

**Northwest Community Healthcare
Paramedic Program**

SINUS RHYTHM AND DYSRHYTHMIAS

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Reading assignments:

SOP: Bradycardia with a pulse
Narrow QRS Complex Tachycardia with a pulse and HR > 100
Procedure Manual: Transcutaneous Pacing

Education standard:

Safely and effectively perform all psychomotor skills within the National EMS Scope of Practice Model AND state Scope of Practice at this level: • ECG interpretation

OBJECTIVES:

Upon completion of the reading assignments, class, and homework questions, reviewing the SOPs, and working with their small group, each participant will independently do the following with at least an 80% degree of accuracy and no critical errors:

1. Describe the possible etiologies and clinical significance of the following rhythms:
 - a) Sinus rhythm
 - b) Sinus tachycardia
 - c) Sinus bradycardia
 - d) Sinus dysrhythmia
 - 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 dysrhythmia
 - 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) Norepinephrine, dopamine
 - c) Glucagon
6. Describe the indications, critical steps, and monitoring priorities for transcutaneous pacing.

NCH Paramedic Program
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I. Definitions as a point of review

- 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 it's normal intrinsic rate, but less than 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)

II. Rhythm interpretation tips

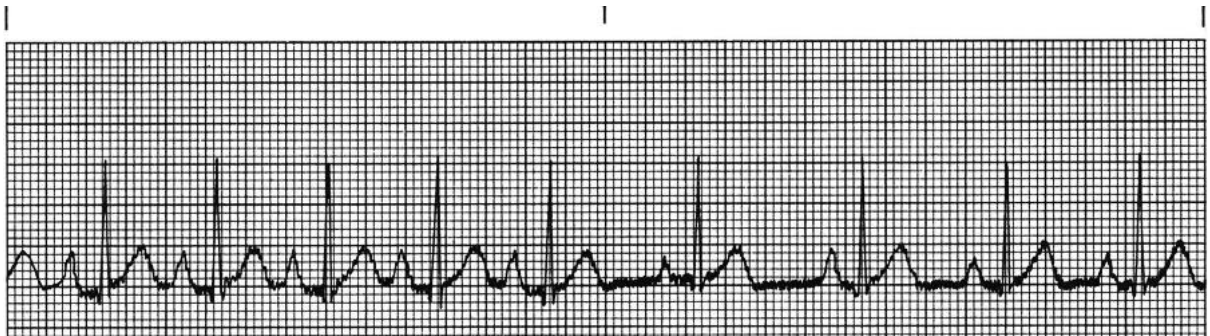
- A. Use a systematic approach - go through all the steps – no shortcuts!
- B. Compare with the rules of the rhythms (characteristics list)
- C. Interpret the dysrhythmia
- D. PRACTICE – PRACTICE - PRACTICE

III. Normal sinus rhythm



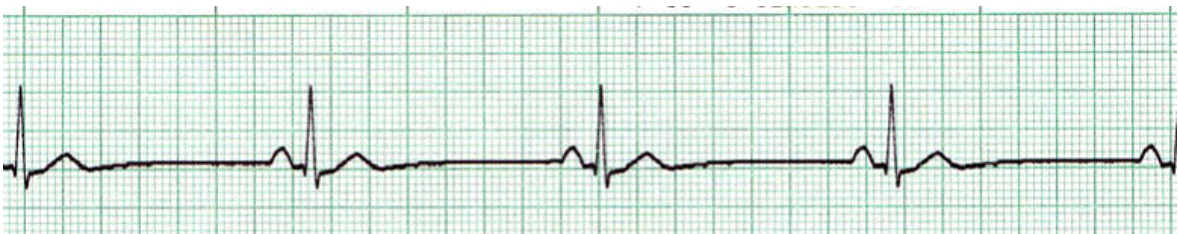
- A. Description: "Normal Rhythm"
- B. Characteristics list
 - 1. Rate: 60 - 100 per minute
 - 2. Rhythm: Regular
 - 3. P waves
 - a. Normal and upright
 - b. One to one relationship with each QRS complex
 - 4. P-R interval: 0.12 - 0.20 seconds and constant
 - 5. QRS complex: 0.04 - 0.10 seconds
- C. 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; especially in children
 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.

