

# Cardiac Arrhythmia & Sleep Apnea

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4. This talk presents material that is related to one or more of these potential conflicts, and the following objective references are provided as support for this lecture:

# Objectives

- What happens during sleep
- Who are the high risk patients
- Arrhythmias in sleep apnea
- What are we monitoring for



# What happens during sleep

2 easily measured parameters of the cardiovascular system

- Heart rate
- Blood pressure



Both controlled by the Autonomic Nervous System

1. Parasympathetic
2. Sympathetic



# What happens during sleep

- Parasympathetic Nervous System
  - Mediated by the Vagus nerve → Cardiac muscarinic receptors
    - Causes bradycardia
    - Decreases cardiac muscle contraction



# What happens during sleep

- Sympathetic Nervous System
  - Impulse carried by the thoracic and lumbar nerves
    - Constriction of blood vessels
    - Increased heart rate → SA node
    - Increased cardiac muscle contraction → direct effect on the cardiac muscle



# What happens during sleep

## At night

- Blood pressure dips by 10%
- Heart rate slows down

(effect of posture, activity level, circadian influence and effect of sleep)

## On awakening

- Blood pressure raises
- Increased heart rate

Gradual steady increases noted  
(effect of increasing activity, postural change)



# What happens during sleep

- Non-REM stages
  - Parasympathetic influence is dominant
    - Heart rate is reduced → bradycardia
    - Sinus pauses (over 2 seconds), AV blocks → due to parasympathetic effect on AV node
    - Respiratory sinus arrhythmia → indicates good cardiac health
      - HR accelerates with inspiration; decreases with expiration
    - No direct effect on Blood pressure
    - Effect most profound in slow wave sleep
  - Sympathetic activity reduced and stable
    - Reduced Blood Pressure





# What happens during sleep

- REM stage
  - Predominant sympathetic state
  - Excitable and unstable state
  - significant fluctuations in heart rate, BP and respiration
  - Swings from tachycardia to bradycardia
  - Parasympathetic activity present but decreased



# What happens during sleep

**Table 2** Autonomic changes during sleep

Sleep stage	Parasympathetic tone	Sympathetic tone
Non-REM	↑	↓
REM	↓	(due to surges) ↑ ↓

REM, rapid eye movement.



# What happens during sleep

- During an arousal
  - spontaneous, secondary to PLMS or respiratory event
    - Has a sympathetic effect (HR and BP)
    - Heart rate increases for 4-5 seconds; noted right before the arousal
    - Bradycardia with and following the arousal



# What happens during sleep

“Normal” healthy individual



- Slower HR in Non-REM
- Variable HR and BP in REM
- Arousals increase HR and BP



# What happens during sleep

Young individuals  
Physically fit/athletes  
Heavy laborers

**Table 1** Normal rhythm changes during sleep in healthy subjects<sup>1 2</sup>

ECG finding	Prevalence
Sinus pause (>2 s)	4–10%
Sinus bradycardia (<40 bpm)	24%
First degree AV block	8–12%
Wenckebach second degree AV block	6–11%

AV, atrioventricular; bpm, beats per minute



What will happen when things are  
“Abnormal”



# Who are the high risk patients

- MI
- Angina
- Heart failure
- Atrial fibrillation
- Long QT syndrome
- Brugada syndrome
- Medications
- Sleep Apnea



# Who are the high risk patients

- MI, Angina
  - Increased risk between midnight to 6 am
  - In Non-REM sleep: reduced BP results in decreased coronary circulation → “non-demand” ischemia
  - In REM sleep: due to increased sympathetic activity → increased heart rate and higher demand from the cardiac muscle → ischemia
  - Post MI: heart function is impaired → arrhythmias
    - (tachycardia, ventricular premature beats);
    - arrhythmia risk decreases after initial 6 months following MI.





# Who are the high risk patients

- Heart Failure
  - Oxygen desaturations can trigger tachycardia and result in arrhythmias
  - Cardiac chambers are remodeled (dilated or hypertrophied)
    - Promote areas for ectopic beats



# Who are the high risk patients

- Atrial Fibrillation
  - Increased risk between midnight to 2 am
  - Risk doubled for A. fib when there is underlying sleep disordered breathing
    - Likely from sympathetic activity during REM
    - Hypoxemia
- Long QT syndrome, Brugada syndrome
  - Lethal ventricular arrhythmias/Torsades de pointes mainly occur at rest and during sleep



# Who are the high risk patients

- Medications:
  - Beta blockers (Metoprolol, Carvedilol..)
  - Calcium Channel blockers (Verapamil, Amlodipine..)
    - They can cross the blood brain barrier and cause violent dreams/nightmares; sleep disruption
    - Induce profound hypotension in Non-REM sleep resulting in coronary ischemia
    - Due to reduced heart rate in Non-REM, other areas conduct a beat → ectopic beats



# Who are the high risk patients

- Medications:
  - Drugs that cause QT interval prolongation
    - ❖ The longer pause can lead to ventricular arrhythmias and fatal events like Torsades de pointes
      - Antibiotics
        - Erythromycin, Clarithromycin, Ketoconazole, Quinine,
      - Antihistamines
        - Diphenhydramine, Hydroxyzine, Loratadine
      - Psychiatric drugs
        - Amitriptyline, Nortriptyline, Despiramine, Clomipramine, Doxepin..
        - Haldol, Ziprasidone, Lithium, Thioridazine
      - Type 3 anti-arrhythmic drugs
        - Amiodarone, Sotalol
      - Type 1 anti arrhythmic drugs
        - Quinidine, Flecainide



