ECG Interpretation Challenge Test Information

CPPN wants to provide you information regarding the new ECG Interpretation challenge option offered to experienced new hires. Two considerations must be met for an experienced hire to be eligible to take the ECG challenge test:

1. Nurse Manager must have approved this as an option they want available to their experienced new hires

2. The experienced new hire needs to self-identify their clinical competence for "dysrhythmia detection and treatment" as competent, proficient, or expert (per Benner's stages of clinical competence).
   - Competent: At least 2-3 years of experience and able to practice independently with minimal support.
   - Proficient: Performs frequently. Able to proactively identify potential issues related to skills, patient condition or equipment.
   - Expert: Practice is intuitive. Able to generate new knowledge related to skills, patient condition or equipment.

**Test Information:** If these two conditions are met the experienced new hire will be given proctored time (not to exceed 2-hours) during CPPN's portion of Nursing Orientation to complete a 60-questions multiple choice test. This will be a closed book/note/resource test with no remediation available. CPPN will provide calipers for the examination. The following packet provides information on the different EKG rhythms that you may have to interpret during the challenge test.

If the experienced new hire scores 84% or higher this will replace attending the 3-day ECG Interpretation course. If the experienced new hire scores less than 84% CPPN will assist them in signing up for the 3-day ECG Interpretation course # DAHS-NADEKGI17-ANCC.
## SINUS RHYTHM AND DYSRHYTHMIAS

### SINUS RHYTHM

- **Rate:** SA node firing at 60-100 bpm
- **Rhythm:** Regular
- **P-Waves:** Present
- **PR Interval:** Normal (0.12-0.20 secs)
- **P:QRS Ratio:** QRS is present after each P wave (1:1 ratio of P’s to QRS’s)
- **QRS Width:** Normal/Narrow <0.12
- **T-Waves:** Present

![Sinus Rhythm ECG](image)

### SINUS BRADY

- **Rate:** SA node firing at <60 bpm
- **Rhythm:** Regularly- Regular
- **P-Waves:** Present
- **PR Interval:** Normal
- **P:QRS Ratio:** 1:1
- **QRS Width:** Normal/Narrow
- **T-Waves:** Present
- **Etiology:** Ischemia/infarction of SA node, hypothermia, cardiac drugs, hypothyroidism, ↑ vagal tone, ↓ sympathetic tone
- **Clinical Significance:** Decreased Cardiac output (CO), predisposes to ectopic beats

![Sinus Brady ECG](image)

### SINUS TACHYCARDIA

- **Rate:** SA node firing at >100 bpm
- **Rhythm:** Regular to slightly irregular
- **P-Waves:** Present, normal unless merged with T-Wave
- **PR Interval:** Normal
- **P:QRS Ratio:** 1:1
- **QRS Width:** Normal/Narrow
- **T-Waves:** Present, may be merged with P-Wave
- **Etiology:** Compensatory response to physiologic stressor (pain, emotions, anxiety, blood loss, hypovolemia, early sepsis, heart failure, allergic reaction, fever, exercise), substances (smoking, alcohol, caffeine, drugs)
- **Clinical Significance:** ↑ oxygen consumption, ↑ workload on heart, ↓ CO if rate too fast for cardiac filling

![Sinus Tachycardia ECG](image)

### SINUS ARRHYTHMIA/DYSRHYTHMIA

- **Rate:** 60-100
- **Rhythm:** Slightly irregular, usually varies with respirations
- **P-Waves:** Present
- **PR Interval:** Normal
- **P:QRS Ratio:** 1:1
- **QRS Width:** Normal/Narrow
- **T-Waves:** Present
- **Etiology:** Common and normal finding in children and young adults, underlying heart disease, digitalis, exercise, mental stress, circadian rhythms
- **Clinical Significance:** Usually none, can make heart more susceptible to ventricular dysrhythmias

