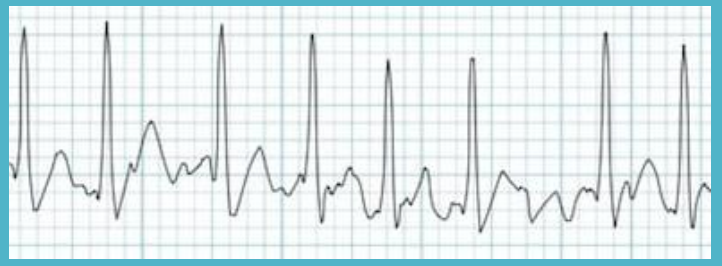
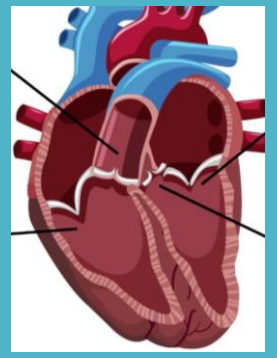
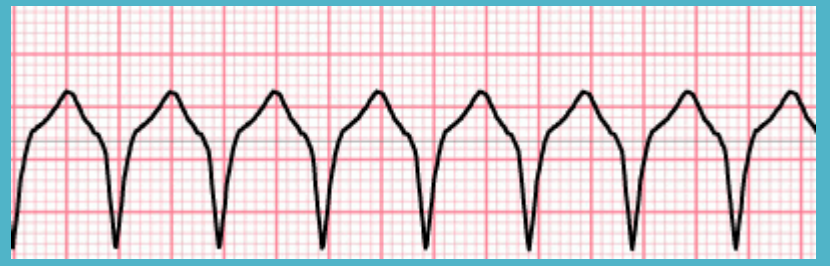




Update on Cardiac Issues:
Afib, Valvular Heart Disease, Ventricular Arrhythmias

Gail Markowski, DNP, ANP-C, ACNP-C, CCRN
State University of New York at Buffalo – DNP/AGNP Program
Catholic Health System of Buffalo - Structural Heart Program

Objectives:

1. Review the diagnosis of atrial fib and discuss pharmacologic and interventional management and stroke prevention
2. Discuss issues caused by Aortic Stenosis and the newest treatment strategies
3. Discuss issues related to Mitral Regurgitation and the newest treatment strategies
4. Review ventricular arrhythmias and treatment measures including pharmacologic and interventional management

Atrial Fibrillation

The most common arrhythmia

2019 – Afib on 184,000 death certificates

Hospitalizations per year 455,000

By 2030, 12.1 million people in the US will have afib, 33.5 million worldwide

Atrial fibrillation occurs when abnormal electrical impulses suddenly start firing in the atria (upper chambers of the heart)

These impulses override the heart's natural pacemaker, which can no longer control the rhythm of the heart. The atria contract randomly and sometimes so fast that the heart muscle cannot relax properly between contractions. This reduces the heart's efficiency and performance and causes a highly irregular pulse rate. Increases risk of stroke 4 to 5-fold

Afib

The root cause of atrial fibrillation is damage to heart tissue or the heart's electrical system. Most of the time, heart damage that results in afib is caused by coronary heart disease or high blood pressure. Many times, the cause of afib remains unknown.

When atrial fibrillation develops, there is loss of the atrial transport factor ("atrial kick"), with consequent decrease of cardiac output. CO declines by 20-30% in normal individuals with loss of atrial kick; the decline in CO is considerably larger in patients with heart disease. In patients with already decreased ejection fraction, this loss can easily put them in acute HF

Signs/Symptoms of afib

Feelings of a fast, fluttering or pounding heartbeat, palpitations
Chest pain
Dizziness
Fatigue
Lightheadedness
Reduced ability to exercise
Shortness of breath
Weakness
Or no symptoms at all

Risk Factors of afib

- Advanced age
- HTN
- Obesity
- DM
- HF
- Hyperthyroidism
- Ischemic heart disease
- CKD
- Moderate to heavy ETOH use
- Smoking

Three types of afib

Paroxysmal Afib: This type of Afib occurs intermittently and stops on its own within seven days

Persistent Afib: This type of atrial fibrillation lasts longer than seven days

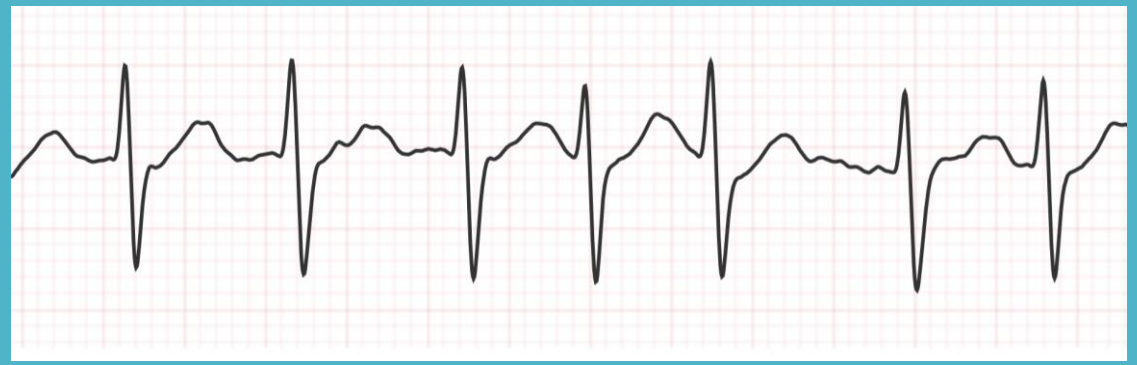
Long-standing persistent Afib: This is similar to persistent Afib but lasts longer than a year.

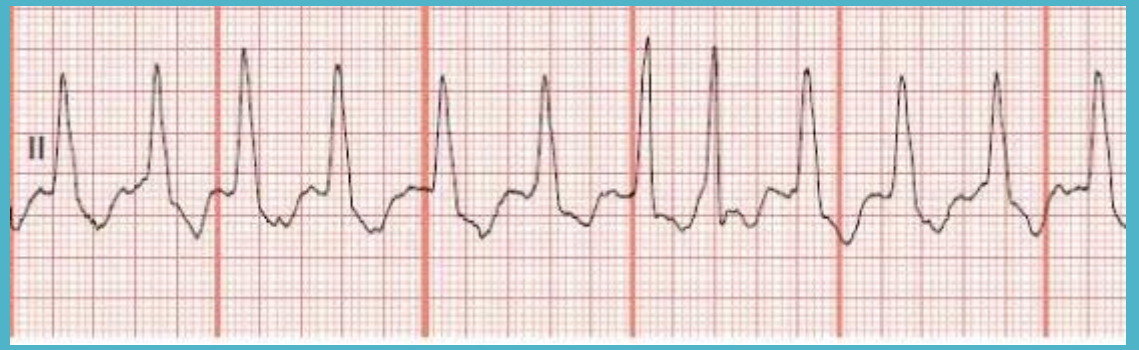
Diagnostic testing

EKG, Holter monitor, MCT, transthoracic echocardiogram, ischemic workup (stress test)

Lab work – CBC – check for anemia, chemistry, thyroid panel (r/o hyperthyroidism)

Is the rate controlled?





Treatment of afib

The goals of atrial fibrillation treatment are rate control, maintenance of sinus rhythm, and prevention of thromboembolism.

Treatment depends on:

How long you've had Afib. Afib begets afib

Symptoms

The cause

Atrial fibrillation treatment may involve:

Medication

Therapy to reset the heart rhythm - cardioversion.

Surgery or catheter procedures.

Rate and Rhythm Control

The goals of heart rate control in atrial fibrillation are to minimize symptoms associated with elevated heart rates and to prevent tachycardia-associated cardiomyopathy.

The benefit of rhythm control over rate control has been controversial in terms of mortality. The current guidelines recommend restoration and maintenance of SR in patients with symptomatic Afib. There is data showing a significantly lower in-hospital mortality rate when initial Afib was successfully converted to SR either by drugs or electrical cardioversion in patients with AHF

But maintaining SR is not always possible.

Medications

Beta blockers - help slow the heart rate.

Calcium channel blockers - control the heart rate but may need to be avoided by those who have heart failure or low blood pressure.

Digoxin - This medicine may control the heart rate at rest, but not as well during activity. Most people need additional or alternative medicines, such as calcium channel blockers or beta blockers.

Antiarrhythmics to control the heart rate and rhythm, Example – Amiodarone. More side effects than other medicines

Blood thinners = anticoagulants prevent blood clots and reduce the risk of stroke. include warfarin (Jantoven), apixaban (Eliquis), dabigatran (Pradaxa), edoxaban (Savaysa) and rivaroxaban (Xarelto)

Cardioversion

The success rate of cardioversion with atrial fibrillation is generally better than 90 percent. (42-53% are still in SR after 1 year, 27% at 4 years) Chances of success are lower when the atrial fibrillation has been present for more than several months or when the left atrium is very enlarged.

Chemical cardioversion - drugs, such as Dofetilide or sotalol are used by EP, patients require hospitalization, tele, renal function monitoring.

Electrical cardioversion - starting at 100J can often restore sinus rhythm if Afib duration is < 30days. An initial energy ≥200J for patients with Afib > 30days and ≥300J if AF > 180 days. Current ACC/AHA/ESC guidelines recommend an initial energy of ≥200J when using MPW and 200J for BPW EC.

There is debate on the number of shocks that maybe administered before labelling AF as refractory to EC. Currently there are no firm data to determine the number of shocks that can be safely delivered during EC.

Prevent Stroke

Recommendations for anticoagulation based on CHADS2 and CHA2DS2-VASc are the same: oral anticoagulation is recommended for a score of ≥ 2. Stroke risk assessment should always include an assessment of *bleeding* risk. This can be done using validated bleeding risk scores, such as the HAS-BLED scores

CHADS2 – VASc Score		
C	Congestive Heart Failure	1
H	Hypertension (>140/90 mmHg)	1
A	Age ≥ 75	2
D	Diabetes Mellitus	1
S_2	Prior TIA or stroke	2
V	Vascular disease (MI, aortic plaque etc)	1
A	Age 65-74	1
Sc	Sex category (Female = 1 pt)	1

File: CHADS2VASc Commons.wikamedia.org

Bleeding risk

The HAS-BLED Score for Predicting Major Bleeding Risk in Anticoagulated Patients With Atrial Fibrillation

Afib patients are subdivided into 3 risk stratifications, in which a score of 0 indicates low risk, 1–2 indicates moderate risk, and ≥3 indicates high risk.

HAS-BLED

Letter	Clinical Characteristic	Points
H	Hypertension	1
A	Abnormal Liver or Renal Function	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INR	1
E	Elderly (age > 65)	1
D	Drugs or Alcohol	1 or 2
Maximum Score		9

File: Hasbled Commons.wikimedia.org

Anti-arrhythmic drug therapy

There are two main types of channel blockers:

Sodium Channel Blockers - slow your heart's ability to conduct electricity

Flecainide (Tambocor)

Procainamide (Procanbid)

Propafenone (Rythmol)

Quinidine

Because these drugs slow down how fast electrical signals can travel in the heart muscle, people with CAD or HF cannot use them.

