

## Case Presentation Pulmonary Issues

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## Case Presentation

- 16 yr. old male for third molar surgery
  - 6' 3" 235 lbs
  - athletic: lean – muscular high school football player
- Meds: denies Allergies: denies
- No Surgical history No hospitalizations
- PMH: unremarkable – non contributory
- ASA 1 Mallampati 1
- Treatment plan: excision with general anesthesia in the office

## Monitors for Anesthetic

- Automatic NIBP Q 5 minutes
- Continuous ECG, heart rate, and SpO<sub>2</sub>
- Continuous capnography + stethoscope
  - hear breath sounds
  - see continuous respiratory rate
  - see CO<sub>2</sub> waveform and numerical value
- use both devices because in open airway techniques
  - not unusual to lose capnograph wave but still hear respirations
- Clinical observation by anesthesia team

## Pre operative

- IV in Right ACF: 22 gauge angiocatheter
- IV fluids: LR 500 ml bag
- Patient semi reclined in chair
- Oxygen by nasal cannula 2 L/min
  - CO<sub>2</sub> sampling port + oxygen delivery
- Patient is comfortable, alert, and awake at present

## Vital Signs

- Blood pressure 110 / 60
- Heart rate 50 and regular
- ECG Sinus bradycardia
- Respiratory rate 15 breaths per min.
- SpO<sub>2</sub> 99 – 100
- EtCO<sub>2</sub> 42
- Stethoscope Clear & unlabored breath sounds

## Planned Office Induction

- Midazolam IV 3 mgs – 5 mgs
- Fentanyl IV 50 mcg over 2 doses
- Dexamethasone 8 mg IV
- Glycopyrrolate 0.2 mg IV
- Propofol infusion pump
  - bolus set at 500 mcg/kg ( 300 to 500 mcg/kg )
  - infusion rate set at 120 mcg/kg/min
- Ketamine bolus 25 mg IV with 1<sup>st</sup> Propofol bolus dose
  - repeat ketamine 25 mg IV as needed for 1 to 2 additional doses

## Intra operative Event

- No breath sounds heard from stethoscope
- No tracing on capnograph
  - alarm sounds & is shut off
- Assistant claims patient is breathing
  - sees rhythmic movement of chest & abdomen
- SpO<sub>2</sub> is dropping 99 – 94 - 91

## Monitor



## Differential Diagnosis

- Need to make a diagnosis and provide treatment
- Upper airway obstruction?
  - tongue, tongue + hypertrophic tonsils, other soft tissues
  - inadequate airway management requiring readjustment
- Foreign body aspiration?
  - gauze packing or surgical debris
- Aspiration pneumonitis?
  - irrigation or blood
- Bronchospasm?
- Laryngospasm?

## Laryngospasm

- Most likely diagnosis = laryngospasm
  - light anesthesia
  - patient was moving & moaning in chair
  - surgical stimulation
  - elevator placed on tooth
  - irrigation, blood, and saliva
  - surgeon did not use throat pack
- Diagnosis is made
  - need to provide appropriate intervention

## Management of Laryngospasm

- General management
  - suction the oral cavity and posterior pharynx
  - pull the tongue forward
  - chin lift – jaw thrust or
    - turn the head to one side and pull the tongue forward or jaw thrust
  - laryngospasm notch pressure
- If not relieved by above
  - face mask oxygen + positive pressure
- Spasm is broken, nasal cannula O<sub>2</sub> applied, and SpO<sub>2</sub> returns to normal over 2 minutes
  - patient stable
  - deepen the anesthetic and proceed with surgery

## Case Presentation

- 2 of the impacted teeth are excised
- Patient starts to buck in the chair and you hear crowing
  - another episode of laryngospasm
- Positive pressure oxygen is used to break spasm
  - spasm will not break over 45 seconds
- Succinylcholine is given
  - 20 mgs IV push
  - spasm breaks: total spasm time 90 seconds
  - ventilations by face mask continue until ventilations are spontaneous

## Case Presentation

- Nasal cannula is repositioned under nose
- Surgery is resumed and completed
  - no further intra operative events
- Vital signs are stable & patient is placed in your recovery area
  - IV is left in place
  - Monitors left in place
  - Oxygen is discontinued because patient's SpO<sub>2</sub> are above 97 on room air
  - Patient is stable

## Recovery Area

- 5 mins. post operative
  - patient has sudden onset of dyspnea & tachycardia
  - patient is anxious & responsive to command
  - SpO<sub>2</sub> starts to drop to 90 on room air
- What is your intervention?????

