“The changing face of heart disease 2026”

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Curriculum Vitae

- ***(Why am I qualified to teach this course?)***

- BA Biology Bucknell University, 1973, summa cum laude
- MD Downstate Medical Center, Brooklyn NY 1977, with honors
- Internship and Residency in Internal Medicine 1977-1980
- Irwin Army Hospital, Ft Riley KS 1980-1984. Director of ICU
- Trumbull Mahoning Medical Group, 1984-2013, Warren OH
- Chairman, Medical Ethics, Trumbull Memorial Hospital

Heart Disease:

- Public Health Enemy #1!!!

The leading cause of DEATH is heart disease

- In the US, 1 out of 3 deaths are from heart disease
- In the US, over 900,000 people die annually from heart disease
- By comparison, cancer is the #2 cause of death in the US, about 600,000 deaths per year
- The good news: the RATE of deaths from heart disease has dropped dramatically in the past 50 years, by about 2/3. (From over 550 deaths/100,000 per year to under 250/100,000/year
- Due to declines in the death rate from both heart disease and cancer, the average life expectancy has gone up 10 years since the 1970s (from 68 to 78 in women, 64 to 74 in men), dipping only with the COVID epidemic. (Sidebar: this is one reason Social Security and Medicare are in such financial trouble and most companies have abandoned pensions, with guaranteed benefits, for 401k's.)

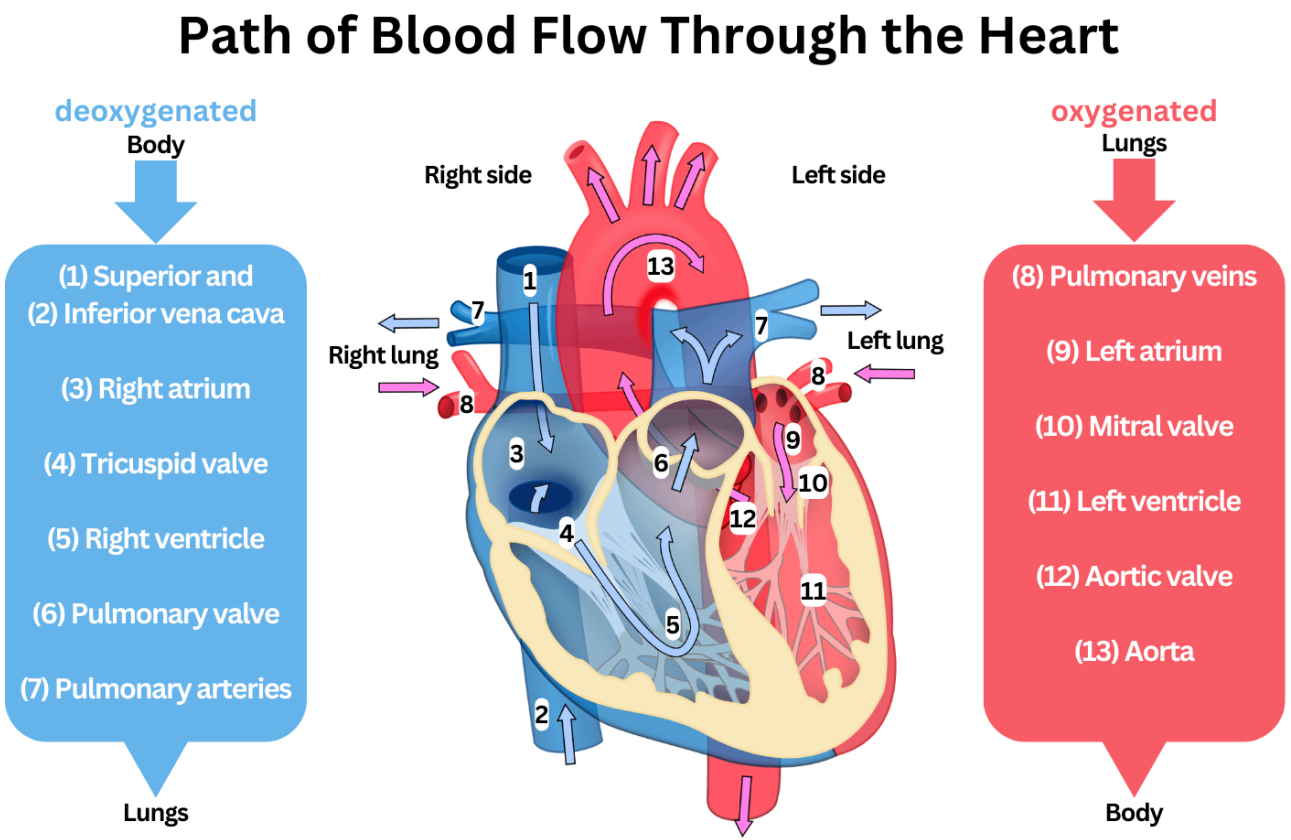
WHY has the death rate dropped?