# Sleep Apnea



# Arrhythmias in Sleep Apnea

- During an apnea increased vagal tone



- Slowed heart rate
- Potential for Bradyarrhythmias



# Arrhythmias in Sleep Apnea

- At the end of an apnea there is hypoxemia, hypercapnia



- Increases sympathetic activity



- Causes elevations in BP and HR



# Arrhythmias in Sleep Apnea

- Apnea ends with an arousal



- Increased sympathetic activity
- Decreased parasympathetic activity

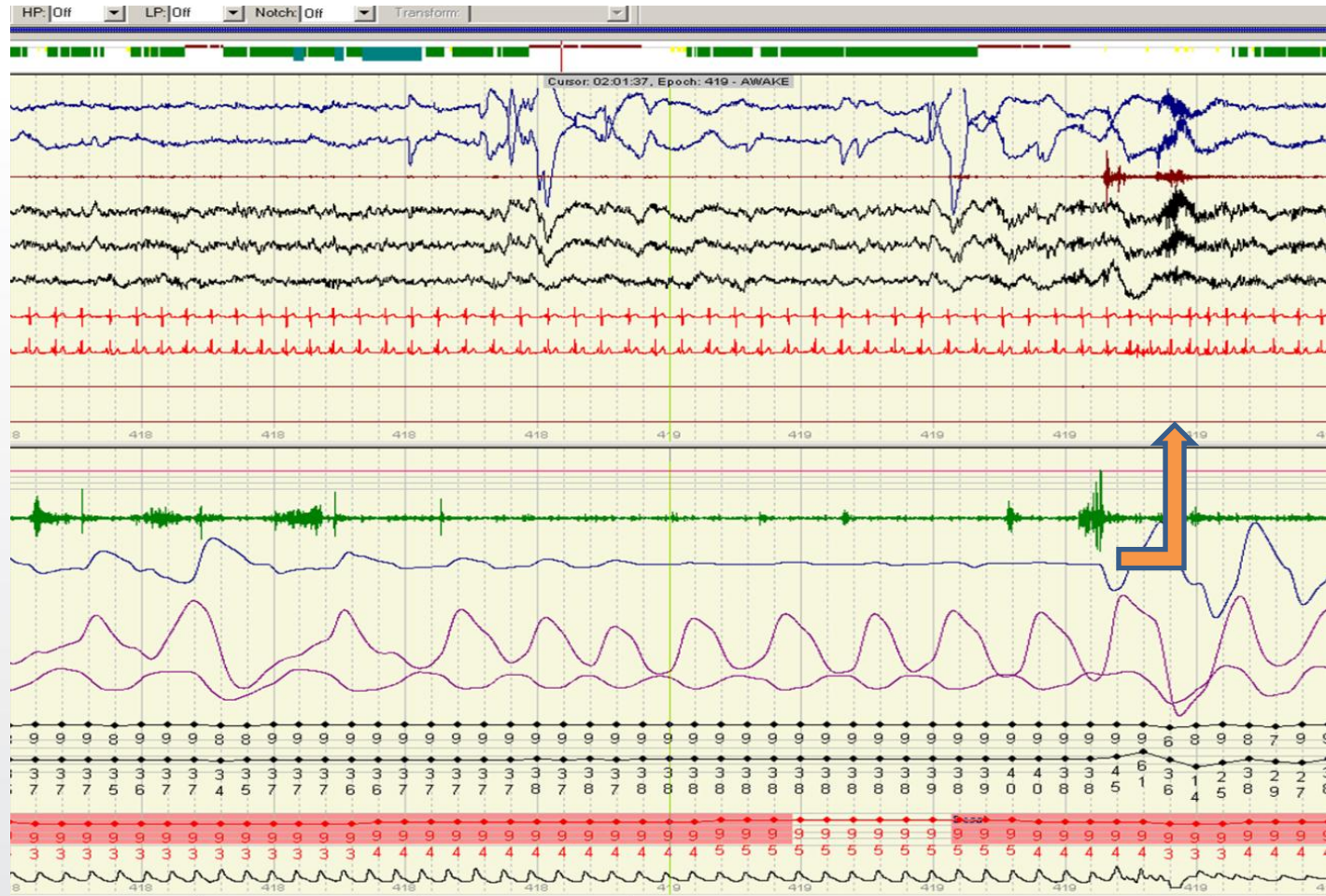


- Increased BP and HR






# Arrhythmias in Sleep Apnea



# Arrhythmias in Sleep Apnea

- Due to sleep disruption from respiratory events
    - Instability in normal sleep stage mediated sympathetic and parasympathetic activity
    - Increased sympathetic response
- 
- Hemodynamic instability (BP and HR)

**Table 2** Autonomic changes during sleep

Sleep stage	Parasympathetic tone	Sympathetic tone
Non-REM	↑	↓
REM	↓	(due to surges) ↑ ↓

REM, rapid eye movement.



# Arrhythmias in Sleep Apnea

- Risk associated with frequency of apnea (moderate OSA) and degree of hypoxemia
- 50% of OSA patients have nocturnal arrhythmias
- OSA patients have 2-4 fold risk of having complex arrhythmia compared to non-OSA patients.



What should be look for



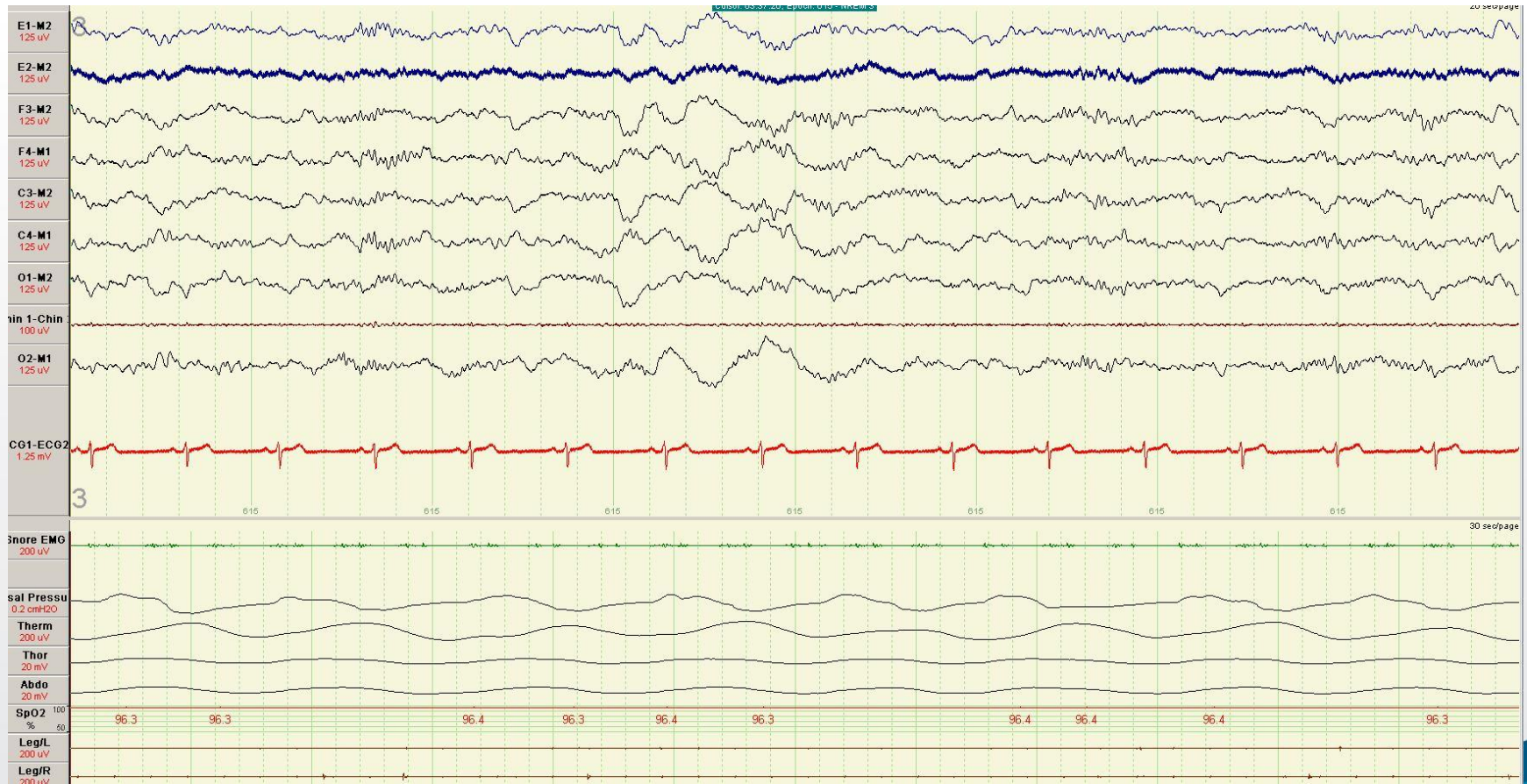
# What should we look for

- AASM Scoring Manual 2012
  - “Recommends” reporting the following adult conditions
    - Sinus tachycardia → sustained HR greater than 90 bpm
    - Sinus bradycardia → sustained HR less than 40 bpm
      - “sustained” means greater than 30 seconds of a stable rhythm
    - Asystole → scored for a pause greater than 3 seconds





