Anesthesia Considerations in Obstetric Emergencies

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August 2023

OBSTETRIC EMERGENCIES

- Maternal Cardiac Arrest
- L.A.S.T
- Total Spinal
- Pulmonary Edema
- Amniotic Fluid Embolism
- Eclampsia
- HELLP
- Placental Abruption

FAILURE TO PREPARE IS PREPARING TO FAIL

- Despite efforts to send high-risk pts to tertiary centers for delivery, **OB emergencies do not discriminate btwn healthy and "high-risk" pts. They can happen ANYWHERE.**
- "Low-risk" healthy pts are still at risk of all OB emergencies, just by nature of being pregnant.
- With that being said, **OB airways** are WELL-KNOWN to be higher risk of: **aspiration, swelling, difficult airway, failed airway, and fatal hypoxemia**.
- Considering an OB emergency requiring airway management is possible in EVERY AND ALL OB pts, **it is critical to consider airway management** in **every single OB pt you care for.**
- Positioning airways appropriately prior to start of c/s by ramping w/ blankets, when needed, will significantly improve success of airway management, and failure to do so can significantly increase airway challenges.
- **OB pts give do NOT tolerate apnea, and do NOT afford time for inefficient, inexperienced airway management.**
- Reviewing **difficult airway management**, intubation techniques, and knowing exactly **WHERE to find, and HOW TO USE** the difficult airway equipment you have available to you at your facility can be the difference between life and death in these pts when an emergency unfolds.
- * This includes knowing HOW to delegate and direct non-anes providers to help you. Can the y find your equipment? Can you explain what you need? Are emergency meds labled? Etc...
- **Assume you may have to intubate every single patient, and plan accordingly.**

Maternal Cardiac Arrest

Maternal Cardiac Arrest

RAPID DELIVERY OF FETUS AFTER MATERNAL ARREST IS CRITICAL

DELIVERY GOAL < 5 MINUTES SINCE ARREST

Maternal Cardiac Arrest

Maternal Cardiac Arrest Causes (USA & UK):

- **Anesthesia complications**: **high neuraxial blocks** from unrecognized spinal catheters or a spinal after failed epidural anesthesia.
  (D'Angelo et al. Anesthesiology 2014;120:1505–12)

- Up to 38% of arrests from antepartum or postpartum hemorrhage/hypovolemia (Mhyre et al. Anesthesiology 2014;120:810–8)

The study provided the strongest evidence to date that timely perimortem c/s delivery improves maternal survival.

The median duration from collapse to delivery was 3 min in survivors **and** 12 min in those who died.

Maternal Cardiac Arrest

BJOG: An International Journal of Obstetrics & Gynaecology homepage

An International Journal of Obstetrics & Gynaecology

Royal College of Obstetricians & Gynaecologists

General Obstetrics

The CAPS Study: incidence, management and outcomes of cardiac arrest in pregnancy in the UK: a prospective, descriptive study

VA Beckett, M Knight, P Sharpe

First published: 24 February 2017 | https://doi.org/10.1111/1471-0528.14521 | Citations: 118

Linked article This article is commented on by JM Mhyre and Bateman, p. 1382 in this issue. To view this mini commentary visit https://doi.org/10.1111/1471-0528.14569. This article has journal club questions by BD Einerson, p. 1383 in this issue. To view these visit http://dx.doi.org/10.1111/1471-0528.14662. This article includes Author Insights, a video abstract available at https://vimeo.com/rcog/authorinsights14521.

Maternal Cardiac Arrest Cause:

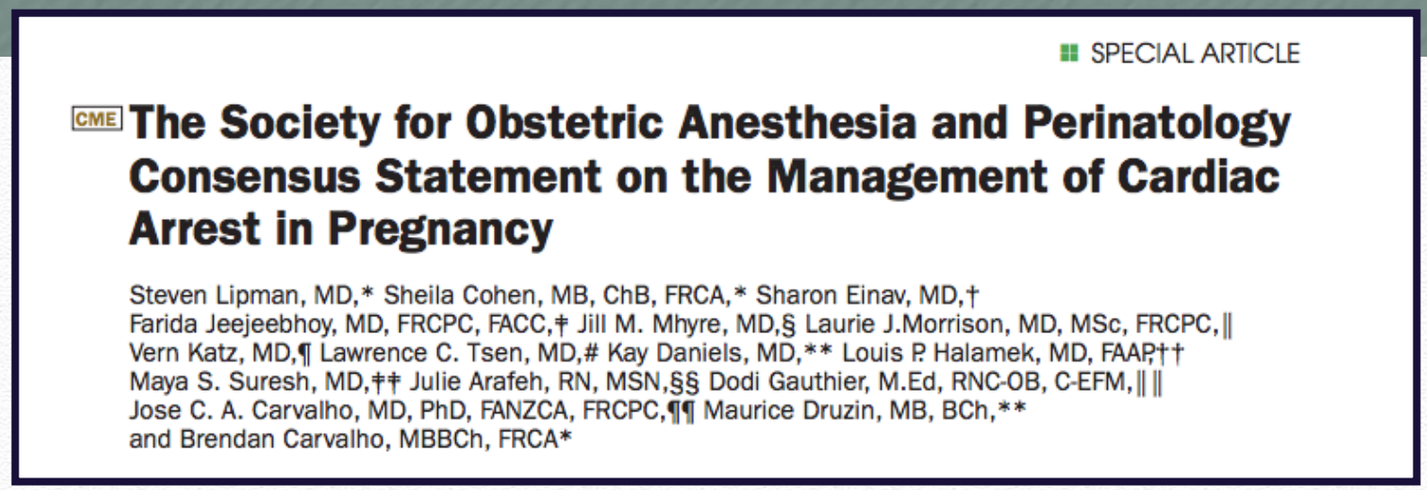
-**25% anesthesia:** Majority total spinal

-**Survival improved with IMMEDIATE CPR and C/S < 5min (perimortem)**

https://www.obgproject.com/2017/05/07/decreasing-mortality-cardiac-arrest-pregnancy/

https://obgyn.onlinelibrary.wiley.com/doi/abs/10.1111/1471-0528.14521

Maternal Cardiac Arrest



SPECIAL ARTICLE

CME **The Society for Obstetric Anesthesia and Perinatology Consensus Statement on the Management of Cardiac Arrest in Pregnancy**

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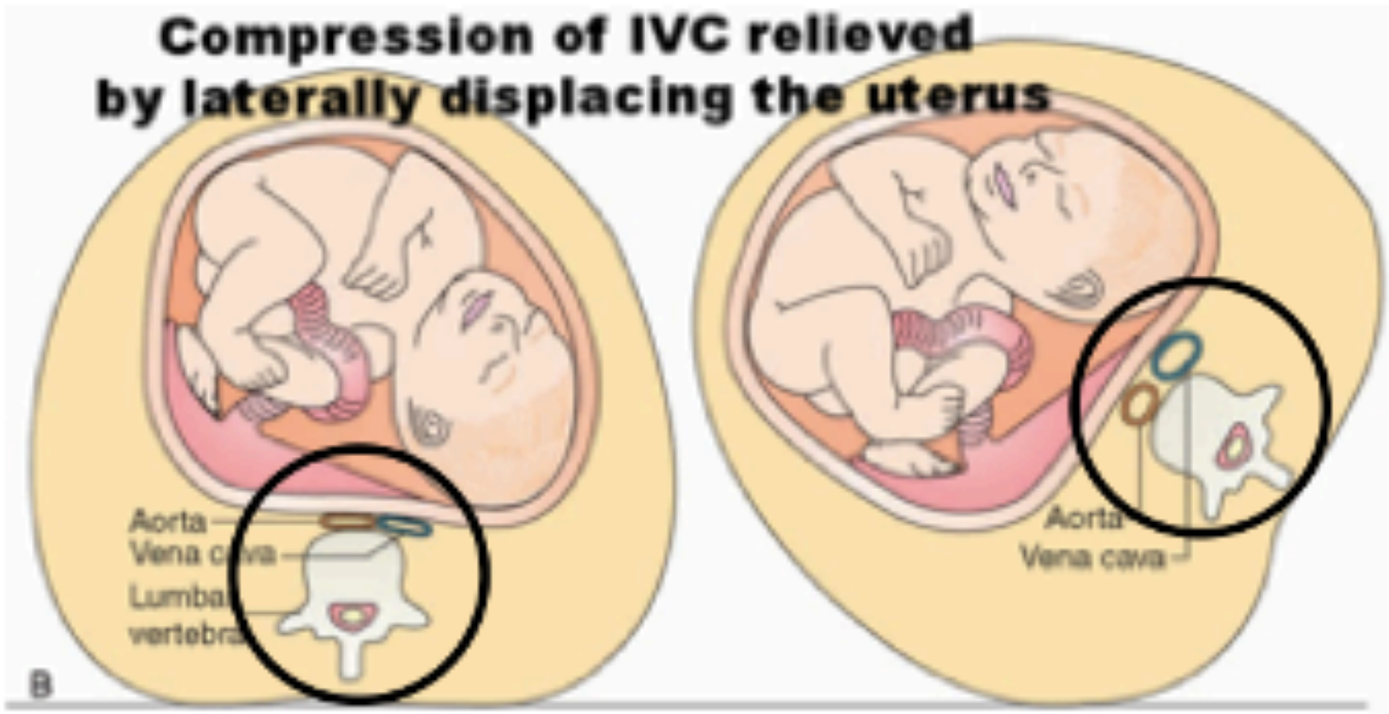
- **CALL FOR HELP:** More anesthesia personnel, Obstetrician(s), surgical and Nursing staff to prepare for emergency C/S.
  - **C/S <u>at site of arrest in < 5 min</u>. DO NOT MOVE PATIENT**
- **Begin CPR immediately:** If Uterus gravid, use Left Uterine Displacement
- Follow ACLS/CPR guidelines: **Same drug doses & defibrillation.**
  - **"Whats good for mom is good for baby"**
  - 100% O2, defibrillation, Epinephrine, Amiodarone, etc.