## Intervention

- Airway is opened
  - oxygen is applied with non rebreathing face mask 6 L/min
  - hear ventilations but labored
  - stethoscope & listen to lungs
- Hear rales over the lung fields
- What is the diagnosis????
- Diagnosis most likely
  - aspiration pneumonitis
  - post obstruction pulmonary edema – negative pressure pulmonary edema

## Review

- Laryngospasm occurred during surgery caused by
  - light anesthesia
  - saliva, blood, or irrigation irritating vocal cords
- Glycopyrrolate should be considered
  - decrease saliva
  - prevent saliva when ketamine is used
  - dose: 0.2 mg IV pre-operative

## Review

- Pulmonary edema after laryngospasm
  - rare event overall
  - more likely to occur to adolescents & young adults
  - not due to fluid overload in healthy patients
  - can be life threatening if left untreated
  - patients need transported to hospital for treatment
  - usually resolves after 24 hours unlike aspiration pneumonitis

## Post Obstruction Pulmonary Edema

## Post obstructive Pulmonary Edema

- Post obstructive pulmonary edema POPE
- Negative pressure pulmonary edema NPPE
- Laryngospasm induced pulmonary edema
- Acute obstructive pulmonary edema
- Post laryngospasm pulmonary edema PLPE
- Athletic pulmonary edema
- Acute post operative pulmonary edema

## Epidemiology of NPPE

- Rare event but can be life threatening if untreated
  - irreversible hypoxic cerebral damage
  - death
- Mortality rate without proper management
  - 10 to 40%
- Mortality rate when managed properly
  - ~ 2%

## Epidemiology of NPPE

- Men have higher incidence: 2 times greater risk
- Athletes have a higher risk
  - well developed thoracic muscles
  - generate very high negative intra-thoracic pressures against an airway obstruction
  - “athletic pulmonary edema”
  - anabolic steroids increase muscle mass
- Elderly are low risk due to muscle atrophy
- Neonates & infants very low risk
  - unless ET tube is kinked or obstructed

OOO 95: 2 2003

## Epidemiology of NPPE

- No case reports for moderate sedation
  - monitored anesthesia care ( MAC )
- Moderate sedation patients that require urgent airway intervention
  - ET tube or LMA
  - can develop NPPE
- Any patient undergoing surgical manipulation of the upper airway is at risk

## Patients At Risk

- Difficult intubations
- Nasal, oral, or pharyngeal surgery
- Obesity & OSA
- Short, thick neck patients
- Perioperative opioids
- Vocal cord dysfunction or paralysis
- Premature extubation

Perez. Current Surgery. 61: 5, 463-465. 2004

## Non intubated Cases

- 2 most common causes of UAO in mask general anesthesia
  - tongue & laryngospasm
- Oral surgery cases
  - oral secretions, blood, irrigation, debris, & light anesthesia in the presence of stimulation or pain

Pediatric Emergency Care Vol 21 November 2005

## Types of POPE

- Type I
  - forceful inspiratory effort against an upper airway obstruction
  - adults: 50% cases are due to laryngospasm
  - children: 75% cases are due to croup & epiglottitis
  - no chronic airway disease is present
- Type II
  - develops after surgical correction of a chronic, partial airway obstruction
    - adenotonsillar hypertrophy & airway tumors
    - OSA surgical correction