Potassium Channel Blockers **- slow the electrical signals in the heart that cause Afib**

Dofetilide (Tikosyn)

Sotalol (Betapace)

AntiArrhythmics con’t

Amiodarone (Cordarone, Nexterone, Pacerone) is both a sodium channel blocker and a potassium channel blocker. It's by far the most effective anti-arrhythmic drug available, possibly as much as 75%. Side effects include pulmonary toxicity (with a fatality rate of about 10%), visual disturbances as well as hepatic, cardiac and thyroid toxicities.

Dronedarone - (Multaq) was designed to be like amiodarone without the side effects. It does have fewer side effects, but clinical trials showed that it didn't keep people in sinus rhythm very well. It may not prevent Afib, but it might prevent some of the symptoms of Afib, perhaps by blunting fast heart rates. This is for maintenance of SR (do not give in perm afib)

Afib ablation

Catheter ablation uses radiofrequency to destroy a small area of heart tissue that is causing rapid and irregular beats

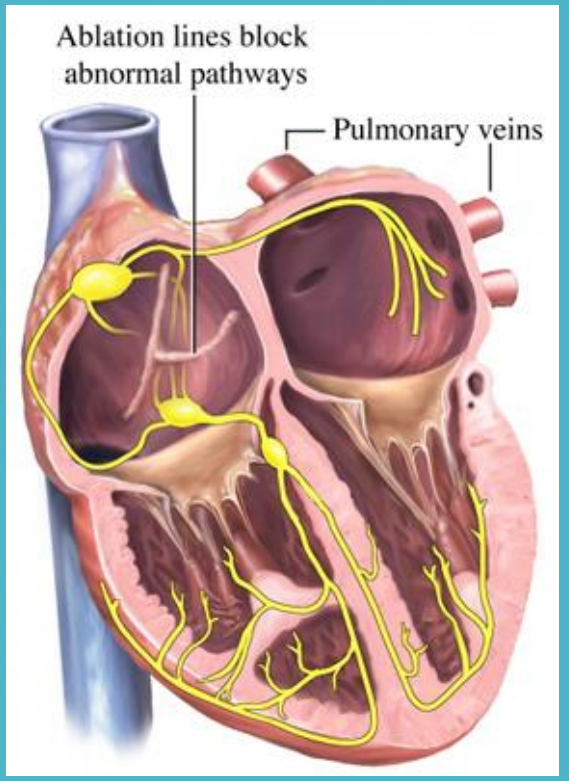
Pulmonary vein isolation ablation (PVI) In some patients, fibrillation is triggered by extra electrical currents in the pulmonary veins. A catheter tip can be used to destroy that tissue. In many cases, SR returns.

AV node ablation with pacemakers. Sometimes, the trigger for Afib occurs in the atrioventricular node. The catheter tip is placed near the AV node to destroy a small area of tissue. A pacemaker is implanted to restore and maintain the heart's normal rhythm.

Surgical procedures

Maze is a surgical procedure used to treat afib. A surgeon creates a pattern (maze) of scar tissue in the atria using a scalpel or a device that delivers heat or cold energy. Scar tissue doesn't conduct electricity, so the maze interferes with stray electrical heart signals that cause atrial fibrillation.

There are different types of Maze procedures.



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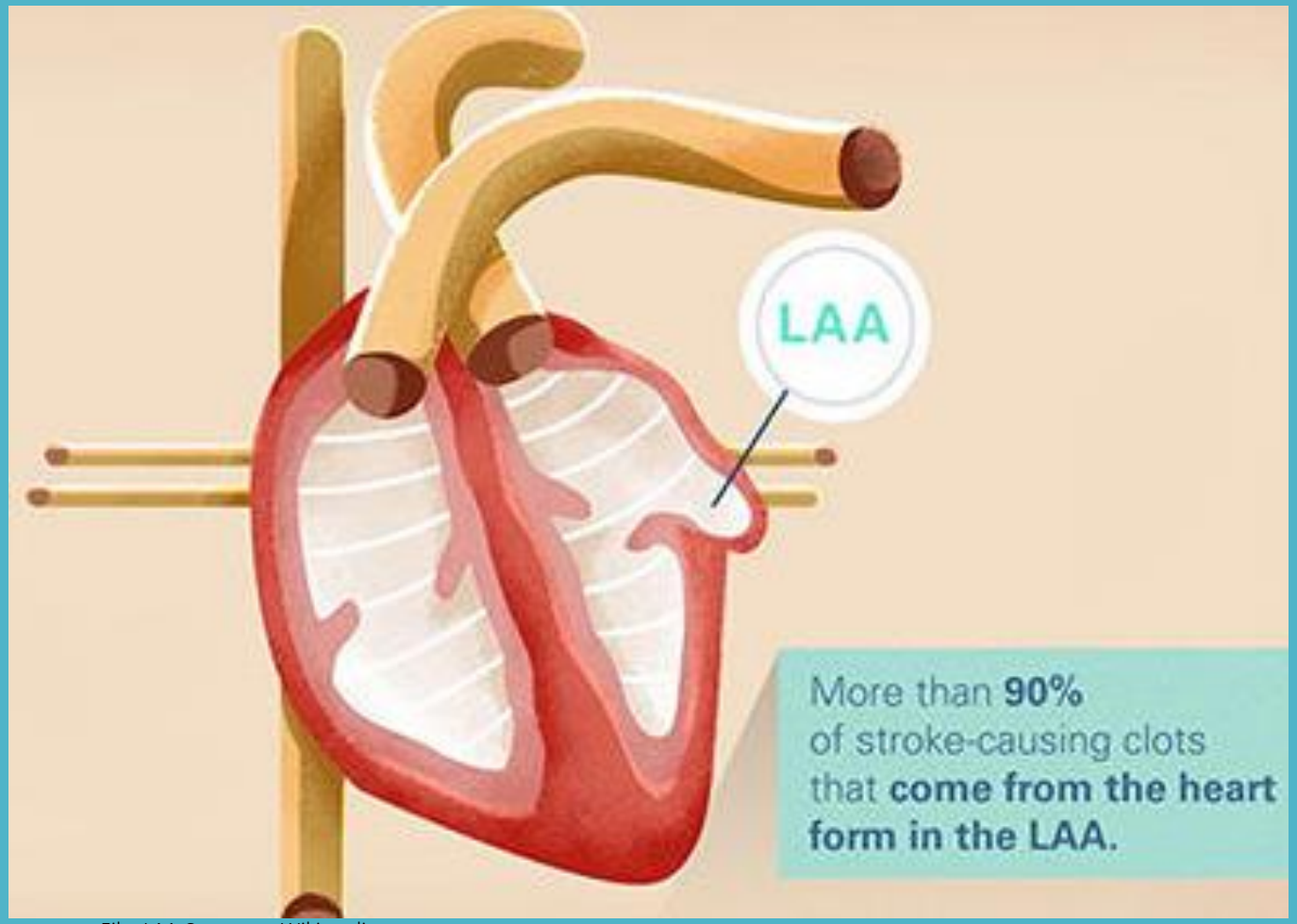
The left atrial *what*??

The Left Atrial *Appendage*

Why is this important?



File: LAA Commons.Wikimedia.org

LAA

Over 90% of embolic strokes are caused by thrombi originating from the left atrial appendage (LAA).

What can we do?

Close it, ligate it, clip it, occlude it, just get rid of it

It can be clipped during open-heart surgery such as valve replacement or CABG

LAAC or Watchman

Using a standard percutaneous technique, a guidewire and vessel dilator are inserted into the femoral vein.

The implant procedure is performed with fluoroscopy and transesophageal echocardiography (TEE). The interatrial septum is crossed using a standard transseptal access system.

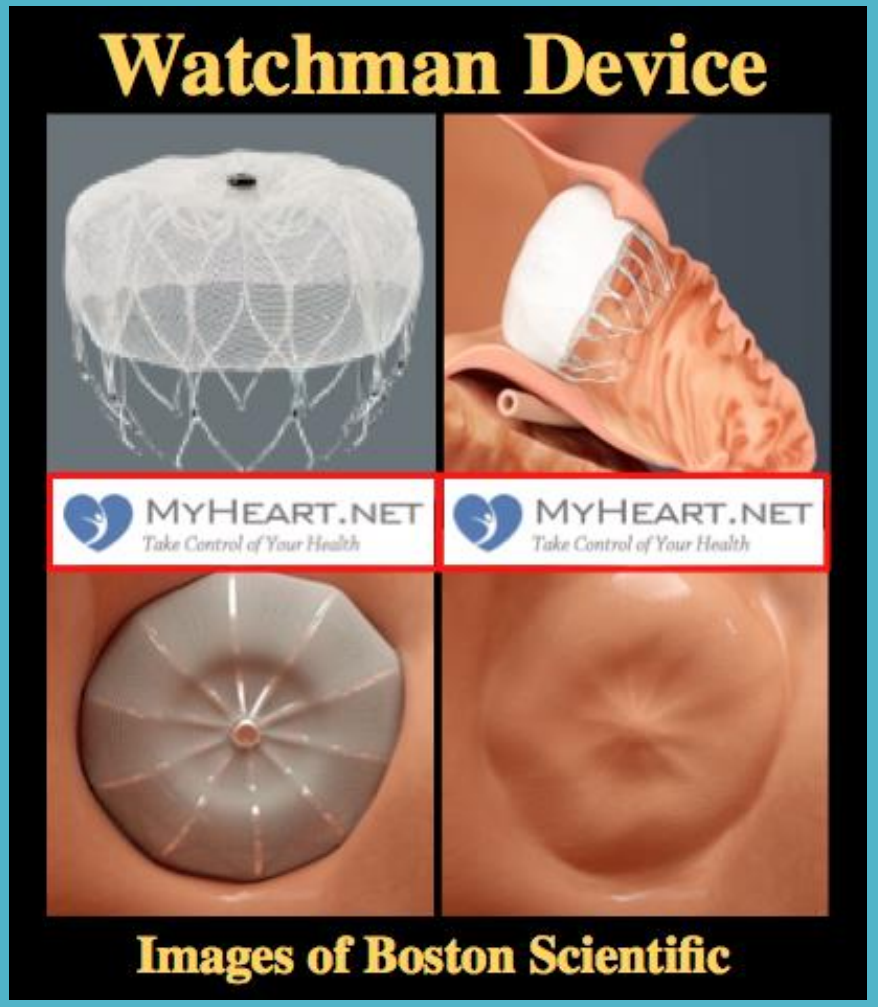
The access sheath is advanced over the guidewire into the left atrium and then navigated into the distal portion of the LAA over a pigtail catheter.