Maternal Cardiac Arrest

CPR CONSIDERATIONS IN PREGNANCY



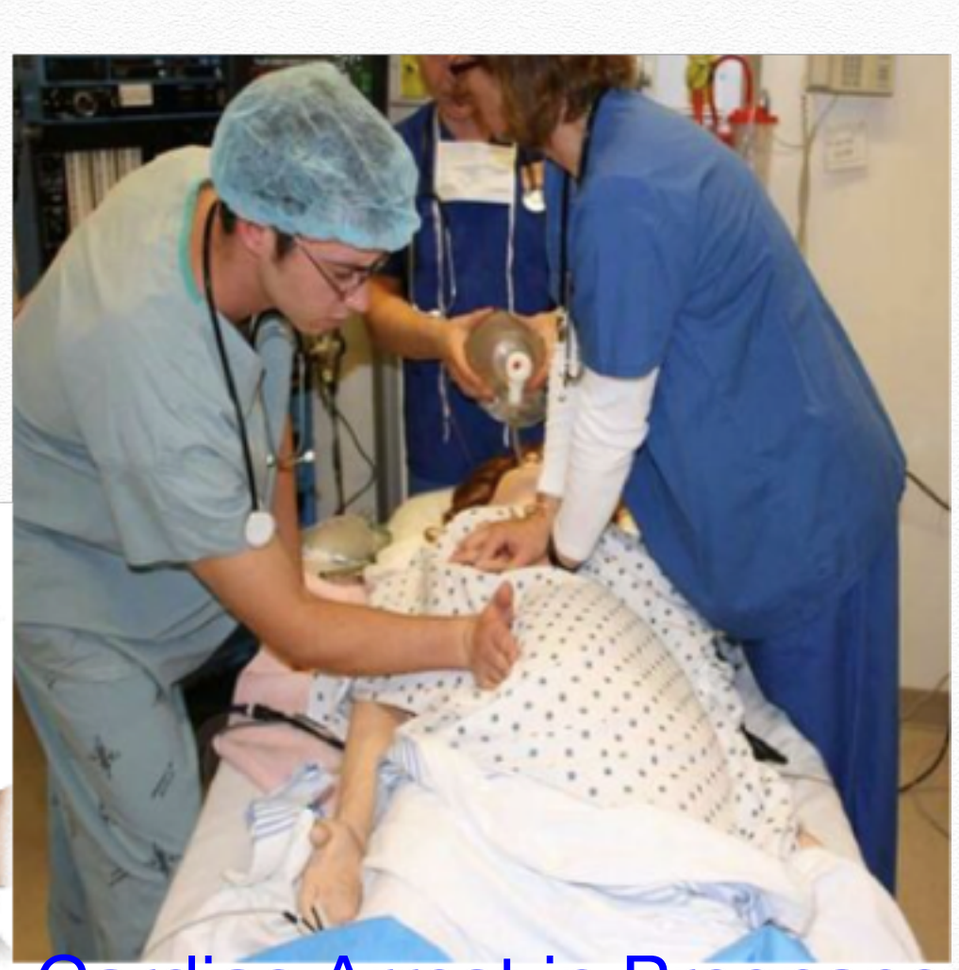
HTTP://WWW.PAT-WHEN-HOW.COM/NURSING/NORMAL-PREGNANCY-MATERNAL-AND-NEWBORN-NURSING/PART-4/

https://www.australiawidefirstaid.com.au/resources/cpr-guide-pregnancy

MANUAL left uterine displacement reduces compression of aorta & vena cava to improve maternal circulation.

Maternal Cardiac Arrest

- Leave patient supine and flat during CPR.
- A firm backboard should be used to improve compressions.
- Use **manual left uterine displacement**
  - NOT tilt the backboard
  - NOT using blanket/roll under left hip.
    - The 30 degree tilt decreases force & effectiveness of chest compressions. (AHA, 2015)



[Cardiac Arrest in Pregnancy]

Maternal Cardiac Arrest

- **BEGIN CPR with LEFT LATERAL DISPLACEMENT**

- If no ROSC in 4 minutes – Emergent C/S no matter where you are – (L&D room, hallway, OR, PACU, CT scanner, triage bay, in a bed or on the floor.)

- **PERIMORTEM C/S OCCURS AT SITE OF ARREST. DO NOT MOVE PATIENT.**

- **BEGIN CRASH C/S AT 4 MINUTES, FETUS DELIVERED < 5 MIN AFTER ARREST OCCURS.**

  - C/S is to facilitate Maternal CPR & resuscitation for maternal survival, not for fetal benefit, although < 5mins from arrest to delivery has improved fetal outcomes.

  - Transverse or vertical incision (whatever OBGYN feels they are faster at), placenta can stay in situ until ROSC, abdomen can be packed and stapled until ROSC.

  - C/S only considered if fetus > ~22-24 wks, as prior to this, fetal gestational size is not large enough to compromise maternal circulation in CPR (primary concern), and viability is unlikely, so best outcome is just restored maternal perfusion to restore fetal perfusion.

Maternal Cardiac Arrest

Obstetric Considerations:

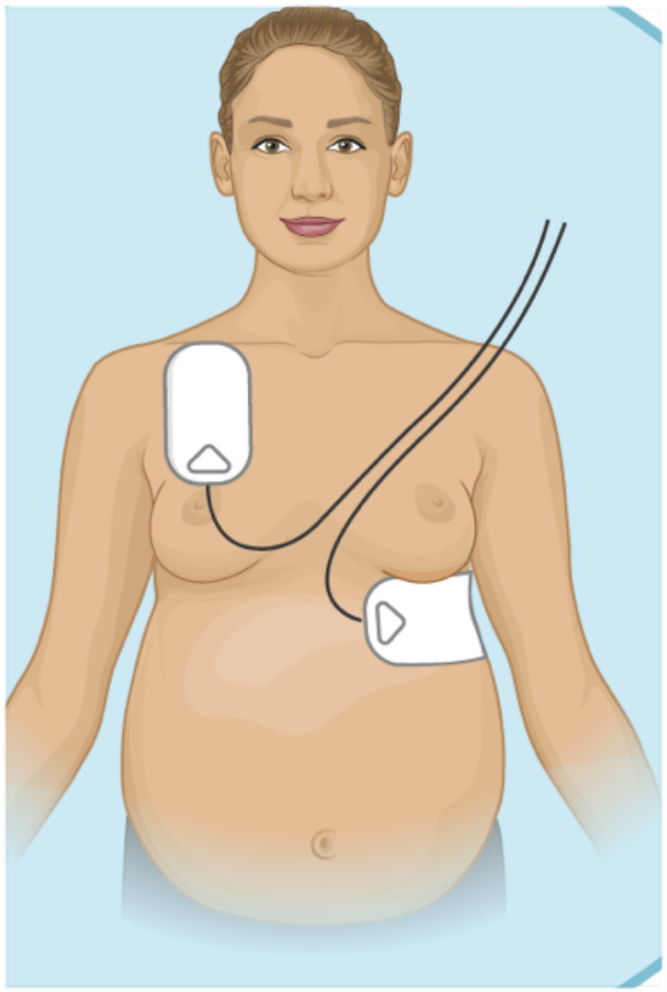
- **Left Uterine displacement** if Gravid Uterus
- If L.A.S.T suspected, give INTRALIPID therapy
- If on Magnesium, **STOP Magnesium - Give Calcium**:
  - Calcium gluconate 3g IV push (30 mL of 10% solution)
  - Calcium Chloride 1g IV push (10mL of 10% solution)
- Anticipate Difficult Airway