## POPE Type I

- **laryngospasm:** 50% of adult cases
- hanging or choking a victim
  - not an approved anesthetic technique
  - *just like in the old days of 100% nitrous, but it leaves bruises*
- epiglottitis and croup infections
- foreign body airway obstruction – displaced gauze
- post extubation subglottic edema
- constriction of ET tubes
- post extubation laryngospasm
- airway manipulation
- OSA

## Duration of Laryngospasm

- studies have shown that 10 to 30 seconds of UAO is all that is needed to precipitate pulmonary edema

Ped Emerg Care. 21: 2005

## Physiology of Normal Inspiration

- Intercostal muscles & diaphragm contract
- Diameter of thorax increases
- Intra-thoracic pressures decrease
  - go from + 3.5 cm to - 6.0 cm
- Alveolar pressure decrease from atmospheric pressure to - 3.0 cm
- Create a pressure gradient of 3 cm to allow air to pass into lungs

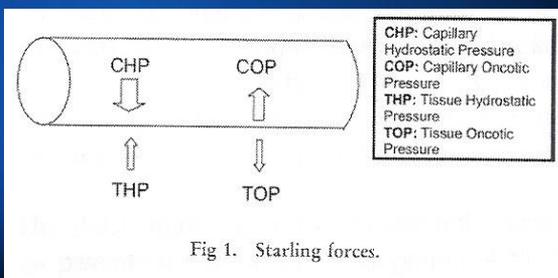
## Physiology of Normal Exhalation

- Inspiratory muscles relax
- Thorax size decreases
- Intra-thoracic pressures increase from - 6.0 cm to + 3.5 cm
- Alveolar pressure equilibrates to atmospheric pressure
- Air flows from lungs

## Starling Forces

- 4 physiologic forces that govern movement of fluid across capillary walls
- 2 of the forces favor movement of fluid out of the capillary
  - capillary hydrostatic pressure ( CHP )
  - tissue oncotic pressure ( TOP )
- 2 of the forces oppose outward flow of fluid
  - capillary oncotic pressure ( COP )
  - tissue hydrostatic pressure ( THP )

## Starling Forces



J. PeriAnesth Nursing, 22(2): 132-135, 2007

## Pathophysiology of NPPE

- 4 events occur during NPPE
  - 1<sup>st</sup> is the generation of large negative intra-thoracic pressures
  - 2<sup>nd</sup> hypoxia develops
  - 3<sup>rd</sup> hyperadrenergic state
  - 4<sup>th</sup> possible stress failure of the alveolar capillary membranes
    - leads to post obstruction pulmonary hemorrhage

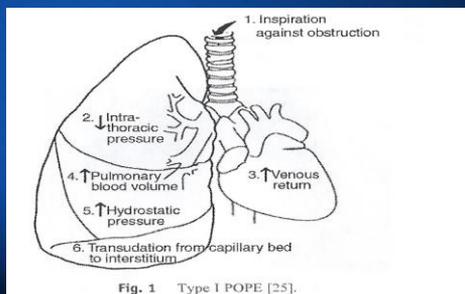
## Pathophysiology of Type I NPPE

- Mueller Maneuver
  - forceful inspiration against a glottic obstruction
  - causes baseline negative intra thoracic pressure of -4.0 cm of water to drop dramatically
  - negative pressures can approach -140.0 cm of water
- Increase venous return to right side of the heart
- Decreased cardiac output secondary to increased afterload

## Pathophysiology of Type I NPPE

- Overall end result is
  - increased pulmonary blood volume and pressure
  - increased pulmonary hydrostatic pressure
  - low protein fluid leaks out of capillaries into the lung interstitial tissues
    - develop pulmonary edema
  - if capillary walls break down
    - develop pulmonary hemorrhage & hemoptysis = rare event

## Pathophysiology of Type I NPPE



J. Critical Care. 25: 508e1-508e5. 2010

## Type I NPPE

- Fluid accumulates in interstitial tissue & alveoli
  - gas exchange deteriorates
  - develop hypoxia
- Hypoxia causes release of catecholamines
  - systemic hypertension develops
  - afterload is increased
  - decrease in myocardial contraction from hypoxia
  - may also alter the integrity of the capillary walls
  - results in more pulmonary blood volume and pressure
    - more fluid in lung tissues

## Type I NPPE

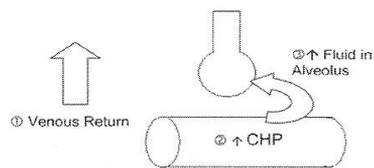


Fig 2. Negative pressure pulmonary edema mechanism.

