Electrolyte Physiology

Something in the way she moves me...
Electrolyte Movement

- CONCENTRATION GRADIENT
- ELECTRICAL GRADIENT
- DRIVING FORCE
- NERNST NUMBER (E-ion)
- CONDUCTANCE (G-ion)
- PERMEABILITY
  - CHANNELS: small ions
  - PORES: medium-sized molecules (sweat)
  - TRANSPORT PROTEINS
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- **Depolarize**: to become positive from baseline
- **Overshoot**: more positive than the threshold potential
- **Repolarization**: to become negative from a positive potential
- **Hyperpolarization** (or undershoot): to become more negative than baseline potential
Sodium Channels

Cyto

PLASMA

gNa

↑gNa

G-Na

INACTIVATED

RESET
DEP

$\frac{0}{gNa}$

REP

$\frac{3}{gk}$

\[ \frac{4}{gNa} \]

AUTOMATICITY
\[ P_{\text{seg}} \xrightarrow{Q} S_{\text{ST seg}} \xrightarrow{T-U} \]

\[ \text{PR int} \qquad \text{QT int} \]

\[ \text{Height} = \text{Voltage} \]

\[ \overline{\text{Width} = \text{Duration}} \]
HEART BLOCKS

- NORMAL PR-interval: <0.2sec
- FIRST DEGREE HEART BLOCK: fixed and prolonged PR-interval
  - Problem is AT the SA node or BETWEEN the SA node and the AV node
  - NO treatment necessary
  - Speeding up the heart rate (exercise) will make the block disappear
HEART BLOCKS, cont

- SECOND DEGREE HEART BLOCK
  - MOBITZ 1: progressive lengthening of PR-interval until QRS is dropped
    - Early ischemia at the AV node
    - Also called WENCKEBACK’S
    - Put in pacemaker if symptomatic; do nothing if asymptomatic
MOBITZ II: PR-interval is normal; QRS complexes are dropped erratically

- Late ischemia at the AV node
- Some cells are negative; some cells are positive
- ALL must have a pacemaker
HEART BLOCKS, cont

- THIRD DEGREE HEART BLOCK
  - COMPLETE AV DISSOCIATION
  - AV-node has INFARCTED
  - P-waves and QRS complexes have NO relationship
  - ALL must have a pacemaker
QRS COMPLEXES

- Premature ventricular complex (PVC)
  - No P-wave; wide QRS complex; a pause following the QRS complex
  - **BIGEMINY**: A PVC every other beat
  - **TRIGEMINY**: A PVC every third beat
  - **VENTRICULAR TACHYCARDIA**: three or more consecutive PVCs with a minimum heart rate of 150
  - **VENTRICULAR FIBRILLATION**: NO recognizable QRS complexes
VENTRICULAR TACHYCARDIA

- IF PATIENT STABLE: treat with medication
- IF PATIENT UNSTABLE:
  - SHOCK with 200 joules
  - SHOCK with 300 joules
  - SHOCK with 360 (max) joules
  - LIDOCAINE
  - SHOCK
  - BRETYLIUM or AMIODORONE
VENTRICULAR FIBRILLATION

- **EPINEPHRINE**
- **TREAT LIKE VENTRICULAR TACHYCARDIA**
ATRIAL ARRHYTHMIAS

- Premature atrial contraction (PAC)
- Multifocal atrial tachycardia
- Paroxysmal supraventricular tachycardia
- Atrial flutter
- Atrial fibrillation
  - If ACUTE and STABLE: treat with medication
  - If ACUTE and UNSTABLE: DEFIBRILLATE
  - If CHRONIC: treat medically; put on coumadin
  - May defibrillate after minimum 2 weeks on coumadin