https://soap.memberclicks.net/assets/docs/CPR-consensus-statment.pdf

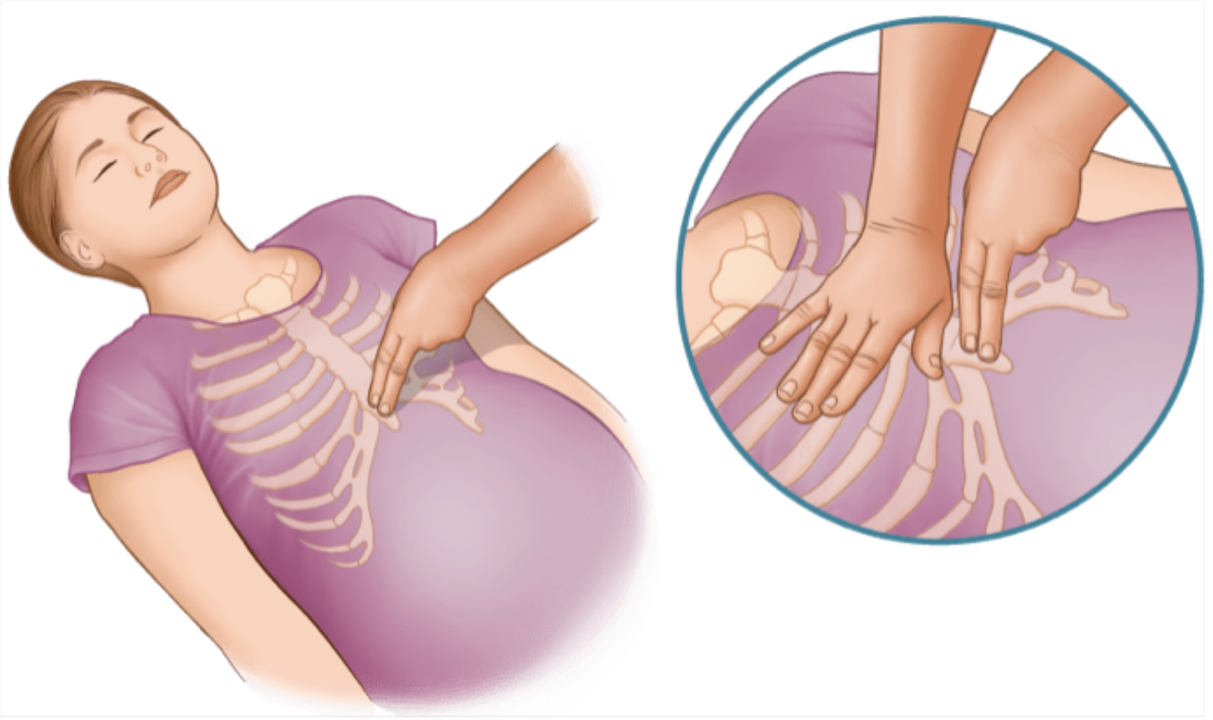
Maternal Cardiac Arrest

CPR CONSIDERATIONS IN PREGNANCY

AED pad placement



Same hand placement 2 fingers above xyphoid process

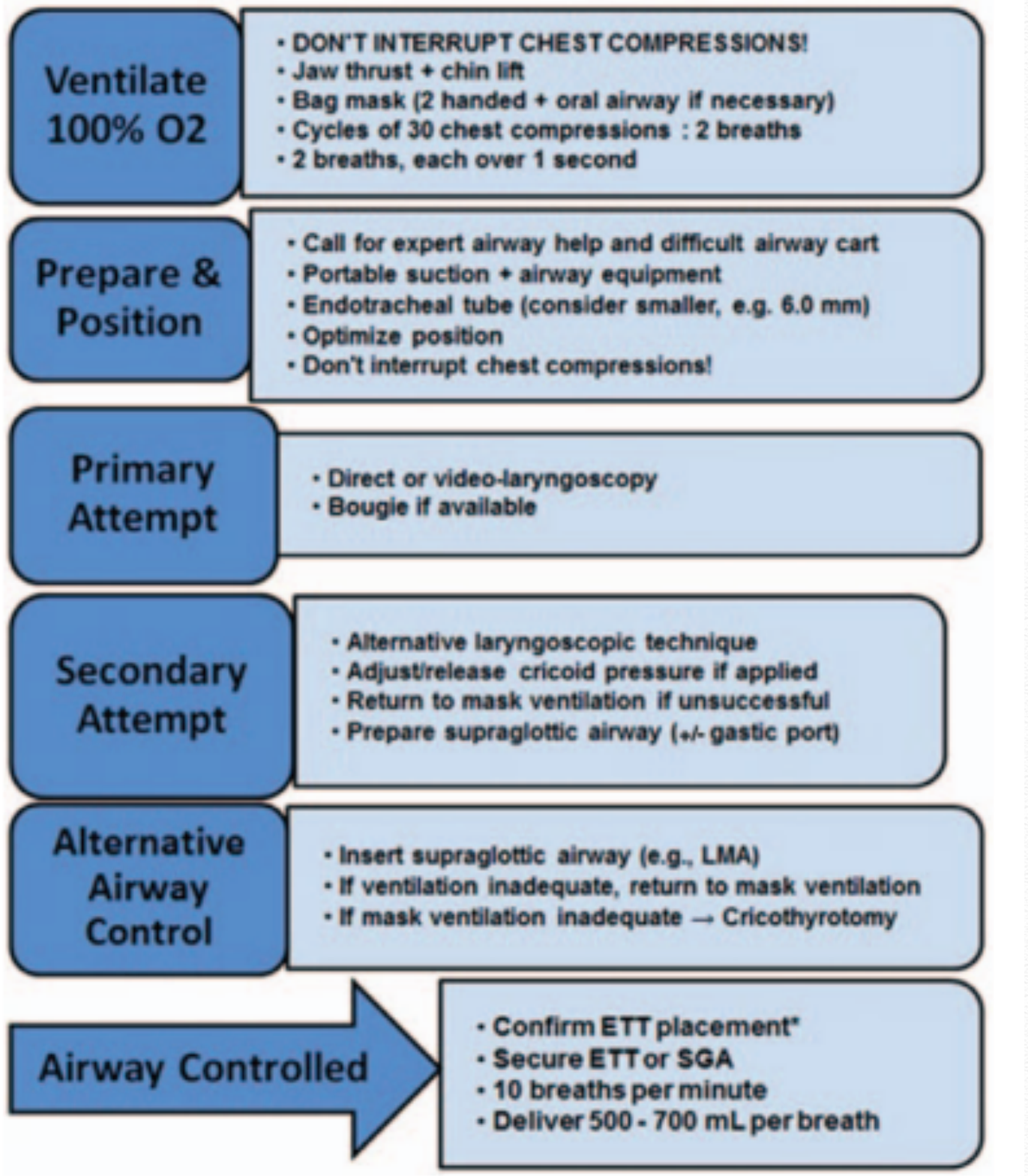


https://www.australiawidefirstaid.com.au/resources/cpr-guide-pregnancy

Maternal Cardiac Arrest

S.O.A.P

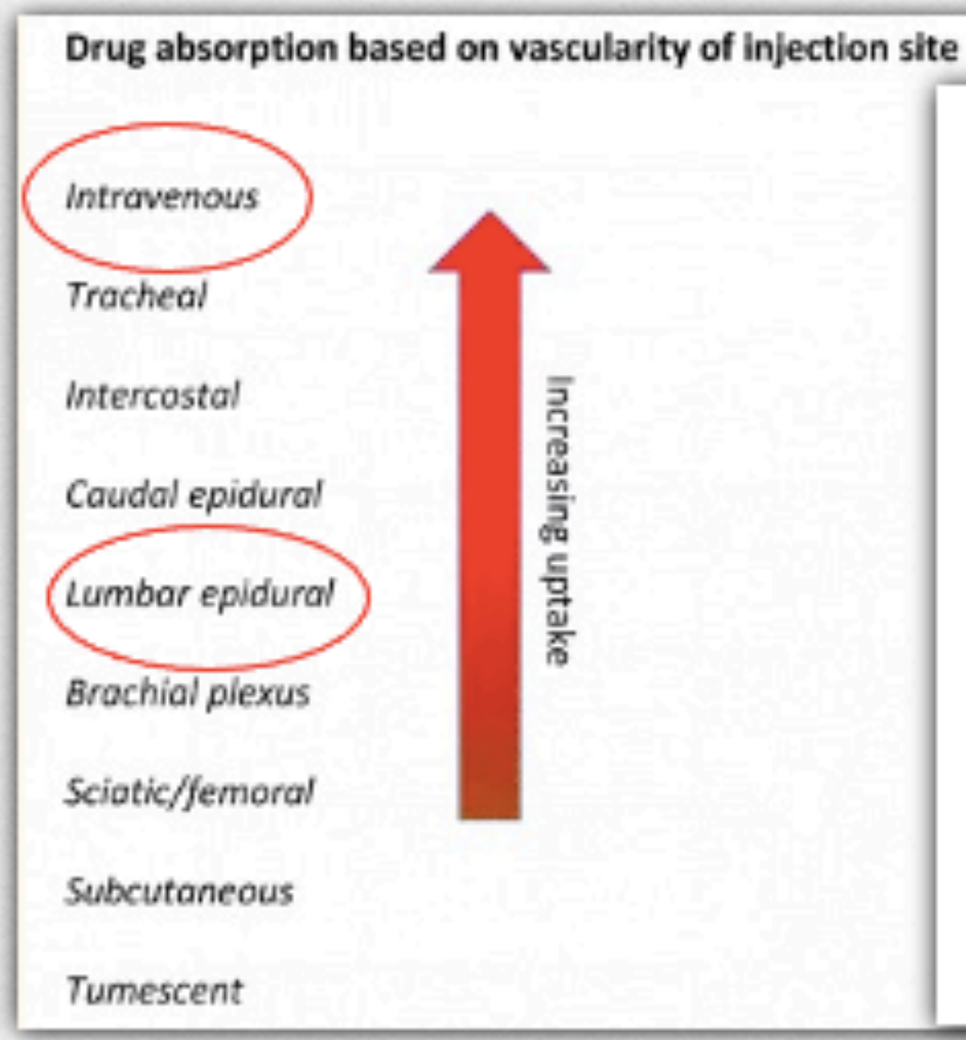
Algorithm for Difficult Airway During CPR



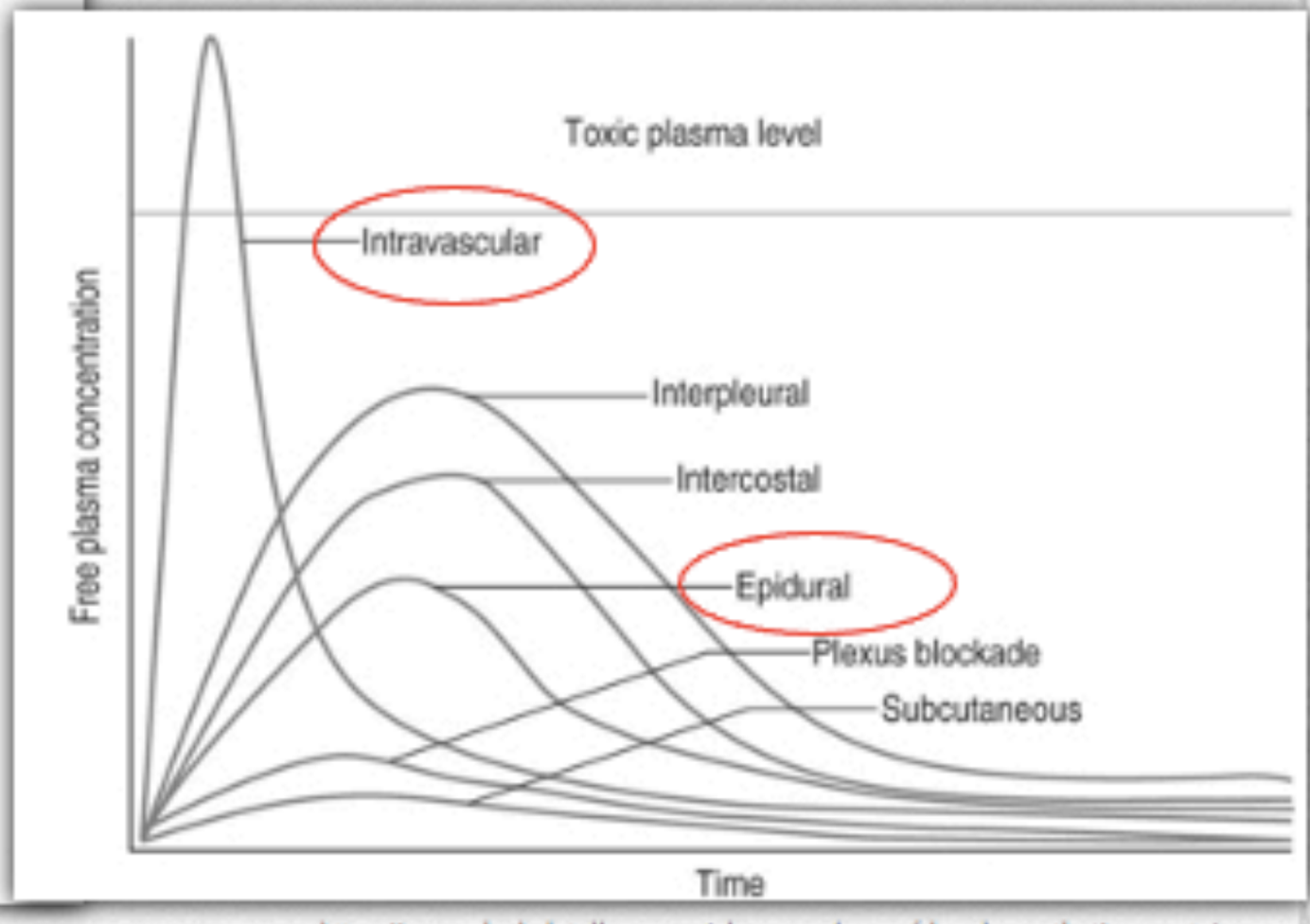
L.A.S.T

L.A.S.T

- LAST most likely with LA lidocaine bolus to convert labor epidural for C/S, and catheter is in VEIN/IV and not in Epidural space.
  - [20cc 2%lido w/ epi 1:200,000 (=400mg)]
- This dose > max dose **IV lido**, and pregnancy physiology makes women more sensitive to LA toxicity.
  - (decreased A_1 Acid glycoprotein = more free fraction of LA)