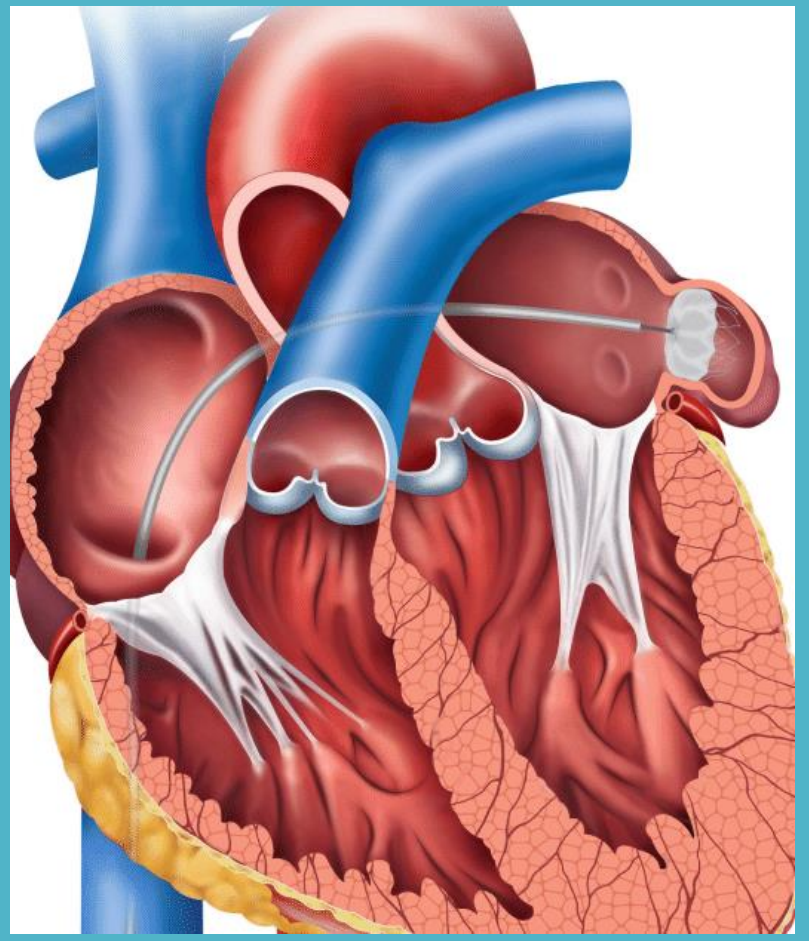
WATCHMAN device is then deployed and released in the LAA.

Heart tissue grows over the WATCHMAN implant and the LAA is permanently sealed. Patients remain on an OAC for at least 45 days post-procedure or dual anti-platelet therapy (DAPT). ASA indefinitely.

Watchman

LAAC

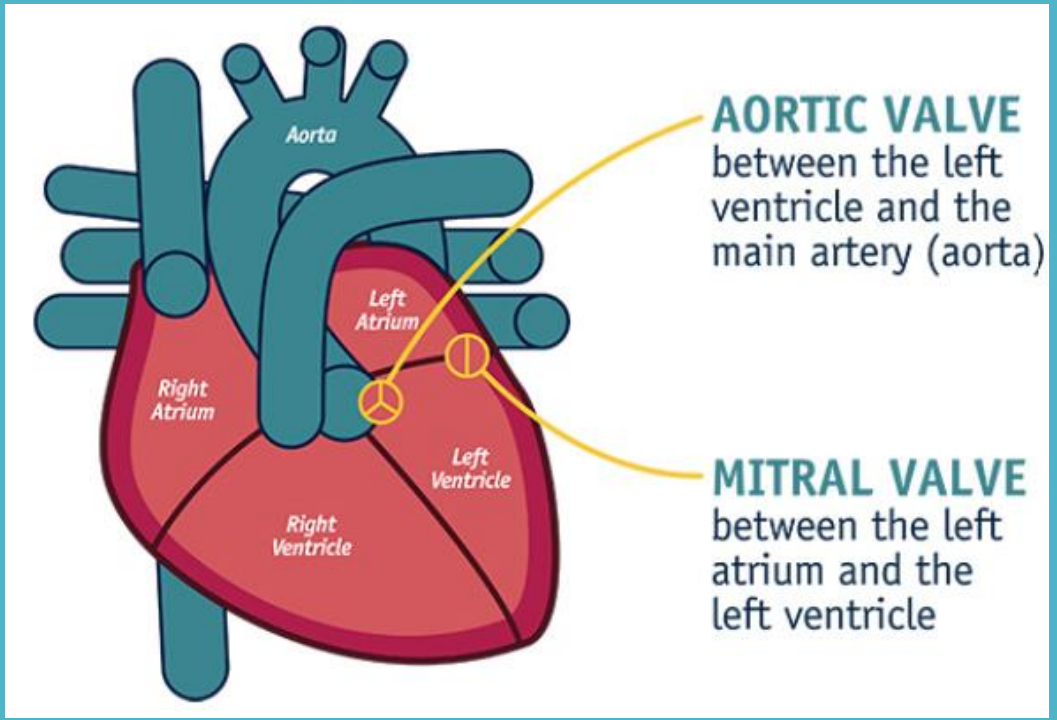




www.bostonscientific.com

Valvular Heart Disease

Mitral regurgitation (MR) is the most common valve defect, followed by aortic stenosis (AS) and then aortic regurgitation (AR). Degenerative disease is the most common etiology of MR, AS, and AR, though these forms of VHD also can be caused by congenital valve defects, systemic inflammatory diseases, endocarditis, and other conditions.

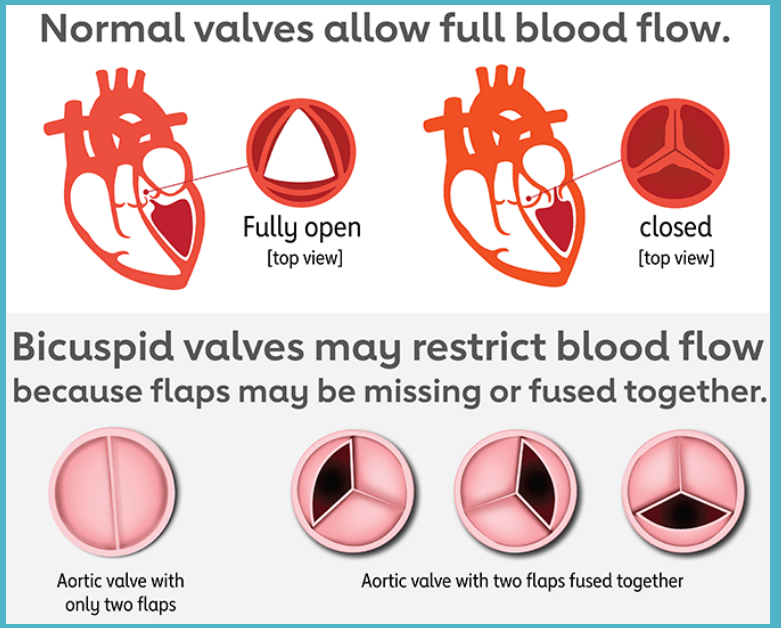


Aortic Stenosis

The # of pts will increase globally as our population ages.

The aorta is the main artery that carries blood out of the heart to the rest of the body. Blood flows out of the heart and into the aorta through the aortic valve. In aortic stenosis, the aortic valve does not open fully. This decreases blood flow from the heart.

The aortic valve has 3 leaflets except in some people that have a bicuspid AV.



File: bicuspid valve Commons.Wikimedia.com

Aortic Stenosis

As the aortic valve narrows, the left ventricle has to work harder to pump blood out through the valve. LV walls become thicker (diastolic dysfunction)This can lead to chest pain & diastolic HF

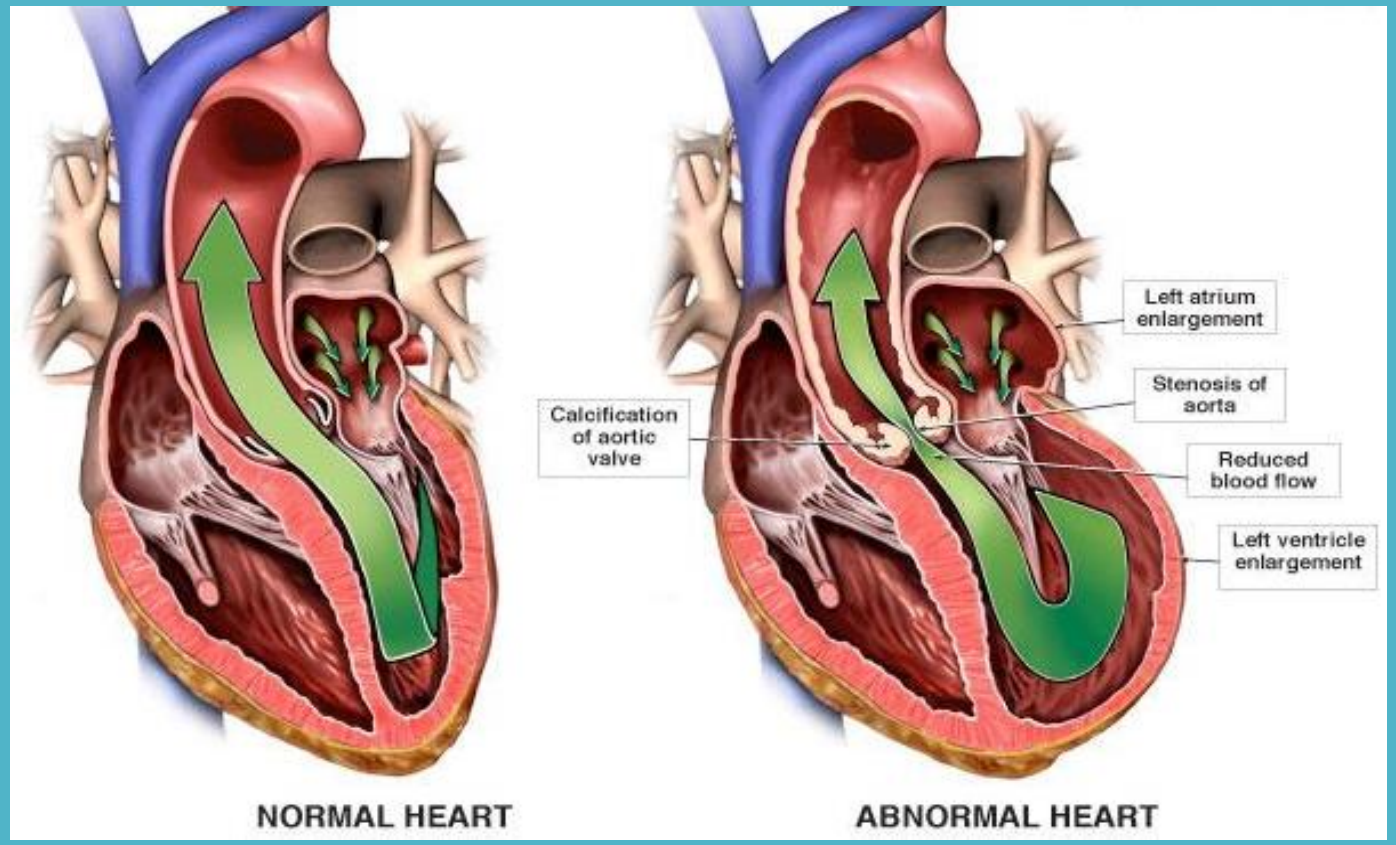
As the pressure continues to rise, blood may back up into the lungs. Severe aortic stenosis can limit the amount of blood that reaches the brain and the rest of the body. Dizziness or syncope may occur.

Aortic stenosis mainly occurs due to the buildup of calcium deposits that narrow the valve. This is called calcific aortic stenosis. The problem mostly affects older people.

