Can We Mitigate Cardiovascular Aging?

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Learning Objectives Class 3: By the end of the session, the learner will be able to:

- Identify the two roles of the myocardial myocytes.
- Name the components of the conduction system.
- Describe how the electrical impulse is transmitted through the myocytes.
- Contrast atrial fibrillation and ventricular fibrillation.

Class Outline:

Class 3: It Takes a Spark: Mitigating Alterations in the Conduction System (Word search: https://wordsearchlabs.com/view/704988

- Differences in Myocytes (muscle cells)
    - Cardiac
        - Located in walls of the heart
            - Striated (striped); usually 1 nucleus
            - Involuntary control (SNS, PSN)
    - Skeletal
        - Located in the muscles connected to the skeleton
            - Striated with multiple nuclei
            - Voluntary control
    - Smooth muscle
        - Located in the walls of hollow organs (stomach, intestines, etc.)
            - Non-striated with a single nucleus
            - Involuntary control
- Types of Cardio Myocytes
    - Myocardial Contractile Cells
        - 99% of the cells in the atria and ventricle
        - Conduct impulses
        - Responsible for contractions that pump blood
    - Myocardial Conducting Cells
        - 1% of the cells in the atria and ventricle
        - Form the conduction system of the heart
        - Generally, much smaller than the contractile cells and have few of the myofibrils or filaments needed for contraction
- Conduction System
    - Both rate and rhythm are vital to cardiac output (systemic perfusion)
    - Rate: heartbeats per min; usually 60 – 100
        - Slow rates (brady), especially less than 50 BPM, may take too much time filling with less time for ejection

        - Fast rates, (tachy), less time for ventricular filling leading to reduced volume during ejection
    - Rhythm: predictability; allows for simultaneous ejection of the chambers which enhances left ventricular filling; synchrony
        - Regular irregular: not normal, but predictable
        - Irregularly irregular: not predictable

- Conduction System Components
    - Sinoatrial (SA) node: pacemaker node of the heart
        - Located in the upper wall of the right atrium, at the junction where the superior vena cava enters
        - Wave of excitation spreads via gap junctions across both atria, resulting in atrial contraction (atrial systole) – with blood moving from the atria into the ventricles
        - Rate at which the SA node generates impulses is influenced by the autonomic nervous system (usually 60 – 100 bpm)
            - SNS increases rate
            - PNS decreases rate
    - Atrioventricular (AV) node
        - After impulses spread across the atria, they converge at the atrioventricular node –located in the back of the right atrium within the ventricular septum, near the opening of the coronary sinus
        - AV node and conduction fibers surrounding it, including Bundle of HIS are referred to as the AV junctional tissue
        - AV node also has automaticity at a rate of 40 – 60 bmp should the SA node be injured
        - The AV node acts to delay the impulses by approximately 120ms, to ensure the atria have enough time to fully eject blood into the ventricles before ventricular systole; known as atrial kick (20%-30% of atrial blood volume)
        - The wave of excitation then passes from the atrioventricular node into the atrioventricular bundle.
    - AV bundle/Bundle of His/Common Bundle (rate 20 – 40)
        - Shaft of conducting tissue
        - Penetrates the septum and bifurcates into left and right branches
            - Right Bundle Branch: continuation of Bundle of HIS
                - Conducts impulse downward to the right of the RV apex and terminates at the base of the anterior papillary muscle of the ventricle; long and thin
            - Left Bundle Branch: perpendicularly from the HIS bundle
                - Divides into 2 fascicles to carry the impulse to the LV
                - LA fascicle vulnerable to ischemia as it is dependent upon LAD for blood supply
    - Purkinje fibers
        - Originate from the 3 ends of the bundle branches and over the subendocardial ventricular surfaces
        - They are abundant with glycogen and have extensive gap junctions
        - Rapidly transmit cardiac action potentials from the atrioventricular bundle to the myocardium of the ventricles.