- Improvements in early detection and intervention
- Greater emphasis on prevention and risk factor modifications, especially cholesterol
- Marked improvement of the death rate (almost 90% decline in the past 50 years) from an ACUTE heart attacks (myocardial infarction)
- The downside: since more people are surviving acute heart attacks, the rate of OTHER, later complications like heart failure and cardiac arrhythmias (abnormal heart rate and rhythm) have increased by over 80%

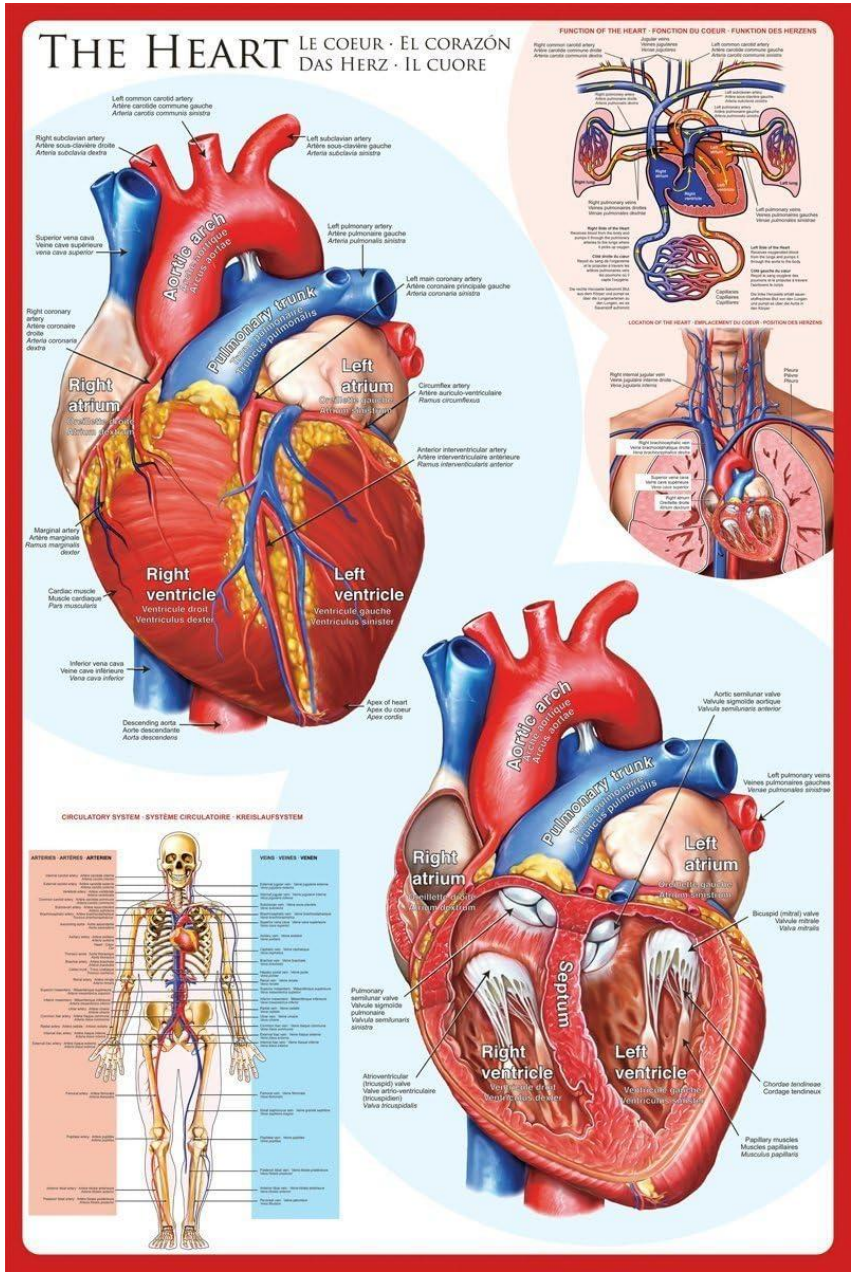
3 common Cardiac conditions

- 1. Coronary artery disease, also know as atherosclerosis of “hardening of the arteries”
- 2. Atrial fibrillation, a very common irregularity of heartbeat
- 3. Congestive Heart Failure

Blood flow


Path of Blood Flow Through the Heart
deoxygenated
Body
(1) Superior and
(2) Inferior vena cava
(3) Right atrium
(4) Tricuspid valve
(5) Right ventricle
(6) Pulmonary valve
(7) Pulmonary arteries
Lungs
Right side
Left side
Right lung
Left lung
1
2
3
4
5
6
7
8
9
10
11
12
13
oxygenated
Lungs
(8) Pulmonary veins
(9) Left atrium
(10) Mitral valve
(11) Left ventricle
(12) Aortic valve
(13) Aorta
Body

Coronary Anatomy



Coronary Artery Disease

- The heart is a muscle that pumps blood through the body.
- Unlike other muscles, the heart muscle NEVER “takes a break”, it beats 60-90 times per minute, every minute of every day
- Like any muscle, the heart muscle needs a steady and reliable source of blood to bring it the oxygen and nutrients it needs to pump
- The heart’s blood supply comes through 3 coronary arteries, that come off the aorta (the main artery leading out of the heart) as soon as it comes out.

What is the coronary artery anatomy

- The heart muscle is supplied by 3 main arteries. The Left main artery, which then divides into 2 major branches, the Left Anterior descending and Circumflex, and the Right.
- The LAD supplies the front portion of the heart, which provides about 70% of the pumping action. The circumflex supplies about 10% of the pump on the left side of the heart, so the left main supplies (together) 80% of the muscle, which is why it is so critical.