![Sinus Arrhythmia/Dysrhythmia ECG](image)
### SINUS ARREST/SINUS PAUSE

- **Rate:** Varies (due to the sinus arrest/pause)
- **Rhythm:** Irregular (due to break in rhythm)
- **P-Waves:** Present except for pauses
- **PR Interval:** Normal
- **P:QRS Ratio:** 1:1
- **QRS Width:** Normal/Narrow
- **T-Waves:** Present except for pauses
- **Etiology:** Hypoxemia, ischemia, diseased SA node, drugs (digoxin, beta blockers), cardiac disorders, vagal stimulation
- **Clinical Significance:** Depends on patient’s symptoms, if short and infrequent the patient may be symptomatic. If patient is symptomatic can have symptoms of ↓ CO

### SICK SINUS SYNDROME

- **Rate:** Varies (bradycardia to tachycardia)
- **Rhythm:** Irregular
- **P-Waves:** Varies, present but some may be junctional escape beats and won’t have a P-wave
- **PR Interval:** Varies with rhythm
- **P:QRS Ratio:** Varies
- **QRS Width:** Normal/Narrow
- **Etiology:** Coronary artery disease, fibrosis of SA node, Trauma to SA node, antidysrhythmic drug side effects (digoxin, beta blockers, calcium channel blockers)
- **Clinical Significance:** Loss of reliable pacemaker, ↑ workload on heart, ↓ CO

### ATRIAL DYSRHYTHMIAS

#### PREMATURE ATRIEL CONTRACTION (PAC)

- **Rate:** Beat occurs prematurely during underlying rhythm
- **P-Waves:** P-Wave for PAC is usually abnormally shaped or buried in preceding T-Wave
- **PR Interval:** Changes with the premature beats
- **QRS Width:** Normal/Narrow
- **Clinical Significance:** Depends on frequency, if more then 5-6/min then it is frequent and more significant, could ↓ CO. Predisposes to other dysrhythmias.

#### ATRIAL FLUTTER

- **Rate & Rhythm:** Depends on the AV conduction ratio- regular if consistent ratio (2:1, 3:1, 4:1), irregular if variable ratio
- **P-Waves:** None, rather there are flutter F-Waves at a rate of 250-350 bpm, “sawtooth”
- **PR Interval:** None
- **QRS Width:** Normal/Narrow
- **T-Waves:** Not able to distinguish d/t F-Waves
- **Etiology:** Enlarged atrial tissue, elevated atrial pressures, digoxin toxicity, pulmonary embolism, ETOH abuse, following open heart surgery
- **Clinical Significance:** Abnormal movement of electrical depolarization in reentry circuit, ↓ CO, blood stasis=increased incidence of clotting, ↑ myocardial oxygen consumption
| **ATRIAL FIBRILLATION** | Rate: Depends on AV conduction. Controlled <100, Uncontrolled/RVR >100  
Rhythm: Irregularly irregular  
P-Waves: None, rather there are fibrillatory f-Waves at a rate of 400-600 bpm  
PR Interval: None  
QRS Width: Normal/Narrow  
T-Waves: Not able to distinguish d/t f-Waves  
Etiology: Acute MI, digitalis toxicity, long standing hypertension, left atrial enlargement  
Clinical Significance: Loss of atrial kick, ↓ filling time, ↓ CO |
| **SUPRAVENTRICULAR TACHYCARDIA (SVT)** | Rate: 151-250  
Rhythm: Regular  
P-Waves: usually buried in QRS or T-Wave  
PR Interval: Not measurable  
QRS Width: Normal/Narrow unless distorted by buried P-Waves  
T-Waves: Present  
Etiology: Structurally related, heart failure, thyroid disease, excess of stimulants, pregnancy, surgery, chronic lung disease  
Clinical Significance: usually benign and self-limiting when the cause is removed, ↓ filling time, ↓ CO |
| **WANDERING ATRIAL PACEMAKER** | Rate: <100  
Rhythm: Irregularly irregular (sites of impulses vary)  
P-Waves: Different shapes (minimum of 3)  
PR Interval: Varies  
P:QRS Ratio: 1:1 if antegrade  
QRS Width: Normal/Narrow  
T-Waves: Present  
Etiology: ↑ vagal tone, digoxin toxicity, heart disease, COPD, Respiratory failure, atrial enlargement, electrolyte abnormalities, stimulants  
Clinical Significance: Can be normal in young pts and common in athletes, no clinical significance if patients are asymptomatic, correct underlying condition |
| **JUNCTIONAL DYSRHYTHMIAS** | Rate: Underlying rhythm  
Rhythm: Regular except the PJC is a premature beat  
P-Waves: 3 different p presentations possible 1) antegrade: p before QRS, 2) retrograde: p after QRS, and 3) p wave buried in QRS. Can be positive or negative deflection (inverted).  
PR Interval: <.12 if antegrade, none if retrograde or buried in QRS  
P:QRS Ratio: 1:1 if antegrade  
QRS Width: Normal/narrow unless distorted by p wave  
T-Waves: Present  
Etiology: Digoxin toxicity, excessive caffeine intake, inferior MI, rheumatic heart disease, valvular heart disease, hypoxia, swelling of AV junction  
Clinical Significance: depends on frequency and if patient is symptomatic, ↓ CO, may have a pause after a PJC |
### Junctional Rhythm/Junctional Escape Rhythm

