CPPN provides experienced new hires an opportunity to complete the ECG Interpretation Challenge Exam during Nursing Orientation. Two considerations must be met to be eligible to participate in the ECG Challenge Exam:

1. Nurse Manager has approved the ECG Challenge Exam as an opportunity for new hires in their department.
2. The experienced new hire self-identifies 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 provided proctored time (not to exceed 2-hours) during CPPN's portion of Nursing Orientation to complete a 60-question multiple choice exam. This is a closed book/note/resource test with no remediation available.

A score of 83% or higher will satisfy attending the ECG Interpretation course and fulfill the “Basic Dysrhythmia Detection and Treatment” skill checklist. If a score less than 83% is received, CPPN will provide information on how to register for the ECG Interpretation course if required by the new hires department.

The following packet provides additional exam information for participants.
**EXAM FORMAT**

The format of the ECG Interpretation Challenge Exam consists of fill in the blank, multiple choice and select all that apply questions. CPPN will provide calipers for the examination. The exam will cover a variety of concepts, including but not limited to:

- Interval measurements: e.g. PRI, QRS, QT interval
- Atrial and ventricular rhythm regularity and rate
- Nursing considerations: e.g. assessment, interventions, treatment
- Rhythm interpretation

**SINUS RHYTHM AND DYSRHYTHMIAS**

**SINUS RHYTHM/NORMAL SINUS RHYTHM**

- Rate: SA node firing at 60-100 bpm
- Rhythm: Regular
- P-Waves: Present, similar morphology
- PR Interval: Normal (0.12-0.20 secs), consistent
- P:QRS Ratio: QRS is present after each P-wave (1:1 ratio of P’s to QRS’s)
- QRS Width: Narrow (≤.11) or Wide (≥.12)
- T-Waves: Present

**SINUS BRADYCARDIA**

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

**SINUS TACHYCARDIA**

- Rate: SA node firing at >100 bpm
- Rhythm: Regular
- P-Waves: Present, normal unless merged with T-Wave, similar morphology
- PR Interval: Normal, consistent
- P:QRS Ratio: 1:1
- QRS Width: Narrow or Wide
- 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 ARRHYTHMIA/DYSRHYTHMIA**

- Rate: Usually around 60-100 but can be slow or fast
- Rhythm: Slightly irregular, usually varies with respiration (inspiration/expiration)
- P-Waves: Present, similar morphology
- PR Interval: Normal, consistent
- P:QRS Ratio: 1:1
- QRS Width: Narrow or Wide
- 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 ARREST/SINUS PAUSE**

- Rate: Single or multiple pauses can occur
- Rhythm: Irregular (due to break in rhythm)
- P-Waves: Delayed or non-conducted (after a pause there may or may not be a P-wave)
- PR Interval: Normal except during the pause event
- P:QRS Ratio: Usually 1:1 except during the pause event
- QRS Width: Narrow or Wide
- T-Waves: Present
- 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. Pause may be followed by a Junctional Escape Beat (JEB) to prevent cardiac standstill.
### SICK SINUS SYNDROME
- **Rate:** Variable (bradycardia to tachycardia)
- **Rhythm:** Irregular
- **P-Waves:** Variable, can be delayed or non-conducted
- **PR Interval:** Variable
- **P:QRS Ratio:** Variable
- **QRS Width:** Narrow or Wide
- **Etiology:** Coronary artery disease, fibrosis of SA node, Trauma to SA node, antidyssrhythmic drug side effects (digoxin, beta blockers, calcium channel blockers)
- **Clinical Significance:** Loss of reliable pacemaker, ↑ workload on heart, ↓ CO

### ATRIAL DYSRHYTHMIAS

#### PREMATURE ATRIAL CONTRACTION (PAC)
- **Rate:** PAC is a single complex. May occur in patterns: pairs, every other beat or every three beats.
- **Regularity:** Ectopic beat makes it irregular
- **P-Waves:** P-Wave for PAC is usually abnormally shaped or buried in preceding T-Wave, different morphology than underlying P waves
- **PR Interval:** Changes with the premature beat(s), PRI with be ≥.12
- **QRS Width:** Narrow
- **Etiology:** Stimulants, stress, anxiety, electrolyte imbalances, hypoxia, drug toxicity, coronary heart disease, fatigue, fever
- **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:** Usually Atrial: 200-400 bpm, Ventricular: 100-300. Rate can be slower, depends on the AV conduction ratio.
- **Rhythm:** Variable presentation. Regular if consistent ratio of F-waves:QRS (2:1, 3:1, 4:1), irregular if variable ratio
- **P-Waves:** Absent, rather there are flutter “F-Waves” with a “sawtooth” appearance, F-waves are consistent
- **PR Interval:** None
- **QRS Width:** Narrow or Wide
- **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, loss of atrial kick, ↓ filling time, ↓ CO, blood stasis=increased incidence of clotting (clot dislodgment can cause pulmonary embolism or stroke), ↑ myocardial oxygen consumption