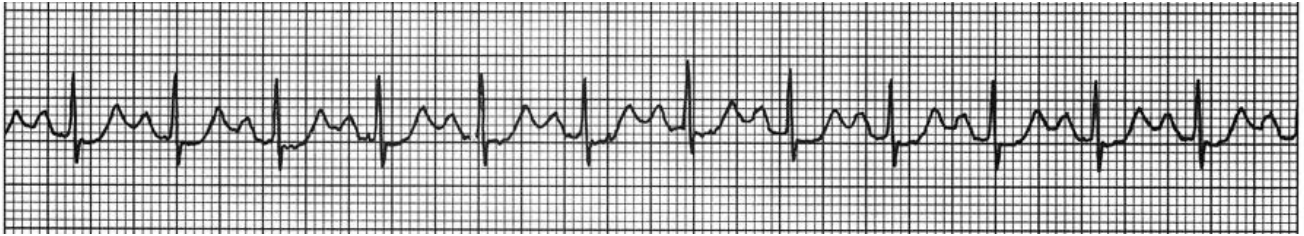
E. Treatment – See Bradycardia with a Pulse SOP

In all cases – **treat the patient, NOT the monitor!** Further ALS interventions are unnecessary unless patient is 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.
Correct rate problems first unless VT/VF.
2. **Assess/treat for possible underlying causes:** Hypoxemia, cardiac ischemia, OD, vasovagal episode, etc.
3. **IMC:** Support ABCs; determine need for advanced airway; O₂ if hypoxemic
Anticipate need for pacing; apply pads while attempting vascular access
4. Obtain, review, and transmit 12-lead ECG per ACS SOP (don't delay therapy)
5. If possible ACS & alert; + gag reflex; stable: Treat ischemia/pain per ACS SOP with **ASA & fentanyl**
6. **Lower acuity: NONE to MILD cardiorespiratory compromise:** Alert, oriented, well perfused, SBP ≥ 90 (MAP ≥ 65)
 - a. Ongoing assessment for hemodynamic and rhythm stability
 - b. Place transcutaneous pacing (TCP) electrodes in anticipation of clinical deterioration in pts w/ acute ischemia or MI associated with severe sinus bradycardia, asymptomatic 2° AVB Mobitz type 2, asymptomatic 3° AVB; or new onset Bundle Branch Block (BBB) or bifascicular block with AMI

