BASIC DYSRHYTHMIA RECOGNITION

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OBJECTIVES
Following this lecture the learner will be able to:
• Describe the underlying principles of cardiac electrophysiology
• Accurately determine heart rate and rhythm from ECG strip
• Analyze ECG strips in a systematic manner
• Identify cardiac dysrhythmias
• Explain the nursing measures used in management of patients with cardiac dysrhythmias
• Evaluate appropriate medical management for dysrhythmias discussed
• Discuss the use of pacemakers in the treatment of cardiac dysrhythmias

CONDUCTION SYSTEM

EKG TRACINGS

ELECTROCARDIOGRAM
Records cardiac electrical currents (voltages, potentials) by means of skin electrodes placed on the surface of the body. Electrodes on the skin measures electrical activity as the current reaches the skin surface.

ELECTRICAL STIMULATION

WAVE FORMS

DEPOLARIZATION - Electrical stimulation of the cardiac muscle cells.

• POSITIVE
  - Electrical stimuli moving toward positive electrode

• NEGATIVE
  - Electrical stimuli moving away from a positive electrode

• BIPHASIC
  - Electrical activity located central between positive and negative electrode

• ISOELECTRIC
  - No electrical activity

REPOLARIZATION - The return of the cardiac muscle cells to their resting state after electrical stimulation
EKG LEADS USED FOR MONITORING

- Lead II
- MCL 1

EKG PAPER

- Paper speed - Usually 25mm/second.
- Strip markings - Usually in 1 or 3 second markings.
- Standardization - Usually 1mV = 10mm deflection
- Waveforms - P, QRS, T, ST Segment (sometimes)

EKG WAVEFORMS

- P wave - Represents
- PR interval - Represents the time it takes for the impulse to spread from the atria to the ventricles.
- QRS complex - Represents ventricle depolarization.
- T wave - Represents ventricle repolarization.
- ST segment - Indicates early ventricular repolarization. Area to look at if suspect AMI.

QRS COMPLEX

- Q wave is the negative wave preceding an R wave
- R wave is the first positive wave deflection
- S wave is a negative wave following an R wave

NORMAL TIME DURATIONS FOR WAVEFORMS

- PR interval < .20 seconds (.12-.20)
- QRS interval < .12 seconds (.08-.12)
TIME AND VOLTAGE

METHODS TO CALCULATE RATE

- **SMALL SQUARES**
  - Count the small squares between 2 consecutive “R” waves or “P” waves and divide by 1500 (1500 small squares in 1 minute)

- **LARGE SQUARES**
  - Count the large squares between 2 consecutive “R” waves or “P” waves and divide by 300 (300 large squares in 1 minute)

- **6 - SECOND STRIP**
  - Count the number of QRS complexes within a six second strip. (Use for irregular rhythms)

NATURAL PACEMAKERS

- **SA NODE (atrial)**
  - 60-100 bpm

- **AV NODE**
  - 40-60 bpm

- **VENTRICLES**
  - 30-40 bpm

STEPS IN ANALYSIS OF EKG WAVEFORMS

- RATE
- RHYTHM
- P waves
- PR Interval
- QRS Complex
- ST Segment

NORMAL SINUS RHYTHM

NSR represents a regular discharge from SA node.
No treatment is necessary
SINUS BRADYCARDIA

CAUSE: Decrease in the rate of discharge from the SA node that follows the normal conduction path. May be normal (athletes), as a result of medications (digoxin, antihypertensives, sedatives), or with ischemia.

TREATMENT: Asymptomatic – no treatment
Symptomatic - treat cause / atropine / pacemakers

SINUS TACHYCARDIA

CAUSE: Represents an increase in the rate of discharge from the SA node that follows the normal conduction path. May be secondary to multiple factors (exercise, fever, anxiety, hypovolemia, injury, pain, hypotension, etc.)

TREATMENT: Treat underlying cause if sustained

SINUS DYSRHYTHMIA

CAUSE: Characterized by an irregular rhythm that originates in SA node. Results from an ↑ in rate with inspiration and a ↓ in rate with expiration. Common in children usually reflects cardiac structural damage in adults.

