Intro to Critical Care Series Part 3 of 3

Telemetry

By Kreek't Rebaño (PT)

Telemetry

(Cardiac Monitoring)

- Tele = "remote"
- Metron = "measure"
- Consists of:

Why this skill?

Determine when it is appropriate to **begin** an exercise program, **continue** with treatment, or **terminate** the therapy session/exercise.

What is exercise?

n. Activity requiring physical effort, carried out especially to sustain or improve health and fitness

v. engaging in physical activity to promote or improve muscular strength, health, and fitness

- **Repetitive**
- **Demands increased muscular response**
- **Demands increased oxygen consumption**
- **Planned, structured, for the purpose of conditioning**

Objectives

1. Correctly place electrodes on a standard telemetry setup.
2. Correctly connect the device base to monitors or tele-box.
3. Identify the basic components of an ECG tracing.
4. Indicate HR based on triplicate or 6-sec strip methods.
5. Identify and correct for presence of motion artifact.
6. Identify rhythms on a monitor output.
7. Identify pacing origin, and rhythms:
    - **sinus** rhythms, normal or bradycardia vs tachycardia
    - abnormal: **atrial** versus **ventricular** rhythms
    - presence of PACs, **PVCs**, pacemakers, **heart blocks**
    - **rhythm switching**, paroxysmal bursts
8. List 3 reasons to terminate a session (based on cardiac or Pt monitoring)

Who gets monitored? (ACC/AHA)

Practice standards based on expert consensus (not RCTs)

Class I:

- at risk of an immediate, life-threatening arrhythmia—typically ICU appropriate
- Pts in first 48 hours of ACS or with high grade lesions awaiting tx
- acute heart failure
- 2nd and 3rd degree AV block
- temporary pacing
- long QT syndrome
- WPW w/rapid anterograde conduction
- IABP
- post cardiac arrest/code blue
- post cardiac surgery (CABG, MVR)
- post-PCI or ablation with complication
- post pacemaker placement with pm dependence
- conscious sedation (MAC)

Class II:

- chest pain syndromes
- syncope
- known & on active arrhythmia meds titration
- heart failure
- post-PCI, post-ablation or post-pacemaker placement without complications

Not indicated in **Class III**:

- includes rate-controlled afib
- chronic PVCs
- ESRD on HD
- low risk post-surgical patients

Signs of cardiac compromise

Quiz-mo game!

Signs of cardiac compromise

Signs of heart failure

Shortness of breath, DOE or when lying down
Fatigue, weakness
Swelling (edema) in your legs, ankles, feet, abdomen
Rapid or irregular heartbeat
Reduced ability to exercise
Persistent cough, wheezing (white or pink blood-tinged phlegm)
Increased need to urinate at night
Sudden weight gain from fluid retention
Lack of appetite and nausea
Difficulty concentrating or decreased alertness
Chest pain if your heart failure is caused by MI
Heart rate drop with exertion
Syncope

Signs of decreased CO

variations in BP
tachycardia
presence of gallop rhythm
fatigue and weakness
dyspnea, tachypnea, chest pain
crackles (rales)
restlessness, change in mental status
dizziness, syncope
diminished or absent peripheral pulses
cool extremities
pallor or cyanosis of skin
capillary refill time greater than 3 seconds
oliguria
hemodynamic abnormalities (increased PAP, PCWP,CVP)
significant decrease in oximetry results
dysrhythmias

INPATIENT EXERCISE GUIDELINES

	DON'T START EXERCISE IF:			TERMINATE EXERCISE IF:		
VARIABLE	ACSM*	AACVPR**	APTA***	ACSM	AACVPR	APTA
Pulse Rate	> 120 b/min	-----	< 40 b/min > 130 b/min	Post MI 20 b/min above rest	Post MI 30 b/min above rest	Med/Surg > 130 b/min
Pulse Rate	-----	-----	-----	Post CABG 30 b/min above rest	Post CABG 30 b/min above rest	Med/Surg > 130 b/min
Respiration	-----	-----	> 30 b/min	-----	-----	Inability to converse
SBP	> 200 mm/Hg	-----	> 250 mm/Hg	> 220 mm/Hg	10 mm/Hg drop	> 250 mm/Hg 10 mm/Hg drop
DBP	> 110 mm/Hg	> 110 mm/Hg	> 120 mm/Hg	> 110 mm/Hg	> 110 mm/Hg	> 120 mm/Hg
SPO_2	< 88 % (request supplemental oxygen))	-----	< 85% (request supplemental oxygen)	Titrate to maintain @ ≥ 90%	-----	Change from rest of 3–5% (document)
Fever	-----	-----	> 100° F	-----	-----	-----
Arrhythmias	30% of complexes in 1 min^{-1}	-----	> 6 min^{-1}	30% of complexes in 1 min^{-1}	-----	> 6 min^{-1}
RPE				13	13	

