The Nightmares Course

Session 2:

Integrative and Enhanced Difficulty Scenarios

Scenarios by: Filip Gilic, CCFP-EM

Scenario layouts by: Ana Blagojevic, MD

Kristin MacLeod, MSc

Queen's University

Department of Family Medicine

Note to Instructors:

Welcome to the 2nd session of the Nightmares Course. In the initial session, we practiced the basic skills and then full scenarios that focused on a particular resuscitative topic. Now, we push the realism by facing the resident with scenarios where they don't know what kind of problem the patient will have. The aims of this session are threefold:

- **To simulate important scenarios that do not fit neatly into one of our core topics, such as DKA and hyperkalemia**
- **To practice problem solving where elements of several core topics are simultaneously present**
- **To keep the residents operating at a high level of cognitive load**

To this end, we provide two levels of difficulty- Regular and Enhanced Difficulty. Each resident can choose his or her level of difficulty but stress that the aim of the session is for them to be at the edge of their cognitive capacity. If they are flying through the regular strength ones, do not hesitate to switch to the tougher ones.

Case 1: Hyperkalemia due to obstructive renal failure

John Sutton

82 year old man with weakness for a week and progressive shortness of breath over the last 2 days, severe this morning

No cough, sputum, fevers or chest pain.

If prompted will tell about slight suprapubic discomfort

If prompted will tell that he hasn't eaten or peed much in a few days

PMH: CABG 7 years ago (no MI), HTN, BPH

Meds:

Flomax, HCTZ, Ramipril, ASA

Conscious but weak

Pupils 3mm reactive

Chest clear, heart sounds normal

Weakness in all limbs, can barely lift them up

Suprapubic tenderness

Initial vitals:

BP 95/65

HR 80

RR 18

O2 sat 88%

T 36.7

EKG:

Hyperkalemia

max severity

ON DEMAND:

Labs

CBC: 11/115/350

lytes: 135/8.2/105

creat: 1980

CK, Trop: 65, 0.11

VBG: pH 7.42/pCO2 52/HCO3 30

Foley drains 1.2 L if inserted. Urine negative for leuks and nitrites

IV fluid bolus: BP goes to 110/80, HR 80

O2 NP or NRB: sats to 100%

CaCl or Ca gluconate 1g IV: QRS narrows into normal QRS- sinus with frequent PVCs on monitor

No Calcium given by minute 5:

HR 20, BP undetectable, unresponsive

If no Calcium given in another 2 minutes:

HR does not respond to pacing or drugs and patient dies

Insulin drip 0.1 u/k and **Ventolin** 10 mg neb:

HR 95, PVCs reduced

Sodium Bicarbonate:

No change in status

Needs urgent dialysis. Internal Medicine or Nephro will take him urgently if contacted

Learning objectives:

1. Recognition of hyperkalemia
2. Need to stabilize the membranes with Calcium
3. Shifting of K with insulin and Ventolin
4. Need for urgent dialysis