Calcium buildup of the valve happens sooner in people who are born with abnormal aortic or bicuspid valves. In rare cases, calcium buildup can develop more quickly when a person has received chest radiation

Aortic Stenosis

As the stenosis worsens, there is significant LV outflow destruction to cause symptoms. LV responds to high afterload, LV wall thickness increases to offset the pressure overload keeping wall stress normal, BUT…losing LV contractility. We see significant reduction in SV



File: aorticstenosis Commons.Wikimedia.org

Aortic Stenosis

Treatment – no medications can improve the valve function. Research continues to try to *prevent* AS

Pathophysiology – following endothelial damage (DM, HTN) which initiates the AV injury, lipids such as lipoproteins & oxidized low-density lipoprotein cholesterol infiltrates the valve. It starts the inflammatory response triggering valve calcification….

Trials with different statin therapy – no change

Testing is being done with ace inhibitors. The ACE is upregulated in calcific AV disease – it did slow progression but not statistically significant

Aortic Stenosis

Once symptomatic, “50% pts die within 5 years, with HF 2 years”

There is no pharmacological treatment for aortic stenosis.

You can treat the symptoms – CHF – diuretics – But it is critical to remember that patients with aortic stenosis are preload dependent and require aggressive fluid support to maintain their cardiac output. There is concern that vasodilators may lead to a reduction of the coronary perfusion pressure. The use of ACE-Inhibitors in aortic stenosis is “contraindicated” but not all agree with this.

Afib and non-sustained VT are common arrhythmias associated with aortic stenosis. Pts often have VT during syncopal episodes.

Afib typically causes more severe symptoms with aortic stenosis due to the stiff left ventricle and the resulting greater need for atrial contraction.

Aortic Stenosis

The criteria for “severe” AS has evolved over decades:

AVA </= 1.0 cm2 (nl 3 – 4 cm2)

Mean AV pressure gradient >/= 40 mmHg (nl <5 mmHg)

Peak AV velocity (jet velocity) >/= 4.0 m/s (nl 1.0 m/s)

With the progression of moderate to severe AS, there is *significant* reduction in SV = increased risk of CV events

Risk assessment - primarily dependent on evaluation of symptom status.

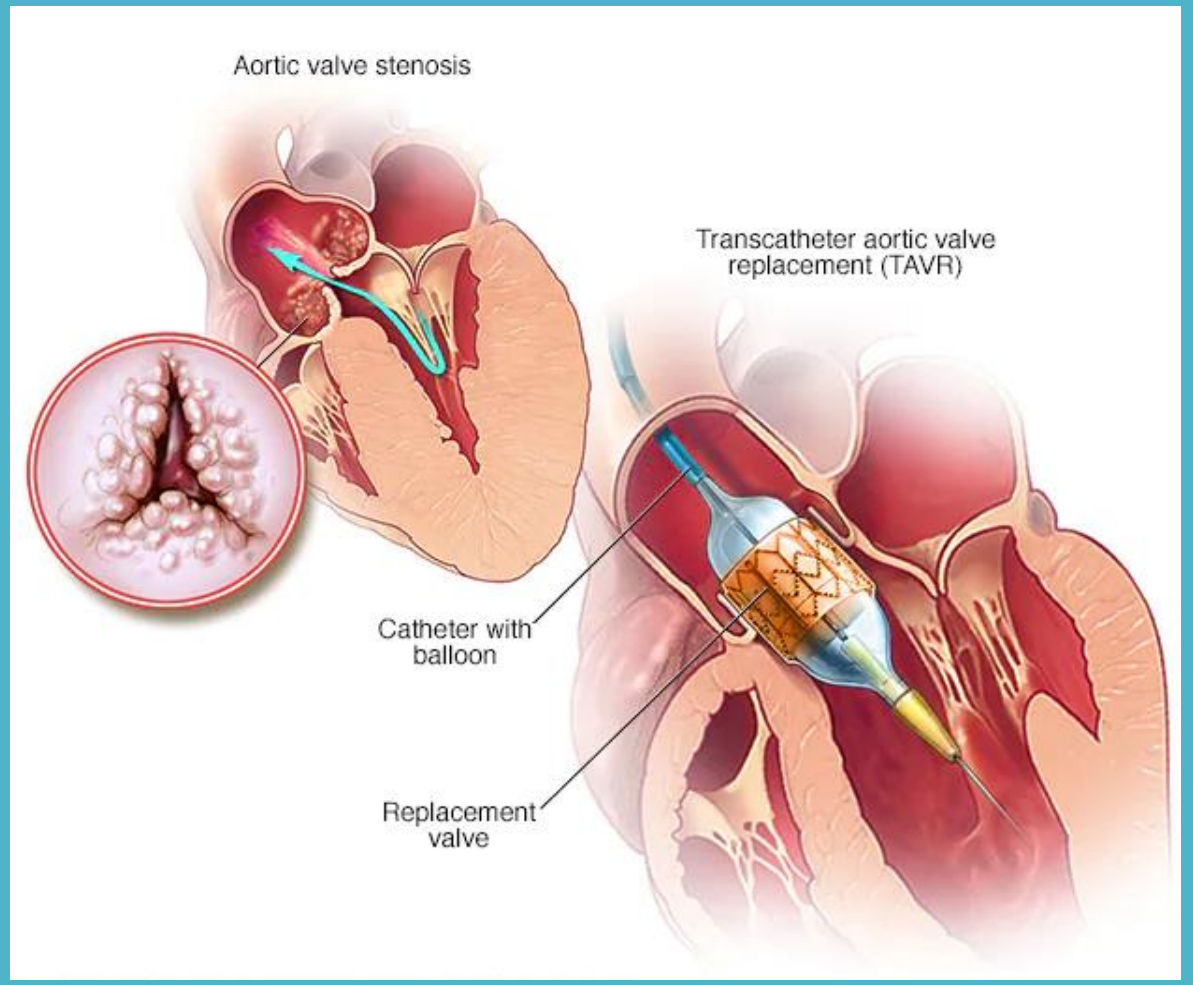
Patients with symptomatic, severe AS is Class I indication for AVR

Many of these pts are elderly and frail. This is often a disease of aging.

How well do you think this patient would do with a sternotomy?

Joe, age 86

iStock



www.Edwards.com

Enter Transcatheter Aortic Valve Replacement (TAVR)

In 2019 in the US, # TAVRs > # SAVRs

Initially approved only for the inoperable or high surgical risk patient

TAVR

The replacement valve is delivered via one of several access methods: transfemoral, transapical, subclavian, direct aortic, and transcaval, among others.

Risks – death, MI, CVA 0.5-2%, vascular/nerve injury, bleeding, renal injury, conduction problems necessitating a permanent pacemaker (10-15%), paravalvular leak

Workup includes: TTE, Cardiac angiogram, CTA chest/abdomen/pelvis, pre-op labs, EKG, CXR, dental clearance If echocardiographic numbers do not meet criteria, pts may undergo a Dobutamine Stress Echo to check for low flow, low gradient severe AS

Commonly used TAVR valves

Supra-annular self-expandable valve – Medtronic's CoreValve/Evolut

Intra-annular balloon-expandable valve – Edwards' Sapien 3

www.Medtronic.com

www.Edwards.com

Valve-in-Valve TAVR

Valve-in-valve transcatheter aortic valve replacement (ViV TAVR) has become an alternative to re-operative surgery for those pts who have had a bioprosthetic AVR

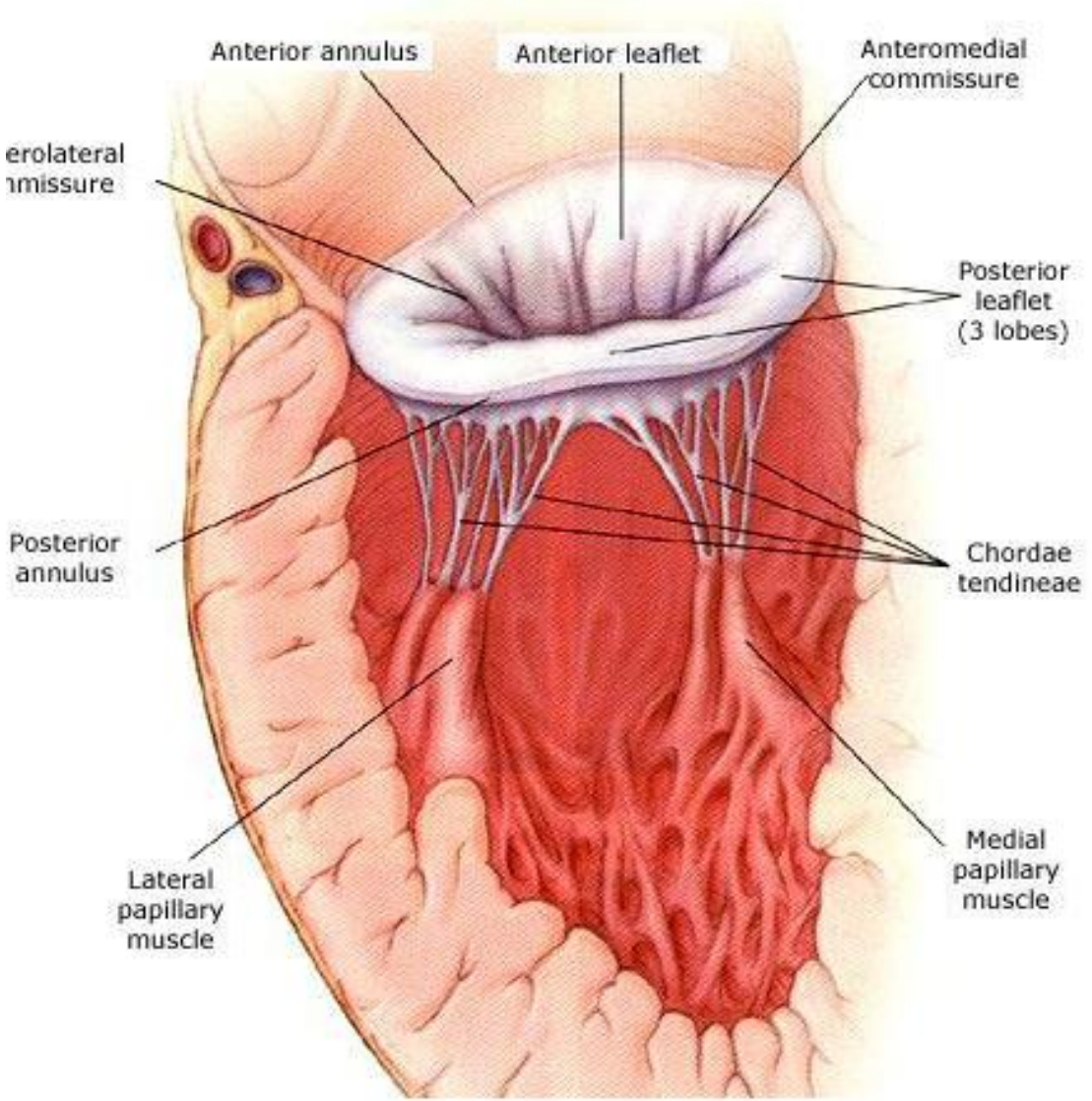
If a patient has a degenerated bioprosthetic mitral valve, there is a transcatheter mitral valve replacement (TMVR) option for some patients. The gold standard is still a redo surgery; but many patients are considered inoperable or high risk.