Increased capillary hydrostatic pressure causes fluid to leak into the interstitial tissues and alveoli

J. Perianesth Nursing. 22(2): 132-135. 2007

## Type I NPPE

- Hyperadrenergic state
  - increases blood return to the heart
  - increases the afterload
  - possible myocardial ischemia which alters left ventricular function
- Stress Failure & Hemorrhage
  - causes breaks in capillary walls
  - increases fluid release
  - develop hemoptysis & focal hemorrhage in lungs
    - rarely seen event
    - severe cases of NPPE

## Type I NPPE

- Auto PEEP
  - positive end expiratory pressure
  - positive airway pressures are generated when try to exhale against an obstruction
  - opposes the release of fluids out of the capillaries into the interstitial tissues & alveoli
  - once obstruction is relieved
    - increased venous hydrostatic pressures are now unopposed
    - fluid is released from the capillaries
    - release if very rapid

## Diagnosis of NPPE

- Resolved upper airway obstruction
- Rapidly developing pulmonary edema
  - usually within mins after the obstruction is relieved
  - time range is 0 to 90 minutes
  - rarely see a delayed reaction
    - if it occurs, it will present within 12 to 24 hours
- Clinical and radiographic resolution in 12 to 24 hours

## Signs & Symptoms

- Dyspnea & hypoxia
  - SpO<sub>2</sub> < 90 reported ranges of 50 to 90
- Hypercarbia & tachypnea
- Tachycardia
- Agitation & restlessness
- Frothy, pink sputum
- Cough & rales
- May see hemoptysis from capillary wall breaks & alveolar hemorrhage

OOO. 2002; 93: 4-6

## Chest X ray

- bilateral interstitial edema
  - on rare occasions a unilateral edema is seen
  - xray findings are found simultaneously with clinical signs
- normal heart size
  - no cardiomegaly, no signs of acute CHF
- CXR usually is clear within 12 to 24 hours

## Pulmonary Edema



## Differential Diagnosis

- Aspiration pneumonitis
  - very high on the differential
- Anaphylaxis
- Pulmonary embolus
- Iatrogenic fluid overload
- Cardiogenic pulmonary edema

## Aspiration versus POPE

- Aspiration: dyspnea, wheeze, & desaturation
- Aspiration: CXR may see positive findings, but they lag behind the clinical signs & symptoms
  - 50% of patients with CXR findings take 3 or more days to resolve
- POPE: CXR findings are present as soon as the edema is clinically evident
  - usually resolves within 24 hours
  - rare to take days for resolution

Anesth & Intensive Care Vol 28 Feb 2000

## Cardiogenic Pulmonary Edema

- Patient may have history of chronic CHF
- New onset LV dysfunction may be due to
  - acute ischemia, injury, infarction, or dysrhythmias
  - echocardiogram aids in diagnosis
- Distended jugular veins & peripheral edema
- Gallop is heard with stethoscope
- CXR
  - enlarged heart & bilateral Kerley's B lines
- ECG
  - dysrhythmias, ST changes, & conduction defects

CHF case

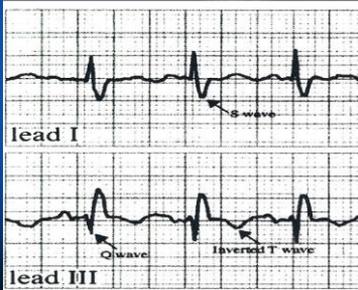
## Pulmonary Edema from Anaphylaxis

- Perioperative allergen exposure
  - neuromuscular blocking agents, anesthetics, latex, antibiotics, contrast dye
- Rash, urticaria, & swelling
- Bronchospasm
- Hypotension
- ECG: variable findings

