**Preceptor Guide: Zoll ECG simulator session**

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**Simulation Resources**:

1. Companion Power Point Slide Deck (resident facing)
2. Zoll ECG Simulator (can plug in directly to Zoll R Series monitor/defibrillator)
   1. Options: VT, VT-HI, VT-LO, VF, AFIB, AFLTR, NSR, ASYS, SBrady, 1st AVB, 2nd I AVB, 2nd II AVB, 3rd AVB, TdP, Convert.

**A white box with blue buttons

AI-generated content may be incorrect.**

1. Zoll R Series Monitor/defibrillator

A close-up of a medical device

AI-generated content may be incorrect.

**Audience**: IM Interns

**Duration**: 20-30 minutes hands on scenarios

Case List (Clickable)

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# **Case 1: (Asystole)** “Can’ Shock What You Can’t See”

**Scenario:**

* 70M with history of ESRD found unresponsive on the floor. No pulse.
* CPR in progress, IV access obtained.

**Initial Zoll Rhythm**: Asystole

**Management Steps:**

* Confirm asystole in >1 lead
* Continue high-quality CPR (2 minutes) then pulse check
  + Consider H/Ts + Labs (ppt slide with resulted labs available)
* Give **Epinephrine 1 mg q3–5 min**
* **Repeat until ROSC or called.**

**Possible Branch Points:**

* If team confirms asystole in 2 leads: Continue CPR + Epi q3–5 min.
* If team does not confirm (and it's fine VF): Opportunity to shock missed → worsening prognosis.
  + Fine VT ppt (slide available, discuss Zoll settings
    - Consider *resident role-play how to* ***communicate lab findings quickly*** *to influence ACLS management.*
    - *General Lab Interpretation* 
      * ***Severe metabolic acidosis*** *(low pH, HCO₃⁻), likely from lactic acidosis and hypoperfusion.*
      * ***Hyperkalemia*** *(7.2) → treat urgently*
      * *Mild* ***hypocalcemia****, which may contribute to cardiac instability.*
* If hyperkalemia identified and appropriately treated 🡪 Convert to organized rhythm + **absent pulses**
* After additional round of ACLS 🡪 ROSC

**Teaching Pearls:**

* No shock for asystole
* Review zoll settings & charging
* Review dosing of epinephrine
* Always confirm rhythm isn't fine VF (ppt slide)
* ?Discuss importance of ETCO₂ and reversible causes (e.g., hyperkalemia in ESRD).

# **Case 2: (VF**) “Pulseless but Not Powerless”

**Scenario:**

* 58F with history of CAD s/p remote DES, CABG now admitted for UTI with sepsis. Telemetry alarms "code blue" on hospital day 2. On initial assessment, pulses are absent.

**Initial Zoll Rhythm:** VF

**Management Steps:**

* Immediate CPR
* Charge and shock ASAP (defibrillation)
* Resume CPR
  + ***Labs available on ppt***
* Rhythm check: VF 🡪 Shock
* **Epinephrine q3–5min + Resume CPR**
* Rhythm check: VF 🡪 Shock
* **Amiodarone 300 mg IV + Resume CPR**
* Continue algorithm until ROSC

**Possible Branch Points:**

* If team shocks within 2 min: Higher ROSC likelihood.
* If shock delayed >5 min: Worse outcomes, prolonged downtime.
* If Epi then Amiodarone given: VF may organize into a perfusing rhythm.
* If ROSC achieved:
  + Hypotension (SBP 80s) occurs → **post-ROSC care** (fluids, vasopressors, possible TTM if comatose).
  + ***EKG Post ROSC available 🡪 Anterior STEMI 🡪 discuss next steps***
* If team fails to reassess rhythm every 2 min: Missed VF → PEA ensues.

**Teaching Pearls:**

* Time to first shock is critical in VF
* Emphasize compression quality, minimizing pauses and rhythm checks every 2 minutes.
* Role of antiarrhythmics is adjunctive, not primary
* Quick Prioritization in Practice:
  + Think **acute MI**, **electrolyte derangements**, and **hypoxia** first in the setting of sudden VF arrest.
  + In peri-arrest or monitored settings (e.g., during a procedure), **drug toxicity** or **iatrogenic causes** may be more likely.

# **Case 3: (Pulseless VT / VT-HI)** “No Pulse, Wide QRS”

**Scenario:**

* 45M with HFrEF collapses in clinic waiting room. Code is called. Initial assessment reveals absent pulses.