Mitral Valve Anatomy

The left atrium (LA) is connected to the left ventricle (LV) via the mitral valve, which opens during diastole to allow blood to flow from the LA to the LV.

During ventricular systole, the MV closes and prevents backflow to the LA.

The normal function of the MV depends on its six components, which include the LA wall, the annulus, the leaflets, the chordae tendineae, the papillary muscles, and the LV wall.



File: mitralvalve Commons.Wikimedia.org

Mitral Regurgitation/Insufficiency

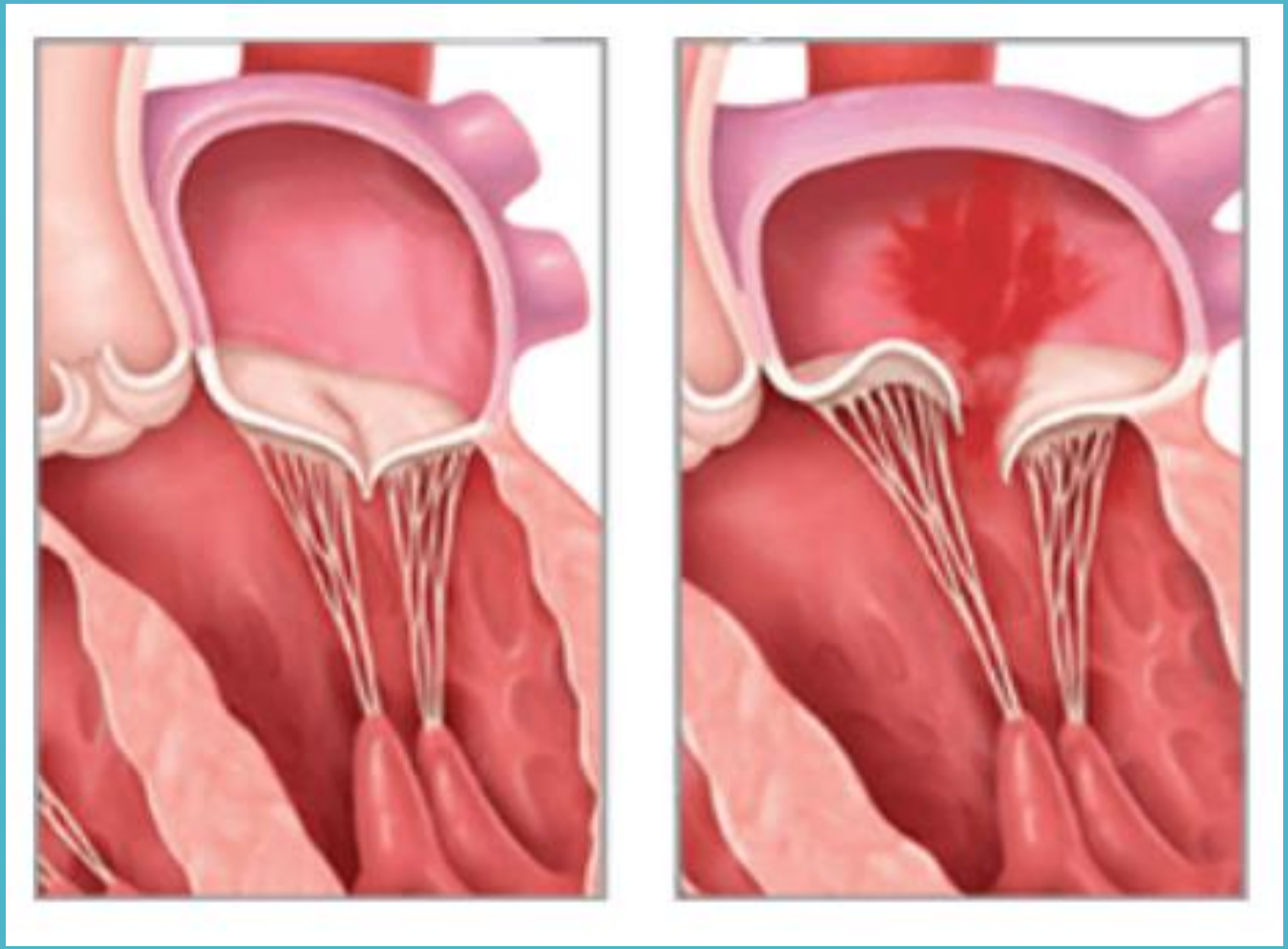
MR is a common valvular abnormality, occurs in 10% of population

Primary mitral regurgitation (MR) or degenerative MR is caused by a primary abnormality of one or more components of the valve apparatus (leaflets, chordae tendineae, papillary muscles, annulus)

Secondary Mitral Regurgitation – also referred to as functional or ischemic MR. This occurs when there is a problem(s) in the left ventricle so blood moves backward through the valve. It is a consequence of left ventricular (LV) dysfunction with normal mitral valve leaflets and chords. LV dysfunction may be due to coronary heart disease or (nonischemic) cardiomyopathy

Normal versus Abnormal MV

MV regurgitation



File: mitralregurg Commons.Wikimedia.org

Mitral Regurgitation

Complications of mitral regurgitation include:

Heart failure and related symptoms (i.e., shortness of breath)

Atrial fibrillation

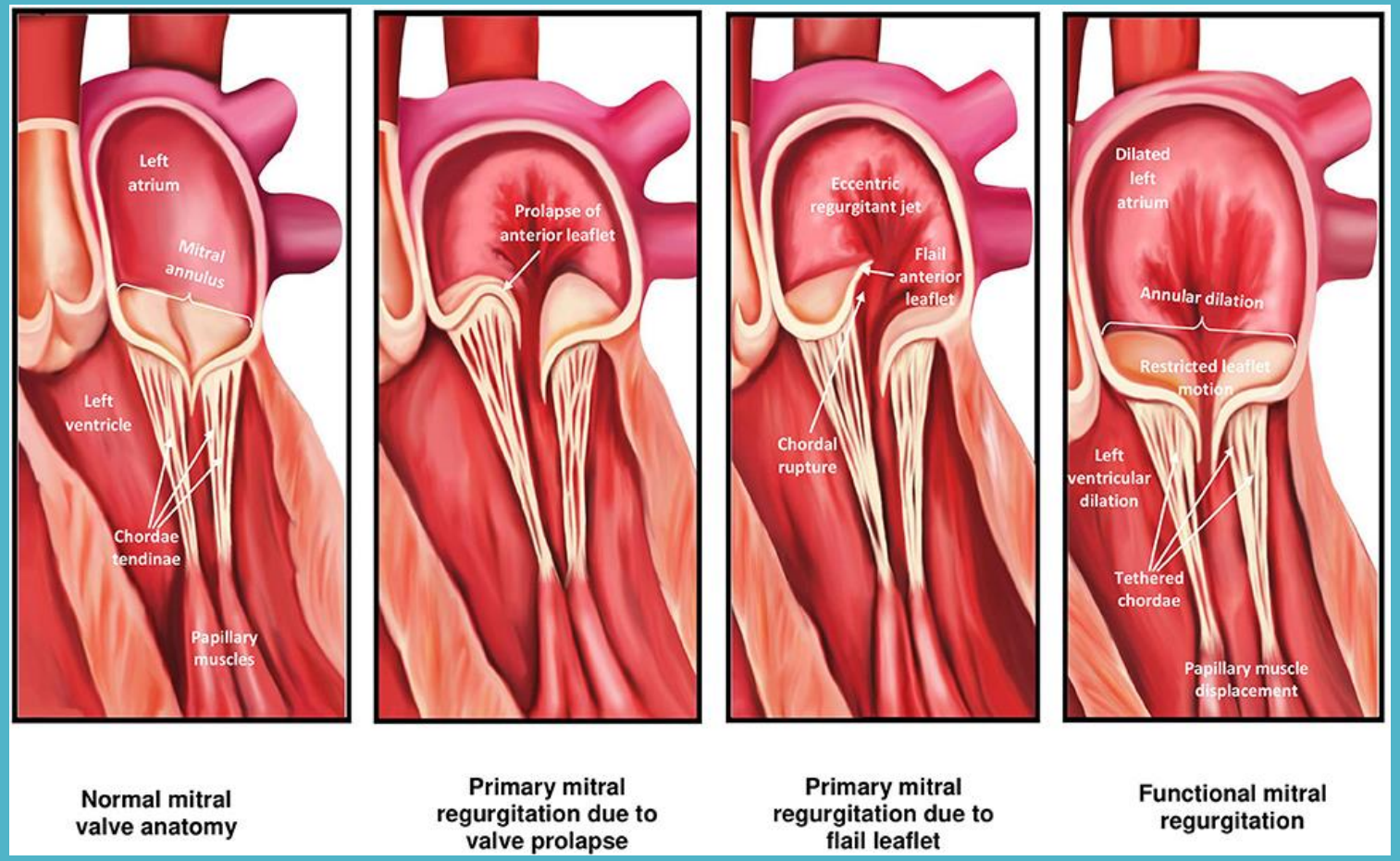
Stroke due to arrhythmias

Pulmonary artery hypertension

Dilation of the heart and cardiomegaly

Mitral Valve

Anatomy



Thekeyholeheartclinic.org

Pathophysiology of Secondary MR

MR is a retrograde flow from the left ventricle into the left atrium. MR leads to LV volume overload due to increased stroke volume, caused by an increase in blood volume within the left atrium and an increased preload delivered to the LV during diastole.

In chronic progressive MR, ventricular remodeling occurs, allowing a stable cardiac output and initial increase in EF. Over time, there is a positive feedback loop where volume overload from MR causes ventricular dilatation, widening of the mitral annulus, and diminished coaptation of leaflets and worsening of MR. Eventually, volume overload becomes so severe that wall stress-related afterload on the LV leads to dilatation and decreased contractility resulting in a reduction of EF

As with severe AS, many of these pts are frail, elderly and with significant comorbidities.

Acute versus Chronic MR

Acute Mitral Regurgitation - findings associated with a significant decline in cardiac output and possibly cardiogenic shock, pulmonary edema.

Acute MR is typically related to either papillary muscle rupture from ACS or destruction of the valve secondary to acute bacterial endocarditis

Chronic MR - often asymptomatic until late in the course. Clinical findings include fatigue, DOE, orthopnea, PND, weight gain, edema, and JVD, syncope or near syncope, cyanosis, anasarca, hepatomegaly, ascites and pleural or pericardial effusions. These latter findings are reflective of the development of pulmonary hypertension with right ventricular systolic dysfunction.

Treatment

Medical treatment can include ace inhibitors, loop diuretics, symptom mgt

The decision for surgery is dependent on the underlying cause of MR. Patients with valvular damage due to chordal or papillary muscle rupture or infective endocarditis require surgery. Functional causes of MR, such as ischemia, require revascularization. Patients with acute, symptomatic MR, or deterioration of LV function require surgical intervention.

Pts diagnosed with primary severe MR require surgery when they are symptomatic with an ejection fraction over 30% or asymptomatic with an EF of 30% to 60%. Mitral valve repair has two aims: have an acceptable surface area of mitral valve leaflet coaptation and correct annular dilatation.