**Rate:** 40-60 bpm  
**Rhythm:** Regular  
**P-Waves:** 3 different p presentations possible 1) antegrade: p before QRS, 2) retrograde: p after QRS, and 3) p wave buried in QRS. Can be positive or negative deflection (inverted).  
**PR Interval:** <.12 if antegrade, none if retrograde or buried in QRS  
**P:QRS Ratio:** 1:1 if antegrade  
**QRS Width:** Normal/narrow unless distorted by p wave  
**T-Waves:** Present  
**Etiology:** Sick sinus syndromes, vagal stimulation, electrolyte imbalances, digoxin toxicity, inferior wall MI, rheumatic heart disease  
**Clinical Significance:** ↓ HR and ↓ CO

### Accelerated Junctional Rhythm

**Rate:** 60-100 bpm  
**Rhythm:** Regular  
**P-Waves:** 3 different p presentations possible 1) antegrade: p before QRS, 2) retrograde: p after QRS, and 3) p wave buried in QRS. Can be positive or negative deflection (inverted).  
**PR Interval:** <.12 if antegrade, none if retrograde or buried in QRS  
**P:QRS Ratio:** 1:1 if antegrade  
**QRS Width:** Normal/narrow unless distorted by p wave  
**T-Waves:** Present  
**Etiology:** Digoxin toxicity, hypokalemia, hypercalcemia, inferior and posterior MI, rheumatic heart disease, valvular heart disease  
**Clinical Significance:** loss of atrial kick and ↓ filling time if retrograde, ↓ CO

### Junctional Tachycardia

**Rate:** 100-130 bpm  
**Rhythm:** Regular  
**P-Waves:** 3 different p presentations possible 1) antegrade: p before QRS, 2) retrograde: p after QRS, and 3) p wave buried in QRS. Can be positive or negative deflection (inverted).  
**PR Interval:** <.12 if antegrade, none if retrograde or buried in QRS  
**P:QRS Ratio:** 1:1 if antegrade  
**QRS Width:** Normal/narrow unless distorted by p wave  
**T-Waves:** Present  
**Etiology:** inferior or posterior MI, SA node disease, hypoxia, ischemia, digoxin toxicity, ↑ vagal tone  
**Clinical Significance:** loss of atrial kick and ↓ filling, ↓ CO, predisposes to other dysrhythmias, less dependable pacemaker, predisposes to heart failure
# CARDIAC CONDUCTION BLOCKS

## FIRST DEGREE HEART BLOCK

- **Rate**: Underlying rhythm rate
- **Rhythm**: Regular
- **P-Waves**: Present
- **PR Interval**: Prolonged >.20*
- **P:QRS Ratio**: 1:1
- **QRS Width**: Normal/narrow
- **T-Waves**: Present
- **Etiology**: Ischemia, myocardial infarction, rheumatic heart disease, CAD, cardiac medications (digoxin, beta blockers, calcium channel blockers)
- **Clinical Significance**: Not dangerous by itself, typically no symptoms, it can progress to something worse.