# Sinus Bradycardia

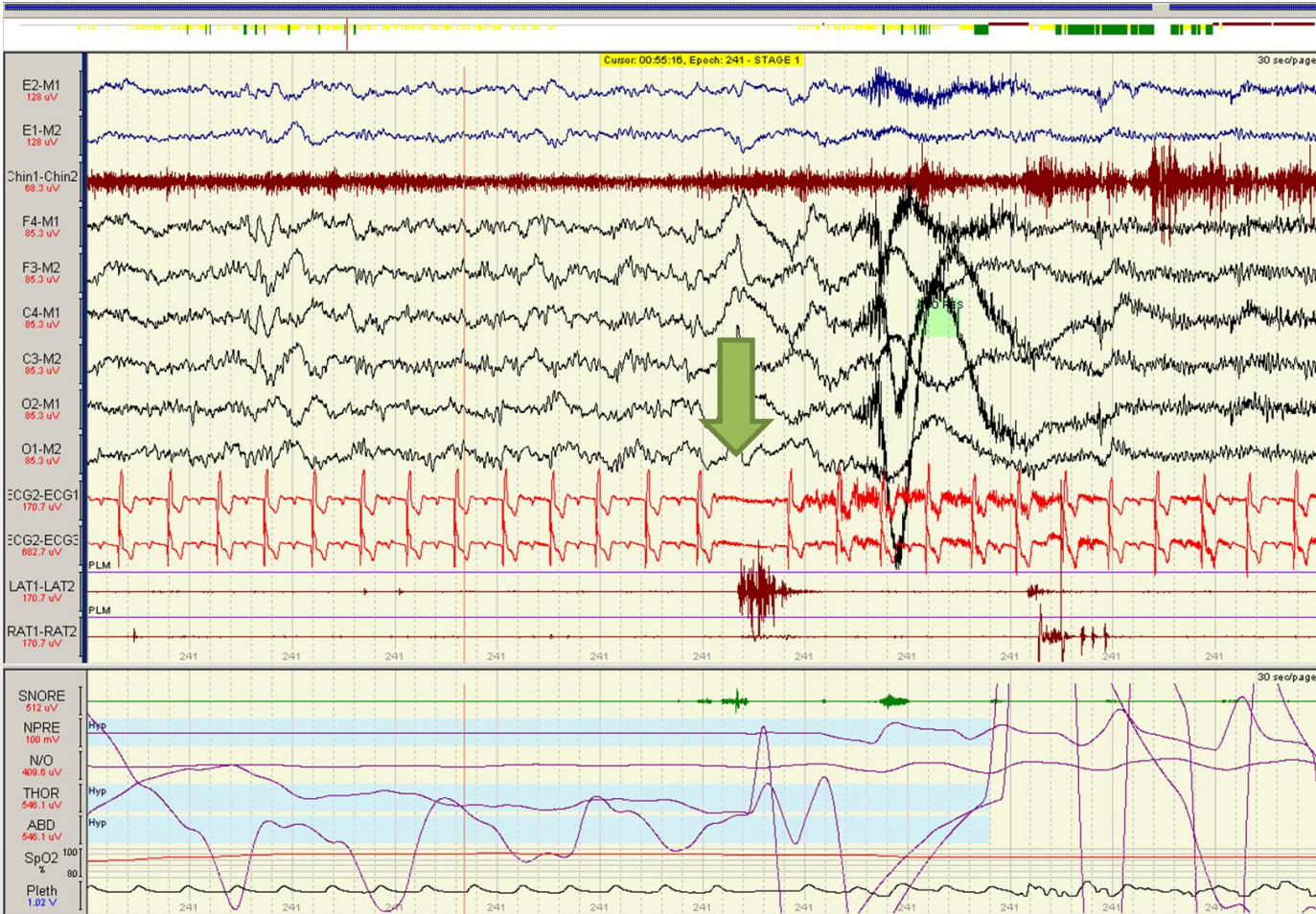


# Asystole

- Less than 3 seconds: normal
- 3-5 seconds pauses: no intervention required.
  - But if patient is symptomatic → Assess patients use of beta blockers or calcium channel blockers
  - Contact on call physician
- 5 -10 second pauses: assess for symptoms; check BP.
  - Assess patients use of beta blockers or calcium channel blockers
  - If patient qualifies for CPAP therapy initiate it.
  - Notify on-call physician.