*American College of Sports Medicine; **American Association of Cardiovascular and Pulmonary Rehabilitation; ***American Physical Therapy Association – Acute Care Section

ECG Fun Facts

Willem Einthoven (1860 – 1927)

- Dutch doctor and physiologist
- Inventor of the first practical (ECG or EKG) in 1903
- Received the Nobel Prize in Medicine in 1924 👍👌🏾👏🏻

Photograph of a Complete Electrocardiograph, Showing the Manner in which the Electrodes are Attached to the Patient, In this Case the Hands and One Foot Being Immersed in Jars of Salt Solution

- required water cooling
- required 5 people to operate
- weighed 270 kg (595 lbs)

Monitors

Monitor Tech Station

ICU overhead monitor

Monitors

Transport/Portable Monitor

Portable Tele

Telemetry Box, Wires, Electrodes

*CPO

continuous pulse Oximetry

Camera Exercise

Views and Views and Views

(+) vs (-)

12-lead ECG (overview only)

"V" = vector?
= ventricle?
= voltage?

***MUSE** Marquette Universal System for Electrocardiography

12-lead ECG (overview only)

- **10 electrodes** (4 limb, 6 precordial)
- **12 views of the heart**
- **Diagnosis of:**
  - rates and arrhythmias
  - heart blocks
  - bundle branch blocks
  - enlargement/hypertrophy
  - heart failure
  - pericarditis
  - PE/tamponade
  - congenital defects
  - heart valve defects
  - Myocardial Ischemia
  - Myocardial Infarction

Electrode Placement (12-lead)

cECG
Conventional
ECG

modified
Mason-Likar
placement

Reduced distortion from limb movement

Reduced interference from muscle potential

Provide rapid acquisition of ECGs

Dower **EASI**
placement

12 lead ECG using only 5 electrodes

Increased Pt mobility

Chest unencumbered - easier for imaging

Electrode Placement (5-lead/tele)

Standard

- *dysrhythmias*

EASI

- *ischemia monitoring*
- *derive 12-lead ECG*

Placement Mnemonic (+Pacemaker)

Conduction System

For active image, visit https://epsavealife.org/resources/heart-concepts/

ECG Waveforms

P wave
- precedes the QRS
- "small bump"
- amp: 1.5-2.5 mm
- PR duration: 0.12-0.20 sec

QRS complex
- most prominent
- duration: <0.12 sec
- amp: criteria specific
- ddx: narrow vs wide, variable forms
- ST segment: elev/dep

T wave
- comes after the QRS
- "long, higher bump"
- amp: <5 mm
- QT duration: <440(m) <460(f) ms

* < half before next RR, QTc
- ddx: inversion, peak

Deflections (QRS complex)

P 1st upward before QRS

T 1st upward after QRS

Q 1st downward

R 1st upward
2nd upward: R' (R prime)

S any downward after R that crosses isoelectric line

Examples:

QRS | RS | RR' | RSR' | QS

The presentation of the QRS complex is valuable in identifying disease and conduction deficits.

ECG tracings

duration vs voltage
BBB vs IVB
wandering pacemaker

Identify the waves/complexes

Identify the waves/complexes

3+ consecutive PVCs = VT

Identify the waves/complexes

Identify the waves/complexes

Rates (tachycardia vs bradycardia)

300 - 150 - 100 - 75 - 60 - 50 - 43 - 37 - 33 - 30 - 27 - 25 ...

Rates

6 sec. strip x10

count the QRS complexes or number of RR intervals

Rhythms (regular vs irregular)

Technical Problems

Muscle Artifact

- high frequency, low amplitude
- poorly supported/ tense muscles in the arms and legs
- loose, dry or outdated electrodes or lead wire
- poor patient cable connection
- poor skin prep

Wandering Baseline

- low frequency, high amplitude artifact
- observed during deep inhalations and exhalations

Technical Problems

Electromagnetic Interference (EMI)

- interference from electronic sources such as power cords
- near-by use of a hand held radio, mobile devices
- AC powered equipment (switch to battery supply)
- noisy environment

Artificial Pacemaker / Pacer Spikes

- seen when implanted pacemaker is firing
- thin upright spikes (one is marked with "**x**")
- QRS complex following represents ventricles depolarizing
- "(artificial) pacemaker captures" when it is able to successfully depolarize its intended target.
- If a pacing spike is not followed by its intended response, we say that it has "failed to capture" ("**z**")