- The right coronary artery supplies the right side and bottom portion of the muscle, about 20%.
- The anatomy and pump supply of these arteries is critical in determining the best treatments for blockage, to be discussed later.

What is Coronary Artery Disease

- Over time, the coronary arteries (which supply blood to the heart muscle) may get blocked.
- Once partially blocked, many (but not all) patients may experience symptoms like chest pain or pressure (called “angina”), or shortness of breath, especially with exertion.
- Exertion increases the demand on the heart muscle to pump blood, and with blockage, not enough blood and oxygen gets through for the muscle to keep up.
- If an artery becomes COMPLETELY blocked, so the muscle is getting NO blood flow, that can cause a heart attack (“myocardial infarction”), where a piece of heart muscle dies.

What blocks the arteries?

- The arteries become blocked by fatty deposits over time.
- The fatty deposits are mostly made of cholesterol, a necessary substance for many normal body functions. Especially troublesome is a form of cholesterol called LDL (low density lipoprotein).
- Another substance called lipoprotein A makes the LDL more likely to “stick” to the walls of the arteries.
- Fatty deposits cause inflammation over time, which can attract platelets (parts of blood cells that help form clots, necessary to stop bleeding), which can then cause a clot to completely block the artery (leading to heart attack).
- Over time, the fatty deposits attract calcium, which can show up on CT scans and other cardiac tests.

Risk Factors for coronary artery disease (I)

- Risk factors make it MORE LIKELY that certain patients will develop blocked arteries
- Age – older people get more blockage, especially after age 70
- Male gender – men tend to get blockage 10 years earlier, on average, than women. Estrogen provides partial protection, so blockage in women is much less common before menopause (unless they have diabetes). This is a major part of the reason women live, on average, 4 years longer than men
- High Blood pressure (hypertension)
- High cholesterol (especially high LDL “bad” cholesterol)
- SMOKING!