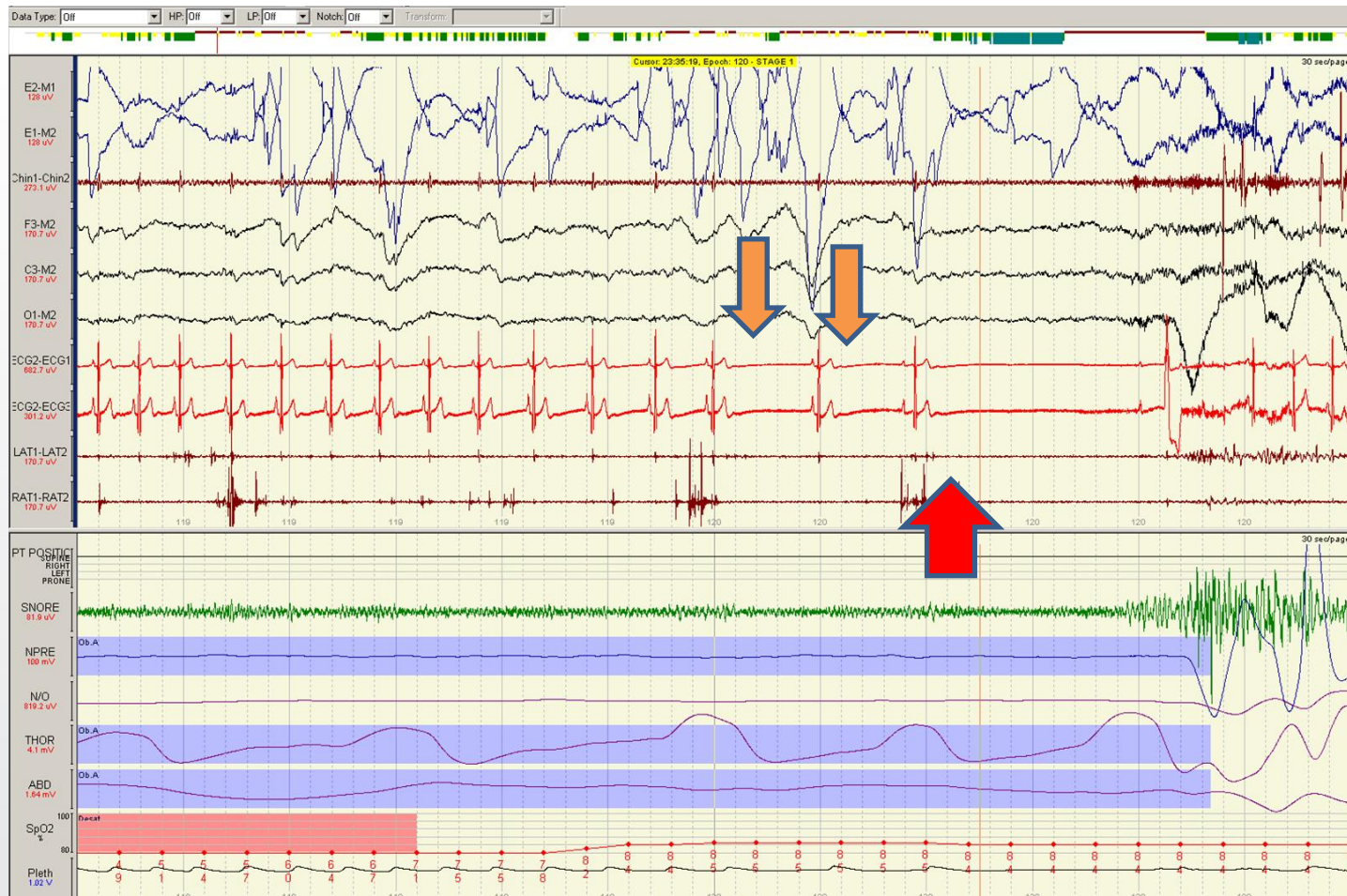


# Sinus pause??

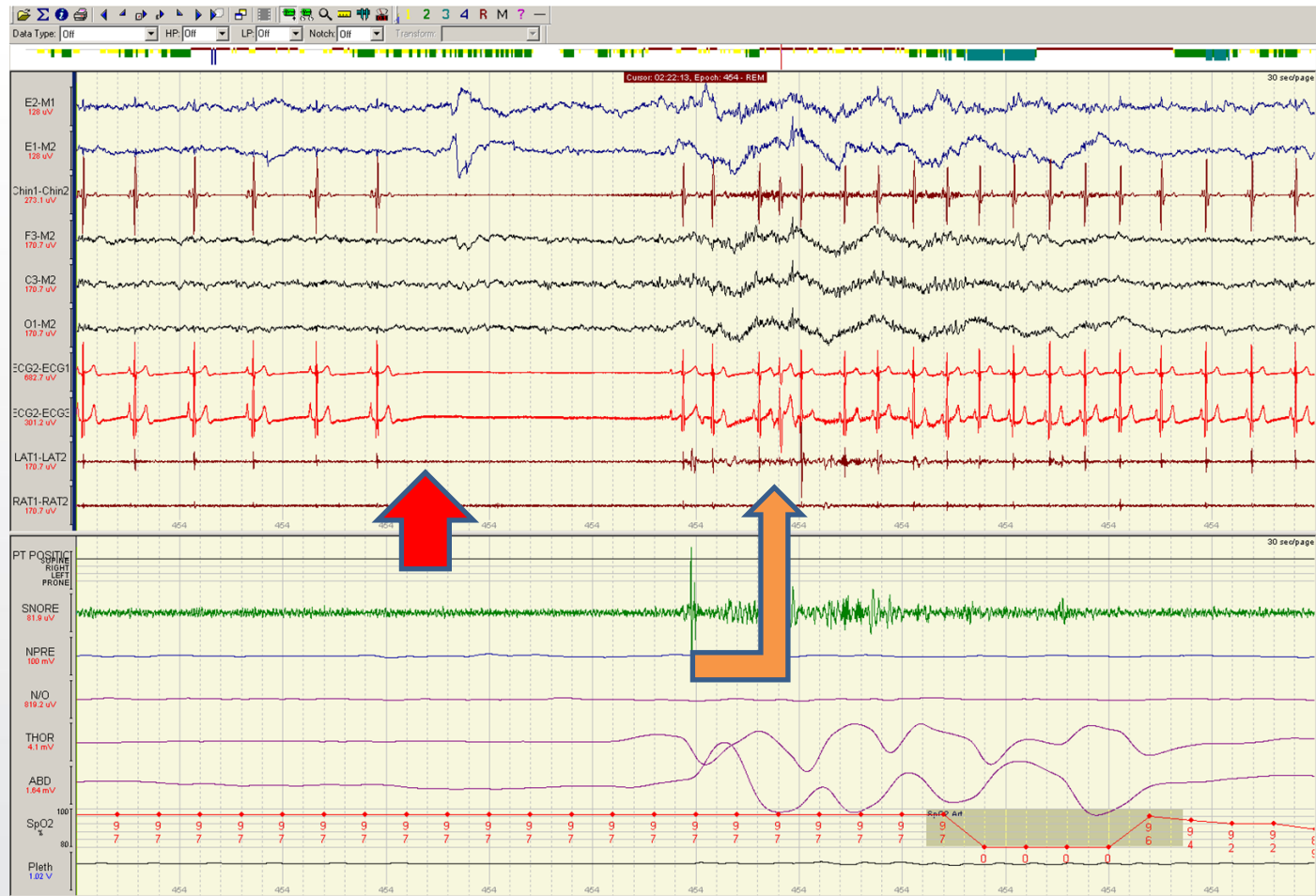




# Asystole



# Asystole



# What should we look for

- AASM Scoring Manual 2012
  - “Recommends” reporting the following adult conditions
    - Wide complex tachycardia: consecutive 3 beat minimum; rate greater than 100 bpm; QRS greater than 120 milliseconds.
      - Sustained vs. Non-sustained
    - Narrow complex tachycardia: consecutive 3 beat minimum; rate greater than 100 bpm; QRS duration less than 120 milliseconds