## SECOND DEGREE AV BLOCK TYPE I (MOBITZ I, WENCKEBACH)

- **Rate**: Ventricular rate is less then atrial rate d/t dropped QRS complexes
- **Rhythm**: Atrial regular, ventricular irregular d/t non-conducted beat
- **P-Waves**: Present
- **PR Interval**: Varies, *progressively gets longer* before* dropping a beat
- **P:QRS Ratio**: Variable, P>QRS
- **QRS Width**: Normal/narrow
- **Etiology**: Ischemia, MI, CAD, drug toxicity such as digoxin, rheumatic fever, myocarditis, transient after heart surgery, cardiac medications (beta blockers, calcium channel blockers)
- **Clinical Significance**: Depends on the frequency of dropped QRS complexes, ↓ CO, predisposes to progression to more serious block

## SECOND DEGREE AV BLOCK TYPE II (MOBITZ II)

- **Rate**: Ventricular rate is less then atrial rate d/t dropped QRS complexes
- **Rhythm**: Atrial regular, ventricular regular or irregular depending on number of blocks
- **P-Waves**: Present
- **PR Interval**: Normal and consistent
- **P:QRS Ratio**: Ratio of conducted beats to dropped beats. P>QRS
- **QRS Width**: Normal/narrow
- **Etiology**: Septal wall necrosis, anterior septal MI, myocarditis, CAD, drug toxicity, cardiomyopathy
- **Clinical Significance**: Depends on the frequency of dropped QRS complexes, ↓ CO, predisposes to progression to more serious block, unpredictable rhythm
### Third Degree AV Block (Complete Heart Block)

- **Rate:** Atrial rate usually 60-100 and ventricular rate 40-60 if AV node, 20-40 if Purkinje fibers as pacemaker cells
- **Rhythm:** Regular
- **P-Waves:** Present
- **PR Interval:** Consistent, if marched out can possibly find hidden p waves in T-wave and QRS complexes
- **P:QRS Ratio:** No correlation between p-wave and QRS, more P waves then QRS
- **QRS Width:** Narrow or Wide
- **Etiology:** MI, digoxin toxicity, acute myocarditis, degenerative changes in the heart, calcium channel blockers, beta-adrenergic blockers, cardiac surgery
- **Clinical Significance:** Slow ventricular rate, ↓ CO, unreliable pacemaker, predisposes to lethal dysrhythmias, severity of symptoms depends on ventricular rate

### Bundle Branch Block

- **Rate:** Abnormal conduction in ventricle, can be right or left side of the pathway.
- **QRS Width:** Wide, ≥ 0.12
- **Right BBB:** V1-Rabbit ear appearance with RSR’ and inverted T-Wave; V6-widened S-Wave and upright T-wave
- **Left BBB:** V1- Wide S-Wave and positive T-Wave; V6-notch at top of QRS, inverted T-Wave
- **Etiology:** Right—congenital, anterior MI, CAD, PE. Left—hypertension, aortic stenosis, heart disease, CAD
- **Clinical Significance:** LBBB more serious than RBBB, examine V1 to determine if right or left, need 12 lead to diagnose

### Ventricular Dysrhythmias

#### PVC

- **Rate and Rhythm:** Underlying
- **P-Waves:** None with the PVC
- **PR Interval:** None with the PVC
- **QRS Width:** Early, wide, ≥ 0.12 and larger than normal QRS
- **T-Waves:** Usually in the opposite direction than the QRS of the PVC, wide and bizarre
- **Etiology:** Electrical irritability, hypoxia, MI, ischemia, electrolyte imbalance, acid base imbalance, stimulants, drugs, stress and anxiety
- **Clinical Significance:** Depends on frequency, can lead to more serious dysrhythmias, ↓ CO, PVCs do not perfuse adequately, can cause R-on-T phenomenon
- **Terminology:**
  - Bigeminy: PVC every other beat
  - Trigeminy: PVC every three beats
  - Couplet: 2 PVCs in a row
  - Unifocal: coming from one focus, same shape and size
  - Multifocal: coming from more than one irritable foci, different shapes and sizes
## VENTRICULAR TACHYCARDIA

- **Rate:** 100-250 bpm
- **Rhythm:** Regular
- **P-Waves:** Absent
- **PR Interval:** None
- **P:QRS Ratio:** Absent
- **QRS Width:** Wide and bizarre
- **Etiology:** Myocardial ischemia, MI, CAD, valvular heart disease, heart failure, cardiomyopathy, electrolyte imbalances, drug intoxication, stress, anxiety
- **Clinical Significance:** Very unpredictable and dangerous. Unreliable pacemaker. ↓ ventricular filling time, ↓ CO, quickly deteriorate to V Fib and complete cardiac failure. There are 3 presentations—
  - Stable VT with pulse
  - Unstable VT with pulse
  - VT without a pulse