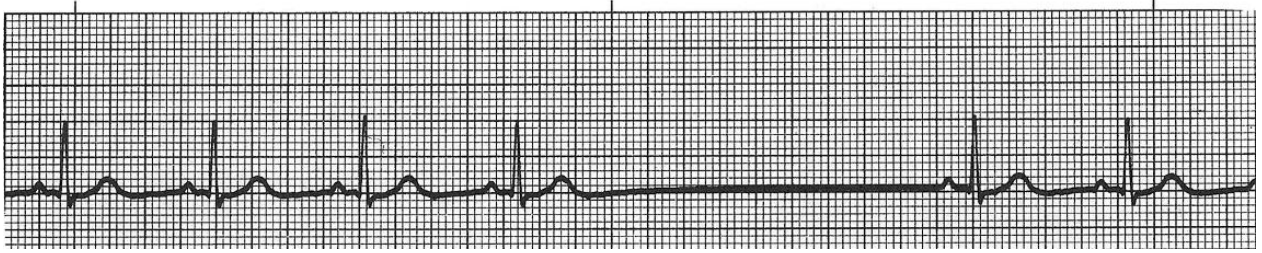
7. **Emergent to Critical: MODERATE to SEVERE cardiorespiratory compromise:** Instability related to slow rate: Acutely altered mental status, SBP < 90 (MAP <65), chest discomfort or pain, SOB, poor peripheral perfusion, weakness, fatigue, light headedness, dizziness and presyncope or syncope, pulmonary congestion, HF or pulmonary edema, escape beats, frequent PVC.
- a. **ATROPINE 0.5 mg rapid IVP/IO** unless contraindicated [AVB 2° Mobitz type 2 or 3° w/ wide QRS; transplanted hearts (lack vagal innervation) – See drug profile.
Repeat **ATROPINE 0.5 mg rapid IVP/IO** q. 3-5 min to a max of 3 mg IVP if bradycardia with ↓ BP persists.
- b. **If atropine ineffective or contraindicated**
NOREPINEPHRINE 8 mcg/min; maintain SBP ≥ 90 (MAP ≥65). See drug profile.
- c. **If atropine & norepinephrine ineffective or contraindicated or no vascular access:**
Transcutaneous external cardiac PACING (TCP) per procedure manual
- Select starting rate of 60 BPM. May adjust rate up to 70 BPM based on clinical response.
 - Increase mA until mechanical capture confirmed (palpable femoral pulse) or a maximum of 200 mA used. Evaluate BP once mechanical capture is achieved.
 - If mechanical capture present: CONTINUE PACING ENROUTE; do not turn off
 - **Assess need for sedation / analgesia:** : If SBP ≥ 90 (MAP ≥ 65):
If agitated: **MIDAZOLAM** as below .If condition deteriorating and critical, omit sedation.
MIDAZOLAM 2 mg increments slow IVP q. 2 min (0.2 mg/kg IN) up to 10 mg IVP/IN titrated to pt response. If IV unable and IN contraindicated: IM dose 5-10 mg (0.1-0.2 mg/kg) max 10 mg single dose. All routes: may repeat to total of 20 mg prn if SBP ≥ 90 (MAP ≥ 65) unless contraindicated. ↓ total dose to 0.1 mg/kg if elderly, debilitated, chronic diseases (HF/COPD); and/or on opiates or CNS depressants.
If pain: FENTANYL 1 mcg/kg (max single dose 100 mcg) IVP/IN/IM/IO. May repeat once in 5 min: 0.5 mcg/kg (max dose 50 mcg). Max dose per SOP: 150 mcg (1.5 mcg/kg)
Elderly (>65) or debilitated: 0.5 mcg/kg (max single dose 50 mcg) IVP/IN/IM/IO.
Additional doses require OLMC: 0.5 mcg/kg q. 5 min up to a total of 3 mcg/kg (300 mcg) if indicated & available.
- d. If on beta blockers & unresponsive to atropine, norepinephrine (dopamine), and pacing: **GLUCAGON** 1 mg IVP/IN/IO/IM.

VI. **Sinus tachycardia**



- A. **Description:** Increase in rate of sinus node discharge to HR > 100
- 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 HR 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 NARROW QRS Complex Tachycardia w/ pulse & HR > 100**
1. Assess for physiologic stimulus (pain, fever, anemia, anxiety), hypoperfusion and cardiorespiratory compromise
 2. **IMC:** Support ABCs; determine need for advanced airway management
 - a. Identify rhythm; obtain, review and transmit 12 L ECG
 - b. IV NS TKO in proximal vein (AC/external jugular)
 - c. If unconscious: defer vascular access until after cardioversion
 3. **Consider/treat for possible underlying causes:** cardiac ischemia, OD, compensation for other pathology etc.
 - a. **Rate problem:** Tachycardia w/ or w/o coordination between atria & ventricles is reducing CO - use this SOP
 - b. **Pump problem:** HR > 100 & LV failure: - see HF/Pulmonary Edema/Cardiogenic Shock
 - c. **Volume problem:** See Hypovolemic Shock
 - d. **Metabolic problem:** See Glucose Emergencies, Drug OD, & Renal emergencies
 4. If possible ACS & alert + gag reflex; stable: Treat ischemia/pain per ACS SOP with ASA & Fentanyl