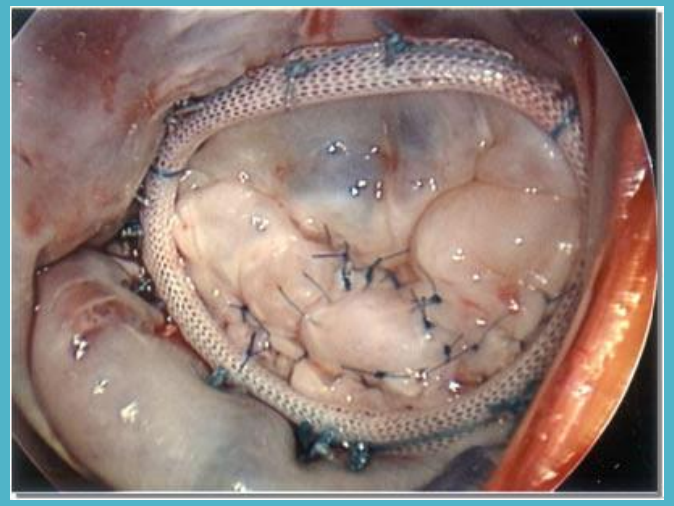
The ACC/AHA recommend MV repair over replacement due to decreased recurrence of MR after repair. Some data saw a decrease in morbidity and mortality after surgical repair over replacement. MVR is favorable over repair when there is extensive tissue destruction such as in IE in some cases of infective endocarditis.

MV Annuloplasty

Annuloplasty is the most common surgical repair performed to treat MR following the 3 principles of surgical MV repair: preserve leaflet mobility, increase leaflet coaptation surface and avoid progressive annular dilation

This technique is widely used as a stand-alone procedure in FMR or added to leaflet repair or chordal implantation in degenerative MR

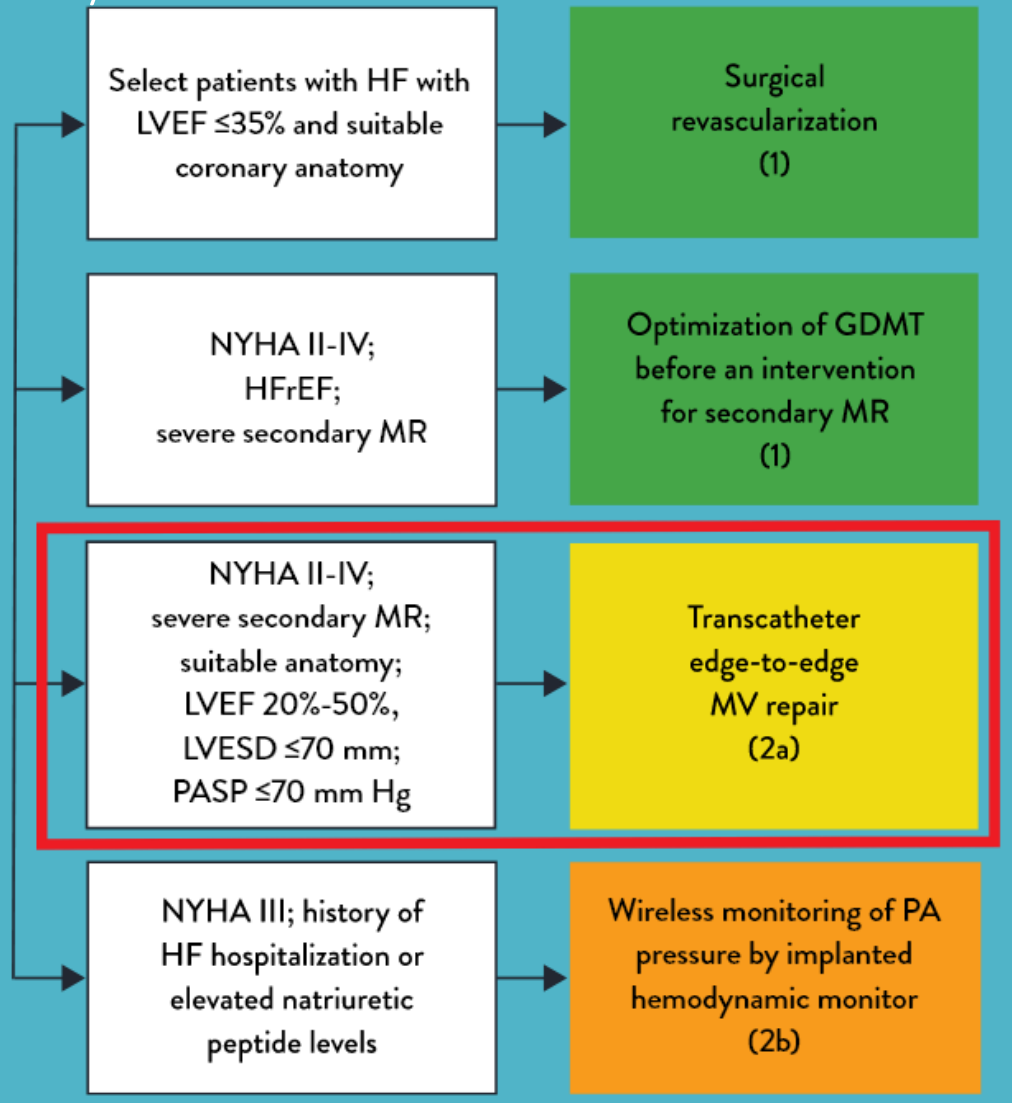
As open-heart surgery, minimally invasive surgery and now the direct percutaneous mitral annuloplasty addresses the underlying mechanisms of functional MR with a less invasive, catheter-based approach.



Heartvalvesurgery.com

Enter Percutaneous Treatment

2022 AHA/ACC/HFSA Guideline for the Treatment of Heart Failure



Risks/Complications

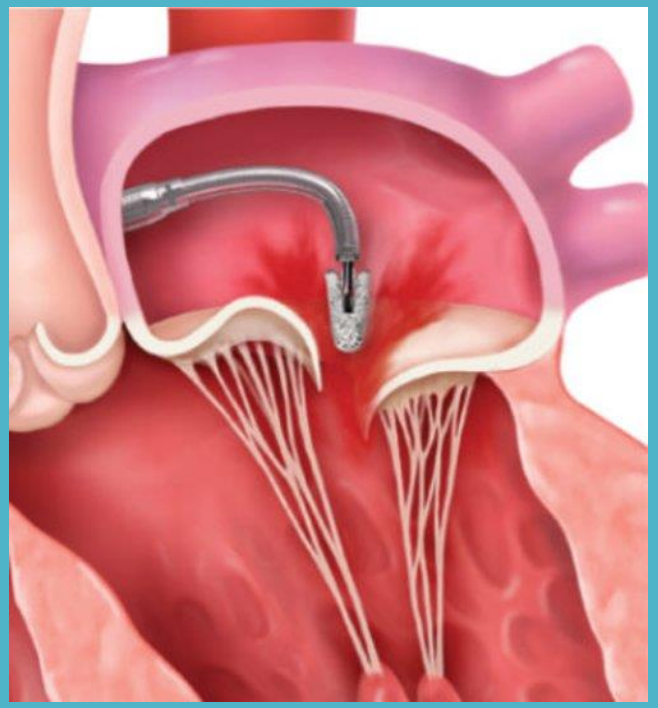
Multiple device-specific considerations distinguish TMVR from TAVR. The mitral valve annulus is significantly larger than the aortic annulus, and therefore will require larger valve mounted on larger delivery system.

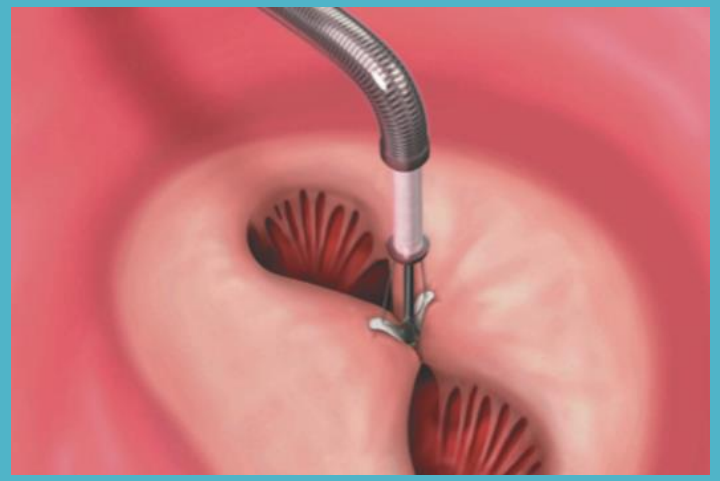
Short term complications ***(peri-procedural)***	**Long term complications** ***(post-procedural)***
Valve embolization or late migration	Severe PVL/Hemolysis
Need for second valve/Reintervention	Valve Thrombosis/Dysfunction
Damage/interference with other structures • LV perforation • LV pseudoaneurysm • Mitral annular disruption • LCx occlusion • MV leaflet/Chordal disruption • Pulmonary vein perforation	Residual moderate to severe MR
Conversion to open heart surgery	Cerebral embolic events (clinical or subclinical)

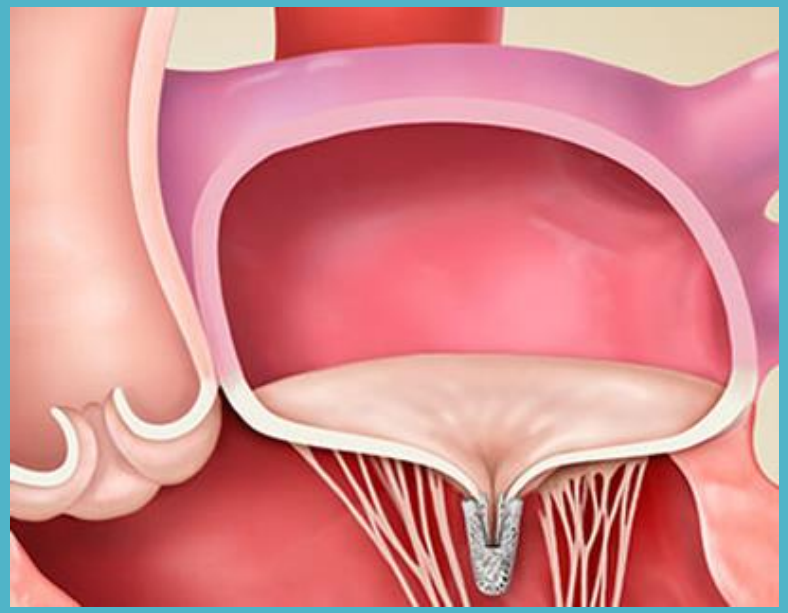
File: TMVR Commons.Wikimedia.org

Transcatheter edge to edge repair (TEER) of the MV

A minimally invasive, non-surgical treatment option often using a MitraClip device. This technique is used to treat high-risk patients who have severe MR