Risk Factors (II) - Diabetes

- Diabetes, both type I (insulin requiring, used to be called “juvenile”, and type 2 (strong familial factors, used to be called “maturity onset”).
- Diabetics are, in many ways, 10 years older physiologically in terms of developing heart disease risk than non-diabetics.
- Even very MILD diabetes, with slight increases in fasting blood sugar and A1C levels, are associated with higher risks of heart disease.
- Many physicians no longer believe there is such a condition as “prediabetes”. New treatment guidelines encourage starting many patients on medication to lower blood sugar and A1C at levels much lower than were considered “acceptable” 10-20 years ago.

Risk Factors (III)

- Triglycerides (TG): another blood fat. High triglycerides closely linked to diabetes/elevated blood sugar. High triglyceride patients frequently have“high normal” blood sugars and/or family history of diabetes.
- Most experts now believe high TGs are not really an “independent” risk factor but really reflect underlying glucose intolerance (“mild or Stage 0 diabetes), and THAT increased risk. (It’s a MARKER of risk, not a CAUSE.)
- FAMILY HISTORY: A big one, especially for men. “Premature” heart disease is defined as a family member YOUNGER than age 70 with serious heart disease, bypass surgery, or death. One thing physicians are now realizing, and emphasizing, is earlier and more intensive intervention for OTHER risk factors, especially blood pressure and cholesterol, for people with bad family histories.

Risk Factor Modification

Modifying things we KNOW increase the risk of blockage actually WORKS, and lowers the likelihood of getting serious blockage.

1. Blood pressure: The new “normal” or “target” has been lowered from 140/90 to 120/80. This lowers the risk of heart attack, stroke, and kidney failure. And, it’s much easier to achieve in many patients, due to wide variety of blood pressure medications. 20+ years ago it was very challenging for doctors to lower BP enough without the meds causing intolerable side effects.
2. STOP SMOKING! The biggest single factor reducing cardiac death and increasing life expectancy in the last 50 years is the decline in the rate of smoking. (Smoking rate was 40% in the 1970s; now, 10-11% smoke cigarettes with another 6-7% using another form of tobacco, like vaping.) Life insurance companies ALWAYS ask about smoking, and, under Obamacare, the ONLY medical question allowed to set rates was smoking.

Risk Factor modification – Blood Sugar

- Lowering blood sugar, even from “modest” levels. Normal blood sugar is under 100 fasting, with 95-100 being “high normal”. A1C, a measure of long-range blood sugar (past 3 months), should be under 5.5, 5.5-6 “high normal” and over 6 diabetic. The goal in treating diabetics is to get the A1C from 7 or 8 to 6.5 or less.
- New weight loss drugs for diabetes (like Ozempic) help patients lose weight, lower sugar and A1C, and reduce the risk of heart attack. (The drug itself may have some direct benefit in addition to lowering weight and sugar). It’s one reason there has been a push for insurance companies and Medicare to cover weight loss drugs; it lowers COSTS overall by preventing heart problems. Also, losing weight just by diet and exercise becomes MUCH more difficult for many (if not most) patients over age 70, those at greatest risk for heart disease.

Risk Factor Reduction - Cholesterol

- Physicians used to say a total cholesterol level of 200 is “normal”, and LDL (‘bad”) cholesterol should be under 150.
- There is also a substance called HDL (“good”) cholesterol, that “soaks up” bad cholesterol in the blood and prevent blockage. HDL should be over 40, only exercise currently increases HDL (no meds yet).
- The new “targets” for cholesterol – especially for people with other risk factors (like male gender or bad family history) – are 120-130 for total and 60-70 for LDL. For “at risk” patients, “NORMAL” is too high. The GOAL is a LOW level and, for almost all patients not willing to adopt a vegetarian diet and fanatical exercise routine, that means medication.

Cholesterol (II)

- The most commonly used drugs to lower cholesterol are the “statin” drugs. Some cardiologists go so far as to say that statins should be in the water!
- Lowering cholesterol – and especially LDL – to these “low” levels has been proven to be very effective in lowering the risk of heart attack and death.
- Previously, cholesterol levels hadn’t been emphasized until age 50. More recent data shows marked effectiveness in reducing the risk of heart disease in patients in their 40s and even some in their 30s, especially males and those with bad family histories.