VII. **Sinus block and/or 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. Atropine, dopamine, pacing if bradycardic and hypotensive

ATROPINE

Classification	Pharmacologic: Anticholinergic (Parasympatholytic)
Action	<ul style="list-style-type: none"> - Competes with the neurotransmitter acetylcholine for receptor sites, blocking the stimulation of parasympathetic nerve fibers. This blocking action enhances both sinus node automaticity and AV node conduction to indirectly increase HR (remove the brake to the heart). - ↓ GI motility - Dries secretions - Dilates bronchioles
Indications	<ul style="list-style-type: none"> - Symptomatic supraventricular bradycardia (Class I) unless contraindicated - AV blocks at the nodal level - 1° AVB or 2nd° Mobitz I (Class IIa; acceptable, probably helpful) - Cholinergic poisonings (organophosphates/WMD) - Neurogenic shock
Contraindications	<ul style="list-style-type: none"> - Known hypersensitivity - Infranodal AV block: 2° MII or 3° AVB with wide QRS complexes (Class III) - Unlikely to be effective in pts w/transplanted heart - Cushing's response in TBI - Avoid in hypothermic bradycardia
Packaging	<p>Preload for most uses</p> <p>DuoDote Auto-injector: antidote for cholinergic chemical weapons poisoning</p>
Dose & Route	<p>Symptomatic bradycardia: 0.5 mg rapid IVP. Repeat 0.5 mg rapid IVP/IO q. 3-5 min to max of 3 mg IVP if bradycardia & ↓ BP persists</p> <p>Cholinergic poisoning: 1 mg rapid IVP/IM. Repeat q. 3 minutes until reduction in secretions. May need large doses – usual dose limit does not apply. Cholinergic poisonings cause an accumulation of acetylcholine. Atropine blocks acetylcholine receptors, thus inhibiting parasympathetic stimulation. Also see Chemical Agents SOP.</p> <p>Peds: 0.02 mg/kg IV/IO Min. 0.1 mg</p> <p>Max doses</p> <p>Child single dose: 0.5 mg; total dose: 1 mg</p> <p>Adolescent single dose 1 mg; total dose 2 mg</p>
Side Effects	<ul style="list-style-type: none"> - CNS: Sensorium changes; drowsiness, confusion (mad as a hatter); headache - CV: tachycardia; rarely VT or VF; ↑ myocardial O₂ demand - Eyes: dilated pupils (not fixed), blurred vision - GI: dry mouth. (dry as a bone) - Skin: warm, dry, flushed (red as a beet) - Drying of secretions (mouth, nose, eyes, bronchioles)
Precautions	<ul style="list-style-type: none"> - Push as fast as possible. Slow administration (resulting in low dose) or dose < 0.1 - 0.5mg may cause paradoxical bradycardia d/t central effect. - Use with caution in suspected ACS/AMI as HR is a major determinant of O₂ demand; excessive tachycardia can worsen ischemia or ↑ the area of infarction - When given to pts with nonsymptomatic bradycardia may produce adverse effects - If atropine is given with other anticholinergic drugs, additive effects may occur - Antacids slow the absorption of anticholinergic drugs

GLUCAGON (Glucagen)