## Pulmonary Edema from Pulmonary Embolus

- Awake patients most common findings
  - dyspnea, anxiety, cough, tachycardia
- Anesthetized patients most common findings
  - hypotension & tachycardia
  - hypoxia & hypocarbia
  - V/Q mismatch with shunting of blood
- Shunting of blood causes
  - overperfusion of the rest of the lung
  - leads to pulmonary edema & hemoptysis

## Pulmonary Edema from Pulmonary Embolus



### CVS Findings

1. Hypotension
2. Tachycardia
3. Gallop rhythm
4. S<sub>1</sub> Q<sub>3</sub> pattern
5. S<sub>1</sub> Q<sub>3</sub> T<sub>3</sub> pattern

J. Clin Anesthesia. 23: 153-165. 2011

## Management

- Primary goal is to correct hypoxia
  - non rebreather face mask at 10 L/min is first step
  - CPAP by face mask at 4 to 25 cm water
  - possible BIPAP
  - mask ventilations with PEEP at 5 to 10 cm water
    - positive end expiratory pressures
- Most patients will respond to the above treatments
- Monitor SpO<sub>2</sub> keep > 90
- Suction the pink, frothy secretions
  - amount determines mask versus intubation

Laryngoscope 116 Sept 2006

JOMS. 2002; 60: 1503-5

## Management

- if SpO<sub>2</sub> can not be maintained above 90
  - re intubate the patient
  - PEEP at 5 to 10 cm water
  - may need to maintain PEEP for 8 to 24 hrs until resolved
- Diuretics: furosemide ( Lasix )
  - 20 mg IV bolus                      10 to 20 mgs Q 6 h prn
  - controversial: these patients are not fluid overloaded
  - possible to induce hypovolemia & hypotension especially in the elderly
  - insert Foley to monitor output if using multiple doses

Laryngoscope. 116. Sept 2006

Arch Facial Plastics. 8: 815. 2006

## Lasix

- furosemide
- biphasic drug action
- 1<sup>st</sup> phase action
  - ~ 5 mins after injection IV
  - ↑ venous capacitance & ↓ venous tone
  - causes the veins to dilate
  - get ↓ LV filling volume & ↓ preload to heart
  - positive effects of lasix
- 2<sup>nd</sup> phase action
  - onset of diuresis in 10 to 15 mins after injection of drug
  - negative effects of lasix

## Management

- Hospitalized patients: IV fluids at maintenance
- Steroids very controversial
  - sometimes used if see hemoptysis
  - Decadron 6 to 8 mgs Q 6 h
- **Inhaled beta agonists useful**
  - increases the rate of alveolar fluid clearance

Anesthesiology. 113(1): 200-7. 2010  
 J. Crit Care. 25: 508e1-5. 2010  
 Clin Med Res. 9(2): 88-91. 2011

## Office Surgery & UAO

- **Monitor the patient post operatively for at least 60 to 90 mins if laryngospasm occurred**
- Monitor for dyspnea
- Auscultate the lungs
- SpO<sub>2</sub> > 92 on room air
- Any positive findings -- transport to hospital

Anesthesia Progress. 56: 49-51. 2009

## OMFS Case

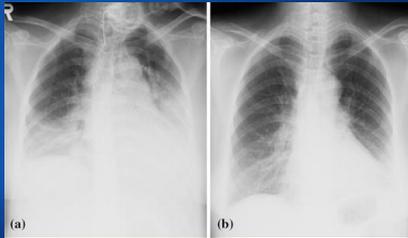
- 21y.o. ASA I; bilateral fractured mandible
- ORIF; nasal intubation, SpO<sub>2</sub> > 97; Sx 150 mins
- Extubated in the OR ; immediate drop in saturation to 75; pt was cyanotic
- Laryngospasm was diagnosed ; positive pressure oxygen by mask without success; succinylcholine was needed to break spasm; resolved in 1 minute
- SpO<sub>2</sub> returned to 95 in the operating room
- Transported to PACU