Drugs for cholesterol lowering

- Statins – there are 7 widely available statin drugs. They all work the same, by blocking an enzyme in the liver that makes cholesterol. They all lower LDL (“bad”) and raise HDL (“good”) cholesterol and lower the risk of heart attack. The chemical names all end in “statin” and they are the first line of treatment. Generally well tolerated although some people have side effects, especially muscle aches and some liver problems. If you have side effects from one statin, another one may be tolerated better.
- Zetia – prevents cholesterol absorption in the intestine. Not as effective as statins, used in patients that can’t tolerate statins.
- Cholestyramine/colestipol – bind cholesterol in gut, many GI side effects.
- Niacin – can cause distressing flushing, not well tolerated.

Other cholesterol meds

- Repatha – injectable monoclonal antibody that targets PCSK9, a protein that interferes with liver's ability to clear LDL out of the blood. Taken after 2-4 weeks. Used for familial hypercholesterolemia and patients where a statin doesn't lower the cholesterol enough.
- Nexletol, a non-statin oral drug that blocks LDL production and can be taken instead of or with a statin.
- Leqvio, a twice a year injectable
- New drugs are being developed that targe Lipoprotein A (which makes LDL cholesterol stick to the arteries)

Sudden cardiac death

- Many – but not all – patients have “warning” symptoms, like chest pain/pressure or shortness of breath, especially with exertion.
- For women, in particular, the “warning” symptoms may be atypical or unusual. Because doctors have been trained that heart disease is less common in women, often “warning” symptoms in women may be dismissed.
- For 10% or more, sudden cardiac death is the FIRST symptom of heart disease, with NO warning.
- Sudden death is usually an arrhythmic (“electrical”) cause and not a blockage (“plumbing”) problem, especially in younger patients. Many older patients DO have some degree of blockage.

Early detection/prevention in asymptomatic patients

- Because some patients have NO warning symptoms, tests have been developed to detect signs of cardiac blockage earlier.
- These tests can be used for screening, especially for patients at higher risk due to other factors.
- The Calcium cardiac CT scan depicts calcium deposits in the coronary arteries. The test is simple, noninvasive, quick, widely available, and inexpensive ($100 although frequently not covered by insurance). This makes it very suitable as a screening test for large numbers of patients.
- The calcium score runs from 0 (none) to 1000 (you needed open heart surgery months ago).

Early detection (II)

- An elevated calcium CT score can lead to:
  - More thorough testing, such as stress test or cardiac catheterization to determine the exact degree of blockage and functional capacity
  - More aggressive risk factor reduction (higher doses especially of statin drugs to get the cholesterol and LDL to very low levels)
  - Another, more specific and detailed test like a CT coronary angiogram. This is a test that requires an injection of dye in a vein to outline the arteries directly, but is less invasive than a catheterization (where the dye is directly injected into the arteries and heart). In some high risk patients, the doctor may go directly to this and skip the calcium CT scan.
  - Among my circle of acquaintances in Cincinnati, I am aware of at least 4 people who had NO symptoms who had blockage bad enough to need a stent or bypass that was originally detected by the calcium CT scan.

What to do if we find blockage?

- The appropriate treatment for blocked arteries depends on many factors, including the number, degree, and location of blockages.
- For many asymptomatic or minimally symptomatic patients without severe or critical blockages, treatment with medication has a good an outcome as more invasive options.
- Treatments include beta blockers (which slow the heart rate and protect the heart muscle), daily baby Aspirin (which prevents platelets from forming clots in the arteries leading to worse blockage) and risk factor reduction, especially smoking, blood pressure control, cholesterol lowering and blood sugar lowering.

Cardiac intervention for blockage

- Patients with more serious and advanced blockages may benefit from interventions like angioplasty (“roto-rooter”) with placement of stents to keep the arteries open, or bypass surgery (CABG).
- Every case is individual. In general, bypass surgery is preferred when there are more than 2 or 3 blockages to open up and stent.
- Blockages in critical locations – like the Left main artery or proximal Left anterior descending artery (LAD) – may benefit more from bypass surgery. These critical blockages put a greater amount of heart muscle tissue at risk if there is a complete blockage (and heart attack) and have worse outcomes.
- Angioplasty and bypass also require follow up care and risk reduction to prevent recurrences.