V-paced

Sinus Rhythms

P waves
P "married" to R
P morphology constant
P-P constant, R-R constant

Normal Sinus Rhythm (NSR)

"normal" only if 60-100
normal PR-QRS intervals

Sinus Tachycardia (ST) >100 bpm

sinus rhythm criteria
rates >100 (i.e. 101+)

Sinus Bradycardia (SB) <60 bpm

sinus rhythm criteria
rates <60 (i.e. 59-)

Sinus Arrhythmia

normal physiological phenomenon
rate varies due to reflex changes in vagal tone
inspiration: HR, expiration: <HR

Normal Sinus Rhythm	Only referred to as "Normal" if the rate is between 60-100. If there are other abnormalities, just "sinus rhythm"	**NO SPECIFIC TX REQUIRED**	
Sinus Tachycardia (ST)	**> 140** difficult to identify P from T. If Pt is symptomatic, try vagal maneuver to distinguish from an SVT	**Normal Response to sympathetic tone, fevers, dehydration, hypovolemia, PNA**	**• ischemia compensation • cardiogenic shock(+MI) • hypotension, hypoxia • POTS (exag. response)**
Sinus Bradycardia (SB)	May be seen in: athletes, sleep, hypothermia, increased ICP, seizures, medications, HF	**Can cause decreased cardiac output, and may lead to atrial, junctional, or ventricular ectopic rhythms**	**Monitor Pt for lightheadedness, hypotension, vertigo, syncope, or BRADYCARDIA with exercise**
Sinus Arrhythmia	It can be naturally occurring or in the elderly due to heart damage or digoxin	**P-P variation, but PR is constant.**	

Atrial Rhythms

Odd "p"
usually narrow QRS
(QRS duration <0.12)
may have WCTs

SVTs include several different types of arrhythmias, such as atrial fibrillation (RVR), atrial flutter (RVR), atrial tachycardia, AVNRT, and AVRT (WPW).

Atrial Fibrillation (afib / AF)

no discernible P
extremely irregular QRS
>100 bpm = RVR

Atrial Flutter (a-flutter / AFL)

"saw-tooth" / flutter waves
regular or irregular QRS
>100 bpm = RVR

Premature Atrial Complex (PAC)

occurs early
different looking P
regular or irregular QRS

Paroxysmal SVT

occurs suddenly
v rate usually 150-250
responds to vagal maneuvers, carotid massage

Sketchpad

Atrial Fibrillation (afib / AF)	may cause no symptoms often associated with palpitations, fainting, chest pain, or CHF, irr-irregular pulse rate	**paroxysmal AF of > 48 hr duration is unlikely to spontaneously revert to sinus rhythm and anticoagulation must be considered**	**"RVR" can cause decreased CO, Tx may include cardioversion**
Atrial Flutter (a-flutter)	often in individuals with CVD (HTN, CAD, cardiomyopathy), DM. can occur spontaneously in healthy hearts	**Typically not a stable rhythm, frequently degenerates into atrial fibrillation (AF)**	**Tx by cardioversion, ablation. Can cause clots, SUDDEN DEATH if with pre-existing conduction deficits**
Premature Atrial Complex (PAC)	another region of the atria depolarizes before SA node, triggers a premature heartbeat	**often completely asymptomatic, some get "palpitations". no treatment usually, but beta blockers can reduce the freq PACs**	**PACs can indicate heart disease or an increased risk for other cardiac arrhythmias, underlying cause must be treated**
Paroxysmal SVT	one of two mechanisms: 1. re-entry (SNRT, AVNRT, JRT, WPW) 2. automaticity (EAT, MAT, AF, AFL)	**SVT is generally not life threatening, though it may cause worsening heart function if prolonged**	**Pounding heart, SOB, CP, Tachypnea, Dizz, LOC (in only the most serious cases)**

Practice!

On your worksheets, identify sinus and atrial rhythms and indicate their rates (any method)

Junctional Rhythms

inverted or no p waves
rate is 40-60
usually narrow QRS
(because normal conduction pathway through ventricles)

- "back up intrinsic pacemaker"
- usually no P wave (SA not active)
- may or may not have retrograde P

Sketchpad

Ventricular Rhythms

wide QRS
(QRS duration >0.12)
needs immediate care

Ventricular Fibrillation (coarse)

(VF or v-fib)
disorganized, no pattern
can degenerate to fine VF

Ventricular Fibrillation (fine)

both types VF
can quickly degenerate to asystole

Ventricular Tachycardia

organized, "sustained" >30s
wide complex QRS
can degenerate to VF

Premature Ventricular Complex (PVC)

occurs early
wide, bizarre "QRS"
3+ in a row considered v-tach

Sketchpad

Ventricular Fibrillation (fine)