        - This rapid conduction allows coordinated ventricular contraction (ventricular systole); Fans out to conduct impulses from the inside of the endocardium to the outside of the myocardium
- Cardiac Action Potential: The Spark
    - Major ions involved: K+, NA+, Ca2+:
        - Higher concentrations of potassium (K+) inside of cell
        - Higher concentrations of NA+ and CA2+ outside of the cell
    - When conducting cells are resting, they are negatively charged on the inside compared to the outside (more K+ inside); cell membrane impermeable to sodium NA+ & Ca2+ = *polarized*
    - For contraction to occur, the muscle cell must become less negative requiring a positive ion shift into the cell
    - When hit with an electrical impulse, the cell membrane becomes more porous to NA+ & Ca2+ which enters the cell, making the inside of the cell less negative and contraction occurs = *depolarization*
    - After contraction, the cell must then begin relaxation and return to a polarized state awaiting a stimulus = *repolarization*
- Repolarization (example: flushing the toilet)
    - Relaxation: Repolarization occurs in a "transmural" pattern, meaning it starts at the outer layer of the ventricular wall (epicardium) and progresses towards the inner layer (endocardium).
    - <u>Steps to repolarization:</u>
        1. Calcium channel closes reducing the influx of positive ions into the cell
        2. Potassium channel opens: allowing potassium ions to rapidly move out of the cell, creating a negative charge inside the cell.
        3. Return to resting potential with the outward movement of potassium, completing repolarization
    - Preparation for the next contraction
    - Crucial for coordinated heart rhythm
- Cardiac Cycle
    - Both rate and rhythm are vital to cardiac output (systemic perfusion)
    - Rate: heartbeats per min; usually 60 – 100
        - Slow rates (brady), especially less than 50 BPM, may take too much time filling with less time for ejection
        - Fast rates, (tachy), less time for ventricular filling leading to reduced volume during ejection
    - Rhythm: predictability; allows for simultaneous ejection of the chambers which enhances left ventricular filling; synchrony
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    - Taking the Picture: EKG/ECG
        - P wave: atrial depolarization
        - PR- delay at the AV node
        - QRS: ventricular depolarization

        - ST- ventricles depolarized
        - T wave: ventricular repolarization
    - Heart Rate & Cardiac Output Relationship
        - Both rate and rhythm are vital to cardiac output (systemic perfusion)
        - Rate: heartbeats per min; usually 60 – 100
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- The Aging Conduction System
    - Decline in the cardiac autonomic nervous function
    - Decrease of the sinoatrial node response
    - Decline in the number of pacemaker cells and ion channels in the sinoatrial node (by age 75, the number is less than 10% of what it was in a young adult)
        - Reduced conduction system automaticity and lower HR
    - Decline of beta-and alpha-adrenergic (receptors in heart and blood vessels) regulation of cardiovascular function
    - Incidence of arrythmias increases during aging
    - Decreasing heart rate variability (HRV): indicator of arrhythmic complications and a predictor of sudden death: maximum heart rate decrease with age
    - Increased adipose tissue deposition, collagen and amyloid leading to lower HR (fatigue, exercise intolerance)
    - Decrease of electrical waves propagation throughout myocardium even without evidence of disease
    - Increase in the prevalence, density, and complexity of ectopic (extra) beats, both atrial and ventricular
    - Slowing the conduction of the depolarization wave and contributing to the age-related arrhythmic process
    - Slower myocardial contractions and less complete ventricular relaxation compared with younger adult individuals
    - Left side of the cardiac skeleton can calcify, which can affect the atrioventricular (AV) node, AV bundle, and other parts of the conduction system
- Cardiac Cell Aging: a Picture

- A Rose by any other Name
    - Arrhythmia and dysrhythmia: terms for an irregular heartbeat
    - Arrhythmia = "without rhythm“; any variation from a normal heartbeat rhythm
    - Dysrhythmia ="bad rhythm“; disturbances of rate and conduction
- Causes of dysrhythmias: Delay in impulse formation
    - Abnormal automaticity: disorder of impulse formation: changes in the influx of NA+ and CA2+;
        - Ischemia (lack of O2 to the cells), acidosis, excess catecholamines, excess K+ ion shifts
    - Triggered activity: An abnormal response to a previous stimulus, an action potential causes oscillations in the membrane potential, can occur during or after repolarization, can cause heart cells to contract twice after only being activated once.
        - Hypoxia, mechanical injury, antiarrhythmic meds, hypokalemia, intracellular CA2+ overload, Digitalis intoxication, high catecholamines
- Causes of dysrhythmias: Disorder in Impulse conduction
    - Reentry: most common mechanism of arrhythmia and does not require abnormal cellular electrophysiology
    - Normally once an impulse is generated, it stimulates all available receptive cells and once availability is exhausted, it dissipates
    - Impulse encounters an area of resistance—anatomic (due to scar) or functional (due to heterogeneity in electrophysiologic properties of the myocardium)
    - Supply never ends
    - One stimulus may be able to re-excite and reinitiate cardiac action
    - Primary role in several types of tachydysrhythmias (rapid rhythms)
        - Acute ischemia, myocardial infarction (MI), myofibrosis
- Common arrhythmias: bradycardias
    - Bradycardia: Resting heartbeat is slower than 60 bpm (normal for athletes)
        - More common in elderly as SA node less effective
        - Symptoms of fatigue, dyspnea, dizziness, confusion, and frank syncope due to reduced forward blood flow
        - May occur with blocks in the AV conduction system; rate 20 - 40
        - Various medications may cause bradycardia
        - Treated depending on type of symptoms
        - Sinus bradycardia