Acute intervention for heart attacks

- 50 years ago, when I was an intern and resident, the treatment for a heart attack was as follows. We admitted the patient to the CCU; we put them on a heart monitor to watch for abnormal beats; we put them on oxygen; we started an IV and gave pain medication; and we hoped they wouldn’t die. Stay in the hospital was 10-14 days and cost under $50,000.
- Now, when a patient presents to the emergency room with a heart attack, they go directly to the heart catheterization lab, get a clot busting drug IV, get a heart cath with a stent placed in the blocked artery. Hospital stay is 3-5 days and the bill is $250-500,000.

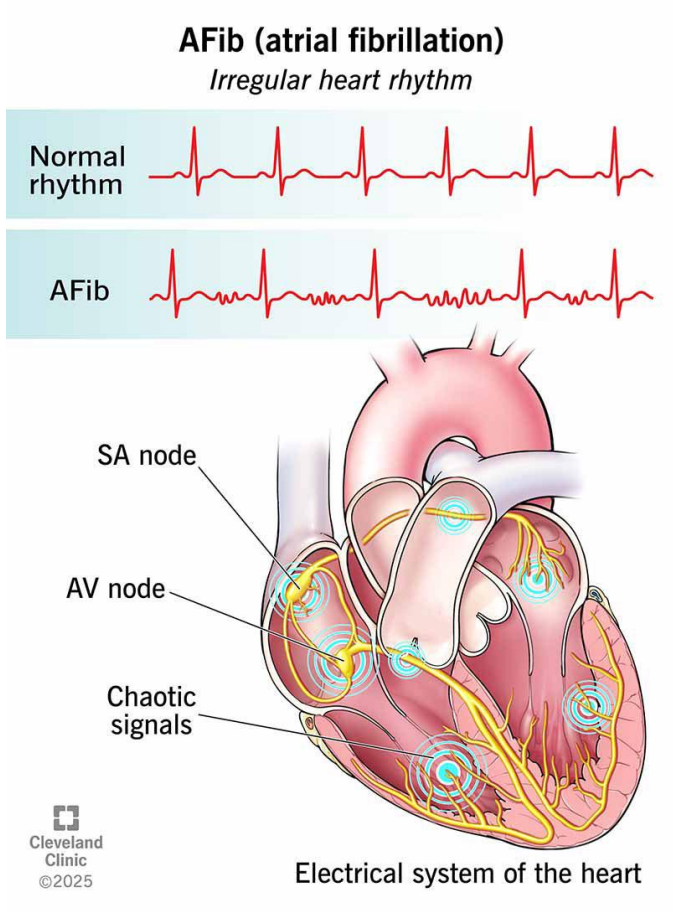
Acute intervention – is it worth it?

- So why do we do this, is it worth the trouble and expense.
- 50 years ago, the in-hospital mortality from an acute MI was 10% and the 1 year mortality, an additional 10%.
- Now, the in-hospital mortality is 1% and the 1 year mortality another 1%. This is a 90% reduction in death rate from heart attack.
- And now you can hopefully understand why medical care is so expensive and Medicare is going broke. Advances in medical care improve outcomes dramatically, but the cost increases are also dramatic.

Atrial Fibrillation

- Atrial fibrillation (A fib) is a common irregularity of the heartbeat. The beat is rapid and irregular, and the pumping action of the heart is “out of sync”.
- Risk factors for developing A fib include
  - Increasing age
  - Hypertension
  - Alcohol consumption (more than 1-2 per day, “binge” drinking)
  - Hyperthyroidism
  - SLEEP APNEA

A fib on EKG


AFib (atrial fibrillation)
Irregular heart rhythm
Normal rhythm
AFib
SA node
AV node
Chaotic signals
Cleveland Clinic ©2025
Electrical system of the heart

A fib – intermittent/persistent

- A fib can be intermitent (on and off, sometimes for very brief periods of time) or persistent.
- A fib may be asymptomatic, especially when brief
- Symptoms include palpitations, dizziness, shortness of breath, and passing out
- It used to be very difficult to diagnose intermittent A fib, since the episodes may be brief, scattered, and hard to “capture”. Only when A fib was persistent or symptomatic would the patient seek treatment.
- The Apple watch (and other brands) have made A fib much easier to recognize. The watch will sense A fib and notify the patient. Physicians are seeing many more patients with A fib who discovered it incidentally on their watch!