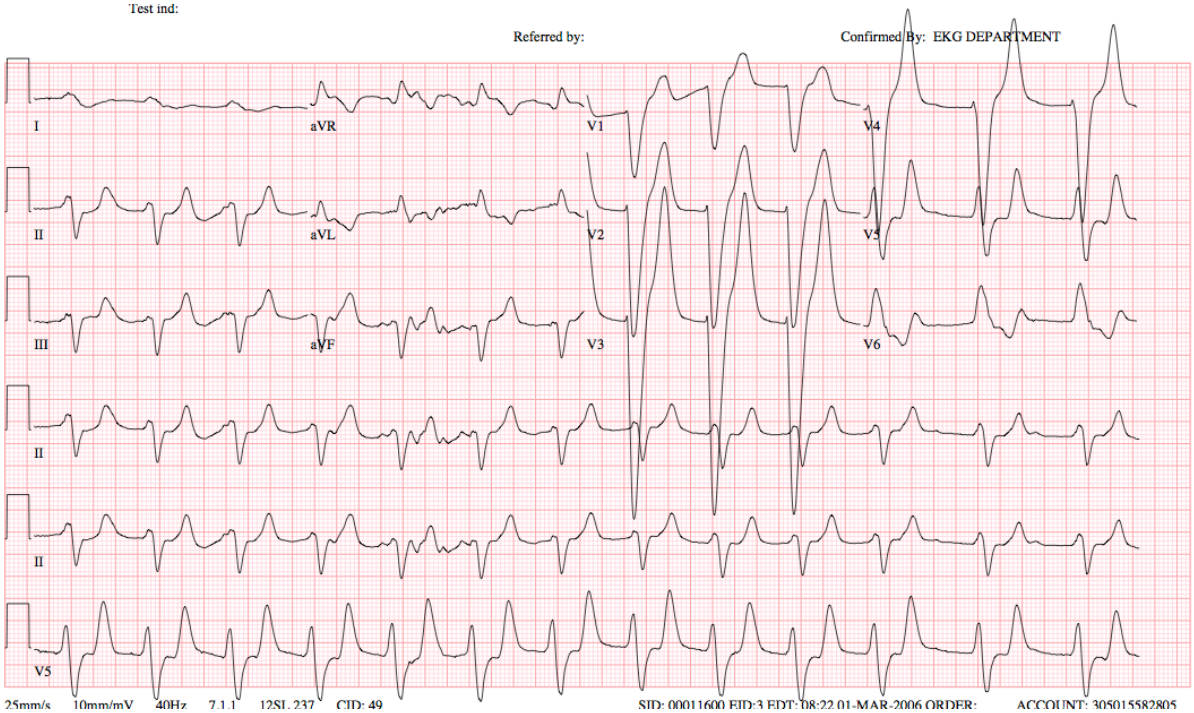
Pro tips:

1. Think of Hyper K if wide and
    a. Too slow to be VTach
    b. Does not look like LBBB
2. Sodium Bicarb is only useful if hyperkalemia is present together with acidosis, otherwise it has no measurable effect
3. If you do need to use Bicarb, can give 2-3 amps and then make a drip using 3 amps in a liter of D5W (each amp has 50 meq sodium, so 3 amps make it isotonic), run at 200-250 and hour.
4. Ventolin dose is twice the dose normally used for nebulising

Test ind:

Referred by:

Confirmed By: EKG DEPARTMENT



25mm/s 10mm/mV 40Hz 7.1.1 12SL 237 CID: 49

SID: 00011600 EID:3 EDT: 08:22 01-MAR-2006 ORDER:

ACCOUNT: 305015582805

Case 2: DKA with abdominal pain and SOB

John Beddard
20 yo male, brought in by mother because of SOB and vomiting. Feeling unwell for a week. Nausea after meals started 5 days ago, intermittent vomiting over last 2 days; more than 10x yesterday. Today, breathing **fast and deep**
PMH: None.

Meds: None.

GCS 13, but confused at times
PERLA 3 mm
Neurological exam intact
Chest clear on auscultation, abdo mildly tender, finger prick glucose is HIGH

Initial vitals:
BP 100/80
HR 120
RR 32
O2 sat 98%
T 36.6

EKG:Sinus tach

ON DEMAND:
Labs
CBC: 14/145/350
lytes: 125/5.2/95
creat: 125
glucose: 35

VBG
pH: 7.32
pCO2: 25
HCO3: 15
pO2: 45

Fluid bolus containing IV hydration

Insulin drip
-0.1 u/kg/hr +/- bolus of 0.1 u/kg

Frequent lytes monitoring

Potassium supplementation
in IV when K falls below 5 and he starts urinating

Repeat blood work at 2 hr
Glucose: 24.5
Lytes: 130/4.2/105
VBG:
pH 7.15
pCO2 30
HCO3: 12

When trying to place the patient in the ICU, the ICU physician argues that since the patient had an essentially normal pH, he does not need an ICU but just a Family Medicine hospitalist bed. Will only agree to take the patient if the resident presents a convincing case

Learning objectives:

1. Recognition of diabetic ketoacidosis
2. Recognition of deep and fast breathing as a sign of metabolic acidosis
3. Recognizing risks of low K+
4. Therapeutic Insulin dosing

Pro tips:

1. DKA is not defined by low pH. In fact, severe DKAs can often present with initial pHs close to normal.
2. The primary metabolic problem is a metabolic acidosis caused by the body switching to ketone production in the absence of endogenous insulin. This will cause a compensatory respiratory alkalosis (breathing fast and deep) which will nudge the pH closer to normal. The profound dehydration from sweating, glycosuria, vomiting and poor oral intake can then cause a metabolic alkalosis (losing free water, but retaining bicarbonate) which can push the pH close, or back to fully normal.
3. Anion gap is the most reliable measure of DKA presence and severity. However, to calculate it accurately, we must account for fictitious lowering of Na serum concentration by the high serum glucose concentrations which bring more free water into the intravascular space, diluting the sodium.
4. Correction is to increase the measured Na by 3 points for each 10 glucose above 15. In the case, glucose is 35. That is 20 points above 15, so we should raise the sodium from the measured 125 to 131 (6 points). This gives us an anion gap of 21 (131-95-15).
5. Correction of metabolic contraction alkalosis by re-hydration will often unmask the true extent of metabolic acidosis
6. Potassium if artificially elevated due to lack of insulin and acidosis, even though the body potassium stores are depleted due to high urinary output due to glycosuria. Add potassium to IV fluids once K is within normal range and the patient is producing urine.

Case 3: Rapid atrial fib >48 hours requiring rate control

Mr John Billiard
68 yo male, presented with a sense of his heart beating fast.
PMH: DM2, HTN, high cholesterol, ex-smoker (quit 5 years ago).
Chest discomfort over last 3-7 days, sometimes worse with exertion. No chest pain right now. No SOB. Never had chest pain before. Previous palpitations possible.

Meds:
Metformin 500 mg
Lipitor 20 mg
MCTZ 50 mg
Metoprolol 25 mg

Initial vitals:
BP 140/90
HR 180
RR 15
O2 sat 95%

EKG: Afib

ON DEMAND:
Labs
CBC: 8.3/145/350
lytes: 135/4.0/105
creat: 105
CK/Trop: 45/0.02

After 3-5 min: If no action taken to decrease HR, BP drops to 100/40, O2 sat drops to 90%, patient becomes confused.

HR not controlled by 10 minutes, crushing chest pain

Amiodarone/Procainamide or Cardioversion
-converts but has stroke 48 hours later

Metprolol /Diltiazem

-If patient receives <10 mg Metprolol or <30 mg Diltiazem total, BP 120/80 and HR 120

-If patient receives >10 mg of Metprolol or >30 mg Diltiazem OR Metprolol <10mg PLUS <30 mg, BP 120/80, HR goes to 90. Patient feels fine.

-If patient receives >10 mg Metprolol PLUS >30 mg Diltiazem, HR drops to 50 and patient feels nauseas and weak.

Adenosine
-HR slows for 30 sec to atrial fib 110
-patient feels nauseated with increased chest pressure during that time

Learning objectives:

5. Recognition of a fib requiring rate control
6. Recognizing stable vs. unstable
7. Recognizing contraindication to rhythm control (duration>48h)
8. Recognizing when adequate rate control has been established (resting HR<110)

Pro tips:

1. Established Afib is often quite hard to rate control.
2. Significant amounts of rate-control medications might be needed
3. Co-administration of BB and CCBs is an option. Most textbooks will tell you not to do it, but as long as you respect the peak serum concentrations (20 min for Metoprolol, 2 min for Diltiazem) and space them out, you should be fine. If you overshoot, give 0.25-0.5gr of Calcium to reverse the CCB

Case 4: A fib with Wolff-Parkinson-White syndrome

John Branson
35 yo male. Racing heart for 1 hr; started in a meeting. Never had this before, no precipitations. No drugs, but drank a lot of alcohol last night.
PMH: None.