        - Nodal/junctional bradycardia
        - Heart block
- Acute Treatment of life-threatening bradycardia
    - Emergency meds:
        - Atropine
        - Epinephrine (Adrenaline)
        - Dopamine
    - Temporary Transcutaneous pacing
        - Catheter directed pacing wire
        - External generator
    - Correct the underlying condition
- Permanent Pacemaker
    - Devices placed surgically
    - Leadless; small, size of a pill; implanted with a catheter procedure directly to the inner layer of the heart with no need for leads
    - Single lead pacer or double lead pacer
    - Biventricular: 3 leads that allow pacing of both ventricular chambers and the RA; provides synchrony of chamber response; resynchronization therapy
        - Placed either with a catheter or through a small surgical incision; generator is inserted into a “pocket” under the skin of your abdomen.
        - Batteries last 10 – 15 years depending on type
        - Regular device check with wand
        - Avoid magnets/cell phones/headphones/smart watches
        - Medic alert bracelet; photo of the device card
        - Airport security should be alerted
        - $20,000 – 100,000 without Medicare
- Common arrhythmias: Ectopic Beats
    - Premature or extra beats:
        - Normal rhythm should be regular and predictable
        - Ectopic beats: Heart skips a beat (palpitation) because the electrical impulse comes sooner than normal.
            - Premature atrial contraction, PAC (no/hidden ‘P’ wave’)
            - Premature ventricular contraction, PVC (no ‘P’ wave, QRS is wide & bizarre)
            - Brief pause, after which there is a stronger than normal beat
        - May occur in healthy hearts: caffeine, stress alcohol, decongestants and antihistamines, smoking

        - Diseased hearts: ischemia, hypertrophy, street drugs
        - Rarely treated unless excessive
- Supraventricular tachycardia (SVT)
    - Supra (above) the ventricles:
    - Initiated above or at the AV node (re-entry event)
    - Rate above 100 BPM (tachycardia) when at rest
    - No ‘P’ wave, narrow QRS, regular, with rate 180 – 220BPM
    - Medications, caffeine, alcohol, physical or emotional stress, or cigarette smoking can trigger SVT
    - Women 2X risk; elderly 5X risk
    - S/S: anxiety, palpitations, chest discomfort, lightheadedness, syncope, or dyspnea. In some cases, a patient may present with shock, hypotension, signs of heart failure, lightheadedness, or exercise intolerance.
- Subtypes of SVT
    - Paroxysmal supraventricular tachycardia: occurs most often in younger, healthier people during exercise and causes extra heartbeats as the signal travels from the upper to the lower chambers; abrupt onset, 150-250BPM, spontaneous cessation
    - Atrial flutter: rate around 250–350 BPM, with the upper chambers beating more frequently than the lower chambers.
        - Normal QRS but not with every p-wave; may be regular or irregular,
        - P waves saw-toothed/jagged
    - Risk factors: Older age, heart disease, history of thyroid disorders, diabetes, COPD, and alcohol consumption
- Atrial fibrillation (Afib)
    - The most common subtype,
    - 12.1 million in US with Afib by 2030 (1 in 22 adults); “growing epidemic”
    - Rate may be as high as 400 BPM
        - Electrical activity is erratic, disorganized, and too rapid
        - Muscle in the wall of the atria cannot keep up with the storm of impulses = atria wall doesn’t eject, it quivers (fibrillates)
        - Loss of atrial contraction / atrial kick = less filling of blood in the ventricle, leading to less blood to stretch the ventricular wall, and leads to less blood being ejected
        - AV node overwhelmed, some impulses connect with the ventricle leading to irregular beating
    - ***Patients with afib have 5X increased risk for stroke***
- Common symptoms
    - May be unnoticed until a physical exam or routine EKG/ECG

    - 1st sign may be stroke caused by a clot from the atrium
    - S/S: fatigue, shortness of breath (SOB), rapid & irregular heartbeat
    - Sensation of “fluttering”, “pounding”, “extra beats” = palpitations
    - Symptoms of less effective heart function:
        - Chest pain or pressure
        - Dizziness, fainting, or feeling like you are about to faint
        - General weakness, tire easily
        - Swollen feet or ankles
- Types of atrial fibrillation
    - Paroxysmal (intermittent) afib
        - Episodes come and go
        - Stop without medical intervention within 7 days (most within 24 hr)
    - Persistent(continuous) afib
        - Lasts more than a week
        - Needs medical attention to stop (meds or cardioversion/shock)
    - Longstanding persistent
        - Lasts more than a year (treatment did not work)
    - Interventions focus on keeping a lower rate, not on converting the rhythm
    - The longer afib lasts, the risk of it getting worse increases; may cause damage to the heart itself
- Risk factors for Afib
    - Age: increases with age, 10% or more people over 80 have Afib
    - Gender: more men develop afib and tend to be diagnosed about a decade earlier; women tend to have worse symptoms and QOL as their afib is more likely to lead to stroke
    - Family history: impacts only a small minority of people
    - Alcohol: heavy consumption leads to damage of the heart muscle
    - Cardiac risk factors: hypertension, defective heart valves, coronary artery disease, other
    - COPD, chronic kidney disease, diabetes, obesity, sleep apnea, overactive thyroid
- Treatment: medications
    - Preventing Stroke
        - Anticoagulants: decreases risk of stroke in afib by 50 – 80%
        - Work by decreasing clot formation (not thinning the blood)
        - Increased risk of unwanted internal bleeding (1 extra event per 100 people at 1 year)