A fib and sleep apnea

- One of the more common associated causes of A fib is sleep apnea.
- Sleep apnea is a condition where the air passages in the mouth and nose close off (obstruct) during sleep. This disturbs the sleep cycle and wakes the patient up (briefly) to breathe.
- Episodes of sleep apnea may happen dozens of times an hour without the person even being aware. This recurring sleep disruption makes the person tired and sleepy during the day. It also causes very loud snoring
- Sleep apnea is more common with advancing age, for males, and for overweight (obese) people.
- So, we are seeing many cases of people who don’t know they have sleep apnea presenting because their watch showed A fib!

Complications of A fib

- A fib affects about 10 million people in the US, and is responsible for over 150,000 deaths a year, mostly from stroke
- A fib increases the risk of stroke by at least 5x
- Blood clots can form in the left atrium (upper portion) in patients with either intermittent or persistent A fib. These clots can break off and travel to the brain where they cause a stroke
- A fib also increases the severity of a stroke and the mortality (by almost double)
- The stroke risk makes the use of anticoagulants (blood thinners) essentially mandatory. Anticoagulants markedly reduce stroke risk from A fib.
- Anticoagulant use may be permanent (rest of life) unless the patient undergoes special treatment (ablation to prevent a fib or a Wartchman implant)

Treatment of A fib

- Medication: to keep heart rate regular, prevent A fib, and slow down A fib rate if it occurs
  - Beta blockers – first line but not always well tolerated, can cause fatigue/sluggishness
  - Digoxin
  - Diltiazem/Verapamil – calcium blockers, useful for patients who can't tolerate beta blockers
  - Flecanide/amiodarone – powerful antiarhythmics to prevent A fib
  - ANTICOAGULANTS to prevent stroke – a MUST
  - Cardioversion – for persistent symptomatic A fib, converting the heart back to a regular rhythm with either medication or electric shock. Patient must be anticoagulated before cardioversion to prevent stroke.

Newer/more aggressive treatment options

- Ablation – the cause of A fib is “faulty wiring”, where electrical conduction pathways in the heart are not working properly and triggering A fib.
- Ablation is an invasive procedure where a wire is placed in the heart, the abnormal conduction areas are identified, and they are then destroyed (by ultrasound waves, burning or freezing the tissue.)
- The success rate of ablation varies from 35% to 80%, more likely to succeed with intermittent than persistent A fib. The procedure may be repeated 2 or 3 times, after 3 failed attempts the likelihood of success is low (and many insurances won’t cover more than 3).
- Anticoagulants usually continued after an ablation for 6-12 months to prevent stroke if the A fib recurs. After that period, many patients can successfully stop blood thinners, one of the attractions of an ablation.

Newer treatments for A fib (II)

- When is ablation considered?
  - Symptoms are bothersome to patient
  - Medication isn’t controlling A fib or symptoms well, or patient is having difficulty tolerating meds
  - A fib is persistent, having frequent or severe recurrences that necessitate frequent emergency room visits to control
  - Patient has difficulty tolerating anticoagulants OR is at increased fall risk. Falling while on anticoagulants can cause bleeding in the brain, which can be serious or even fatal.

