Reading assignment:
Aehlert: Vol. 1 pp 765 – 770
SOP: Bradycardia with a pulse
SOP: Narrow QRS Complex Tachycardia

OBJECTIVES:
Upon reading the text assignments, completion of the class and study questions, reviewing the SOPs, and working with their small group, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Describe the possible etiologies and clinical significance of the following rhythms:
   a) Sinus rhythm
   b) Sinus tachycardia
   c) Sinus bradycardia
   d) Sinus arrhythmia
   e) Sinus block/arrest

2. Identify on a 6-second strip the following rhythms:
   a) Sinus rhythm
   b) Sinus tachycardia
   c) Sinus bradycardia
   d) Sinus arrhythmia
   e) Sinus block/arrest

3. Systematically evaluate each rhythm for the following criteria:
   a) Rate,
   b) Rhythm: Regular/irregular,
   c) Presence/absence/morphology of P waves,
   d) R-R Interval, P-P Interval,
   e) P-QRS relationship, and
   f) QRS duration.
   g) Pacemaker origin

4. Correlate the cardiac rhythm with patient assessment findings to determine the emergency treatment for each rhythm according to NWC EMSS SOPs.

5. Discuss the classification, action, prehospital indications, contraindications, dose and route, and side effects of the following:
   a) Atropine
   b) Glucagon
   c) Dopamine

6. Describe the indications, critical steps, and monitoring priorities for transcutaneous pacing.
Definitions
A. Tachycardia: Rate greater than 100
B. Bradycardia: Rate < less than the minimum for that pacemaker site
C. Intrinsic firing rate: normal rate range for a particular pacemaker site
D. Isoelectric line: Baseline where no electrical activity is present. Measured during the T-P interval.
E. Accelerated rhythm: A rhythm that fires above its normal intrinsic rate, but < 100
F. Ectopic beat: A beat originating outside the normal pacemaker’s control
G. Pacemaker: The location responsible for originating the rhythm
H. Depolarization: Electrical firing of the cells of the heart (caused by ions crossing membrane)
I. Repolarization: The time during which the cells recharge (reset) after depolarization
J. Refractory period: The time during repolarization when cells rearm and may or may not be able to accept another stimulus (depolarize)
K. R – R Interval: The distance between the peaks (apex) of the ventricular depolarization waves (QRS) during each cardiac cycle. Measured for regularity or irregularity.
L. P – P Interval: The distance between the peaks of the atrial depolarization waves (P wave) during each cardiac cycle. Also measured for regularity or irregularity.
M. PR Interval: The distance between the beginning of atrial depolarization (P wave) to the beginning of ventricular depolarization (first deflection of QRS)

Rhythm interpretation tips
A. Use a systematic approach
B. Go through all the steps – no shortcuts!
C. Take your time!
D. Compare with the rules of the rhythms (characteristics list)
E. Interpret the dysrhythmia
F. PRACTICE – PRACTICE – PRACTICE

Normal sinus rhythm
A. Description: "Normal Rhythm"
B. Characteristics list
1. Rate: 60 - 100 per minute
2. Rhythm: Regular
3. P waves
   a. Normal and upright
   b. One to one relationship with each QRS complex
4. P-R interval: 0.12 - 0.20 seconds and constant
5. QRS complex: 0.04 - 0.10 seconds
C. Clinical significance: Assess the patient
D. Treatment: Based on clinical presentation
IV. **Sinus arrhythmia or dysrhythmia**

A. **Description**: SA node discharges impulses at an irregular rate. This causes a phasic or cyclical variation of R-R interval greater than 0.16 seconds.