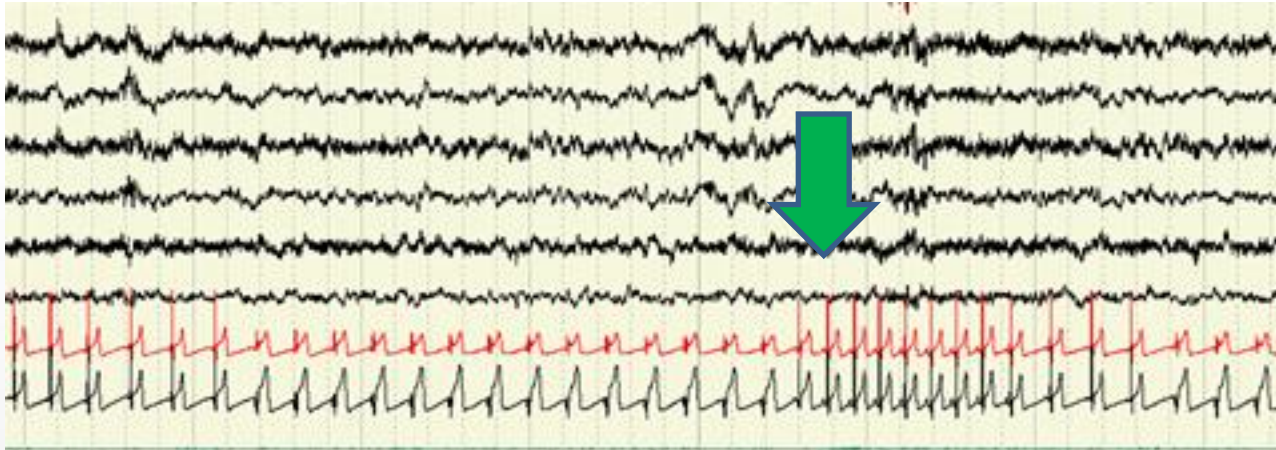


# Wide Complex Tachycardia





# Narrow Complex Tachycardia

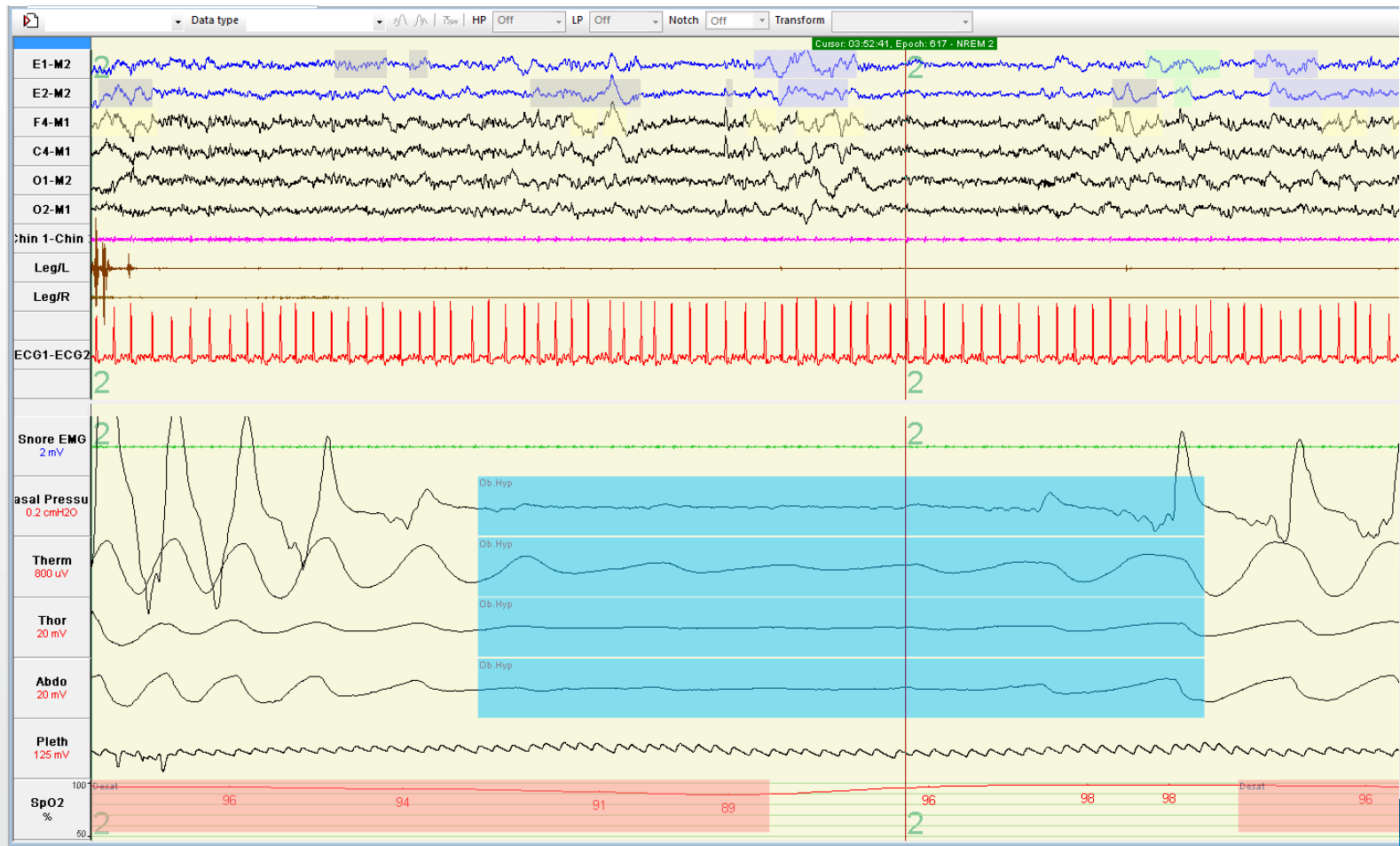


# What should we look for

- AASM Scoring Manual 2012
  - “Recommends” reporting the following adult conditions
    - Atrial fibrillation: irregularly irregular ventricular rhythm; absence of P-waves
      - Does patient have history of it? Is it new onset?
      - Is patient symptomatic from it?
      - Check BP and HR
      - If patient is symptomatic or if unstable send to ER; notify on call physician



# Atrial Fibrillation



# What should we look for

## AV Block:

- AASM expects reporting of AV blocks if quality is sufficient for accurate scoring.
- Atrial beats are not conducting through to the ventricles at the AV node





# AV Block

- 1<sup>st</sup> degree AV block
  - Prolonged PR interval (greater than 0.2 seconds)
- 2<sup>nd</sup> degree AV block
  - Mobitz Type 1 (Wenkebach)
    - Slowly increasing PR interval from beat to beat until a dropped beat (QRS) occurs
  - Mobitz Type 2
    - Randomly dropped beats (QRS)
- 3<sup>rd</sup> degree AV block /Complete heart block:
  - Complete dissociation between atria and ventricles;
    - atrial rate faster than ventricular rate