Initial vitals:
BP 100/65
HR 180
RR 20
O2 sat 95%

EKG: sinus WPW with couplet PVCs with slider to max

No initial distress

BP untreated over 5 min: BP drops to 70/50, patient becomes weak, dizzy, slightly confused

ON DEMAND:
EKG

Sinus tachy with PVCs set to maximum

EKG from previous visit for abdo pain available below

LABS
CBC: 10/140/350
lytes: 135/4/110
creat: 45
CK/Trop: 45/0.02

Procainamide:
(1 g over 1 hr) or **cardioversion**, converts to sinus.

Amiodarone:
->VFIB
-responds to defibrillation by going to sinus if defib done <2 min. Otherwise, goes to asystole.

Adenosine, Metprolol or Diltiazem:
-HR goes to 280 wide QRS irregular, BP to 60/40 and patient passes out.

Immediate cardioversion
-regains consciousness and converts to sinus

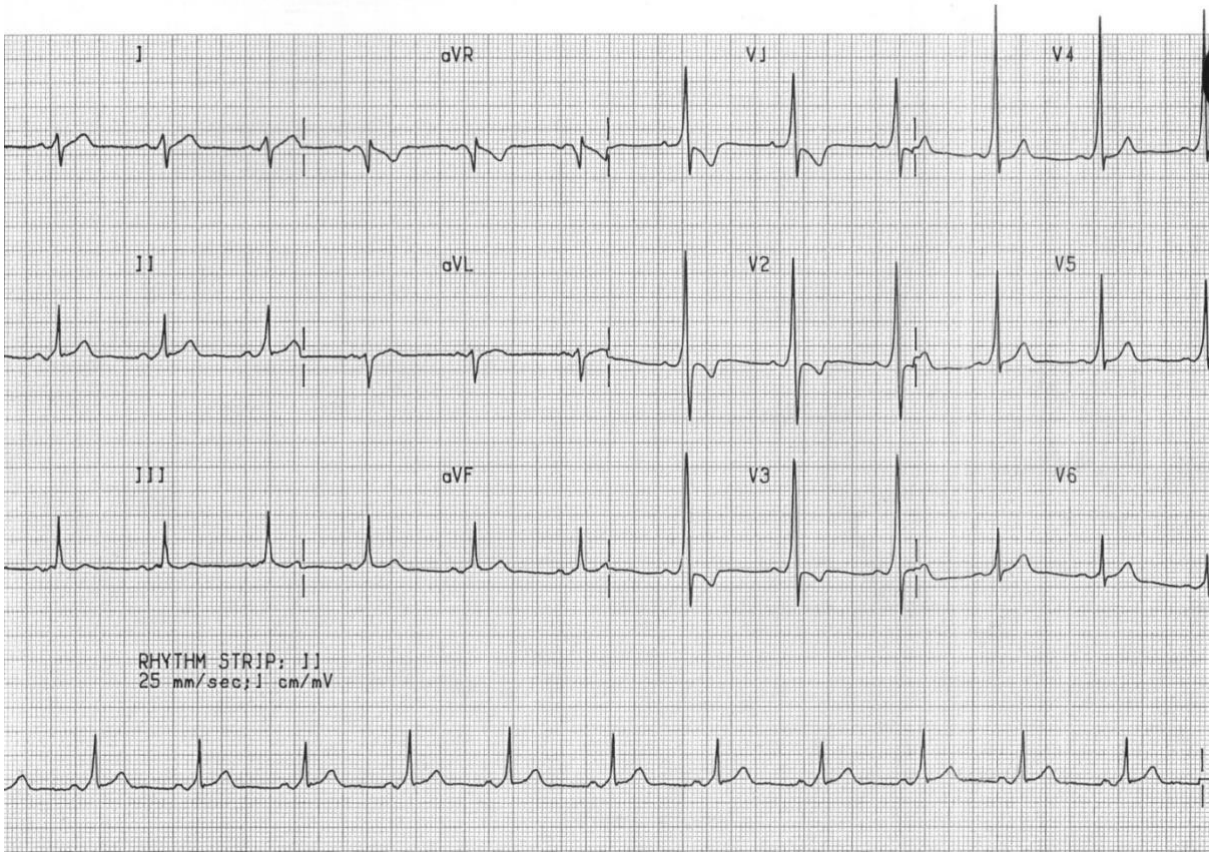
Not cardioverted:
->VFIB
-dies in 5 min

Learning objectives:

9. Recognition of WPW
10. Recognition of Afib with WPW
11. Need for cardioversion
12. Need to avoid AVN blockers

Pro tips:

1. WPW is congenital and usually diagnosed by early adulthood. Afib is more a disease of older people but for some reason 30% of WPW patients develop Afib even in early life
2. Procainamide or cardioversion are the only safe choices in WPW with Aifb. Amiodarone usually led to Vfib in a case series of EPS patients- probably because it has some AVN blocker properties
3. AVN blockers force conduction down the accessory pathway, making the ventricle try to match atrial rates of up to 600 pbm. It leads to VFib and death


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV

Case 5: Septic shock

Violet Dubble
92 yo female (ALC), admitted for failure to cope at home. Decreased eating and more lethargic than usual. Vomited after choking on last meal yesterday.
PMH: Dementia, diabetes (diet controlled), HTN, MI 20 yrs ago, CHF, osteoporosis, severe OA in knees (mostly wheelchair bound). Patient quite kyphotic.

Meds:
Galantamine
ASA
HCTZ
Nitro patch 0.4
Ramipril
Pantoloc
Naproxyn
Tylenol prn

Initial vitals:
BP 80/50
HR 110
RR 15
O2 sat 95%
T: 36.8

EKG: sinus

If no fluid after 3-5 min: BP drops to 70/45, HR 130

Still no fluids: loss of consciousness & cardiac arrest shortly thereafter

NS bolus 500 cc-1 L: BP improves to 89/55, HR 102

After fluids: Urine dip available, nitrites and leuks seen. CXR normal, WBC 3 with 85% neutrophils

If repeated: HR 98, BP stays the same

Nitro patch removed: BP improves by 10 systolic, 5 diastolic

Need **Dopamine/Phenyl infusion** to improve BP to 120/65

Antibiotics
-Cipro 400 mg IV q12h for urosepsis
-if undefined: Piptaz 4.5 g IV q8h or Ceftriaxone 2g until blood cultures and urine come back
-ongoing IV fluids NS

ON DEMAND

Labs
CBC: 3/115/250
lytes: 140/4.5/105
creat: 160
CK/Trop: 250/0.01

Urine
colour: turbid
spec grav: 1.030
pH: 7.2
nitrites: 2+
WBC esterase: 1+

Learning objectives:

13. Recognition of sepsis and septic shock (MAP<65)
14. Use of fluid boluses and pressors
15. Effect of nitroglycerine on BP
16. Rapid need for antibiotics

Pro tips:

1. Usual fluid deficit in sepsis is 4-8 litres
2. Delay in giving antibiotics in septic shock gives an 8% increase in mortality
3. Use pressors early if MAP<65 despite pressure-delivered fluid boluses of 2L+

Enhanced difficulty Scenario 1: Loss of consciousness due to heart block post- STEMI.

Josiah Needle
75 yo male. Presented to ER with episodic chest pains. Response team called to triage when he collapsed. Before collapsing, told nurse he's had episodes of weakness, presyncope and chest pain ~3 min/time over last 3 days. Never had them before.
PMH: HTN, cholesterol, smoker

Meds:
HCTZ 50 mg
Metprolol 50 mg
Lipitor 20 mg

-Pupils equal and reactive 3 mm
-Eyes open to pain, moaning to pain, hand moves towards painful stimuli

Initial vitals:
BP 80/40
HR 40
RR 6
O2 sat 82%
T 37.2

EKG: 3rd degree AV block

ON DEMAND:
Labs
CBC: 11/145/350
lytes: 140/5/100
creat: 65
CK/Trop: 65/3.5

BVM 100% O2: sats go to 88%
BVM/ETT: sats go to 98%

If not paced within 1-3 min
BP 60/40, HR 30, GCS 3

If still not paced in 1-3 min, patient dies.