B. **Etiology**
   1. Respiratory cycle related due to changes in intrathoracic pressure; normal phenomenon
   2. Non-respiratory influenced; normal phenomenon
   3. Enhanced vagal tone

C. **Characteristics list**
   1. **Rate**: Usually 60-100 per minute (varies)
      Respiratory etiology: rate gradually increases with inspiration, decreases with expiration – may become bradycardic at times.
   2. **Rhythm**: Regularly (cyclically) irregular
   3. **P waves** (pacemaker site SA node)
      a. Normal and upright
      b. One to one relationship with each QRS
   4. **P-R interval**: 0.12 - 0.20 seconds and constant
   5. **QRS complex**: 0.04 - 0.10 seconds

D. **Clinical significance**: Normal phenomenon particularly in very young, very old and very healthy

E. **Treatment**: IMC
V. Sinus bradycardia

A. Incidence
1. Common dysrhythmia occurring during the early phases of AMI, particularly frequent in patients with inferior and posterior infarction involving the right coronary artery (that supplies blood to the SA node).
2. 25%-40% of patients with ACS have ECG evidence of SB within the first hour of the onset of symptoms. This declines to 15%-20% four hours after infarction commences.

B. Etiology
1. Any condition that causes slowing of the SA node discharges. This can include increased parasympathetic (vagal) and decreased sympathetic NS tone: carotid sinus hypersensitivity syndrome, sleep apnea syndrome, severe hypothermia, hypothyroidism, and increased intracranial pressure. This is probably protective during AMI as it decreases O2 demand.
2. Activation of the Bezold-Jarisch reflex mediated by the vagus nerves and occurs during reperfusion, particularly of the RCA.
3. Vasovagal reaction causing vascular dilation is commonly seen with vomiting or pain or precipitated by sudden stress and may be intensified by severe pain or hypoxia.
4. Intrinsic sinus node disease
5. Drug effects: Beta blockers, calcium channel blockers, digitalis, quinidine
6. May be normal during sleep and in well-conditioned athletes

C. Characteristics
1. Rate: Less than 60 per minute (Usually between 40 & 60)
2. Rhythm: Regular
3. P waves
   a. Normal and upright
   b. One to one relationship with each QRS
4. P-R interval: 0.12 - 0.20 seconds and constant
5. QRS complex: 0.04 - 0.10 seconds

D. Clinical significance
1. May have none in a healthy athlete. Decreased HR may compromise cardiac output especially if less than 50 BPM.
   a. Hypotension; angina
   b. CNS symptoms: dizziness, lightheadedness, syncopal episode
2. This rhythm may precede more lethal rhythms or lead to atrial ectopic rhythms or beats or escape rhythms from the AV node or ventricles.

E. Treatment – See Bradycardia with a Pulse SOP
In all cases – treat the patient, NOT the monitor! Further ALS interventions are unnecessary unless symptomatic (hypotensive, HF, angina, syncope or AMS).
1. Assess for rate, rhythm, pump, or volume problem, hypoperfusion and cardiorespiratory compromise. Correctly identify the presence & type of AV block.
2. **Assess/treat for possible underlying causes:** Cardiac ischemia, OD, vasovagal episode, etc.

3. **IMC:** Support ABCs; determine need for invasive airway management. Anticipate need for pacing; do not delay TCP while attempting vascular access.

4. Obtain, review, and transmit 12-lead ECG. If possible ACS & alert with gag reflex and stable: Treat ischemia/pain per ACS SOP with ASA & fentanyl.

5. **Intervention: Transcutaneous Pacing (see lab manual)**
   a. Pacing reduces myocardial workload and O₂ demand and increases BP.
   b. **Electrodes:** Pace/defib electrodes are pre-gelled, self-adhesive electrodes that can be used for hands-free defibrillation, ECG monitoring, noninvasive pacing, and synchronized cardioversion.
      1. These electrodes are disposable. Do not reuse.
      2. One electrode set can be used for up to 50 shocks at any energy setting. They can withstand a continuous pacing current for 12 hours and can remain on the patient for 24 hours.
      3. **WARNING:** Do not use pads that are dried out or damaged as this may cause electrical arcing and patient skin burns during therapy. Do NOT use electrodes if they have been removed from the foil package for more than 24 hours. Do not use electrodes beyond the expiration date.
      4. Inspect electrodes to make sure adhesive is intact and undamaged.
      5. Avoid spilling any fluids on the adapters, cables, connectors, or electrodes.
      6. Do not clean the electrodes or their permanently attached electrode cable with alcohol.
   c. **Drugs:** atropine, glucagon, dopamine