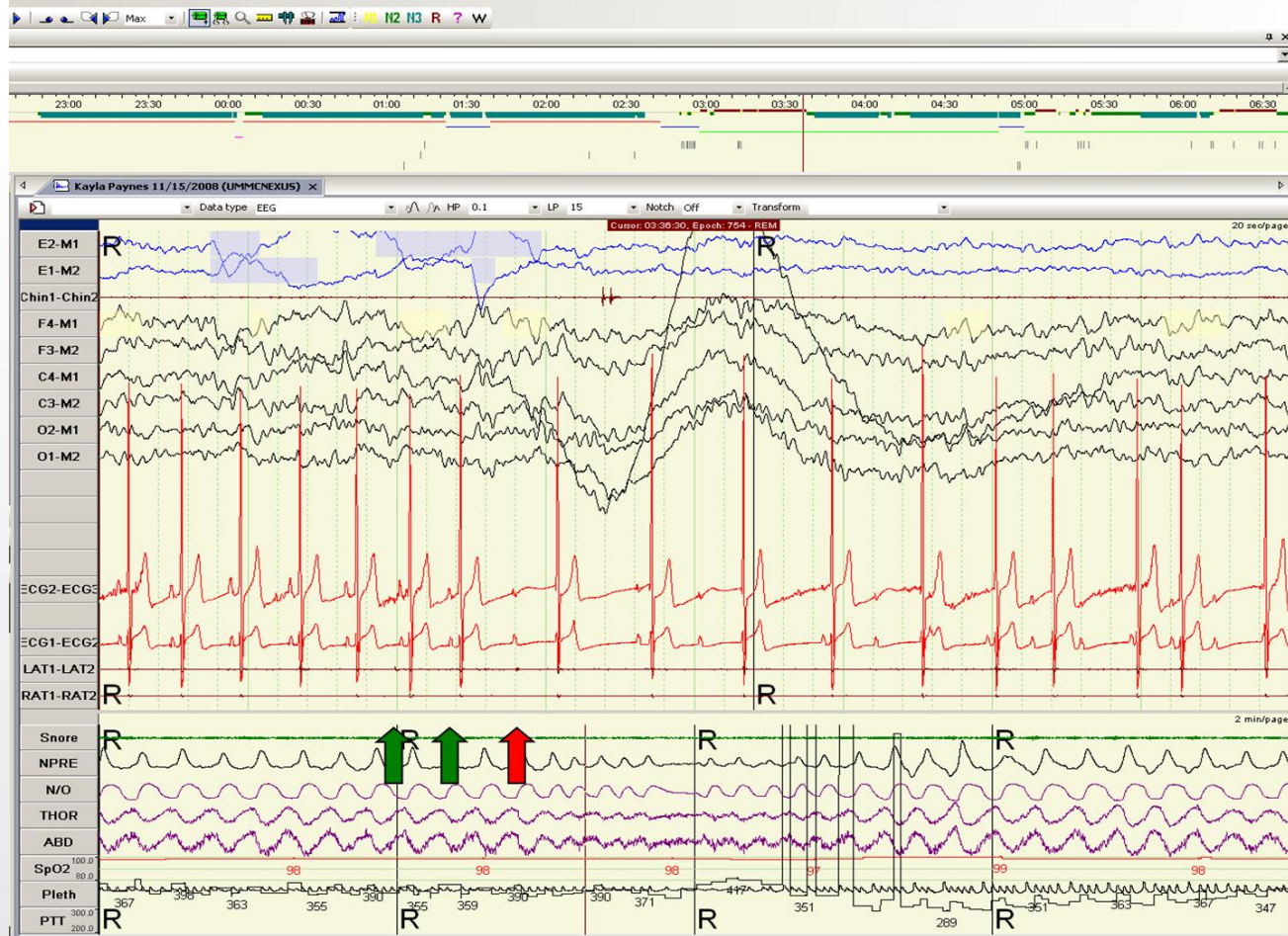


# 1<sup>st</sup> degree AV block

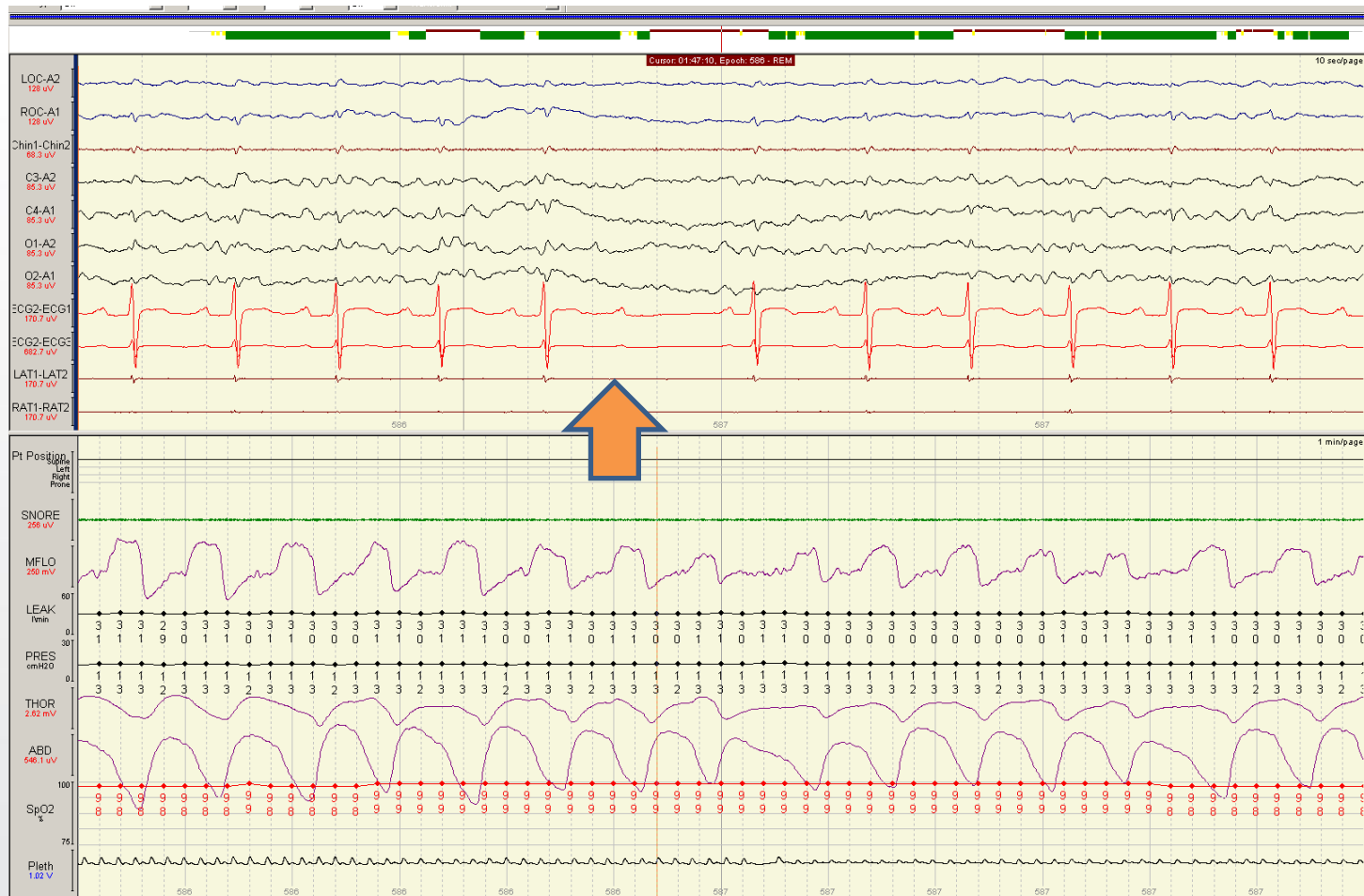
First-Degree AV Block



# 2<sup>nd</sup> degree AV block/Type 1 Mobitz/Wenkebach



# 2nd degree AV block Type 2 Mobitz





# 3<sup>rd</sup> degree AV block

Third-Degree AV Block



# What should we look for

- Ectopic beats: (reported if felt to be clinically significant)
  - Premature atrial contractions
    - P-wave present; narrow QRS
  - Premature ventricular contractions
    - P wave absent; wide QRS complex
  - Premature junctional complexes
    - Originate from the AV junction; will not have a “p-wave” but will have a narrow QRS complex



# What should we look for

- Ectopic beats
  - Couplets, Triplets
  - Bigeminy → N, E, N, E..
  - Trigeminy → N, N, E, N, N, E...
  - Quadrigeminy → N, N, N, E, N, N, N, E...