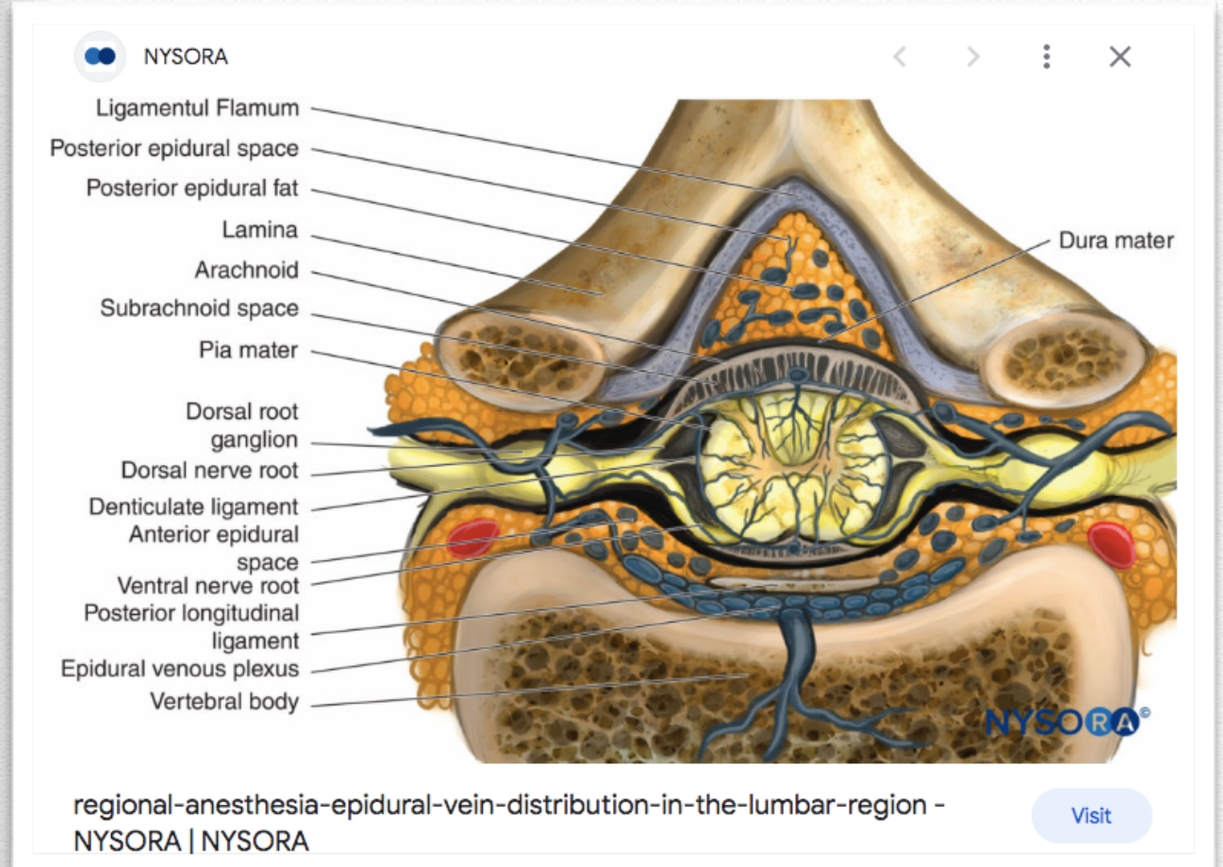
https://emedicine.medscape.com/article/2500068-overview

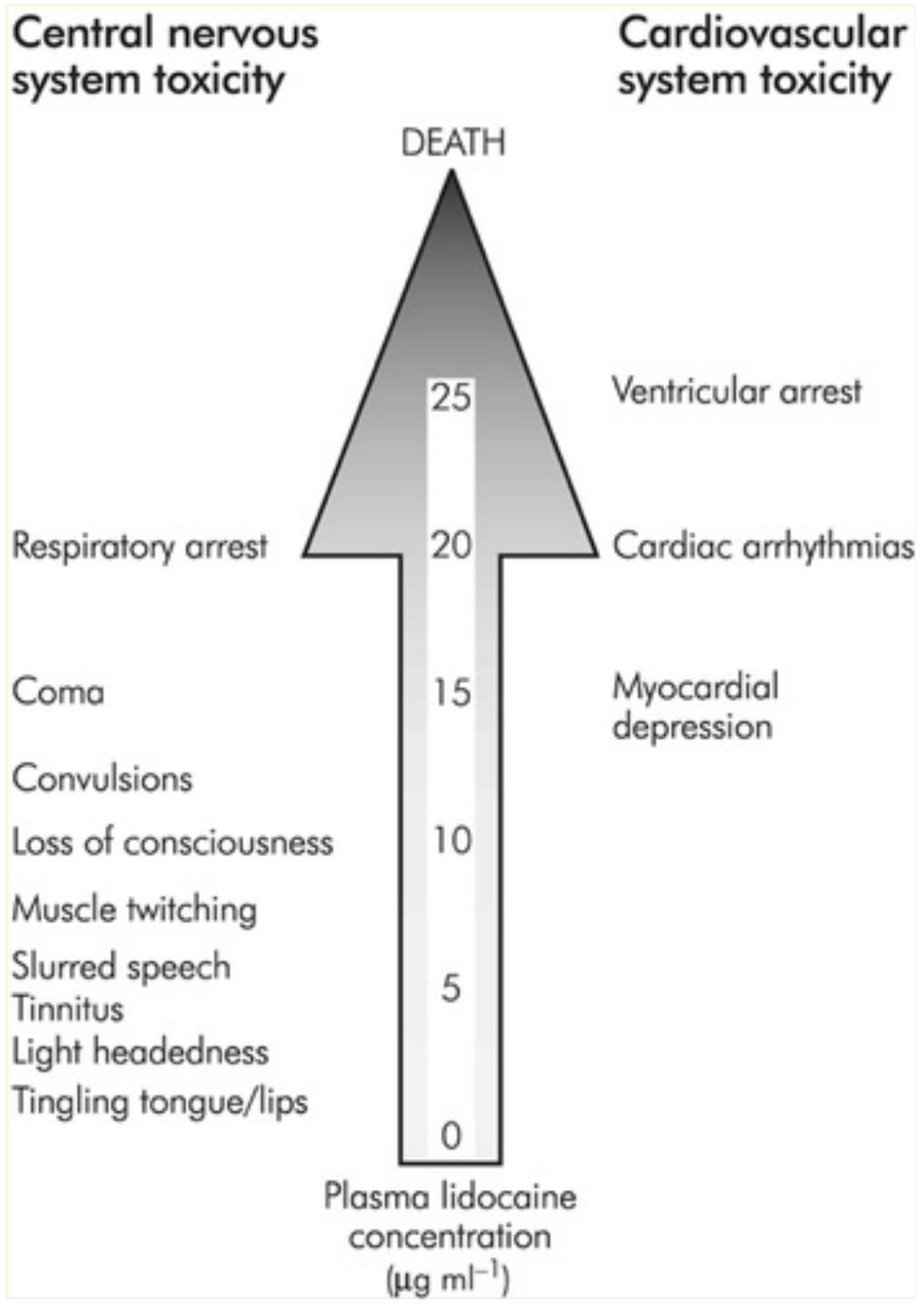


https://musculoskeletalkey.com/pharmacology-of-local-anesthetic-agents/

L.A.S.T

- Intravenous LA causes CV collapse via cardiac myocyte Na+ channel blockade leading to electrophysiological and contractile dysfunction.
- Bupivicaine/Marcaine most cardiotoxic. (Lipophilic profile increases toxicity - higher affinity for cardiac Na+ channels)





https://thejns.org/view/journals/j-neurosurg/132/3/article-p809.xml

L.A.S.T

MECHANISMS OF LAST

NYSORA Learning System

LAs are generally safe and effective in therapeutic doses for tissue infiltration, fascial planes, or near a nerve/plexus of nerves.
However, supratherapeutic plasma levels of LAs can result in **Local Anesthetic Systemic Toxicity (LAST)**.

PLASMA CONCENTRATION FACTORS

High plasma concentration of LAs:

- Partial venous/arterial injection
- Intravascular injection
- Rapid vascular absorption from the highly vascularized injection site

Plasma levels of LA: Proportional to the rate of systemic absorption from the site of therapy.

Rate of absorption:

- Varies among tissues
- Depends on the size of the absorptive surface
- Depends on vascularization of the tissue planes where the injection is made
- ↑ Doses = ↑ plasma levels of LAs, independent of the injection site

LA: MECHANISMS ON THE CELLULAR LEVEL

- **Inhibitory action** on nerve conduction by inhibiting the movement of ions through voltage-gated ionotropic channels at the level of the cell membrane
- **Primary therapeutic target** = voltage-gated sodium channel where inhibition alters the transmission of sensory and motor signals in axons
- **ALSO:** LAs inhibit voltage-gated Ca2+ channels, K+ channels, the Na-K ATPase, and other channels and enzymes
- **Non-ionized vs Ionized molecules:** The inhibition occurs from the intracellular side and requires LAs to cross the lipid bilayer first as unbound, non-ionized free molecules
- **At lower concentrations:** LAs block protein kinase signaling induced by tumor necrosis factor α (TNF-α)
- **At higher concentrations:** LAs can inhibit other channels, enzymes, and receptors, including the carnitine-acylcarnitine translocase in the mitochondria

LA & CARDIAC TOXICITY

Cardiovascular toxicity: Caused by the combination of electrophysiologic and contractile dysfunction

CAUTION: Bupivacaine = lipophilic and greater affinity for the voltage-gated sodium channels, resulting in its uniquely high cardiotoxic profile