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<th><strong>Profile: Atropine for pts w/ bradycardia and hypotension</strong></th>
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<tr>
<td><strong>Classification</strong></td>
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<td><strong>Action</strong></td>
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<td><strong>Therapeutic benefit:</strong></td>
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<td><strong>Indications</strong></td>
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<td><strong>Contraindications</strong></td>
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<tr>
<td><strong>Packaging</strong></td>
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<td><strong>Dose &amp; Route</strong></td>
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<td><strong>Side Effects</strong></td>
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### Profile: Atropine for pts w/ bradycardia and hypotension

<table>
<thead>
<tr>
<th>Precautions</th>
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<tr>
<td>Skin: warm, dry, flushed (red as a beet)</td>
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<td>Doses &lt; the minimum (0.5 mg) may be parasympathomimetic and actually slow the heart rate. Push as fast as possible</td>
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<td>Use with caution in suspected ACS as ↑ in HR may worsen ischemia or ↑ the area of infarction</td>
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<td>When given to pts with nonsymptomatic bradycardia may produce adverse effects</td>
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<tr>
<td>If atropine is given with other anticholinergic drugs, additive effects may occur</td>
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<td>Antacids slow the absorption of anticholinergic drugs</td>
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### Profile: glucagon (Glucagen) for bradycardia w/ hypotension

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<th>Action</th>
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<tr>
<td>Endogenous hormone synthesized by the alpha 2 cells of the islets of Langerhans in the pancreas. Action opposes insulin.</td>
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<tr>
<td>Positive inotropic and chronotropic effects on the heart by ↑ the production of adenylate cyclase which catalyzes the conversion of APT to cAMP and allows the cells to be stimulated in the absence of beta receptor activity. It also initiates a series of enzymatic reactions that promote the breakdown of glycogen to glucose which will raise the blood glucose levels. The degree to which glucagon ↑ blood glucose is dependent on liver glycogen reserves and presence of phosphorylases.</td>
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<tr>
<th>Onset of action</th>
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<td>Maximum activity occurs within 30 minutes</td>
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<tr>
<th>Indications</th>
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<td>Cardiac stimulant for patients with severe bradycardia who are taking ß blockers or Ca channel blockers and are unresponsive to pacing and atropine</td>
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<tr>
<th>Packaging</th>
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<td>Comes packaged as a powder to be mixed with diluent.</td>
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<th>Dose &amp; Route</th>
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<td>1 mg IVP/IN/IO/IM. May repeat every 1-min up to a total dose of 3 mg IVP/IO prn</td>
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<th>Side Effects</th>
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<td>Chest pain, palpitations</td>
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<td>Difficulty breathing</td>
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<tr>
<td>Unusual weakness</td>
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<tr>
<td>Rash; itching</td>
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<tr>
<td>Dizziness or lightheadedness</td>
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<tr>
<td>Muscle cramps</td>
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<td>Nausea/vomiting</td>
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<th>Contraindications</th>
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<td>Adrenal gland dysfunction, malnutrition, chronic hypoglycemia, pancreatic tumors, pheochromocytomas (adrenal gland tumors), liver disease, an unusual or allergic reaction to glucagon, beef or pork products or preservatives (old formulations).</td>
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VI. **Sinus tachycardia**

A. **Description**: Increase in rate of sinus node discharge

B. **Etiology**
   1. ↑ SNS tone: Excitement, exertion, exercise; caffeinated coffee, alcohol, smoking
   2. Fever, infections, septic shock, hypoxia, hypovolemia, hypotension, HF, MI
   3. Pain, anxiety
   4. Drugs that increase sympathetic tone (epi, dopamine, cocaine)
   5. Drugs that decrease the parasympathetic tone (atropine)
   6. Anemia; pump failure; hyperthyroidism

C. **Characteristics**
   1. Rate: 101-150 per minute (may go up to 180)
   2. Rhythm: Regular
   3. P waves (pacemaker site SA node)
      a. Normal and upright
      b. One to one relationship with each QRS
   4. P-R interval: 0.12 - 0.20 seconds
   5. QRS complex: 0.04 - 0.10 seconds (narrow or normal unless IVCD present)

D. **Clinical significance**
   1. May be benign or a compensatory mechanism for decreased stroke volume or increased metabolic demand by tissues.
   2. If rates are faster than 140-150, ventricular filling time may be decreased and myocardial O₂ consumption increased, so may precipitate S&S of chest pain, shortness of breath, and decreased cardiac output. This can produce ischemia or infarct in diseased hearts.