If paced, BP 80/60, HR as set on pacer, RR 12, cognition improves to awake and confused

Need Dopamine support even with pacing. BP 120/60 (cardiogenic shock from STEMI 3 days ago leading to heart block and LV dysfunction)

Atropine transiently increases HR to 60 and BP to 100/60 for 1 min at a time

ACS treatment (ASA, Plavix, Heparin, ?TNK) does not change immediate situation

Patient needs **transvenous pacing and angiogram**

Learning objectives:

17. Recognition of shock due to bradycardia
18. Recognize the need to pace
19. Adjuncts to pacing (inotropes, anticholinergics)
20. Therapeutic use of ACS drugs

Pro tips:

1. 3rd degree AVB is always wide as it is a ventricular escape rhythm
2. Anticholinergics like atropine only affect the rate, inotropes like Dopamine increase contractility too
3. High Troponins and normal CK means he probably infarcted some days ago
4. TNK is most useful within 3 hours form onset of chest pain, and up to 12-24h. This patient has been going for days. Probably best he goes for PCI
5. Bradycardia in ACS is mostly due to infarction of SA and/or AV node. Usually supplied by RCA (80-90% of time)

Enhanced difficulty Scenario 2: Ruptured Ectopic pregnancy.

Meggy Ryan
18 yo female. Presents to ER with pain when she takes a breath, SOB and feeling faint. Woke up feeling like this, not experienced it before. Slowly getting worse all day.
PMH: no recent surgery, no history of leg or lung clots, immobilization or casts. No medical issues. No meds.

NOTE: set the bed up 60 degrees

-Sweaty, pale, extremely anxious
- Heart sounds normal, chest normal
- If lain flat the pain gets worse and moves to left shoulder
-Abdominal cramps for two days before this. Irregular periods; not sure when last one was. No prev pregnancy or STIs. No UTI symptoms.

***If residents query ectopic pregnancy, tell them one the doctors in the ER is licenced in bedside ultrasound and can perform if needed.**

Initial vitals:
BP 80/60
HR 140
RR 18
O2 sat 98%
T 36.7

EKG: sinus

IV fluid bolus: BP increases by 10 systolic and 10 diastolic

Dopamine 20 mcg/kg/min **or Phenylephrine** 300 mcg/min: main BP for 10 min

After 15 min: lose BP and patient goes asystolic unless given blood

4 units O neg blood to stabilize

ON DEMAND:
Labs
CBC: 12/65/320
lytes: 135/4/105
creat: 65
CK/Trop: 65/0.01

Urine pregnancy test positive

D-Dimer negative

Bedside ultrasound positive for free fluid.

Learning objectives:

21. Recognition of ectopic pregnancy
22. Need for fluid and pressors in undifferentiated shock
23. Recognize need for blood (or blood products) in hemorrhagic shock

Pro tips:

1. Real case. Thought it was a PE. Pain got worse when lying flat and went to shoulder which alerted us to possibility of free fluid in the belly
2. When patients are bleeding and in shock, they need blood fast. Saline just dilutes the clotting factors and Hgb and can be used as a temporizing measure only. Max 2L
3. If patient is bleeding and in shock, activate massive transfusion protocols so you can get FFP and platelets too
4. Options for blood (PRBC) are:
    a. O- (females of child bearing age)/O+ (everyone else). Immediately available but limited supply (usually 2-4 units)
    b. Type specific (ABO/Rh matched)- takes 10-15 min to get
    c. Fully cross-matched blood: detects minor antigens besides ABO/Rh. Takes 45 min to an hour to get

Enhanced difficulty Scenario 2: Pneumonia with hypotension.