## TORSADES DE POINTES

- A variant of V Tach
- **Rate:** 150-250
- **Rhythm:** Irregular
- **P-Waves:** Absent
- **PR Interval:** None
- **P:QRS Ratio:** Absent
- **QRS Width:** Wide with *changing amplitudes*
- **Etiology:** Prolonged QT from drugs, myocardial ischemia, electrolyte abnormalities—hypokalemia and hypomagnesemia
- **Clinical Significance:**危险rhythm that can lead to ventricular standstill

## VENTRICULAR FIBRILLATION

- **Rate:** Absent
- **Rhythm:** Irregular
- **P-Waves:** Absent
- **PR Interval:** None
- **P:QRS Ratio:** Absent
- **QRS Width:** None
- **Etiology:** Myocardial ischemia, MI, heart block, untreated V tach, acid base imbalance, electrolyte imbalance, drug toxicity, severe hypoxia, terminal event in many disease states
- **Clinical Significance:** Ventricles are quivering instead of contracting, cardiac output is nonexistent, pacemaker cells are still working, leads to ventricular standstill and cardiac death

## PULSELESS ELECTRICAL ACTIVITY

Any organized rhythm will be present on the monitor but the patient will not have a pulse (with exclusion of pulseless Ventricular Tachycardia)

- **Rate, Rhythm, P-Waves, PR Interval, P:QRS Ratio, QRS Width, T-Waves:** All the underlying rhythm
- **Etiology:** Look at the H’s and T’s: H—hypovolemia, hypoxia, hydrogen ions, hyperkalemia, hypokalemia, hypothermia. T—Tablets, toxins, tension pneumothorax, thrombosis, thromboembolism, trauma
- **Clinical Significance:** Heart muscles loses its ability to contract even though electrical activity is preserved. No heart contraction, no blood flow, no heart rate, no cardiac output
### IDIOVENTRICULAR RHYTHM

- **Rate:** 20-40 bpm, if 40-100 then accelerated idioventricular rhythm, >100 V tach
- **Rhythm:** Regular
- **P-Waves:** Absent
- **PR Interval:** None
- **P:QRS Ratio:** None
- **QRS Width:** Wide ≥ .12
- **T-Waves:** Deflected opposite of QRS
- **Etiology:** Digoxin toxicity, heart disease, pacemaker failure, metabolic imbalance, common following reperfusion therapy
- **Clinical Significance:** Slow ventricular rate and loss of atrial kick reduce CO, progress to more lethal dysrhythmia. If just one idioventricular beat is generated it is called a ventricular escape beat

### ASYSTOLE

- **Rate:** None
- **Rhythm:** None
- **P-Waves:** None
- **PR Interval:** None
- **P:QRS Ratio:** None
- **QRS Width:** None
- **T-Waves:** None
- **Etiology:** MI, severe electrolyte disturbance, massive PE, prolonged hypoxemia, severe uncorrected acid-base imbalance, electric shock, drug overdose, cardiac tamponade, hypothermia
- **Clinical Significance:** Atrial and ventricular activity is at a standstill, terminal rhythm, critical to determine if rhythm is true asystole or a pause, need to look in a minimum of 2 leads

### PACEMAKER RHYTHMS

#### ATRIAL PACED

- **Undersensing:** Pacemaker has failed to sense that the heart has initiated an intrinsic beat (Pacing when it shouldn’t)
- **Oversensing:** Electrical activity is registered as cardiac activity that is not coming intrinsically and is counted as a cardiac beat (Not pacing when it should)
- **Capture:** When the pacemaker delivers an electrical impulse strong enough to result in depolarization
- **Failure to capture:** When the pacemaker delivers an electrical impulse that does not result in depolarization
- **Failure to pace:** No electrical impulse is delivered, complete failure to sense the need or deliver the impulse to initiate pacing

#### VENTRICULAR PACED

#### DUAL CHAMBER PACED