More on bupivacaine: Cardiac toxicity can occur at lower serum concentrations because it can accumulate in the mitochondria and cardiac tissue at a ratio of about 6:1 (or greater) relative to plasma

https://www.nysora.com/topics/complications/local-anesthetic-systemic-toxicity/

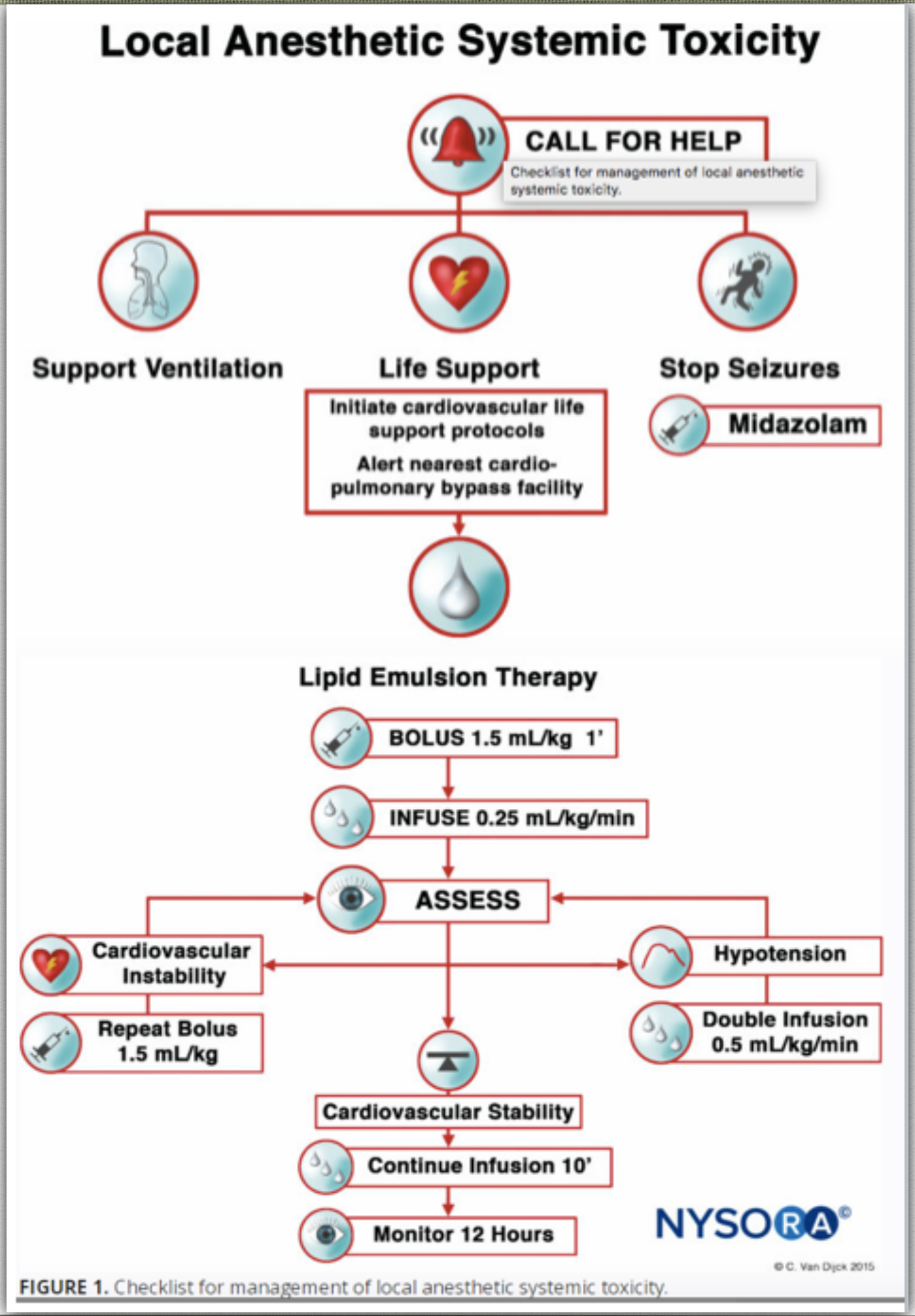


FIGURE 1. Checklist for management of local anesthetic systemic toxicity.

https://www.nysora.com/topics/complications/local-anesthetic-systemic-toxicity/

L.A.S.T

Initial Management of LAST

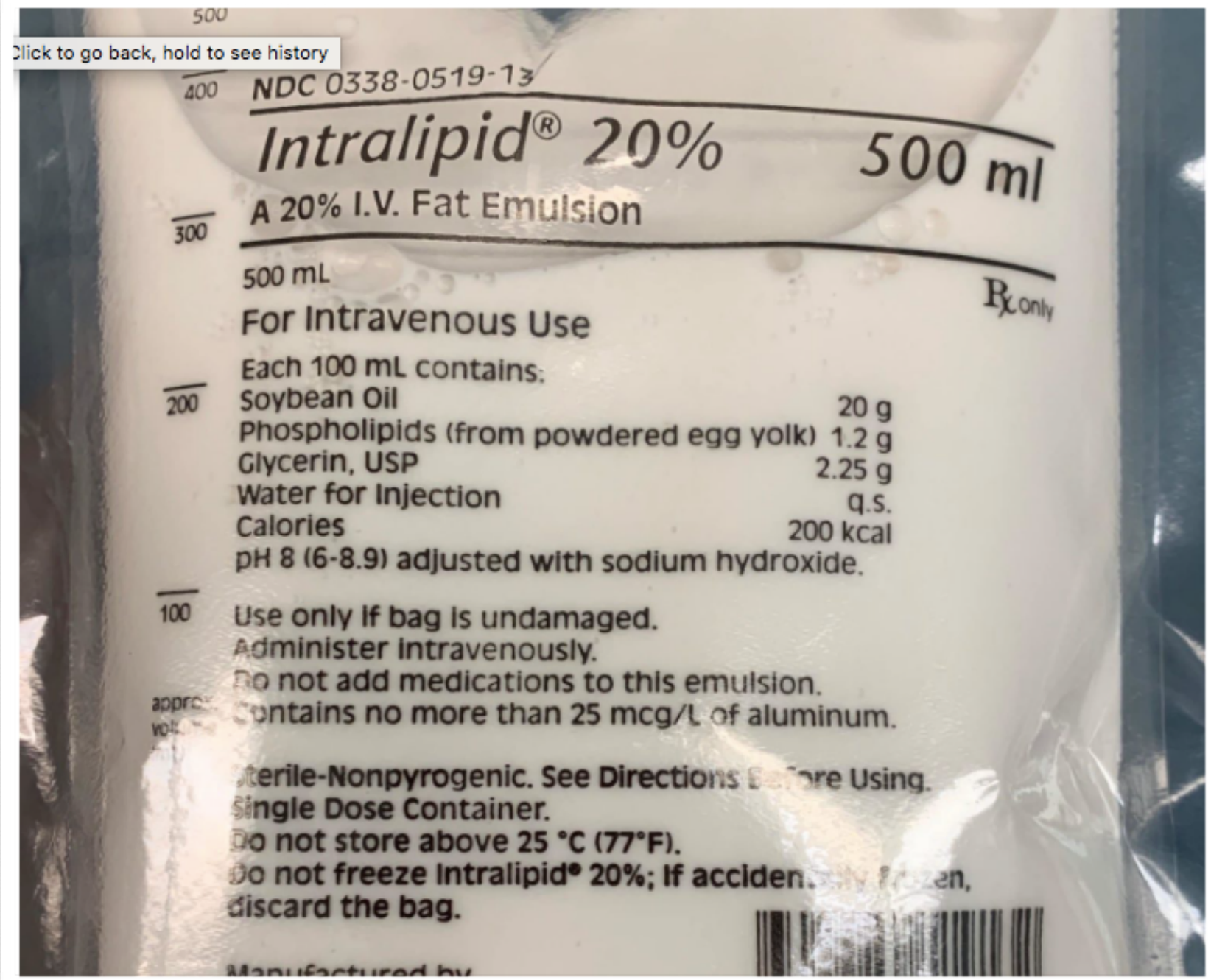
1. Airway management
2. Circulatory support
3. Reduce systemic side effects.

- Immediate ventilation & oxygenation to prevent hypoxia and acidosis can help resuscitation and reduce progression to seizures or cardiac arrest

- If **seizures** occur, immediate administration of **benzodiazepines** recommended, to prevent injury & acidosis.

L.A.S.T

20% Fat Emulsion = INTRALIPID



https://rk.md/2019/intralipid/

L.A.S.T

INTRALIPID 20% FAT EMULSION IS PRIMARY TREATMENT

DOSE:

- **1.5mL/kg BOLUS x 1 min**, then 0.25 mL/kg/min INFUSION
- RE-BOLUS same 1.5mL/kg x 1 min
- if HOTN persists, double INFUSION to 0.5 mL/kg/min

In Cardiac Arrest: —> STAT C/S < 5min

- INTUBATE & **USE CPR TO CIRCULATE FAT EMULSION**
  - Left Uterine Displacement

- Continue CPR until intralipid effective —> ROSC
- NO ROSC? —> Cardiopulmonary Bypass until LA metabolized

L.A.S.T

CRITICAL ACLS DIFFERENCES WITH L.A.S.T

AVOID VASOPRESSIN:

Associated with poor outcomes - may cause pulmonary hemorrhage 2/2 high SVR impairing cardiac output.

Can cause tetanic uterine contraction and further fetal distress

SMALL EPI ACLS DOSES of 1mcg/kg

~ 70-100 mcg for most pts

(Can mix 1mg/cc epi + 9cc NS 0.9%: 1000mcg/10cc = 100mcg/cc)

SEIZURES - GIVE BENZO

-Midazolam**, Lorazepam

(*avoid propofol* - can worsen acidosis & cause cardiac depression/HOTN)

AVOID CCB or BB therapy

- worsens cardiac function.

VENTRICULAR ARRHYTHMIA ---> GIVE AMIODARONE

(300mg 1st dose, then 150mg 2nd dose if needed)

TOTAL SPINAL

TOTAL SPINAL

- Can be sudden LOC, or progress rapidly from a high-level block to a total spinal.

- Happens most often with ***accidental spinal placement of epidural catheter,*** or with ***spinal after failed epidural*** (canal compression, conduit for high dose LA).

TOTAL SPINAL

TOTAL SPINAL

VS.

HIGH BLOCK:

TOTAL SPINAL

TOTAL SPINAL

- **LOC & Respiratory Arrest from brainstem dysfunction from LAs**
  - Can begin with slurred speech & SpO2 desaturation.

- HOTN and BRADYCARDIA (T1-T4 cardiac accelerator fibers, and Benzold Jarisch Reflex*)
  - can progress to complete cardiorespiratory arrest —> CPR & CRASH C/S

- Patients with a total spinal will have fixed, dilated pupils
    - CN2 = optic nerve: pupillary reflex

- UCONSCIOUSNESS REQUIRES AIRWAY SECUREMENT AND HEMODYNAMIC SUPPORT.

TOTAL SPINAL

HIGH BLOCK

[Higher than desired block, no airway compromise, but MAY progress to total spinal]

- Upper extremity weakness
- Hoarse voice/whisper
- Feeling of heaviness in chest "cant take a deep breath."
- HOTN & bradycardia possible from T1-T4 cardiac accelerator fiber blockade
- Nausea/vomiting
- Significant patient anxiety & restlessness.