File: TMVr Commons.Wikimedia.org

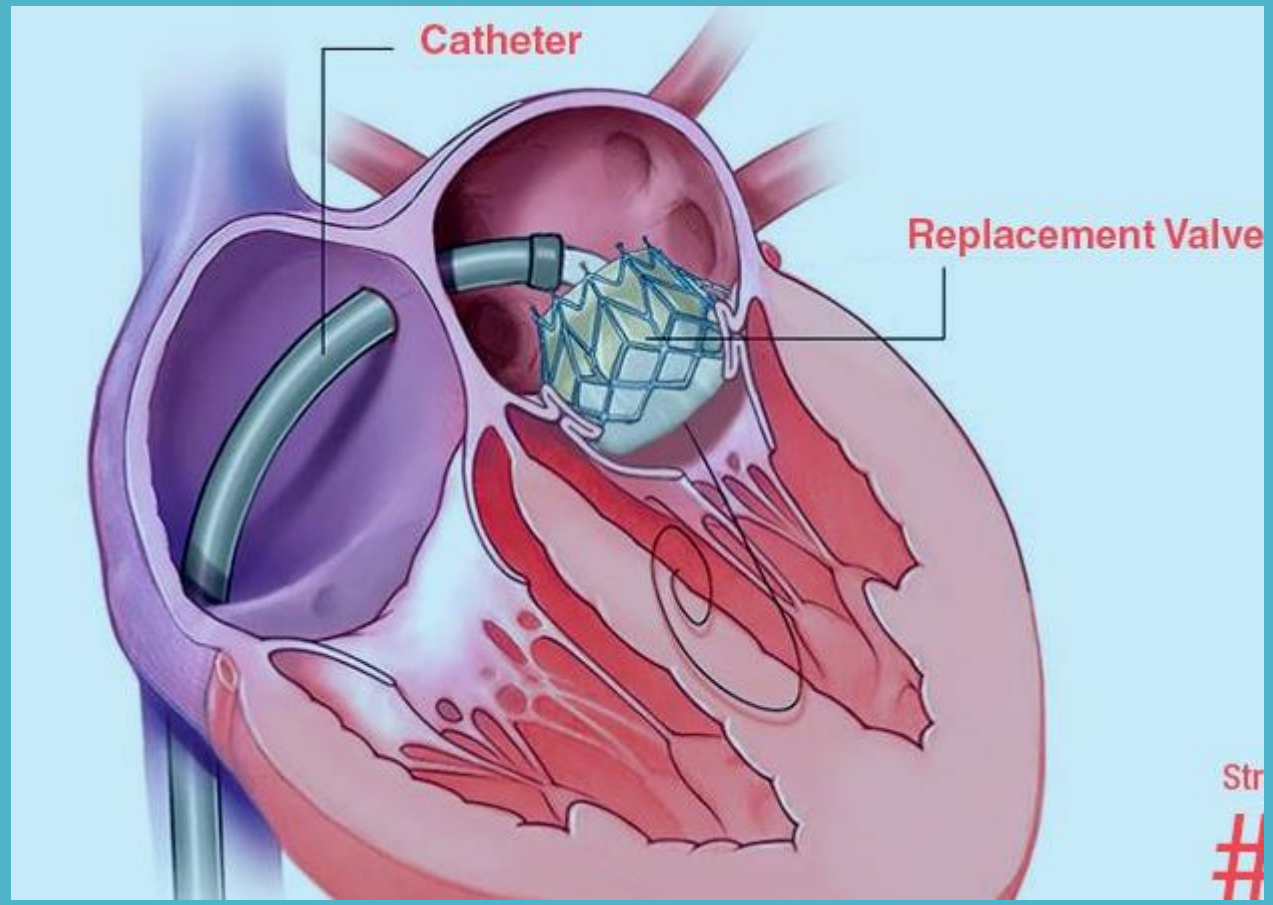
Transcatheter Mitral Valve Replacement

Many anatomies are not suitable for edge-to-edge repair. Transcatheter mitral valve replacement offers several potential advantages over transcatheter repair, most notably a greater and more sustained reduction in MR post-implantation, but also potential disadvantages.

The MV is dynamic, asymmetrical, and heterogenous, with a saddle-shaped annulus and a complex sub-valvular apparatus. Complex anatomy and disease processes presents difficulties for valve delivery, positioning, anchoring and sealing. The valve lies near the aortic valve and left ventricular outflow tract (LVOT); obstruction is a potential complication of TMVR and is associated with poor outcomes.

Transcatheter MVR

TMVR



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What is the difference between TAVR and TMVR?

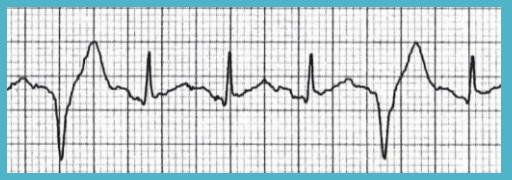
TMVR is much more complex, related to complexities of the MV anatomy, differences in pathology that require MVR as well as the impact that mitral valve replacement has on physiology and cardiac function, irrespective how the mitral valve is replaced.

Importantly, in the case of TAVR, a less invasive method is offered to accomplish the same as the traditional surgical intervention. Valve replacement is not the recommended treatment for the majority of mitral valve disease and is avoided due to surgery giving a shortened life expectancy and increased morbidity with MVR.

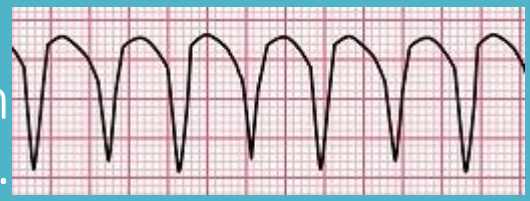
Another distinction between TAVR and TMVR is the etiology and natural progression of the underlying disease that are very different between aortic and mitral valve disease. The primary aortic disease treated has been AS, which has several etiologic factors that cause a similar physiologic dysfunction and risk. AVR leads to improved survival and quality of life. The primary MV disease is regurgitation, which occurs as a primary valve defect and secondary to ventricular dysfunction. Primary MR is treated by valve repair with excellent long-term outcomes. Secondary regurgitation has poor long-term outcomes.

Ventricular Arrhythmias

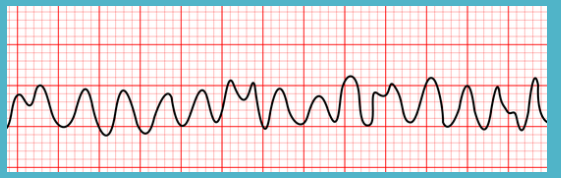
Premature ventricular contractions - PVCs, usually harmless. A key exception is when PVCs happen frequently or for longer periods.



Ventricular tachycardia - Ventricular tachycardia (VT) happens when the lower chambers of the heart beat very fast. In some cases, VT can turn into ventricular fibrillation, which is a more dangerous condition.



Ventricular Fibrillation – wide-complex tachycardia caused by irregular electrical activity and characterized by QRS morphology varying in shape, amplitude, and duration with a prominent irregular rhythm. Vfib ultimately leads to sudden cardiac death (SCD).



Etiology

VF is often linked to underlying structural heart disease.

Three percent to 12% of cases of myocardial infarction (MI) develop VF during the acute phase. MI patients with complete coronary occlusion on an angiogram, anterior wall infarction, atrial fibrillation, and pre-infarction angina are more prone to develop VF.

Common conditions associated with VF include electrolyte abnormalities (hypokalemia/hyperkalemia, hypomagnesemia), acidosis, hypothermia, hypoxia, cardiomyopathies, family history of sudden cardiac death, congenital QT abnormalities, Brugada syndrome, and alcohol use.

Patients with a history of VA especially sustained monomorphic or polymorphic VT may transition to VF in susceptible patients.

VF

VF occurs when parts of ventricular myocardium depolarize erratically in an uncoordinated manner.

The most common presentation for VF is sudden collapse from cardiac arrest leading to SCD from improper ventricular contraction resulting in low cardiac output.

Pts may have signs of acute MI such as chest pain, dyspnea before the event. Pts with a history of CAD or CHF may have worsening of chronic symptoms such as angina, dyspnea, orthopnea, PND and edema.

At the time of presentation, pts are unconscious, unresponsive, and have no palpable pulse. This leads to death within the next few minutes.

Patients surviving VF

- Need a thorough H&P including any family history of unexplained cardiac death.
- Any cardiac history and the med list reviewed for arrhythmogenic drugs.
- Correct reversible causes of VF such as electrolyte abnormalities, acidosis, and hypoxia.
- Evaluate patients for underlying *ischemic heart disease* with an echocardiogram & angiogram. 50+% have significant CAD on angiogram.

Pharmacologic Therapy

Evidence based	Limited evidence
Procainamide, Quinidine, Lidocaine	Ranolazine
Flecainide, Propafenone	Adenosine
Beta Blockers	Digoxin
Amiodarone (combined with BB increases efficacy) High Iodine content, many side effects	Isoproterenol
Dronedarone (non-Iodinated) similar to amio	Ivabradine
Dofetilide, Sotolol	
Calcium Channel Blockers	

Immediate treatment

PVCs/non-sustained VT

- Check K+, Mg+ levels

- Workup needs to include ruling out cardiac ischemia

VT with pulse

- Drugs, *close* observation as this may reoccur or worsen to pulseless VT

VT without pulse/Vfib

- Defibrillate, drugs, CPR

Internal Cardioverter-Defibrillator

The main treatment for anyone who has survived cardiac arrest & used in pts at high risk of sudden cardiac arrest (eg, low EF). It can cardiovert or defibrillate. It records the heart's rhythm and treats dangerous ventricular arrhythmias with a shock. Most defibrillators also work as a pacemaker, should marked slowing of heart rate occur, and can deliver anti-tachycardia pacing should VT occur to terminate it without delivering a shock.

- Anti-tachycardia pacing - an alternative method of stopping ventricular arrhythmias and is available in most ICDs. It involves delivering a short series (eg, 5 to 10) of paced beats. Anti-tachycardia pacing can be very effective for some slower ventricular arrhythmias (eg, 150 to 200 beats per minute), and is often programmed as the initial therapy for these arrhythmias. High-energy shocks are often programmed as the initial therapy for very fast rhythms (eg, more than 200 to 220 beats per minute), or as a rescue therapy if anti-tachycardia pacing fails.