Classification	Hormone produced using rDNA technology
Action	<ul style="list-style-type: none"> - Action opposes insulin; it initiates a series of enzymatic reactions that promotes the breakdown of glycogen to glucose (glycogenolysis) which raises the blood glucose levels. The degree to which glucagon ↑ blood glucose is dependent on liver glycogen reserves and presence of phosphorylases. - Cardiac stimulant (+ inotrope) - causes release of catecholamines & stimulates c-AMP in cells to ↑ cardiac output (allows the cells to be stimulated in the absence of beta receptor activity). - Relaxes smooth muscle of stomach, duodenum, small intestine, & colon
Indications	<ul style="list-style-type: none"> - Symptomatic bradycardia w/ pulse if on β blockers & unresponsive to atropine, pacing, & dopamine - Hypoglycemia w/o IV/IO - Anaphylaxis if on beta blockers & not responding to epinephrine &/or dopamine - β blocker OD if HR < 60 & not responding to epinephrine & dopamine
Packaging	<p>Comes packaged as a powder to be mixed with diluent</p> <p>Glucagen brand: reconstitute by adding 1 mL sterile water for injection</p> <p>Lilly brand: Use only the 1 mL diluent to reconstitute; do not use diluent with other drugs</p> <p>When reconstituting: Roll (don't shake) vial</p>
Dose & Route	<p>Anaphylaxis/bradycardia on β blockers & refractory to usual Rx:</p> <p>Hypoglycemia:</p> <p>≥ 20 kg : 1 mg IVP/IN/IO/IM</p> <p>< 20 kg: 0.03 mg/kg IM/IN/IO up to 1 mg</p> <p>Onset IM: 5-20 min</p> <p>Peaks within 30 min</p> <p>Duration: 60-90 min</p>
Side Effects	<ul style="list-style-type: none"> - GI: Vomiting common (protect airway before glucagon administration) - ↑ HR - Dyspnea
Contraindications	<ul style="list-style-type: none"> - Hypersensitivity - Adrenal gland dysfunction (adrenal insufficiency); adrenal tumor (pheochromocytoma) - Malnutrition, chronic hypoglycemia, pancreatic tumors, liver disease, an unusual or allergic reaction to glucagon, beef or pork products or preservatives (old formulations).
Precautions:	<ul style="list-style-type: none"> - Not as effective in treating hypoglycemia if no glycogen stores: peds, starvation or malnourished states, uremic or those w/ liver disease - Give supplemental carbohydrate ASAP if used for hypoglycemia

Dysrhythmias originating in the sinus node

1. What are the possible underlying causes of bradycardia with a pulse?

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

3. List the following for this rhythm strip:



Rate: _____

R to R (reg or irreg): _____

Pacemaker site: _____

P waves present? _____

PR interval: _____

QRS complexes present? _____

QRS duration: _____

P/QRS ratio: _____

4. Are drugs or pacing indicated for every patient with sinus bradycardia? _____

Why or why not?

5. What signs or symptoms indicate the need for treatment?

6. What is the classification of atropine? _____

7. What is its intended action?

8. Which of these patients should receive atropine?
- A. Alert but weak w/ SOB; BP 118/80; P 60; R 20
 - B. Chest pain and altered mental status; BP 84/50; P 82; R 24
 - B. AMI who is alert; with warm, dry skin; BP 120/90, P 50, R 18
 - D. AMI presenting with lethargy & diaphoresis; BP 84/60; P 50; R 18
9. List 2 contraindications for giving atropine to a bradycardic patient with a pulse.
-
-
10. What is the initial dose of atropine to give to a patient who is bradycardic with a pulse?
-
11. What effect can atropine have on an evolving MI?
-
12. List three common side effects of atropine:
-
-
-
13. What is the maximum dose of atropine for a patient with a pulse who is bradycardic?
-
14. If a bradycardic patient is taking beta blockers and atropine, norepinephrine, and pacing are unsuccessful in generating an acceptable cardiac output, why is glucagon indicated? What is the action of this drug for these patients?
-
-
15. What is the dose and route of glucagon for a bradycardic patient with a pulse?
-
16. How should glucagon be mixed for administration?
-
-
17. An elderly female presents 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 of these is indicated next?
- A. Atropine 0.5 mg IVP
 - B. Transcutaneous pacing
 - C. Amiodarone 150 mg slow IVP
 - D. Transport without further treatment
18. If a pulse is present, but remains slow following atropine and the patient is extremely hypotensive, what drug should be prepared for administration?
-
19. What ECG leads must be applied to a patient in order to pace them? (See procedure manual)
-