- **Symptom management & monitoring for high block - may or may not progress to LOC/total spinal.**

  - **Ability to squeeze hands/thumb strength rules out block higher than C6**
  - **Phonating rules out total spinal (C3-5)— Keep mom talking to assess for neurological changes/apnea etc while you treat HOTN and bradycardia.**
    - (*don't forget Atropine & Ondansetron (if not already given) for Benzold-Jarisch reflex! – [higher block = higher sympathectomy --> reduced preload])

TOTAL SPINAL

ANESTHETIC MANAGEMENT FOR TOTAL SPINAL

Rule out:

- Hemorrhage
- Vasovagal
- Aortocaval Compression
- L.A.S.T
- Emoblism: AFE/PE

TOTAL SPINAL

ANESTHETIC MANAGEMENT FOR TOTAL SPINAL

Call for backup
[ANESTHESIA & OBGYN]

Can progress to Cardiorespiratory arrest/code
—> CRASH C/S

TOTAL SPINAL

ANESTHETIC MANAGEMENT

- AIRWAY securement: *if unconscious - no induction agents needed*

- **Atropine, Phenylephrine, Epi** (~5-10mcg at a time) (epi> ephedrine in this situation – fast-onset & direct-acting)

*Bradycardia can be from blocked T1-T4 cardiac accelerator fibers, AND from Bezold Jarisch reflex (Vagus: tx: atropine & ondansetron: ↓preload offloads LV, HR slows dramatically to allow more diastolic filling, but can slow to asystole. Hypopne/apnea also observed.) (5-HT_3 receptors on vagus nerve implicated in this reflex – reason why higher-dose ondansetron(6-8mg) should always be given BEFORE spinal.

- Manual Left uterine displacement to improve preload

- IVF bolus 500-1L
    - Careful in cardiac or PreE pt – risk of flash pulmonary edema
    - Reverse trendelenburg does not prevent cephalad spread and may cause/accelerate CV collapse from venous pooling (NYSORA)

BEGIN CPR IMMEDIATELY IF LOC w/ PROFOUND BRADYCARDIA & HOTN —-> CRASH C/S. CPR moves medications through circulation.

TOTAL SPINAL

ANESTHETIC MANAGEMENT FOR TOTAL SPINAL

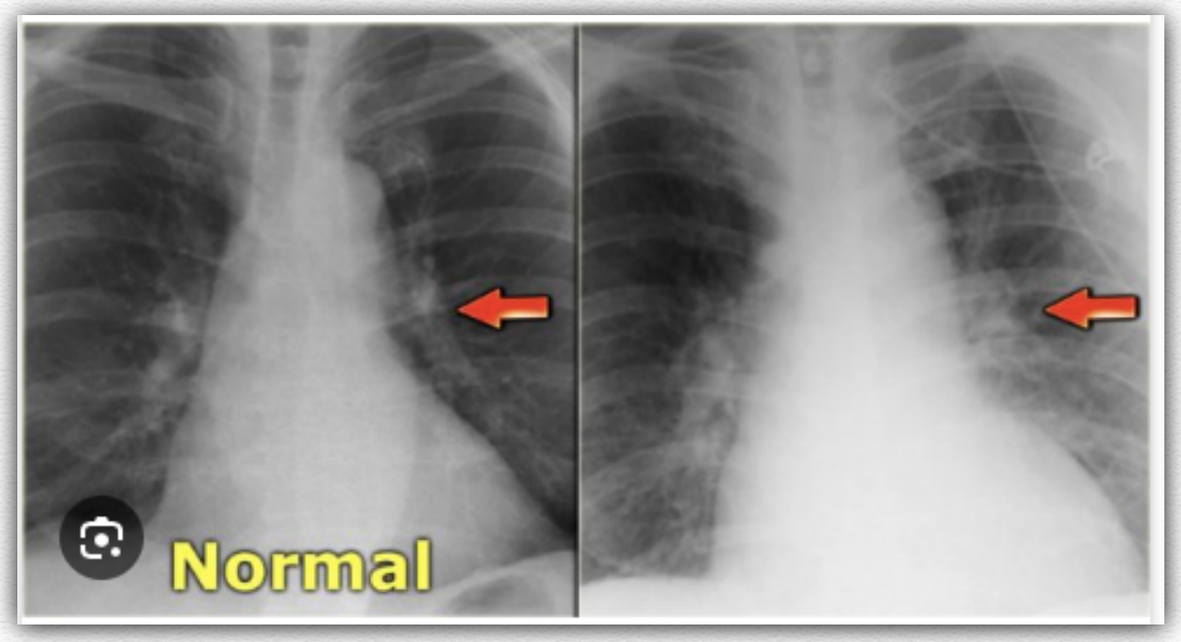
Airway Management & Securement to Prevent Aspiration

- Prepare for emergency/stat C/S
- Have SUCTION – high aspiration risk
- Cricoid pressure should be held while masking/ventilating. Apnea will cause rapid desaturation.
- NO induction agents if unconscious – just intubate.
- Hemodynamic support with fluids/vasopressors
- Remain intubated/on ventilator until able to wean/LA wears off.

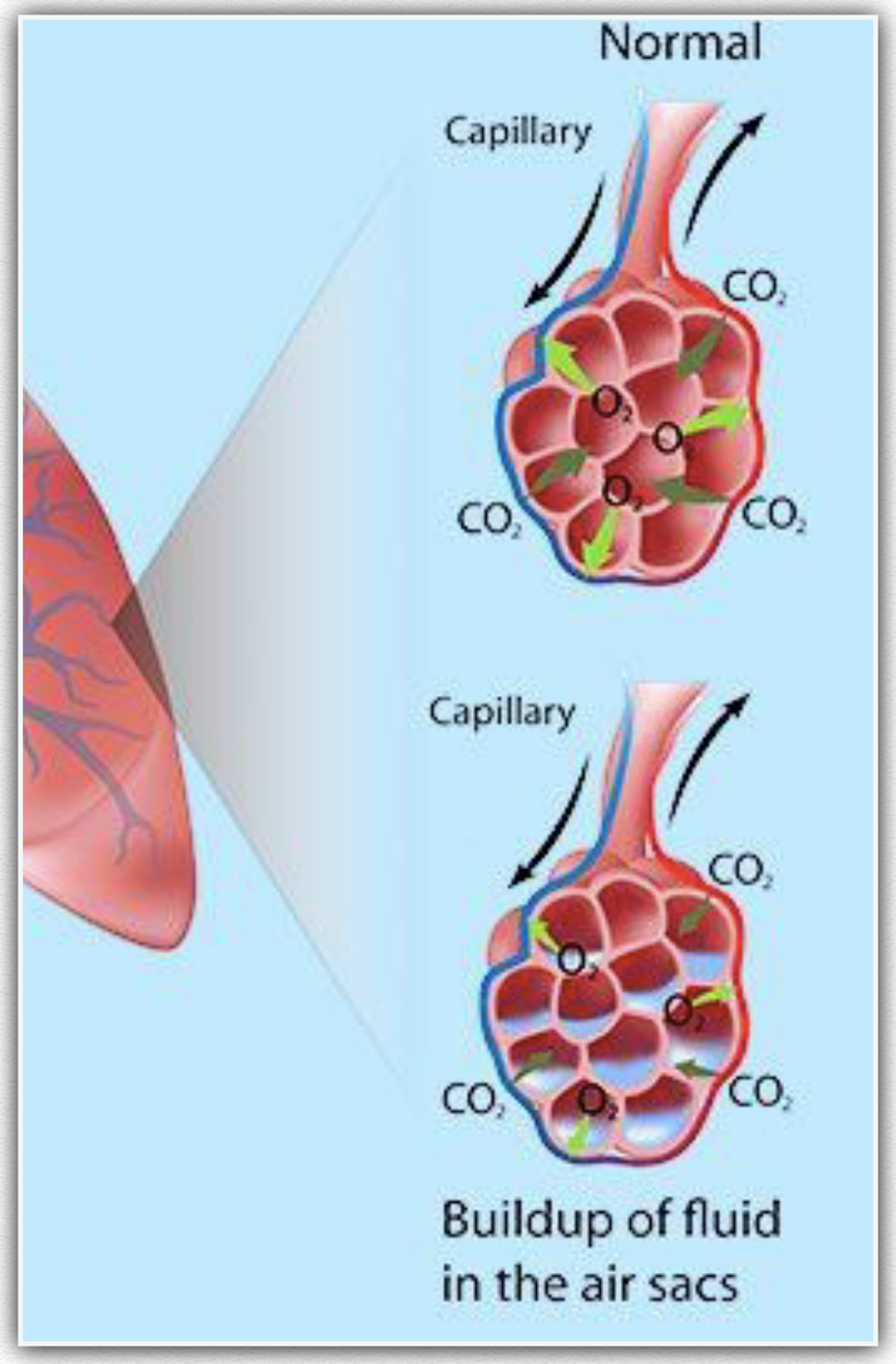
PULMONARY EDEMA

PULMONARY EDEMA

- Acute pulmonary edema 2/2 impaired CV function & fluid flow into pulmonary interstitium.
- Hydrostatic pressures, colloid osmotic pressures & capillary permeability determine fluid in pulmonary interstitium.



https://radiologyassistant.nl/chest/chest-x-ray/heart-failure



https://www.everydayhealth.com/edema/pulmonary-edema/

PULMONARY EDEMA

Life threatening 2/2 hypoxemia from impaired O2 exchange at alveolar-capillary membrane.

<u>Normal physiologic changes of pregnancy place parturients at higher risk:</u>

- Plasma volume increases ~40% by 28wks, ~50% by 35wks
- Decreased serum colloid-osmotic pressure increases membrane permeability and places pregnant pts at higher risk of pulmonary edema:

- Compounded risk in setting of:
  - Additional increases hydrostatic pressure:
    - preload (IVF/autotransfusion)
    - HTN d/o of pregnancy (PreE, HELLP, gHTN)
    - Beta 2 agonist: terbutaline

  - Additional increases in Membrane permeability:
    - PreE 2/2 endothelial damage
    - Mag sulfate —> direct cardiac depression and may cause direct capillary endothelial damage

PULMONARY EDEMA

RISK FACTORS

Category	Specific risk factors
Pre-existing pre-pregnancy conditions	Cardiovascular disease (hypertension, ischaemic heart disease, congenital heart disease, valvular heart disease, arrhythmias, cardiomyopathy) Obesity Increased maternal age Endocrine disorders (phaeochromocytoma and hyperthyroidism)
Specific diseases in pregnancy	Pre-eclampsia Cardiomyopathy Sepsis Preterm labour Amniotic fluid embolism Pulmonary embolism
Pharmacological agents	β-Adrenergic tocolytic agents Corticosteroids Magnesium sulphate Illicit drugs including cocaine
Iatrogenic intravenous fluid therapy	Positive fluid balance > 2000 ml
Fetal conditions	Multiple gestation

PULMONARY EDEMA

- Can occur at any time, most likely peripartum from fluid overload.
    - Highest risk after delivery 2/2 autotransfusion & doubled CO.
        - CO highest 24-48h postpartum (80% above pre-delivery)

- High risk w/ diastolic dysfunction 2/2 cHTN (LVH) or preE (placental inflammatory mediators also affect cardiac myocytes)
    - Impaired LV relaxation ^^ pulm venous pressure & disturbs normal Starling balance of hydrostatic/oncotic pressures.