A fib and the Watchman procedure

- In patient who can't tolerate anticoagulants or who are at a high risk of falling, the Watchman device offers a good alternative for stroke prevention.
- The watchman is a device implanted in the heart by a wire threaded through an artery and is considered minimally invasive
- The watchman device seals off the left atrium (upper portion) where A fib starts and where clots form. It prevents clots from leaving the heart. Most patients can stop anticoagulants after a few weeks or months.
- The watchman doesn't prevent or treat A fib or reduce symptoms, it just prevents blood clots and strokes

Congestive Heart Failure (CHF)

- CHF is a condition that occurs when the heart pump muscle weakens and cannot effectively pump all the blood out
- Blood that can’t be pumped out backs up into the lungs and eventually all the way down into the legs
- Eventually, the blood back-up causes symptoms like shortness of breath and swelling in the legs
- Bloating, weight gain, and loss of appetite may occur

CHF Causes

- CHF is the end-result of a number of heart conditions which weaken the muscle pump over time
- Common causes include
  - Coronary artery disease. Used to cause CHF as a result of multiple heart attacks damaging the muscle, this is less common due to interventions with acute heart attack. But a moderately decreased blood flow over time will weaken the muscle.
  - Hypertension
  - Diabetes
  - Kidney failure
  - Valvular heart disease, congenital or acquired
  - Alcohol abuse
  - Certain chemotherapy drugs (especially Adriamycin)

Stages of CHF

- Stage A – at risk but no signs or symptoms
- Stage B – at risk with evidence of decreased function but not yet symptomatic. Usually detected by testing such as echocardiogram. It's an ultrasound test of the heart that shows the anatomy of the heart, the valves, and assesses effectiveness of heart function by measuring the ejection fraction. The EF is what % of blood is pumped out with each beat. It should be over 50%. 40-50% is mildly to moderately impaired (at risk for CHF), less than 40% is indicative of CHF and usually symptomatic.
- Stage C – symptomatic heart failure
- Stage D – severe heart failure poorly responsive to treatment ("end stage")

Treatment for CHF (Stage A)

- Stage A – risk factor modification to prevent progression. Control blood pressure and diabetes, lose weight, stop smoking, decrease alcohol consumption. Lower cholesterol. Prescribe an ACE or ARB in selected cases.
- (ACE – angiotensin converting enzyme blocker; ARB – angiotensin receptor blocker.) Both drugs lower blood pressure and strengthen the heart muscle. ACE inhibitors include lisinopril, enalopril, captopril etc. ARBs include valsartan, losartan etc. ACE inhibitors can cause a dry cough in many patients, ARBs don't and are usually used when patients don't tolerate ACEs. Both are similarly effective. ACE and ARB drugs are now FIRST LINE in CHF treatment.

CHF Treatment

- Stage B – Risk modification as in Stage A plus ACE or ARB. Low dose beta blocker to lower heart rate and blood pressure and ease the strain on the heart muscle. Aldosterone antagonist (Aldactone) – a mild diuretic that boosts kidney function.
- Stage C – As Stage B plus use of a stronger diuretic (such as furosemide/Lasix), sometimes with fluid and salt restriction, to reduce edema (swelling in the legs due to fluid retention).
- Digoxin/lanoxin which used to be a “mainstay” in heart failure treatment has been largely abandoned, unless the patient also has atrial fib, where digoxin can control the rapid heart rate.

New interventions

- Stage C: a biventricular pacemaker to coordinate the pumping action of the various chambers of the heart. Many Stage C patients with persistent symptoms despite medication note substantial improvement in symptoms and functional capacity after this procedure.
- Stage C: an implantable cardiac defibrillator. Patients with Stage C or D are at increased risk of sudden cardiac death from an abnormal heart rhythm. The defibrillator senses the abnormal rhythm and delivers an electric shock to heart to convert the rhythm back to normal.

Stage D

- Stage D implies CHF not responding to other treatments with severe impairment of heart function. High risk of death, justifies use of “Hail Mary” extreme interventions.
- Best long-lasting option is heart transplant, but there is a shortage of organs for transplant (donor must be brain dead but still have a strong pumping heart). In 2020, there were just 8000 transplants performed worldwide, 3600 of which were in the US.
- Left Ventricular assist device (LVAD) – a mechanical device to help the heart pump blood . Frequently used as “bridge” therapy while awaiting transplant.
- Periodic or continuous infusion of drugs like dobutamine to strengthen the heart muscle pump. Usually of only temporary help.

Any questions?

- Disclaimer – I no longer have an active Ohio medical license and cannot legally render individualized treatment recommendations.