20. What monitor lead setting should a paramedic select when pacing?
- A. Leads I, II, or III
 - B. aVR
 - C. aVF
 - D. Paddles
21. What is the confirmation on the monitor that it is sensing the native R waves?
-
22. What should be the initial heart rate setting for transcutaneous pacing?
-
- What is the maximum HR at which PMs are authorized to pace per SOP?
-
23. When pacing a patient, what observation confirms that the monitor is discharging current at the heart rate selected (What is seen on the ECG)?
-
24. How should electrical capture be confirmed on the ECG?
-
25. How should mechanical capture be confirmed?
-
26. At what mA is the pacemaker set when starting the Transcutaneous pacing procedure?
-
27. What is the upper limit of mA for transcutaneous pacing? _____
28. If there is no mechanical capture at the upper limits of mA, what should a paramedic do to troubleshoot the situation?
-
-
29. If mechanical capture is achieved and the patient is hemodynamically stable but not tolerating the procedure well, what drug (dose & route) should be given to induce amnesia and provide sedation?
-
30. If mechanical capture is achieved and a patient is now hemodynamically stable but in intense pain due to ischemia or the pacing process, what drug (dose & route) should be given for pain?
-
31. List two contraindications for giving that analgesic drug.
-
-
32. List three causes of sinus tachycardia
-
-
-

33. List the ECG characteristics of the following rhythm



Rate: _____

R-R reg or irregular: _____

Pacemaker site: _____

P waves present? _____

PR interval: _____

QRS complexes present? _____

QRS duration: _____

P/QRS ratio: _____

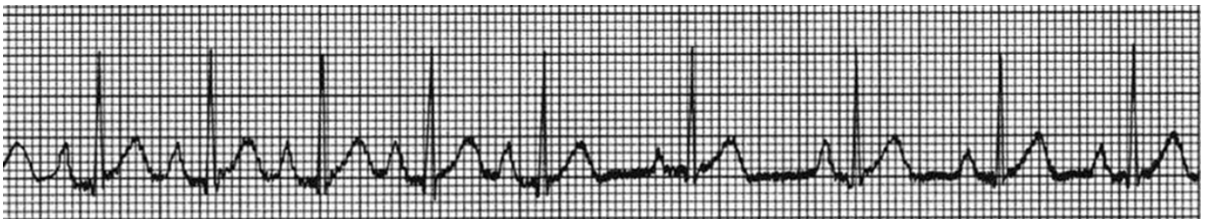
34. List the possible clinical significance of sinus tachycardia:

35. Which SOP gives guidelines for treating sinus tachycardia?

36. What is the goal when treating sinus tachycardia?

37. List one cause of sinus dysrhythmia:

38. List the ECG characteristics of the following rhythm



Rate: _____

Rhythm: _____

Pacemaker site: _____

P waves present? _____

PR interval: _____

QRS complexes present? _____

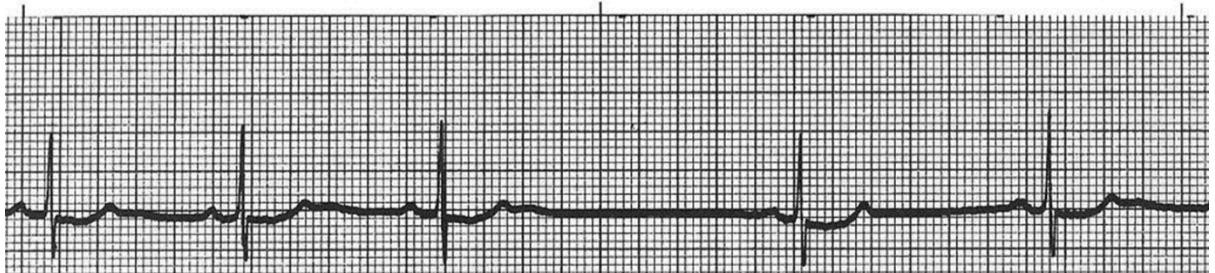
QRS duration: _____

P/QRS ratio: _____

39. Is there any clinical significance to sinus dysrhythmia most of the time? _____

40. List two causes of sinus arrest or block:

41. List the ECG characteristics of the following rhythm:



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 block or arrest?

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