- Pulmonary Edema patients need continuous management x at least 72hours postpartum.
        - CO is highest and cardiopulmonary events/failure are most likely
            - Takes 2+ weeks for CO to return to pre-pregnant levels.

PULMONARY EDEMA

SIGNS/SYMPTOMS:

SOB, agitation, cough, tachycardia, tachypnea, crackles&wheezes, SpO2 drop

TESTS:

Lung US/POCUS, CXR, **BNP, TTE**, ECG, ABG (PaO2)

MEDS:

O2

Lasix/furosemide

Nitroglycerin & Morphine (if HTN)

MANAGEMENT:

PEEP/CPAP/BIPAP/intubation

fluid restriction

PULMONARY EDEMA

Important to distinguish if pulmonary edema is due to:

- fluid overload (can be iatrogenic**)
- capillary leak: sepsis, ARDS, Pre-E endothelial damage
- **…..OR HEART FAILURE!!**
- Pre-E can cause **significant** diastolic dysfunction. ~ 1 in 10 PReE w/ SF develop HFpEF! Can be precipitated by iatrogenic fluid overload.
- Cardiomyopathy can be undiagnosed or misdiagnosed in pregnancy, esp with limited prenatal care or provider experience.

- A **BNP** should help rule in or out cardiac function as a causative factor in pulm edema.
  - Pregnancy BNP cutoffs: (pg/mL)
    BNP: > 50, **NTpro-BNP**: TM 1&2: > 200, TM3 >150
- **High BNP warrants TTE and cardiac workup.**
- Esp in setting of Pre-E where beta blockers are commonly 1st line anti-HTN tx in pulmonary edema, a pt with reduced EF may require combo of inotropes and carefully titrated vasodilators instead, to assist ventricle in moving fluid forward and reducing pulmonary venous congestion.

PULMONARY EDEMA

TWO PULMONARY EDEMA RISK GROUPS IN PREGNANCY:

NON-HYPERTENSIVE

[similar treatment to non-pregnant pt]

HYPERTENSIVE

[PreE is unique to pregnancy w/ special considerations]

PULMONARY EDEMA

NON-HYPERTENSIVE

- May be normotensive or hypOtensive.
- Impaired pulmonary interstitial fluid regulation:
  - Hydrostatic pressure, colloid osmotic pressure & capillary permeability
- Reduced pulmonary reserve (FRC) in pregnancy = reduced tolerance to hypoxia.

CAUSES:

- Tocolysis: β-2 agonist increases CO & hydrostatic pressure
- Sepsis
- Pre-existing CV dx: poor diastolic or systolic function
- Pregnancy-assc CV dx (PPCM, heart failure, ischemic heart disease)
- BBs: reduce myocardial contractility
- AFE w/LV systolic failure
- Aspiration
- Iatrogenic IV Fluid overload
- Steroid use: Celestone = Betamethasone (for fetal lung maturity)
- Magnesium sulfate (for neuropotection in preterm for non-HTN/PreE pt) may cause direct cardiac depression or direct capillary damage

TREATMENT Non-Hypertensive:

Lasix, O2, fluid restriction, PEEP/CPAP/BIPAP/intubation

[Same as non-pregnant patient]

PULMONARY EDEMA

Hypertensive Pulmonary Edema

* **Consideration of delivery if antepartum acute pulmonary edema**.*

Causes:

- cHTN, preE, SIPE, gHTN
- Illicit drug use —> HTN
- Endocrine dx: pheochromocytoma, hyperthyroidism
- **PreE:**
  - Diastolic dysfunction & higher LVEDP (higher hydrostatic pressure)
  - Reduced plasma colloid osmotic pressure, colloid osmotic pressure & increased endothelial permeability
  - Endothelial damage in preE/HELLP increases capillary membrane permeability
  - Mag sulfate for seizure prevention is independent risk factor for pulmonary edema 2/2 theorized direct cardiac depression and direct capillary damage
  - HTN crisis (higher LVEDP/hydrostatic pressure)

PULMONARY EDEMA

Goals of Treatment in HYPERTENSIVE Pulmonary Edema:

- Reduce LV PREload & AFTERload
- Reduce/prevent myocardial ischemia
- Adequate O2 & ventilation w/ clearance of pulmonary edema

PULMONARY EDEMA

Treatment HTN Pulmonary Edema

- O2 & ventilation
- Nitroglycerin & furosemide

Nitroglycerin:

- IV: 5mcg/min, gradually increasing Q 3–5 min, to max 100 mcg/min
- SL spray: (400mcg, 1–2 puffs every 5–10min

Reduce SBP/DBP 30mmHg over 3–5min, then slower reduction to ~140/90mmHg.

- Nitrates benefit: rapidly offload ventricles, but preserve contractility & forward flow.
- Excessive BP drop (esp PreE/PIP) can be tx with small 2-5 mcg epi to preserve contractility (Beta).
- PreE/PIP intravascularly dry - can have excessive response to anti-HTN meds —> compromising fetal perfusion.

Furosemide:

- IV: 20–40mg over 2 min, repeat 40–60mg after ~ 30min if inadequate diuresis (maximum dose 120mg/hr)

2nd line anti-HTN

CCB nicardipine/nifedipine (esp if diastolic dysfunction is diagnosed)

3rd line anti HTN

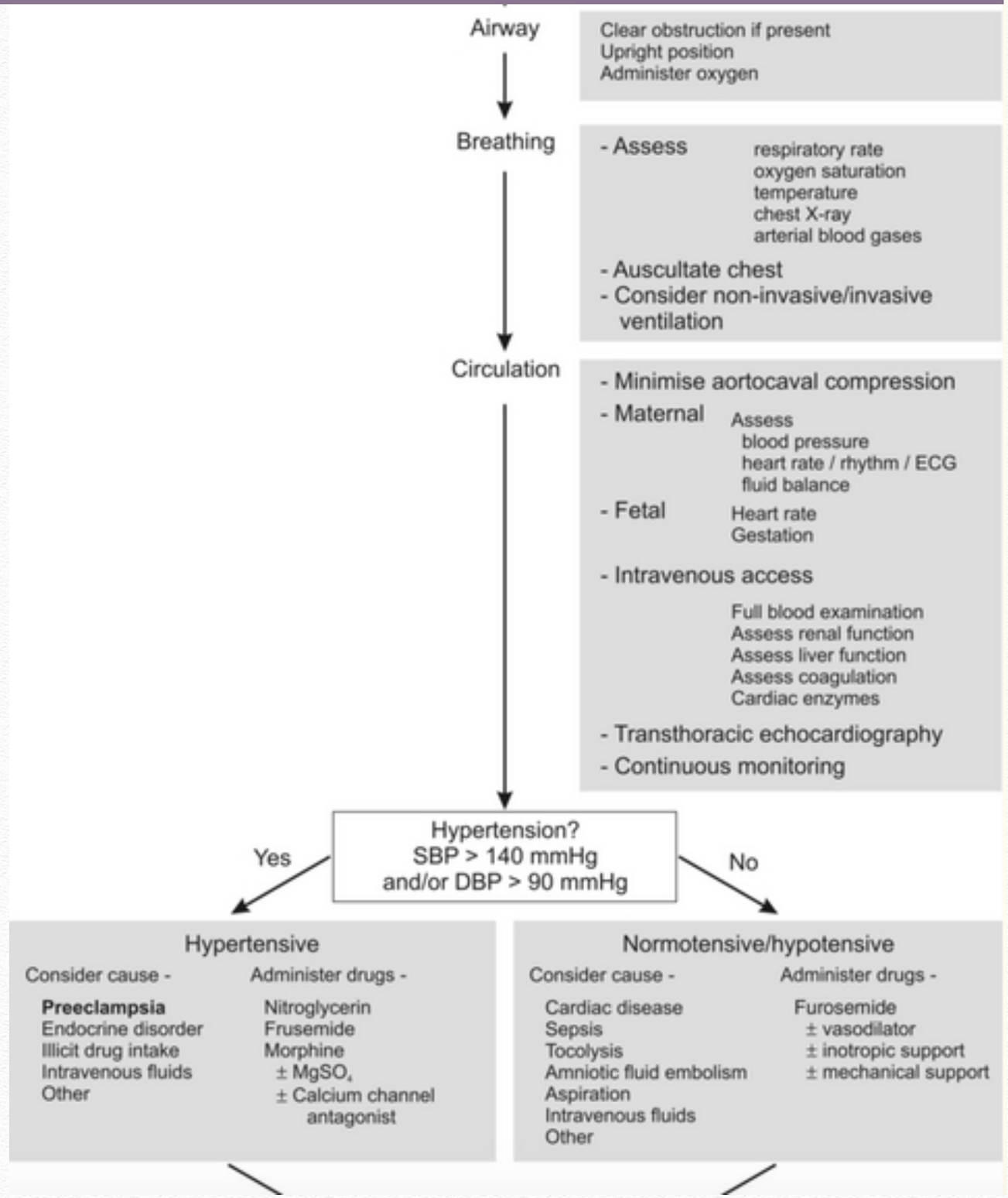
(reduce preload via venous dilaton): Morphine 2-3mg IV

PULMONARY EDEMA

ACUTE PULMONARY EDEMA IN PREGNANCY ALGORITHM

1) **Activate emergency response team (additional anesthesia providers, OBGYNs for stat C/S)**

2) MANAGE AIRWAY



Stabilize, plan for safe delivery, transfer to appropriate level of care with continuous monitoring

PULMONARY EDEMA

- PreE puts women at higher risk of developing DIASTOLIC dysfunction —> pulm edema in pregnancy with fluid shifts, & HFpEF later in life

- Pts with HTN disorders of pregnancy need more specialized attention, as they have higher incidence of ICU-level care.