E. **Treatment - See SOP**
   1. Assess for physiologic stimulus (pain, fever, anemia, anxiety), hypoperfusion and cardiorespiratory compromise
   2. **IMC**: Support ABCs; determine need for invasive airway management
      - Identify rhythm; obtain, review and transmit 12-lead ECG
      - IV NS TKO in proximal vein (AC/external jugular)
      - If unconscious: defer vascular access until after cardioversion
   3. **Consider/treat for possible underlying causes**: cardiac ischemia, OD, vasovagal episode, etc.
      - Rate problem: Beating so fast CO is reduced or beating ineffectively so coordination between atria and ventricles reduces CO- use this SOP
      - Pump problem: HR > 100 & LV failure: - see HF/Pulmonary Edema/ Cardiogenic Shock
      - Volume problem: see Hypovolemic Shock
      - Metabolic problem: see Glucose Emergencies, Drug OD, & Renal
   4. If possible ACS & alert with gag reflex and stable: Treat ischemia/pain per ACS SOP with ASA & fentanyl
VII. Sinus arrest

A. **Description**: SA node fails to discharge for a period of time resulting in the absence of any ECG wave for one or more cardiac cycles. Electrical activity is resumed when either the SA node resets itself and resumes discharge or when a lower latent pacemaker begins to discharge producing escape complex or rhythm.

B. **Etiology**
1. Sinus node ischemia; hypoxia
2. Hyperkalemia
3. Excessive vagal tone
4. Drugs: digitalis (toxicity), beta blockers, calcium channel blockers
5. Degenerative fibrotic disease of the SA node

C. **Characteristics list**
1. Rate
   a. Normal to slow
   b. Depends on frequency and duration of sinus arrest
2. Rhythm: Irregular with pauses that may be followed by escape beats
3. P waves: Normal in basic rhythm, absent during pause. Escape beats are not always preceded by a P wave if originating from the AV node or ventricles.
4. P-R interval: 0.12 - 0.20 (in sinus beats) and constant; absent during pause
5. QRS complex
   a. Normal during regular rhythm.
   b. May be narrow or wide in an "escape beat" generated after the pause. Narrow if originating in the AV node and wide if originating in the ventricles.

D. **Clinical significance**
1. Frequent or prolonged pauses may compromise cardiac output by decreasing rate and producing hypotension, dizziness or syncope.
2. Danger of complete cessation of sinus node activity with no escape beats resulting in cardiac standstill.

E. **Treatment** – Per Bradycardia with a pulse SOP
1. Observe only if patient is asymptomatic
2. Pacing if bradycardic and hypotensive
Study questions - Dysrhythmias originating in the sinus node

1. List two causes of sinus bradycardia:
   ____________________________________________________________
   ____________________________________________________________

2. In what situations may sinus bradycardia be a person's normal rhythm?
   ____________________________________________________________

3. List the ECG characteristics of sinus bradycardia:
   Rate: _______________________________________________________
   Rhythm: ____________________________________________________
   Pacemaker site: ____________________________________________
   P waves present? __________________________________________
   PR interval: _______________________________________________
   QRS complexes present? _____________________________________
   QRS duration: _____________________________________________
   P/QRS ratio: ______________________________________________

4. What is the clinical significance of sinus bradycardia?
   ____________________________________________________________
   ____________________________________________________________

5. Do you need to provide drugs or pacing to every patient with sinus bradycardia? _____________
   Why or why not?

6. What signs or symptoms indicate the need for treatment?
   ____________________________________________________________
   ____________________________________________________________

7. Per the NWC EMSS SOPs, what is the preferred first treatment for patients experiencing bradycardia with a pulse who are lightheaded and hypotensive?
   A. Atropine
   B. Pacing

8. What is the classification of atropine? ________________________________________________

9. What is its intended action?
10. Which patient should receive atropine?
   A. AMI patient who is alert; with warm, dry skin; VS: BP = 120/90, P = 50, RR = 18.
   B. Chest pain patient w/ ↓ LOC; VS: BP = 84/50; P = 82; RR = 26.
   C. Pt. who is alert but weak w/ SOB. VS: BP = 118/80; P = 60; RR = 26.
   D. AMI patient who is lethargic & diaphoretic. VS: BP = 86/60; P = 50; RR = 18.