Ed Dartmouth
72 yo male. Admitted2 days ago with pneumonia, wasn't improving and continued to need Tylenol for fevers. Nurse calls in middle of the night because she feels his breathing is getting worse.
PMH: HTN, cholesterol, MI 5 yrs ago, had stents (but no issues then)

Meds
Ramipril
Lipitor
ASA
Azithromycin and maintenance fluids since admission

EKG: Sinus tachycardia

-Laboured breathing, 2-word dyspnea
-GCS 13, eyes closed, open on command, slightly confused at times
-Sweaty and pale
-PERLA 4 mm
-Lung sounds: rales and crackles bilaterally
-Limbs cool and pale with weak pulses
-Exam otherwise normal

Initial vitals:
BP 80/60
HR 125
RR 35
O2 sat 78%
T 38.6

EKG: sinus

On Demand
Labs
CBC: 22/150/120 (yesterday 14/150/120)
Lytes: 140/4.5/9.3
CK/Trop: 250/0.01
Lactate: 8

ABG
pH 7.2, pO2 85, pCO2 60, HCO3 27 at FiO2 100%
ABG unchanged after 30 min-1 hr on BiPAP

100% O2: O2 sats increase to 82%

BVM/CPAP/BiPAP: O2 sats increase to 88%, RR decreases to 24, 4-word dyspnea

After 30 min, patient tires out and must be intubated

Fluid:
-No fluid given, BP drops by 10 systolic and 5 diastolic every 5 minutes
-If 500 cc - 1 L fluid given, BP stays same but doesn't improve

Phenylephrine 200 mcg/min: HR 120, BP 100/80

Dopamine 10 mcg/mg/min: BP increases to 100/80, HR to 140

Learning objectives:

24. Escalating FiO2 in hypoxic patient
25. Recognize need for PPV
26. Recognizing failure of NIPPV
27. Treatment of shock with fluids and pressors

Pro tips:

1. Pneumonia with septic shock and acidosis is the most likely scenario where NIPPV fails. Use it as a bridge to improve the situation but plan that you will have to intubate them
2. Don't intubate people when they are hypoxic if you can avoid it: time to desat from 90 to 0 is only 2 min in a sick patient and drugs need 1-2 minutes to kick in for RSI- makes for a very stressful intubation that is likely to fail. Optimize them with NIPPV first

Enhanced difficulty Scenario 4: Pulmonary Embolism/shock.

Cherie Gribeaux
24 yo female. Brought to ER by EMS—she collapsed while pouring coffee at work (Tim Horton's). Few blisters on left arm from hot coffee and her arm is red. Best friend/co-worker accompanying. Friend says she's "pretty sure" Cherie is otherwise healthy with no bleeds of any kind, but that Cherie has been SOB last 3-4 days that was getting worse. Cherie complained of chest pain before collapsing.

Meds: UNK
Allergies: UNK

- Pupils equal and reactive to 4 mm
-Does not open eyes to command or pain. Moans if painfully stimulated. Moves arm toward painful stimuli
-Breathing fast

On Demand
<u>Labs</u>
CBC: 12/145/350
Lytes: 140/4.5/105
Creat: 45
CK/Trop: 65/0.3

<u>ABG</u>
pH: 7.15
pO2 (FiO2 100%): 65
pCO2: 28
HCO3: 15

D-Dimer 3.5

<u>Initial vitals:</u>
BP 60/40
HR 120
RR 30
O2 sat 72%
T 36.7

EKG: sinus

O2:
-**if not given** within 3 min, patient desats to 60% and dies
-**if given** O2 only, sats 80-85% for 5 min, then patient desats to 60% and dies

Intubated:
BP 60/30, HR 120, RR 16 on vent, O2 88% with 100% O2 (BP decreases due to V/Q mismatch, exacerbated by positive pressure ventilation)

Need positive pressure ventilation with **BVM or ETT**

2 L fluid (for obstructive shock): BP goes to 80/60

Dopamine/Phenyl: BP goes to 100/70

IV Heparin 5000 u IV bolus, then a drip according to PTT nomogram

TNK: patient slowly improves over 1 hr

***Discussion with ICU/CritiCall suggests TNK is treatment of choice for hemodynamically unstable PE patients with suspected cardiogenic shock and no contraindications.*