**Initial Zoll Rhythm:** Pulseless VT (VT-HI)

**Management Steps:**

* Begin CPR
* Immediate defibrillation
  + ***Consider prompting a scenario where pads are not placed (or placed incorrectly)***
* Epinephrine + Amiodarone per ACLS
* Reassess after each 2 min cycle

**Possible Branch Points:**

* *Immediate defibrillation given*: VT-HI → NSR achieved.
* *If CPR + Epi continue but defibrillation missed*: Rhythm degenerates to asystole.
* *If Amiodarone given after 3 shocks*: Organized wide complex rhythm with ROSC.
  + ***Post ROSC EKG on PPT deck 🡪 Monomorphic VT, HDS***
* *If ROSC achieved but wide QRS remains*: Need to address underlying cause (ischemia, electrolyte imbalance).
* *If no ROSC after 20 min despite ACLS*: Family meeting and consider terminating resuscitation efforts.

**Teaching Pearls:**

* Don't delay shock for rhythm clarification if no pulse.
* Contrast pulseless VT vs stable VT
* Discuss defibrillator pad placement and safety.

# **Case 4: (SVT - PVST/AFL)**: “ Fast and Faint”

**Scenario:**

* 36F admitted for ETOH detox. RRT activated on third day of admission. Tachycardic, HR 180s, dizzy/lightheaded but responsive, pulsatile in all ext.

**Initial Zoll Rhythm:** SVT (PSVT vs AFlutter)

**Management Steps:**

* Assess for stability (hypotension, AMS, CP?)
* If unstable → **synchronized cardioversion**
* If stable (provide EKG on PPT) → vagal maneuvers, then **Adenosine 6 mg**
* Reassess rhythm (may convert or reveal underlying flutter)

**Possible Branch Points:**

* If vagal maneuvers successful: Converts to NSR. Patient asymptomatic. Etiology PSVT
* If vagal fails and ***Adenosine*** 6 mg given rapidly: PSVT terminates but underlying **atrial flutter with 2:1 block** uncovered (rate 150).
  + **See EKG #2 on PPT**
  + **Tx options: BB/CCB +/- Cardioversion (expert consultation)**
* If unstable (SBP <90, AMS, CP) develops: Urgent **synchronized cardioversion** needed (50–100J biphasic).
* If team misidentifies and delays cardioversion: Decompensation → code blue called.

**Teaching Pearls:**

* Review bedside vagal maneuvers.
* SVT vs sinus tach vs AFlutter at 2:1 block.
* Pharmacotherapy options for SVT

# **Case 5: (3rd Degree AVB)**: **“**Slow and Confused”

**Scenario:**

* 82F with bradycardia, confusion, SBP 80s. Monitor shows complete heart block.

**Initial Zoll Rhythm:** CHB

* EKG available on your arrival 🡪 slide deck (Complete AV dissociation, HR 30s on EKG)

**Management Steps:**

* **Atropine 0.5mg bolus. Repeat q3-5min (max 3mg)**
* **Transcutaneous pacing** immediately
* Escalation + expert consultation: prepare for transvenous pacing
* Consider epinephrine or dopamine infusion

**Possible Branch Points:**

* If transcutaneous pacing started early: Capture achieved → BP improves.
* If pacing delayed: SBP drops further, LOC worsens, cardiac arrest risk.
* If Atropine 0.5 mg tried: No improvement (because block is infranodal). Team learns atropine is unreliable here.
* If transvenous pacer not available: Start Epinephrine drip (2–10 mcg/min) while awaiting higher-level intervention.
* If capture maintained: Patient stabilizes, consult cardiology for PPM.

**Teaching Pearls:**

* Complete heart block = AV dissociation.
* Atropine ineffective in infranodal blocks.
* Always have pacing pads in place during rapid responses.
  + Review transcutaneous pacing settings on Zoll

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# **Case 6: (TdP)**: “Twist and Shout”

**Scenario:**

* 24F with recent QT-prolonging meds collapses on telemetry. Monitor shows polymorphic VT.

**Rhythm:** Torsades de Pointes (TdP)

**Management Steps:**

* Pulseless 🡪 Arrest Algorithm
* Pulse+ 🡪 Tachycardia with Pulse Algorithm
* Defibrillation (if pulseless)
* Can consider IV **Magnesium 2 g over 10–15 minutes**
* Discontinue QT-prolonging meds on stabilization

**Possible Branch Points:**

* If pulseless: Immediate unsynchronized shock.
* If pulses present but unstable: Immediate synchronized cardioversion preferred.
* If Magnesium 2g IV given promptly: TdP resolves, underlying QTc prolongation seen.
* If magnesium delayed: consider degeneration to VF → code escalation.
* If QT-prolonging agent (e.g., methadone, ziprasidone) identified and stopped: Risk of recurrence reduced.
* If prolonged bradycardia remains: Overdrive pacing (temporary pacing at HR ~90) considered.

**Teaching Pearls:**

* Magnesium even if Mg level is normal.
* Common reversible causes (meds, hypokalemia).
* Distinguish from monomorphic VT.