11. List 2 contraindications for giving atropine to a bradycardic patient with a pulse.

12. What is the dose of atropine for a bradycardic patient with a pulse?

13. What effect can atropine have on an evolving MI?

14. List three common side effects of atropine:

15. What is the maximum dose of atropine for a patient with a pulse who is bradycardic?

16. If a bradycardic patient is taking beta or calcium channel blockers, what drug should be given if atropine and pacing are unsuccessful in generating an acceptable cardiac output?

   What is the action of this drug for these patients?

   What is the dose and route for this drug in the bradycardic patient?

   How should this drug be mixed for administration?

17. An elderly female presents who is confused and feeling faint. She denies chest pain, nausea, or SOB. She denies any allergies or PMH and doesn't remember the name of her meds. Her skin is pale, cool, and moist. VS: BP 88/62; P 40 and irregular; lungs clear. The monitor shows sinus bradycardia with occasional PVCs. IMC has been completed. Which is indicated next?
   A. Transport without further treatment
   B. Atropine 1 mg IVP
   C. Transcutaneous pacing
   D. Amiodarone 150 mg slow IVP

18. Which ECG leads must be applied to a patient in order to pace them?
19. What monitor lead setting should you select when pacing?
   A. Leads I, II, or III
   B. aVR
   C. aVF
   D. Paddles

20. How do you know that the monitor is sensing the native R waves?

21. When performing TCP, at what heart rate should you start pacing?

   What is the maximum HR at which PMs are authorized to pace per SOP?

22. When pacing a patient, how do you know that the monitor is discharging current at the heart rate you have selected (What indication do you see on the ECG?)

23. How should electrical capture be confirmed on the ECG?

24. How should mechanical capture be confirmed?

25. At what mA is the pacemaker set when starting?

26. What is the upper limit of mA for transcutaneous pacing?

27. If there is no mechanical capture at the upper limits of mA, what should you do to troubleshoot the situation?

28. If mechanical capture is achieved and the patient is hemodynamically stable but not tolerating the procedure well, what drug should be given to induce amnesia and provide sedation?

29. If mechanical capture is achieved and the patient is now hemodynamically stable, but remains in intense pain due to ischemia or the pacing process, what drug should be given for pain?

30. What is a contraindication for giving that drug?
31. If pacing is unsuccessful and the pulse is present, though slow, and the patient is extremely hypotensive, what two drugs should be prepared for administration if not contraindicated?

________________________

________________________

32. List three causes of sinus tachycardia

________________________

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33. List the ECG characteristics of sinus tachycardia
   Rate: ______________________
   Rhythm: ______________________
   Pacemaker site: ______________________
   P waves present? ______________________
   PR interval: ______________________
   QRS complexes present? ______________________
   QRS duration: ______________________
   P/QRS ratio: ______________________

34. List the possible clinical significance of sinus tachycardia:

________________________

35. Which SOP gives orders for treating sinus tachycardia?

________________________

36. What is the goal when treating sinus tachycardia?

________________________

37. List one cause of sinus arrhythmia:

________________________

38. List the ECG characteristics of sinus arrhythmia
   Rate: ______________________
   Rhythm: ______________________
   Pacemaker site: ______________________
   P waves present? ______________________
   PR interval: ______________________
   QRS complexes present? ______________________
   QRS duration: ______________________
   P/QRS ratio: ______________________
39. Is there any clinical significance to sinus arrhythmia?

40. List two causes of sinus arrest or block:

41. List the ECG characteristics of sinus arrest:

   Rate:

   Rhythm:

   Pacemaker site:

   P waves present?

   PR interval:

   QRS complexes present?

   QRS duration:

   P/QRS ratio:

42. What clinical S&S may the patient exhibit with sinus arrest?

43. What would be the indicated treatment for a patient with clinically significant sinus arrest resulting in bradycardia and hypotension?