Learning objectives:

28. Identifying priorities in undifferentiated hypoxia, altered LOC and shock
29. Need for aggressive treatment of BP with fluid and pressors
30. Rapid escalation of FiO2
31. Need for positive pressure ventilation
32. Heparin and TNK use in PE

Pro tips:

1. PEs come in three varieties: massive ones that kills you quickly, submassive ones that make you sick and leave you with RV damage down the line, and small ones that have very little ill effect unless there is re-clotting
2. Heparin does nothing for the current PE, it just prevents re-clotting
3. Absolute indication for TNK/t-PA in PE is sBP<90 for more than 15 min or requiring pressors

Enhanced difficulty Scenario 5: CHF with infarct and cardiogenic shock.

Muriel Down
75yo female. Admitted yesterday for CHF exacerbation. (Admitted to hospital after large Thanksgiving dinner sent her into moderate SOB. Got better overnight until the chest pain started.) Was treated with IV Lasix in ER. Nurse calls because patient is complaining of SOB and chest pain.
PMH: CHF, HTN, smoking, diabetes, 2 MIs previously (last 2 yrs ago, not sure if stented either time.)

Meds:
HCTZ
Metformin
Insulin (26 units morning and 12 units night)
Lipitor
Ramipril
Lasix (normally 40 mg daily po, but received 80 mg IV yesterday)

EKG at admission: sinus at 75 and no ST depression
-Sweaty, anxious, significant work of breathing, complaints that chest pain started 1 hr ago and is getting worse. No recent chest pain
-awake and alert, pupils 4 mm, reactive
-Chest: crackles bilaterally
-JVP: high
-limbs pale with weak pulses, bilateral leg edema 2+

Initial vitals:
BP 70/50
HR 110
RR 30
O2 sat 80%
T 36.7

EKG: sinus, lateral ST depressions

ON DEMAND

Labs
CBC: 8.5/150/400
Lytes: 140/4.5/105
Creat: 120
Creat this morning: 86
CK/Trop: 750/1.2
CK/Trop on admission: 160/0.04

ABG
pH 7.32, pO2 150, pCO2 35, HCO3 24

100% FiO2: O2 sats increase to 86%, with same RR and work of breathing

PPV, BVM or BiPAP: O2 sats increase to 92%, RR decreases to 22, work of breathing progressively decreases

Lasix IV (at least 60 mg): Patient slowly improves over 30 min, but no improvement apparent for first 20 min

Fluid

-if given >250 cc, O2 sats decrease by 10%, RR increases by 5 and breathlessness increases

-if given >1 L, O2 sats decrease by 30%, RR increases by 10 and patient crashes

Must be intubated immediately

Should be given ASA, Plavix or low molecular weight Heparin

Dopamine at 10 mcg/kg/min, HR 140 and BP 100/70

Phenylephrine at 100 mcg/min, HR 140 and BP 50/30. Chest pain increases. If not discontinued in 5 min, patient goes into VFIB

***If called, Cardiologist requests fax of ECG. Says it's not a STEMI but if chest pain doesn't settle within 30 min to send her over for an angiogram*

Learning objectives:

33. Recognition of pulmonary edema and cadiogenic shock
34. Use of NIPPV in pulmonary edema
35. Effect of fluid overload in cardiogenic shock
36. Need for inotropes vs vaspressors in cardiogenic shock

Pro tips:

1. Cardiogenic shock is the one type of shock where fluid will not be very useful and can be harmful is there is evidence of pulmonary edema
2. You need inotropes to improve contractility
3. Pure vasopressors like Phenylephrine will only make it harder for the heart to pump blood out because of increased SVR (and afterload) without an increase in contractility
4. More contractility means more cardiac O2 demand but it also means better perfusion of the coronaries (remember they are part of systemic circulation and their filling pressure is directly related to diastolic BP)
5. PPV usually improves pulomary edema rapidly
6. Cannot use Nitro in a hypotensive patient with CHF