Ventricular Fibrillation (coarse)

Ventricular Tachycardia

Premature Ventricular Complex (PVC)

TX: DEFIBRILLATE IMMEDIATELY (unsynchronized cardioversion)

VF may lead to asystole, and sudden death

***Implantable Cardiac Defibrillators (ICDs) can either deliver "pacing impulses" or a shock.**

Pt: "baseball hitting the back or chest"
Handler/CG: "tingle" or "static shock"

according to morphology, duration (sustained >30 secs), symptomatic/pulseless	**Pt may be asymptomatic. Regardless, need immediate cardioversion**	**TX: Synchronized CARDIOVERSION (with pulse) if no pulse, defib as VT may lead to ventricular fibrillation, asystole, and sudden death**
Be concerned if 30% of complexes occur in 1 min. Example: in a HR of 100, cut off is 33 PVCs	**Pt may be asymptomatic. Possibly reduced CO in Bigeminy, couplets, etc. 3 or more PVCs in a row considered VT**	**"R on T" phenomenon can trigger VTach** **May have CO compromise (I.e, ventricular Bigeminy where rate is 70 but actual PR / beats could be 35**

- Unresponsive
- Pulseless

BEGIN CPR 30:2

SHOCKABLE
defibrillate

High Quality
CPR 30:2

Heart Blocks (Conduction blocks)

1st degree AV block

3rd degree (complete) AV block

2nd degree AVB (I - Wenckebach)

2nd degree AVB (Mobitz II)

1st degree AVB	May be seen in: Athletic training, Mitral valve surgery, Electrolyte disturbances, AV nodal blocking drugs	**NO SPECIFIC TX REQUIRED, except ax of electrolyte levels & drug screens**	**may be at an increased risk of progression to complete heart block if accompanied by BBB or fascicular blocks**
2nd degree AVB (Type I)	incomplete heart block PR increases until dropped beat pattern resets itself	**Usually due to reversible conduction block at AVN** **Usually benign**	**No Tx unless with Sx (Atropine)** **Caused by malfunctioning <u>AVN cells fatiguing</u>**
2nd degree AVB (Type II)	May be seen in: infarction, necrosis, fibrosis at His-Purkinje level below AVN, MI, autoimmune	**TX: Urgent Pacing, Pacemaker Placement**	***High risk progressing to complete block** ***Hemodynamic instability sudden, unexpected**
3rd degree AV block	May be seen in: Inferior MI, AV nodal blocking drugs, progression of Type II blocks	**TX: Urgent Pacing, Pacemaker Placement**	**• SYNCOPE (self-terminating)** **• SUDDEN CARDIAC DEATH (prolonged)**

Sketchpad

Practice!

On your worksheets,
identify heart blocks
and ventricular rhythms
and indicate their rates

Ischemia monitoring (ST-T changes)

Ischemia/Infarctions Diagnosed only with 12-lead ECG

>2 mm depression = ischemic

T wave inversions

i2chemmic

L

Other ECG findings

PEA (Pulseless Electrical Activity)

Asystole / Flatline

no electrical activity
determine if pulseless
check all connections/other lead views

Electrical Alternans

low volt + tachycardia + Alt. QRS amp.
triad of massive pericardial effusion
❤️ swinging inside fluid-filled pericardium

Drips & Drugs

Pressors

*Dopamine (3-10, or 10-20 mcg/kg/min)
*Norepinephrine (0.01-1 mcg/kg/min) *potent
*Epinephrine (0.04-1 mcg/kg/min)
Phenylephrine (0.05-8 mcg/kg/min)
*Dobutamine (0.04-1 mcg/kg/min)
*Vasopressin
*Milrinone

**Inotropes (hard/soft), Chronotropes (slow/fast)*

Sedation

propofol
benzodiazepines
opioids, e.g. morphine, fentanyl
ketamine
tranquilisers, e.g. haloperidol (haldol)

/NM blockade (paralysis)

Vecuronium, Rocuronium, Pancuronium

Antiarrhythmics

Adenosine, Digoxin, Magnesium Sulfate
Amiodarone
Atenolol, Metoprolol, Esmolol (BB)
Lidocaine, Phenytoin
Verapamil, Diltiazem

Antihypertensive

Esmolol, Nicardipine, Sodium Nitroprusside

Take-Away!

Rely on ECG knowledge to assess or anticipate changes in cardiac output but the <u>Patient</u> - not the monitor - is the gold standard.

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