#### ATRIAL FIBRILLATION
- **Rate:** Usually Atrial: 400-700 bpm, Ventricular: Variable. Depends on AV conduction. Controlled if HR<100, Uncontrolled/Rapid Ventricular Response (RVR) HR >100
- **Rhythm:** Irregularly irregular
- **P-Waves:** Absent, rather there are fibrillatory “f-Waves”
- **PR Interval:** None
- **QRS Width:** Narrow
- **T-Waves:** Can be difficult 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, blood stasis=increased incidence of clotting (clot dislodgment can cause pulmonary embolism or stroke)

#### SUPRAVENTRICULAR TACHYCARDIA (SVT)
- **Rate:** 140-250
- **Rhythm:** Regular
- **P-Waves:** usually buried in QRS or T-Wave
- **PR Interval:** Not measurable
- **QRS Width:** Narrow unless distorted by buried P-Waves
- **T-Waves:** Present, can be merged with P-waves
- **Etiology:** Structurally related, heart failure, thyroid disease, excess of stimulants, pregnancy, surgery, chronic lung disease. SVT is a broad term covering many forms of arrhythmias that originate above the ventricles in the atria or AV node (supraventricular = above the ventricles)
- **Clinical Significance:** usually benign and self-limiting when the cause is removed, ↓ filling time, ↓ CO
### Wandering Atrial Pacemaker
- **Rate**: <100 (considered multifocal atrial tachycardia (MAT) if >100)
- **Rhythm**: Irregularly irregular (sites of impulses vary)
- **P-Waves**: Present with variable morphologies. Minimum of 3 different morphologies required.
- **PR Interval**: Variable
- **P:QRS Ratio**: 1:1
- **QRS Width**: Narrow or Wide
- **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

#### PJC
- **Rate**: PJC is a single complex. May occur in patterns: pairs, every other beat or every three beats.
- **Rhythm**: The ectopic beat makes it irregular
- **P-Waves**: Variable for the ectopic beat. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted).
- **PR Interval**: Variable for ectopic beat, <0.12 if P-wave present before QRS
- **P:QRS Ratio**: Variable for ectopic beat, 1:1 if P-wave before QRS
- **QRS Width**: 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, if more then 5-6/min then it is frequent and more significant, could ↓ CO. Predisposes to other dysrhythmias. May have a pause after a PJC

#### Junctional Rhythm/Junctional Escape Rhythm
- **Rate**: 40-60 bpm
- **Rhythm**: Regular
- **P-Waves**: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted).
- **PR Interval**: Variable, short if P-wave present before QRS
- **P:QRS Ratio**: Variable, 1:1 if P-wave before QRS
- **QRS Width**: 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**: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted).
- **PR Interval**: Variable, short if P-wave present before QRS
- **P:QRS Ratio**: Variable, 1:1 if P-wave before QRS
- **QRS Width**: 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
- **P-Waves**: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted).
- **PR Interval**: Variable, short if P-wave present before QRS
- **P:QRS Ratio**: Variable, 1:1 if P-wave before QRS
- **QRS Width**: 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
<table>
<thead>
<tr>
<th>Cardiac Conduction Blocks</th>
<th>Details</th>
</tr>
</thead>
</table>
| **First Degree AV Block** | • Rate: Variable, atrial rate = ventricular rate  
  • Rhythm: Regular  
  • P-Waves: Present, similar morphology  
  • PR Interval: Prolonged > 0.20, consistent  
  • P:QRS Ratio: 1:1  
  • QRS Width: Narrow or Wide  
  • T-Waves: Present  
  • Dropped Beats: No  
  • 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, predisposes to progression to more serious block |
| **Second Degree AV Block Type I (Mobitz I, Wenckebach)** | • Rate: Ventricular rate is less than atrial rate d/t dropped QRS complexes  
  • Rhythm: Variable—atrial is regular, ventricular is irregular d/t non-conducted beat  
  • P-Waves: Present, similar morphology  
  • PR Interval: Variable, progressively gets longer before dropping a QRS complex. The shortest PRI is the one immediately following the dropped beat. The longest PRI is the one immediately before the dropped beat.  
  • P:QRS Ratio: Variable, P>QRS  
  • QRS Width: Narrow or Wide  
  • Dropped Beats: Yes  
  • Etiology: Ischemia, MI, CAD, cardotoxicity 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 than atrial rate d/t dropped QRS complexes  
  • Rhythm: Variable—atrial is regular, ventricular is regular or irregular depending on number of blocked complexes  
  • P-Waves: Present, similar morphology  
  • PR Interval: Normal and consistent  
  • P:QRS Ratio: Variable, P>QRS. There can be a pattern of conducted beats to dropped beats (example: 2:1—2 P waves for every QRS)  
  • QRS Width: Narrow or Wide  
  • Dropped Beats: Yes  
  • 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: Variable. Atrial rate usually 60-100 and ventricular rate 40-60 if AV node, 20-40 if Purkinje fibers are pacemaker cells  
  • Rhythm: Regular, P-waves will march out, QRS complexes will march out. The P-waves and QRS complexes have no correlation.  
  • P-Waves: Present, similar morphology  
  • PR Interval: Variable  
  • P:QRS Ratio: P>QRS, there is no correlation between P-wave and 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 |
### PVC

