beats per minute.

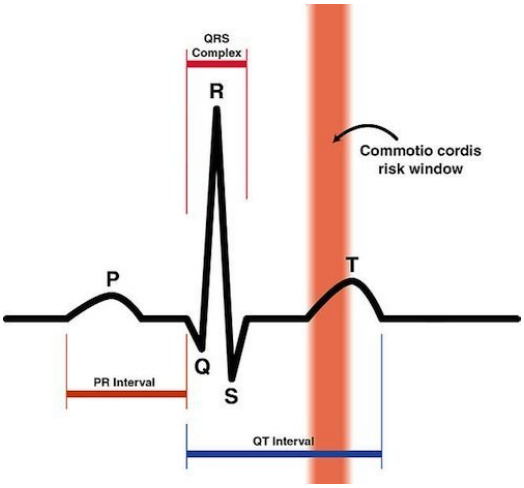


P waves march out in synchrony with the QRS doing the same at a different rate

COMMOTIO CORDIS, latin for "commotion to the heart", results when an object hits the heart causing most commonly ventricular fibrillation. Complete heart block has also been described. The impact is not about size of the object or the velocity of the impact. It is thought that the insult is caused by the trauma impacting the heart during specific periods of repolarization and depolarization. Animal studies demonstrated that precordial blows during repolarization just before the peak of the T wave resulted in Ventricular Tachycardia. Blows at the time of depolarization of the QRS complex resulted in transient Third Degree Heart Block.



COMMOTIO CORDIS is commonly seen with young athletes and is thought to be almost as common a cause of death in young athletes as **Hypertrophic Cardiomyopathy**. Most commonly males are involved. It typically occurs with sports with baseball having the highest incidence. This is followed by softball, hockey and football.



NON CARDIAC DISEASE OF SUDDEN CARDIAC ARREST

Several cause exist that result in SUDDEN CARDIAC ARREST that are not due to an initial insult on the heart. Causes to other organs result in the heart ultimately malfunctioning and causing the arrest.

- Pulmonary embolism
- Intracranial hemorrhage
- Drowning
- Pickwickian syndrome
- Drug induced
- Central airway obstruction
- Sudden infant death syndrome

SUDDEN CARDIAC ARREST IN ATHLETES

About 1:50,000 cardiac deaths occur in young athletes each year. The most common cause are Hypertrophic Cardiomyopathy, Coronary Artery abnormalities and Long QT Syndrome. We can also add other causes that have no underlying abnormality such as commotio cordis, steroid use and stimulants. Males are inflicted more commonly than females and unfortunately, blacks more common than whites. The most common sports are basketball and football in the US and soccer in Europe.