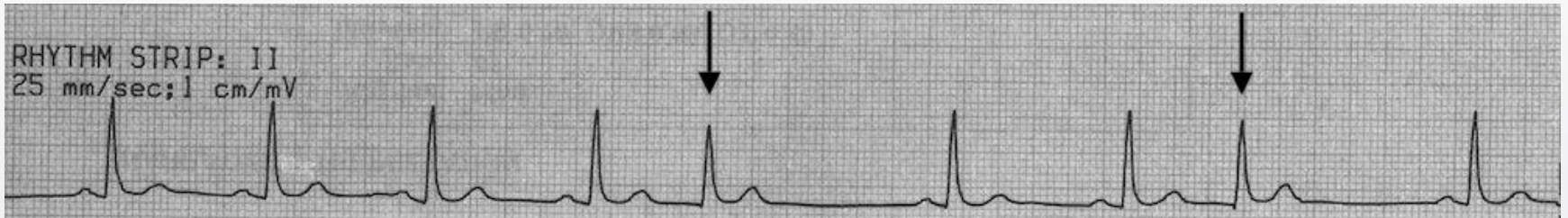


# Ectopic Beats

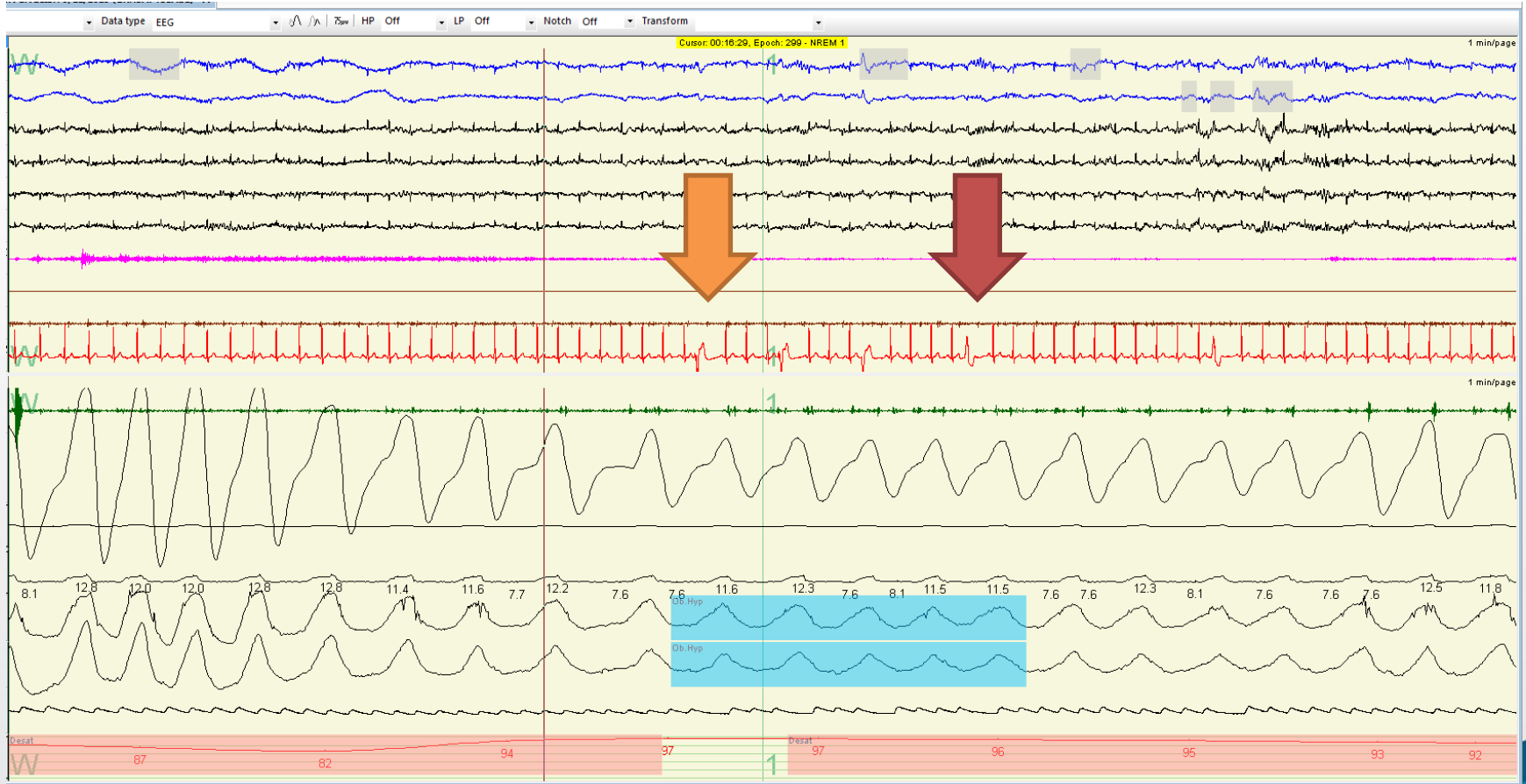




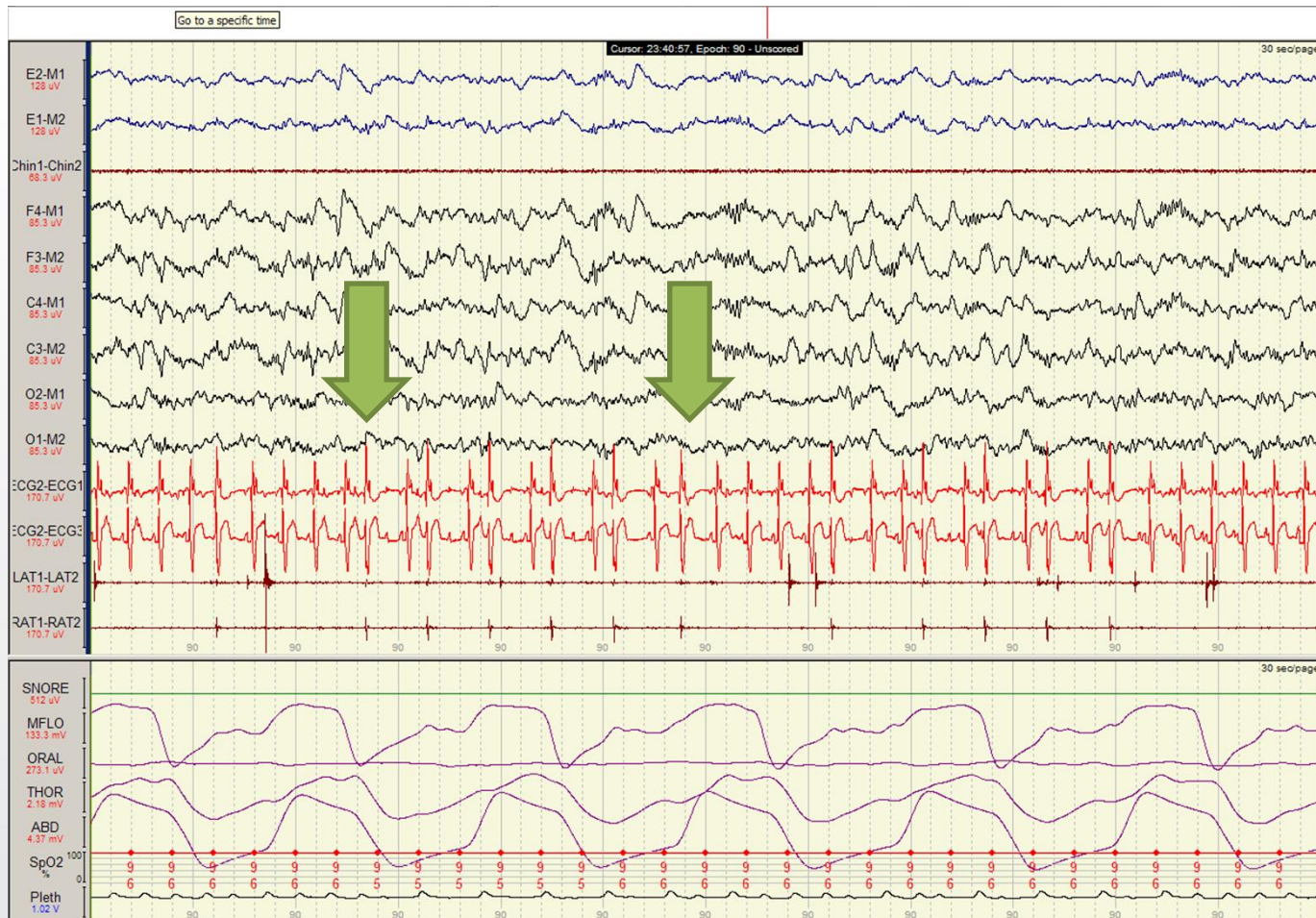
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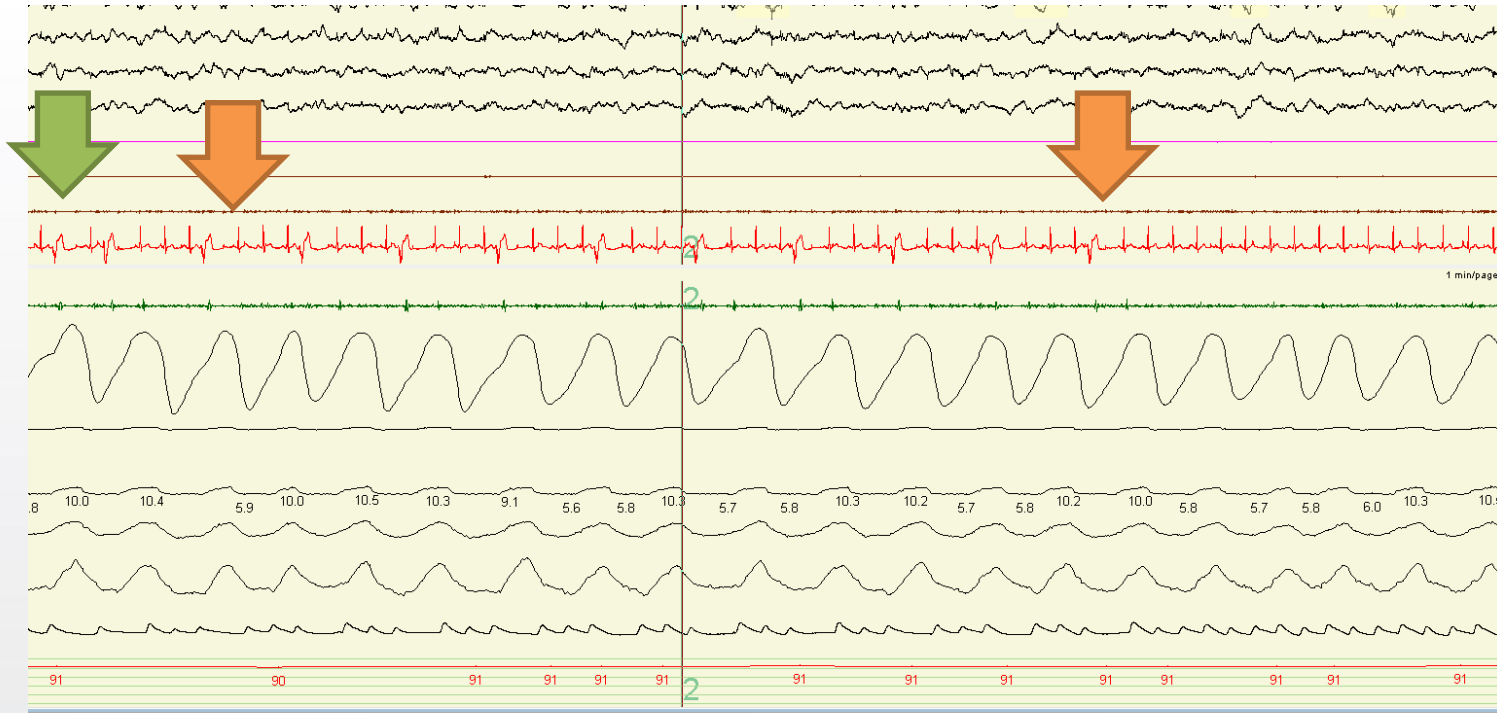
# Ectopic Beats