## OMFS Case

- PACU: SpO<sub>2</sub> 92; heard rales in lungs; CXR showed interstitial fluid – congestion
- Diagnosis by anesthesiologist: aspiration
- Then developed pink, frothy sputum in mouth & continued to desaturate
- Mouth & nose were filled now with pink discharge & patient required ET tube placement & 100% oxygen
- 24 hours later, CXR completely resolved, extubated, & full recovery

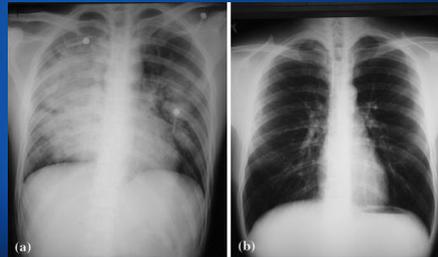
Anesthesia & Intensive Care Vol 25 1997

## X rays of POPE



( a ) is 1 hour post op & ( b ) is 2 days post op almost resolved  
Eur Arch Oto-Rhino 264: 1113. 2007

## X Rays of POPE



( a ) is immediately post op & ( b ) is 3 days post op completely resolved

Eur Arch Oto-Rhino. 264: 1113. 2007

## Unilateral Case of POPE

- Patient was intubated for surgery
  - ET tube was in right mainstem
- Malpositioned ET tube not recognized for several minutes
- ET tube position was corrected
- Pulmonary edema developed
- Which lung????

## Unilateral Case of POPE

- During the case the left lung was trying to ventilate against an obstruction
- Negative intrathoracic pressure was getting more negative in the **left lung**
- Pulmonary edema occurred in the left lung

**FIGURE 1** Immediate postoperative chest radiograph showing pulmonary edema confined to the left lung.

**FIGURE 2** Repeat chest radiograph, 4 hours after the event, showing mild improvement in left lung edema.

**FIGURE 3** Repeat chest radiograph, 20 hours after the event and after discontinuation of mechanical ventilation, showing complete resolution of pulmonary edema.

improvement in left lung edema (Figure 2). Supplemental oxygen and ventilator supports were reduced over the next sixteen hours, the patient's trachea was extubated, and the chest radiograph revealed substantial resolution of the edema (Figure 3). The remainder of the patient's course was unremarkable, and she went home three days after surgery.

**Discussion**  
The patient we describe demonstrated typical features of negative pressure pulmonary edema (NPPE), but its appearance in only one lung distinguished it from the classic presentation. The likely mechanism involved migration of the ETT up to a right endobronchial position (depth noted to be 23 cm). This would obstruct the left main bronchus during forceful inspiratory efforts as the patient emerged from anesthesia, directly obstructing the left lung as significant negative pressure. In the presence of high PEEP, it is possible that increased endotracheal pressure would

Fig 1: immediate post op edema on left side  
Fig 2: 4 hours post op -- improvement on left side  
Fig 3: 20 hours showing resolution

Can J Anesth 2008; 55: 691

## Naloxone Pulmonary Edema

- Naloxone ( Narcan ) is used to reverse opioids
- Naloxone can precipitate sudden & severe sympathetic stimulation
  - severe hypertension & tachycardia
  - ventricular dysrhythmias & sudden death
  - patients with compromised cardiovascular status are especially at risk
  - reports of severe hypertension in healthy patients at doses as low as 0.1 mgs of naloxone

Anesthesiology. 47: 376-378. 1977  
Anesthesiology. 60: 485. 1984

Intensive Care Med. 16: 340-341. 1990  
Anesth Analg. 58: 524. 1979

## Naloxone Pulmonary Edema

- Sympathetic stimulation causes blood to be shifted from the systemic circulation into the pulmonary vasculature
  - increased hydrostatic pressure develops
  - result is pulmonary edema
- Narcan dosing
  - consider a maximum initial dose of  $\leq 0.1$ mg
  - additional doses at 0.1 mg as necessary