TREATMENT: Asymptomatic – no treatment
Symptomatic bradycardia - atropine / pacemaker

PREMATURE CONTRACTIONS

• PREMATURE ATRIAL CONTRACTIONS (PAC’s)
• PREMATURE JUNCTIONAL CONTRACTIONS (PJC’s)
• PREMATURE VENTRICULAR CONTRACTIONS (PVC’s)

PAUSES

COMPENSATORY - Twice P to P interval

NON-COMPENSATORY - < or > twice P to P interval

PREMATURE ATRIAL CONTRACTIONS (PAC’s)

• Early, different looking upright P wave followed by a QRS resembling the QRS of the dominant rhythm.
• Non-compensatory pause follows PAC.
PREMATURE ATRIAL CONTRACTIONS (PAC’s)

CAUSE: Produced when a single irritable area in the atria discharges an impulse before the next regular SA node impulse is due. Can be caused by stimulants such as caffeine, tobacco, stress and fatigue.

TREATMENT: Asymptomatic - no treatment
Symptomatic - remove cause / chronic - medications

UNIFOCAL ATRIAL TACHYCARDIA

- 4-6 or more unifocal PAC's in a row with a regular P-P cycle.
- QRS resembles that of the regular cardiac rhythm.

MULTIFOCAL ATRIAL TACHYCARDIA

- 4-6 multifocal PAC's in a row with an irregular P-P cycle, varying PR intervals.
- Varying P wave configurations

ATRIAL TACHYCARDIA

Atrial tachycardia (AT) 180 bpm or >
Paroxysmal AT (PAT) 180 bpm or >
Supraventricular AT (SVT) 150-180 bpm
Paroxysmal SVT (PSVT) 150-180 bpm

CAUSE: Usually a re-entry phenomena at the AV node. SA node is replaced by an ectopic pacemaker in either atria which is firing very rapidly.

TREATMENT: Meds/ vagal maneuvers/ cardioversion

RE-ENTRY MECHANISM

RE-ENTRY MECHANISM - When the electrical conduction impulse recycles back up the conduction system initiating another impulse.

ATRIAL FLUTTER

- One ectopic focus in the atria fires repeatedly at a rate between 220-350 bpm.
- Flutter waves replace P waves.
- QRS resembles that of regular cardiac rhythm.
- Ventricular response can be 1:1, 2:1, 3:1, etc or can vary.
- TREATMENT: Medications and/or cardioversion
**ATRIAL FLUTTER**

2:1 conduction; Atrial rate 350 bpm / Ventricle rate 175 bpm

4:1 conduction; Atrial rate 280 bpm / Ventricle rate 70 bpm

**ATRIAL FIBRILLATION**

- Multifocal ectopic foci in the atria fire repeatedly at a rate between 350-650 bpm.
- Fibrillatory waves, either coarse or fine, replace P waves.
- Ventricular response is extremely irregular.

**ATRIAL FIBRILLATION**

- Controlled ventricular response Rate = 70 (approx.)
- Rapid ventricular response Rate = 110 (approx.)

**PREMATURE JUNCTIONAL CONTRACTIONS (PJ C’s)**

- Early QRS resembling QRS of dominant rhythm with no visible P wave, or an inverted P wave preceding or following QRS.
- Non-compensatory pause following the PJ C.

**PREMATURE JUNCTIONAL CONTRACTIONS (PJ C’s)**

- Isolated PJ C with non-compensatory pause
- PJ C in bigeminy

**JUNCTIONAL ESCAPE RHYTHM**

- 4-6 or more junctional escape beats in a row at a regular rate below 60 bpm.
- QRS resembles that of the normal cardiac rhythm
**Junctional Escape Rhythm**

Junctional Escape Rhythm with regular R-R intervals ruling out AF. Ventricular rate 40/ min

**CAUSE:** SA node fails to produce impulses properly, the AV node assume the pacemaking function. Suppression of the SA node due to vagal stimulation, cardiac medications, or heart disease are the most common cause of pacemaker failure.

**TREATMENT:** Remove cause / atropine / pacemaker

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**Atrioventricular Blocks**

Impairment of sinus impulse conduction through the AV node due to ischemia, drug effects, or disease of the AV node

- First-Degree AV block
- Second-Degree AV block, Mobitz I (Wenckebach)
- Second-Degree AV block, Mobitz II
- Third-Degree AV block

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**1st Degree AV Block**

NSR with 1st degree AV block - PR interval 0.32

**CAUSE:** Sinus impulse is temporarily delayed at the AV node before being conducted to the ventricles. Ischemia of the AV node secondary to AMI, digitalis, beta and calcium channel blockers can cause this block.