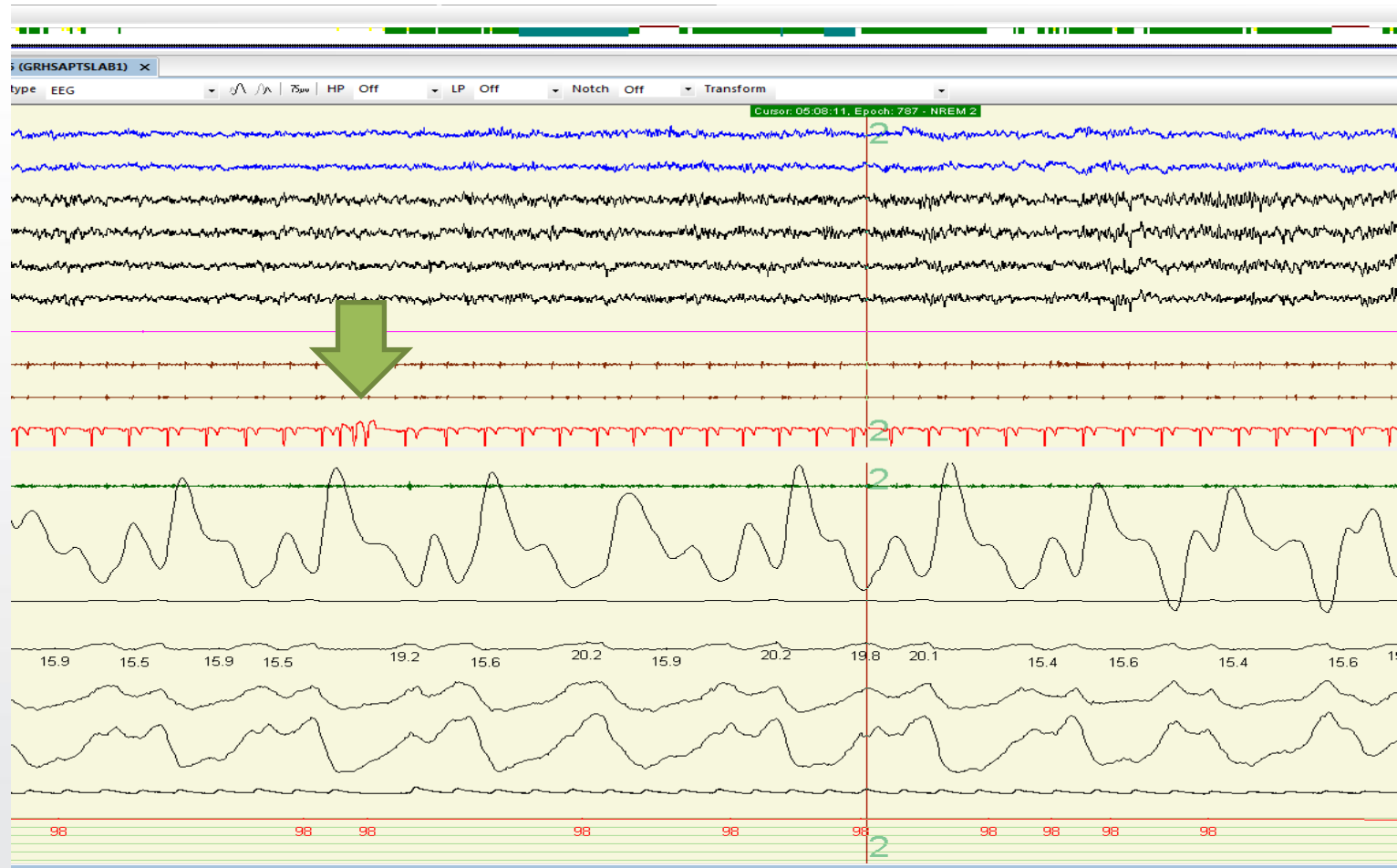
# Ectopic beats



# Ectopic Beats



# Ectopic Beats

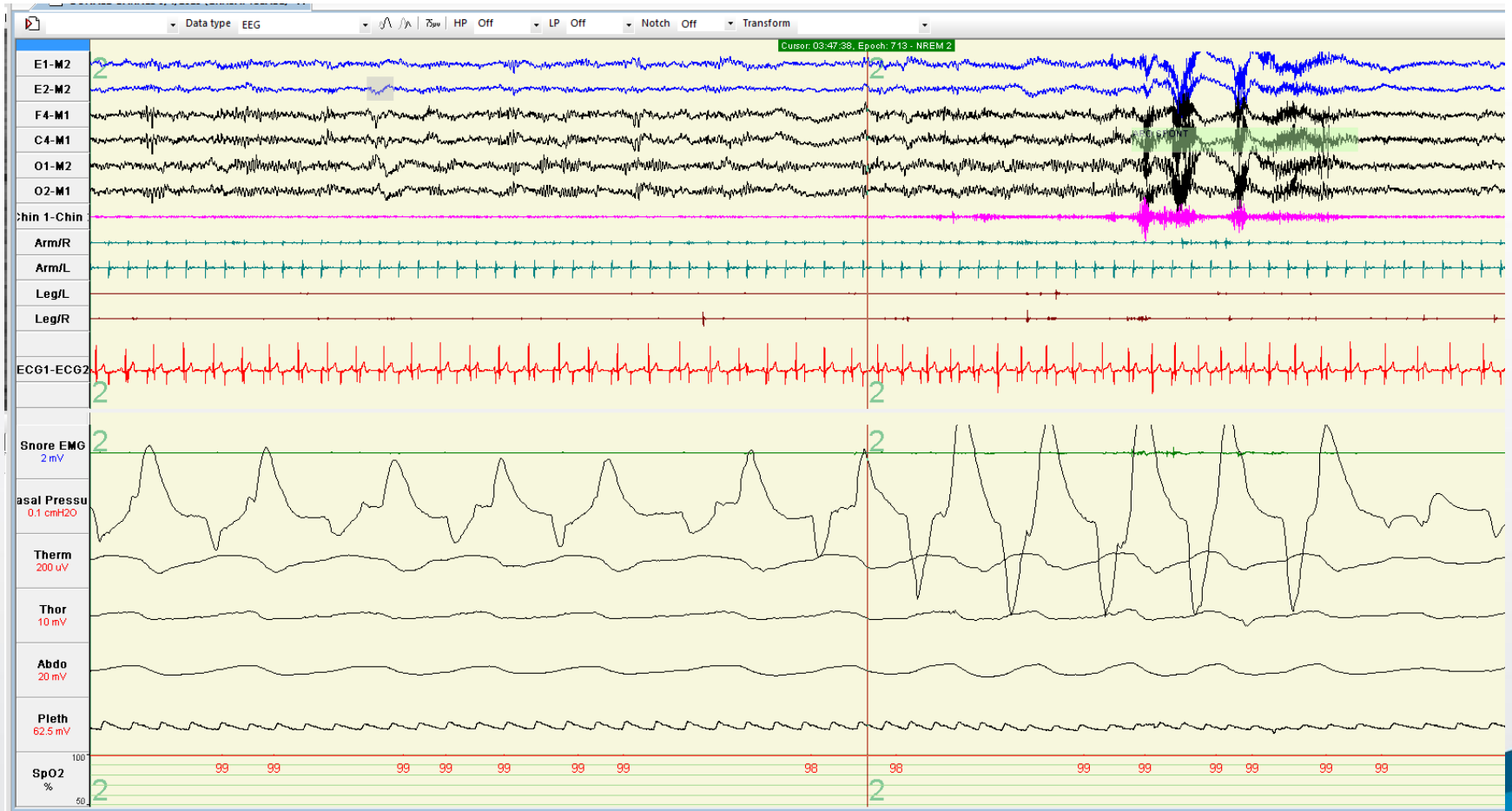




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# References

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