- **Rate:** PVC is a single complex. May occur in patterns: pairs, every other beat or every three beats.
- **Rhythm:** Irregular d/t premature ectopic beat
- **P-Waves:** Absent with the ectopic beat
- **PR Interval:** None with the ectopic beat
- **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
  - Couple: 2 PVCs in a row
  - Unifocal: coming from one focus, same shape and size (the example is unifocal PVCs, they are similar morphology)
  - Multifocal: coming from more than one irritable foci, different shapes and sizes

### VENTRICULAR TACHYCARDIA

- **Rate:** 100-250 bpm (Monomorphic)
- **Rhythm:** Regular (may be slightly irregular at onset)
- **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 filing 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

- **Rate:** 150-250, Torsades is a variant of Polymorphic Ventricular Tachycardiac
- **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:** dangerous 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 (wave forms are visible) but the patient will NOT HAVE A PULSE

- **Rate, Rhythm, P-Waves, PR Interval, P:QRS Ratio, QRS Width, T-Waves:** Variable—All based on 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 it is accelerated idioventricular rhythm, >100 is Ventricular Tachycardia
- **Rhythm:** Regular (ventricular rhythm only, no atrial rhythm)
- **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**

- **Type of Pacemakers:**
  - **Atrial:** Pacemaker spike is followed by atrial depolarization (P-wave)
  - **Ventricular:** Pacemaker spike is followed by ventricular depolarization (QRS)
  - **Dual:** There will be atrial pacing and ventricular pacing

**VENTRICULAR PACED**

**DUAL CHAMBER PACED**

- **Pacemaker Sensing Definitions:**
  - **Undersensing:** Pacemaker has failed to sense that the heart has initiated an intrinsic beat.
    - Pacing when it shouldn’t
    - Pacemaker did not sense the patient’s own intrinsic beat. This causes the pacemaker to send an impulse when it shouldn’t. An impulse is sent, and extra cardiac depolarization can occur. This impulse can occur during the intrinsic beat of the patient, predisposing the patient to R-on-T phenomenon.
  - **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
    - Pacemaker sensed non-cardiac activity and interpreted it as an intrinsic beat. Pacemaker impulse is inhibited as the pacemaker falsely sensed the patient as having an intrinsic beat. An impulse is not sent, and cardiac depolarization does not occur. This leads to an overall decrease in heart rate and cardiac output.

- **Pacemaker Capture Definitions:**
  - **Capture:** When the pacemaker delivers an electrical impulse strong enough to result in depolarization.
    - 100% capture occurs when each pacemaker impulse produces a corresponding depolarization (atrial pacemaker impulse results in a P-wave, ventricular pacemaker impulse results in a QRS Complex). This means that the pacemaker is appropriately capturing/depolarizing.
  - **Failure to capture:** When the pacemaker delivers an electrical impulse that does not result in depolarization.
    - The pacemaker sends an impulse, the pacer spike is visible, however, no corresponding waveform is present following the pacer spike (no p-wave for an atrial pacemaker, no QRS complex for a ventricular).
  - **Failure to pace:** No electrical impulse is delivered, complete failure to sense the need or deliver the impulse to initiate pacing.
    - The pacemaker fails to send a pacing impulse when appropriate and no visible pacing spike is seen.