**TREATMENT:** No specific treatment necessary, monitor for progression to higher degree block.

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**2nd Degree AV Block Mobitz I - Wenckebach**

- Progressive prolongation of PR interval until an impulse fails to be conducted
- QRS resembles that of the regular cardiac rhythm

**CAUSE:** Basically same as with first-degree AV block

**TREATMENT:** Watch for higher block; stop or change medications; possible pacemaker and/or medications to speed heart rate.

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**2nd Degree AV Block Mobitz II**

- Conduction defect is below the level of the AV node in the bundle of His or possibly bundle branches.
- Sinus impulses are blocked at regular intervals, allowing only every 2nd, 3rd, or 4th impulse to conduct.
- PR interval is CONSTANT
- QRS conducted are normal or wide depending on area of defect.
**2nd Degree AV Block (Mobitz II)**

3:1 block - Constant PR interval conducted beats

**CAUSE:** Degenerative/structural changes in the conduction system. Acute blocks can be from AMI.

**TREATMENT:** CHRONIC - observe for progression and then possible pacemaker placement.

ACUTE - treated aggressively with pacemaker and hemodynamic medications if necessary

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**3rd Degree AV Block**

- All sinus impulses are blocked and none reach the ventricles.
- Conduction defect is below the AV node
- Atria and ventricles beat independently.

**CAUSE:** Usually secondary to AMI. Could be degenerative or structural

**TREATMENT:** Permanent pacemaker placement after stabilization

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**Bundle Branch Block**

Wide QRS - usually > 0.12

**CAUSE:** Obstruction occurs in one of the bundle branches causing delayed depolarization in one ventricle. Can be right or left bundle blockage.

Acute cause is AMI, chronic cause is degeneration or scarring along the bundle branch.

**TREATMENT:** Acute - pacemaker. Chronic - usually no treatment necessary

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**Premature Ventricular Contractions (PVC’s)**

- Early, wide and bizarre QRS complex
- QRS usually >0.12
- Opposite T wave deflection following PVC
- Compensatory pause
- No ectopic P wave

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**Unifocal PVC’s**

**Multifocal PVC’s**
PREMATURE VENTRICULAR CONTRACTIONS (PVC’s)

CAUSE: Most common dysrhythmia following AMI. Also associated with hypokalemia, CHF, caffeine, tobacco, alcohol, and other fluid and electrolyte imbalances.

TREATMENT: Treat underlying cause. Lidocaine is drug of choice in acute therapy.

DANGEROUS PVC’s

- FREQUENT
  - > 6/minute
- BIGEMINY
- MULTI FOCAL
- CONSECUTIVE
  - Pairs or couplets
- R on T PHENOMENA
  - PVC strikes on the T wave preceding

R on T PHENOMENON

“R on T” PHENOMENON - PVC’s that falls during the vulnerable repolarization period of the ventricles could precipitate a more chaotic rhythm such as ventricular tachycardia or ventricular fibrillation.

VENTRICULAR TACHYCARDIA

- 4-6 or more PVC’s in a row at a regular rate over 100 bpm
- No atrial activity is present and the QRS complexes are wide and bizarre (0.12 or >)
- May appear as sustained rhythm or short run
- Sustained rhythm leads to ↓ cardiac output

VENTRICULAR TACHYCARDIA

CAUSE: Usually the same as those with PVC’s

TREATMENT: STABLE – same as PVC’s
UNSTABLE – Cardioversion then Lidocaine

REFRACTORY PERIODS

ABSOLUTE REFRACTORY PERIOD: Impulse received will not be able to be conducted.
RELATIVE REFRACTORY PERIOD: Strong impulse can be recognized and conducted.
VENTRICULAR FIBRILLATION

• Chaotically firing of multifocal ventricular ectopic foci at a rate of 150-500 bpm
• No atrial activity or QRS complexes are present

VENTRICULAR FIBRILLATION

CAUSE: May be triggered by PVC or VT or may occur spontaneously without warning. Most common cause of death in patients with coronary heart disease. Death will occur rapidly if resuscitation is not initiated immediately.
TREATMENT: Prompt defibrillation and ACLS protocols

VENTRICULAR ASYSTOLE (CARDIAC STANDSTILL)

CAUSE: Massive coronary event or trauma leading to total absence of electrical activity
TREATMENT: Full code, confirm in more than one lead, transcutaneous pacing, Epinephrine, and Atropine

PULSELESS ELECTRICAL ACTIVITY (PEA)

CAUSE: Electrical activity without any cardiac function. Possible multiple causes. Rhythm may be NSR or any dysrhythmia, but NO pulse.
TREATMENT: Treat underlying cause if known. Full code and ACLS protocol (Epi and Atropine)