TREATMENT OF SUDDEN CARDIAC ARREST

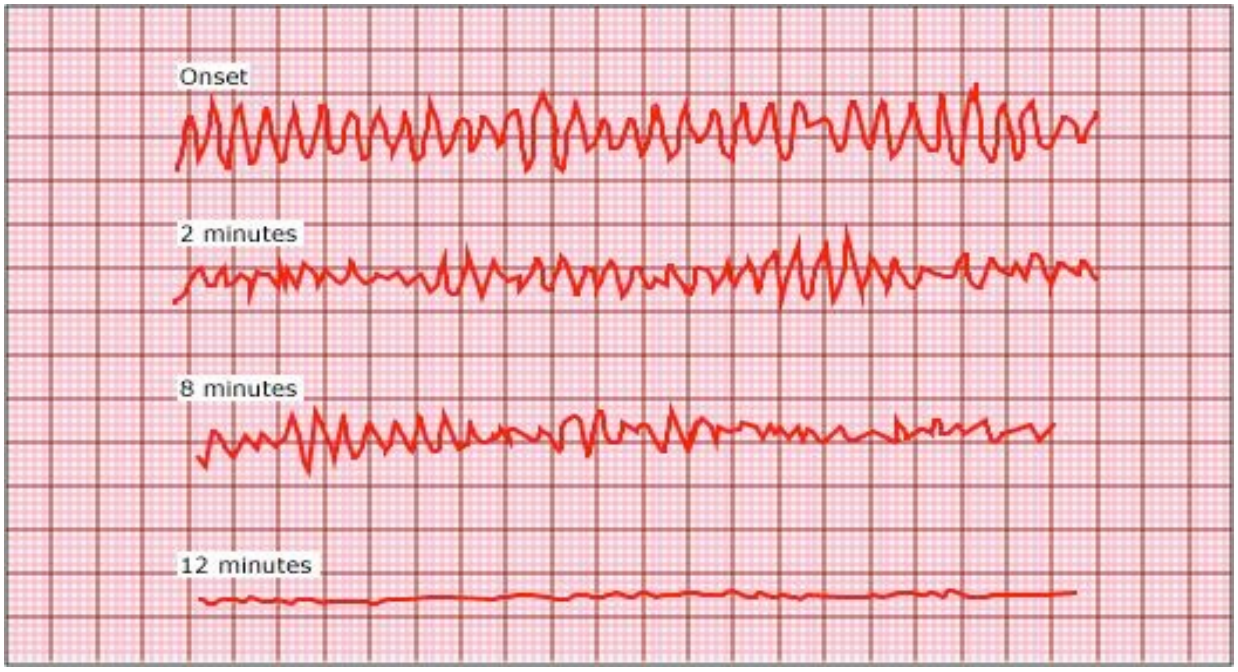
Survival from sudden cardiac arrest has a very small window of success. That is due to the fact that when the heart stops, blood is no longer being pumped to the brain. Lack of oxygenated blood flow to the brain will result in irreversible cerebral anoxia in around ten minutes. **THE TREATMENT THEREFORE IS TO ARTIFICIALLY SUSTAIN CEREBRAL BLOOD FLOW UNTIL CIRCULATION IS RESTORED.**

CPR is presently the modality by which artificial circulation is administered. Studies have shown that bystander CPR improves survival. Interestingly, there has been no significant difference in return to spontaneous circulation between groups that received bystander CPR and those who do not. The difference is seen in neurologic survival where the number that leave the hospital neurologically intact is greater with the bystander CPR group.

Emphasis in the past with CPR had been on oxygenation and compressions. Recently the trend has been toward **CARDIO-CEREBRAL RESUSCITATION** where compressions are prioritized followed by ventilation. This approach appears to be improving outcome. Previously, everyone was worried about getting oxygen to the lungs, but forgot to circulate it to the brain with compressions. CCR focuses on circulating the oxygen that is in the lungs, arteries and even that which is in the venous system. As well, with the compression of the chest, the atmospheric pressure becomes less inside the chest, and with the upstroke, air may will naturally flow into the lungs so as to equilibrate with the outside pressure. Think of it as squeezing a foam ball. When you release, the foam expands because the air moves back into it!

This is not to say ventilation is not important. You always want to optimize the oxygenation and **REMOVAL OF THE POISONOUS CO2**. However hyperventilation is also bad since it couse the all vessels including those going to the brain to vasoconstrict. One study that looked at the rate of breaths given intubated patients during codes in a hospital noted an average rate of 50 breaths per minute. It is hard to slow down the adrenaline but high respiratory rates will quickly alkalinize the body resulting in vasoconstriction of the vessels and diminished cerebral blood flow.

EARLY DEFIBRILLATION is the lynchpin to surviving Sudden Cardiac Arrest. Ventricular Fibrillation rarely spontaneously converts with or without drugs. Electricity can reverse the course and has a much better outcome with course ventricular fibrillation. In fact, there is no proof that epinephrine provides any benefit to survival. It is hypothesized that as the stores of epinephrine in the myocardium get depleted, the ventricular fibrillation takes on a finer characteristic. Therefore, the addition of epinephrine may make it easier to defibrillate the patient in this stage.



The progression of the ventricular fibrillation highlights the need for early electricity. This is why Automatic External Defibrillators are thought to provide the greatest access for recovery of spontaneous circulation and optimal neurologic outcome. Widespread implementation assures that conversion can occur early in the natural progression of the ventricular fibrillation, when it is most susceptible to the defibrillation.