## Succinylcholine

- Can Succinylcholine cause pulmonary edema?
- What would be the mechanism?
- Most likely mechanism
- Develop intraoperative laryngospasm and need succinylcholine to break spasm
- Result is a Type I NPPE
- Most times.....yes      Some times.....no

## Succinylcholine & Pulmonary Edema

- Cook et al, 1981
  - 3 cases of pulmonary edema in infants 3 to 8 weeks old
  - succinylcholine IM 16 to 20 mgs for *intubation*
    - there was no laryngospasm or airway obstruction in these patients
  - pulmonary edema developed within 4 to 8 min after injection
  - no mechanism discussed
  - very rare event

Anesth Analg. 1981; 60: 220-3

## Post Operative Laryngospasm

- Incidence: 0.05 to 1 % of general anesthetics
- Visvanthan et al. 2005 Post operative Complications
  - laryngospasm occurred in 189 of 4000 events
  - occurred usually during emergence
  - resulted in several complications
    - desaturations 61%
    - bradycardia 11%
    - pulmonary edema 4%
    - aspiration 3%
  - 43% of patients required re-intubation
  - 28% required CPAP

## Management of Laryngospasm

- General management
  - suction the oral cavity and posterior pharynx
  - pull the tongue forward
  - chin lift – jaw thrust or
    - turn the head to one side and pull the tongue forward or jaw thrust
  - laryngospasm notch pressure
- If not relieved by above
  - face mask oxygen + positive pressure
- If unsuccessful
  - 10 to 20 mg of succinylcholine IV

## Alternatives to Succinylcholine

- Rocuronium
  - new onset laryngospasm → failed positive pressure → not “blue”
    - 0.6 mg/kg IV → full paralysis in ~ 2 minutes → cords sooner
  - laryngospasm → patient is “crashing”
    - 1.0 mg/kg IV → cord paralysis in ~ 1 minute
  - **need to ventilate patient > 30 minutes**
  - consider reversal with neostigmine / glycopyrrolate before you discharge patient
- Sugamadex: encapsulates and inactivates rocuronium
  - 2-16 mg/kg: dose  $\geq$  4 mg/kg reverses in < 3 minutes ( 1 to 2 min )
  - available in Europe not US → bronchospasm holds up FDA approval

Adlesic – Ganzberg. MH Chapter. Pending publication

## Alternatives to Succinylcholine

- Lidocaine 1%
  - positive pressure fails to relieve laryngospasm
  - inject 1 to 2 ml of 1% lidocaine through the cricothyroid membrane
    - 25 gauge needle 1 – 2 ml 1% lidocaine
  - causes an immediate cough to open cords
  - cords get anesthetized by lidocaine as it is coughed out of the airway
  - reduces risk of recurrent laryngospasm

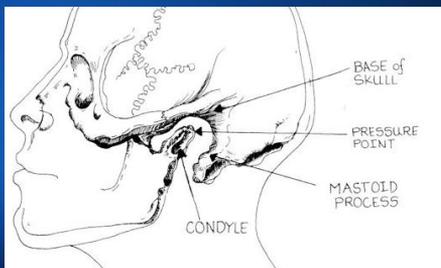
Int Anesthesiol Clin.1997;35(3): 13-31

## Alternatives to Succinylcholine

- Propofol
  - study used low dose propofol for laryngospasm after LMA removal in children
  - **0.8 mg of Propofol IV**
  - 752 LMA – General anesthetics
    - 20 pts had laryngospasm after LMA removed
    - all got positive pressure with 100% oxygen
      - 7 of 20 responded to positive pressure
      - 13 had desaturations to 85% → got Propofol
        - 10 patients responded
        - 3 got re intubated after succinylcholine
    - proposed new study at 1 to 1.5 mg/kg in future

Paediatric Anesthesia.2002;12:625

## Laryngospasm Notch



Pressure behind the condyle

Periosteal pain at styloid process

## Questions?

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