- TX: use synchronized button
ELECTROLYTES AFFECT DEPOLARIZATIONS

- FOUR SPECIALIZED MEMBRANES
  - NEURONS
  - SKELETAL MUSCLES
  - SMOOTH MUSCLES
  - CARDIAC MUSCLE
    - ATRIUM: uses calcium to depolarize
    - VENTRICLE: uses sodium to depolarize; uses intracellular calcium to contract; depends on extracellular calcium to trigger off intracellular calcium release
HYPERMAGNESEMIA

- LESS LIKELY TO DEPOLARIZE
- AFFECTS CALCIUM AND POTASSIUM
- GETS IN THE WAY OF SODIUM
- TX: IV normal saline; loop diuretic
HYPO MAGNESEMIA

- MORE LIKELY TO DEPOLARIZE
- AFFECTS CALCIUM and POTASSIUM
- AFFECTS all KINASES
- TX: magnesium sulphate
HYPERCALCEMIA

- LESS LIKELY TO DEPOLARIZE everywhere except the atrium (more likely)

- SMOOTH MUSCLE: initially less likely (blocks nerve) to depolarize, then more likely to CONTRACT (due to second messenger systems)

- TX: IV normal saline; loop diuretics
HYPOCALCEMIA

- More likely to depolarize everywhere except the atrium (less likely)
- Will affect second messenger systems
- Smooth muscle: initially more likely to depolarize (nerve fires more) followed by less likely to contract (affects second messenger systems)
HYPERKALEMIA

- Initially MORE LIKELY TO DEPOLARIZE
- Potassium will flow into the cell, taking the membrane potential closer to threshold
- Potassium gets trapped INSIDE the cell during repolarization; repolarization therefore takes longer > LESS LIKELY TO DEPOLARIZE
  - Peaked T waves
  - Widened T waves
  - Prolonged QT interval
    - Predisposes to arrhythmias
HYPOKALEMIA

- LESS LIKELY TO DEPOLARIZE
- Potassium will rush out of the cells, making them more negative
  - Cells repolarize even faster
  - Cells repolarize too much
    - Narrow T waves
    - Flat T waves
    - Flipped and inverted T wave
    - The U wave (exaggerated flipped T wave)
HYPERNATREMIA

- MORE LIKELY TO DEPOLARIZE
- SODIUM rushes into the cells, making them more positive
- After sometime, the NA-K ATP-ase kicks into high gear, making the cells more negative (less likely to depolarize)
- TX: IV normal saline; correct slowly
HYPONATREMIA

- MORE LIKELY TO DEPOLARIZE
- SODIUM will now leak out of a cell by Na-K exchange
- When calcium leaks INTO cell in exchange for sodium leaking OUT, cells become more positive
- TX: IV normal saline; correct slowly
  - Use 3% saline if sodium under 120 with symptoms
  - Use fluid restriction if hyponatremia due to SIADH
Hyponatremia
The End: Turn off the lights
Antiarrhythmics

You’re blocking my way!!!
Class 1: Na channel blockers

- 1a
  - Quinidine
  - Procainamide
  - Disepyramide

- 1b
  - Lidocaine
  - Tocainide
  - Mixelitine
  - Phenytoin

- 1c
  - Encainide
  - Flecainide
  - propofenone
Class II: Beta Blockers

- All end in –lol
- Specific beta 1: begins with A thru M, but NOT L or C
- Blocks B-1 and B-2: begins with N thru Z, including L and C
Class II: Beta Blockers

- Propanolol
- Esmalol
- Sotalol
- Timalol
- Butexalol
- Labetalol
- Carvedilol
- Acebutalol
- Atenalol
- Pindalol
Class III: K Channel blockers

- Napa (from procainamide)
- Sotalol
- Bretylium
- Amiodorone
Class IV: Ca Channel blocker

- Verapamil
- Diltiazem
- Nifedipine
- Nicardipine
- Nimodipine
- Femlodipine
- Amlodipine

- Quinidine
- Procainamide
- Phenytoin
IF YOU PLAY WITH LYTES...
You may go down
IN FLAMES