        - DOACs (direct oral anticoagulants): apixaban (Eliquis), dabigatran (Pradaxa), edoxaban (Savaysa), and rivaroxaban (Xarelto)
        - Less interference with other meds and foods
        - Directly inhibit the clotting process
        - 1st line drug of choice
        - Reversable
- Controlling heart rate
    - Rate control: slowing the rate to less than 110 to prevent permanent damage to heart muscle
    - Beta blockers: blocks receptor sites to make heart beat more slowly
    - Atenolol (Tenormin); bisoprolol, carvedilol (Coreg), metoprolol (Lowpressor, Toprol), nadolol (Corgard), propranolol (Inderal)
    - Calcium-channel blockers: slows conduction
        - Diltiazem (Cardizem, Cartia, Tiazac)
        - Verapamil (Verelan)
    - Digoxin (Lanoxin)from foxglove plant; doesn't work well with other meds
- Controlling rhythm: cardioversion
    - Electrical cardioversion
        - Electrical shock to the heart
        - Heart (SA node) is stunned, and no impulse is initiated
        - When heart "wakes up", normal SA pacemaker takes over to restore a regular, coordinated rhythm
        - Patient is deeply sedated
        - Works in 90% of patients
        - Not guaranteed to last, but can be repeated
    - Cardioversion with meds
        - After electrical cardioversion to maintain stable rhythm
        - Amiodarone (Nexterone)
        - Disopyramide (Norpace)
        - Dofetilide (Tikosyn)
        - Sotalol (Betapace)
        - Flecainde
- Procedural/surgical options for treating afib
    - Catheter ablation: minimally invasive in-hospital procedure
        - Uses heat (radiofrequency/RF) or cold (cryoablation) to destroy (ablate) tiny spots of tissues in the atrium to prevent abnormal signals before they cause Afib

        - New method: pulsed field ablation (PFA): destroys abnormal cells without damaging health ones
        - EARLY-AF trial: compared early ablation vs meds: ablation "substantially decreased the chance of afib symptoms returning; prevented hospitalization, and reduced mortality.
    - Maze procedure: surgical technique; makes a particular pattern of scar tissue on the surface of the heart using incisions or RF ablation; disrupts abnormal heart rhythm; usually done in combination with CABG; mini-maze or cryo-maze procedures are occasionally done
    - Left atrial appendage closure
        - Option for patients who cannot take anticoagulants
        - Left atrial appendage (LAA): pocket in the wall of the left atrium, and is often the main source of clots
        - Surgeon can sew it closed or remove it
    - New procedure with catheter to the heart:
        - New device (occluder) small, round plug inserted into the LAA and tissue grows around it, sealing it off to prevent clots
        - Brand name Amulet and Watchman
  - Financial Cost of afib
- Ventricular arrhythmias
  - Often life threatening.
  - <u>Ventricular tachycardia</u>: Involves a fast, regular heartbeat of more than 100 BPM. It may last for a few seconds or go on for a longer period of time.
    - Cause of most cases of sudden cardiac death in the United States, resulting in 300,000 deaths per year
    - More common in men
    - May deteriorate info ventricular fib
  - <u>Ventricular fibrillation:</u> ventricles quiver, but do not contract; non perfusing and lethal
  - Treatment for vtach & vfib
    - VTACH: goal to slow down the rapid heartbeat and prevent future episodes
        - Emergency treatment
            - CPR
            - Antiarrhythmic drugs (e.g., amiodarone, lidocaine, procainamide)
            - Electrical defibrillation

        - Meds: Calcium channel blockers and beta-blockers may be prescribed
        - Implantable Cardioverter-Defibrillator (ICD)
    - V fibrillation: goal to restore blood flow as quickly as possible to prevent brain and organ damage.
        - CPR
        - Defibrillation; may use AED, especially in public
    - Long term:
        - ICD
        - Catheter ablation
- Summary

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