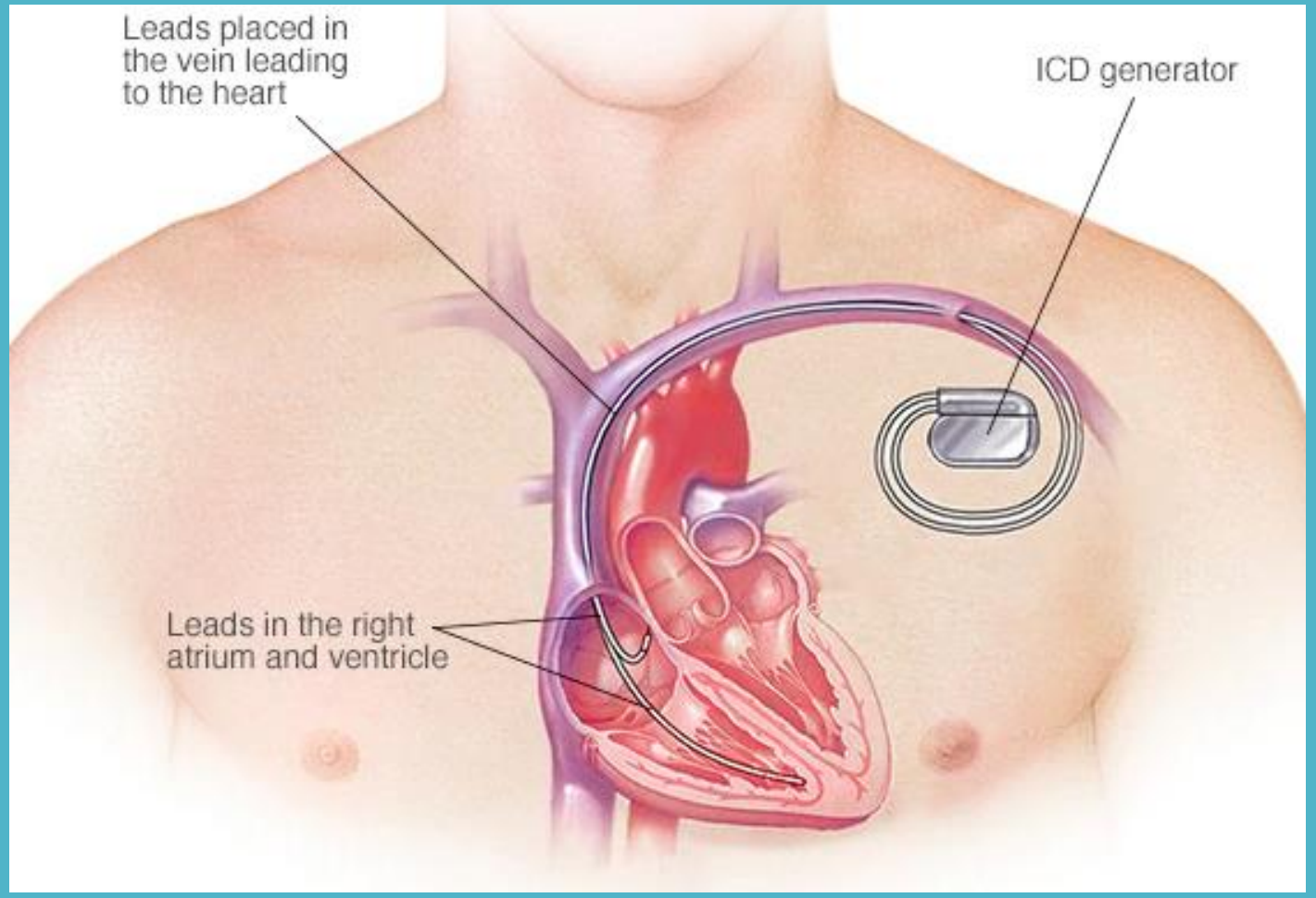
Procedure

In most cases, an ICD is inserted after the person is given a sedative and a local anesthetic (numbing medication) is injected into the skin. Some patients will be given general anesthesia.

An incision is made below one of the collarbones. The leads will be placed into the heart through the vein that runs next to the collarbone. Up to three leads will be placed inside the heart. One lead will be placed in the ventricle, one may be placed in the atrium. A third lead is implanted when cardiac resynchronization therapy is planned.

ICD Placement

ICDs can also be placed subcutaneously



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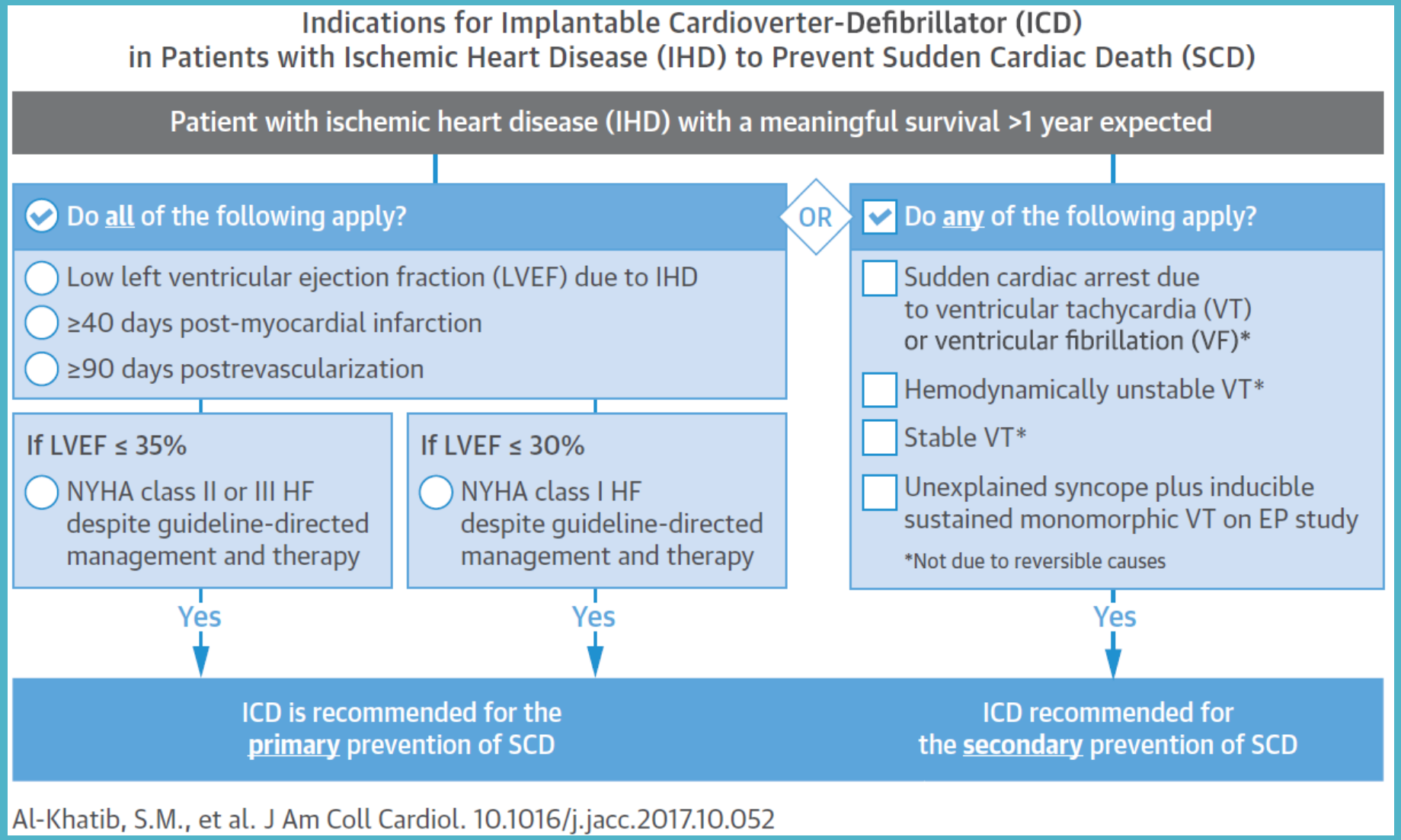
Indications for ICD Implant

ICD Criteria



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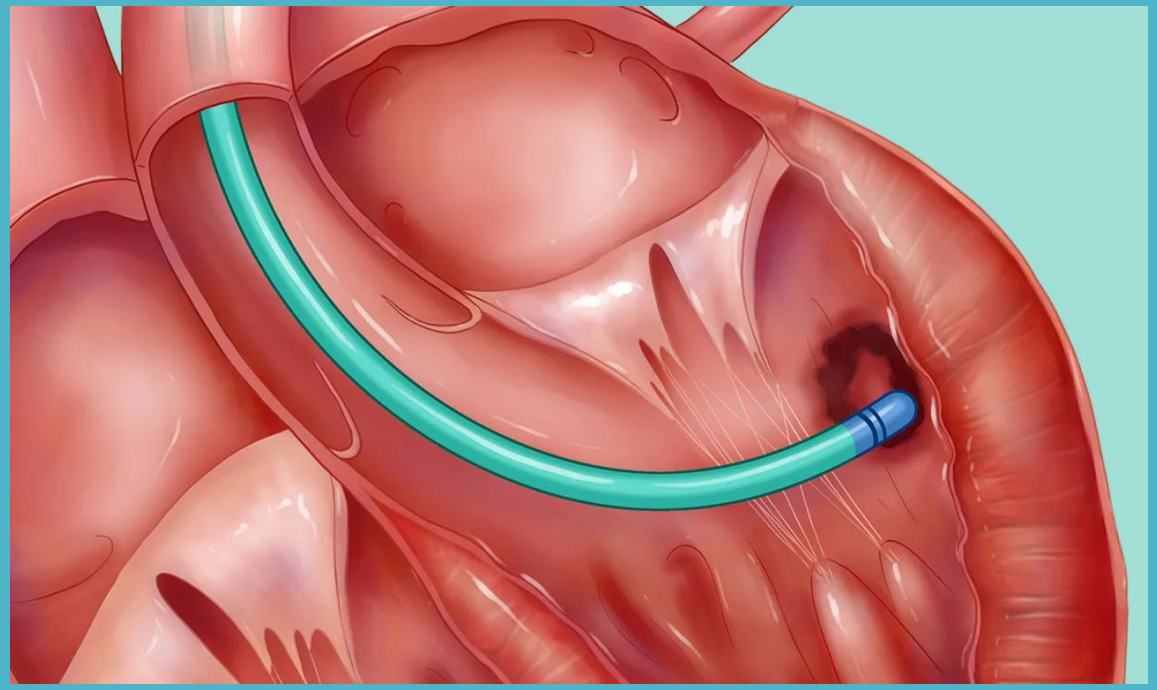
VT Ablation

Ablation of ventricular tachycardia (VT) in structural heart disease is an increasingly used treatment. It is the most effective strategy in controlling arrhythmic burden in VT patients. The approaches are the result of the last 10years of technological advances (Catheters, 3D mapping systems) and the constant study of the mechanisms underlying arrhythmic circuits.

Catheter ablation is currently considered the most effective non-pharmacological approach in reducing recurrence of ventricular tachycardia. Utilizing medications increase the efficacy of ablation.

VT Ablation

The aim of this procedure is to target the abnormal focus of the VT by placing a long, thin wire or catheter into the heart chambers. When the VT focus is identified, radiofrequency energy is applied to a small area (4 to 5 mm in diameter) to destroy the abnormal tissue. The number of burns required to treat the VT varies among patients.



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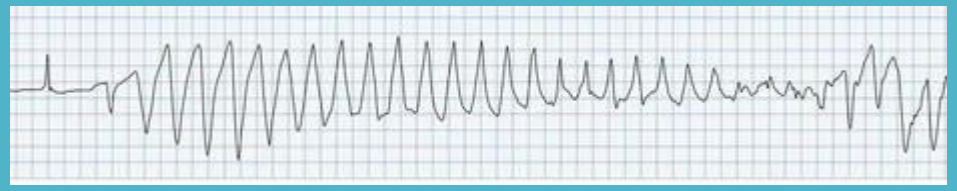
Is there a role for Magnesium



Other than in Torsades?

Magnesium plays an integral role in a variety of functions related to CV disorders. Reduced intake of Mg has been linked with a higher risk of hypertension, atrial fibrillation, ischemic heart disease, and new-onset heart failure and heart failure-related hospitalization.

In *some* studies, a low serum Mg level is associated with up to a 50% higher incidence of new AF, left ventricular hypertrophy, and is a predictor of SCD

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Thank you for your time and attention!

Thank you for all you do, every single day!