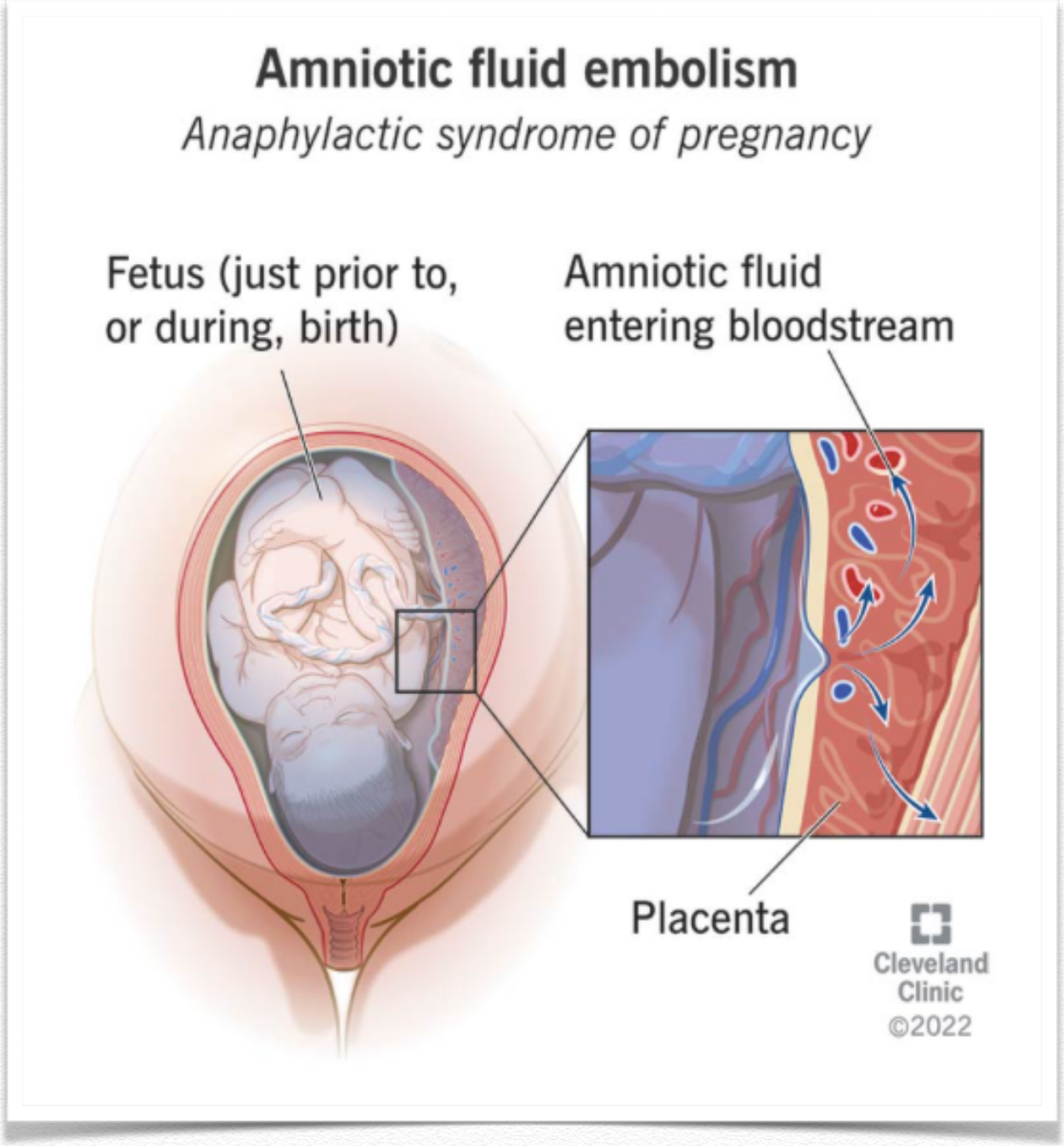
- More PreE w Severe Features pts should probably have ECHO during pregnancy/antenatal. Especially considering long-term implications of CV risk later in life 2/2 PreE.

Amniotic Fluid Embolism

Amniotic Fluid Embolism

- ***Anaphylactoid reaction*** to amniotic fluid (containing unknown fetal antigens) entering maternal circulation.

- Anaphylactoid requires **no prior sensitization**, while anaphylaxis does.
- Anaphylaxis is IMMUNE IgE-mediated to activate histamine, basophils, inflammatory leukotrienes, prostaglandins, PLT-activating factor.
- Histamine: vasodil, smooth muscle constriction, HOTN, tachycardia, erythema, urticaria, edema, angioedema, while leukotriene$_4$ & prostaglandin$_2$ = bronchoconstriction & ^ vascular permeability.
- **Anaphylactoid** directly activates bradykinin +/- complement cascade – directly activating mast cells & basophils
- SAME clinical picture as anaphylaxis – same treatment given.



Amniotic Fluid Embolism

- Amniotic fluid contains cellular elements/other debris (lanugo hair & vernix). Mucin from meconium causes a particularly intense reaction in the pulmonary arterioles. DIC 2/2 AFE does not appear to depend on quantity of amniotic fluid that enters maternal circulation or reaches pulmonary circulation – therefore believed to be patient-dependent anaphylactoid response.

- **This breech may commonly occur in normal pregnancy**, ***but only SOME WOMEN have an anaphylactoid response to it.***

- Some women can have "AFE" or small amounts to amniotic fluid enter their circulation ***and have no response.***

- AFE Signs and Symptoms:
  - Rapid dyspnea, hypoxia —> Cardiovascular collapse, then progresses to DIC

Amniotic Fluid Embolism

Proposed Physiology of CV Collapse

- PLT activation & degranulation releases **serotonin** & **thromboxane A2** (which normally are powerful vasoconstrictors to help hemostasis — but cause massive pulmonary vasoconstriction in AFE.)

- ***Histamine release*** ***of anaphylactoid rxn may also contribute to collapse… warranting anaphylaxis meds (epi, benadryl, hydrocortisone, Pepcid (& vaso +/- methylene blue for vasoplegia)***

- Pulm vasoconstriction/HTN leading to cardiorespiratory failure
  - Causes severe RV dilation & failure. LV compromised from bulging RV –> pulm edema & LV failure
    - **CBP/ECMO/IABP/RVAD**
    - Inhaled N.O.
    - MTP/++cryo/ may need fibrinogen concentrate +/- recombinant F7a for coagulation factor consumption/DIC
    - Vasopressors/inotropes: vasopressin* for vasoplaegia – less increase in PVR in setting of RV failure than with neo, and can try methylene blue as well. Pt **MAY NEED V-A ECMO**
- **Vagal** reflex—> bradycardia & vasodilation.

Amniotic Fluid Embolism

DIC from AFE

- Widespread systemic inflammation causes PLT/clotting factor activation and consumption

- Also Amniotic fluid contains vasoactive and procoagulant products including PLT-Activating Factor, cytokines, bradykinin, thromboxane, and arachidonic acid.

- **Tissue Factor concentrations in amniotic fluid are higher than in maternal circulation, and cause widespread coagulation cascade activation when it enters maternal circulation.** Rapid consumption of clotting factors —> **DIC**

    - Extrinsic pathway activation: [TF & F7a complex]—>common pathway: [(F10 +F5)]—> F2(prothrombin) —> F1(fibrin) (+F13 (fibrin stabilizing factor)

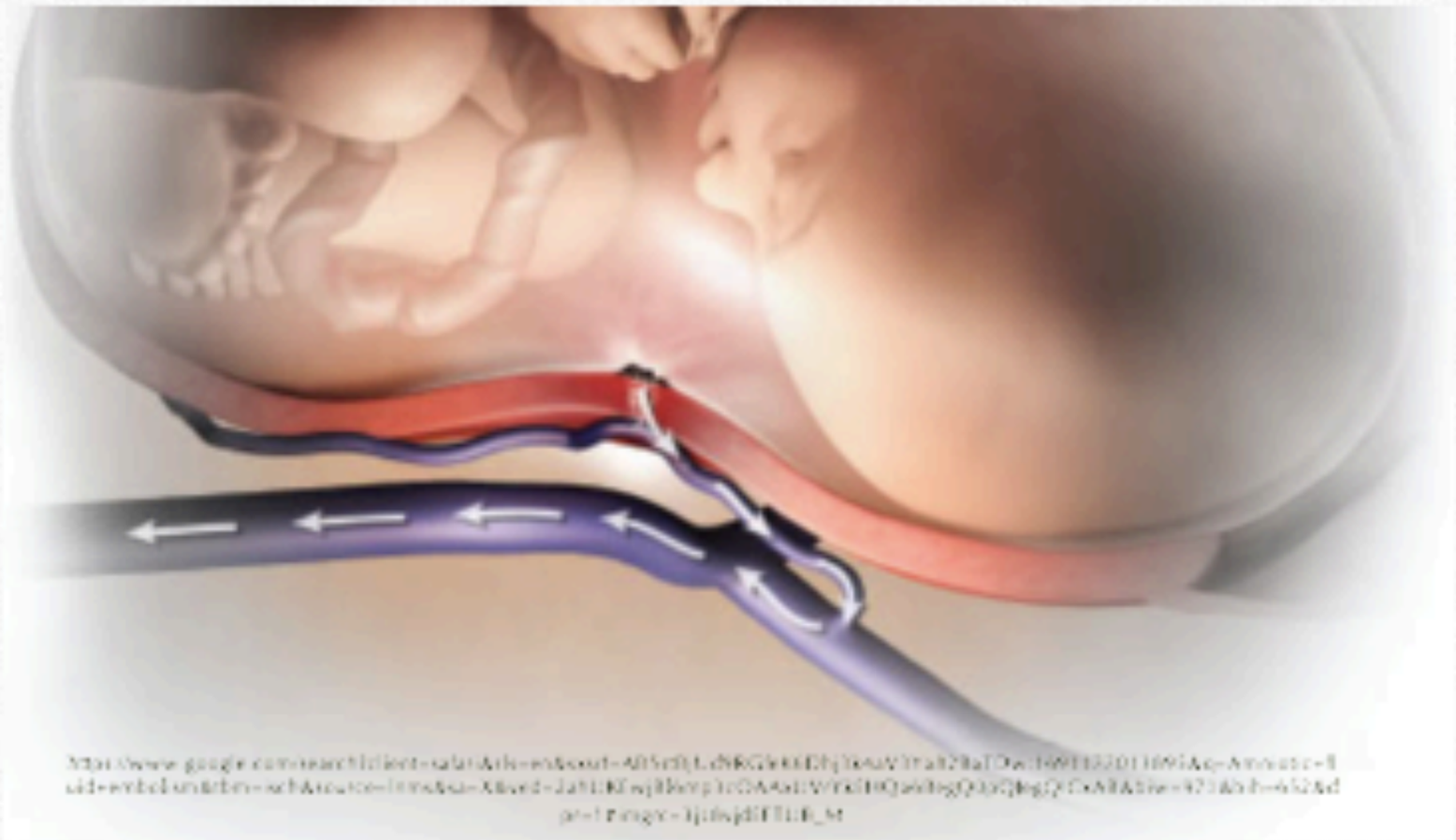
- Possible maternal increases in endothelin when Amniotic fluid enters vasculature. Endothelin has powerful bronchoconstrictive and pulmonary vasoconstrictive properties, which may contribute to CV collapse.

Amniotic Fluid Embolism

AFE CAN OCCUR:

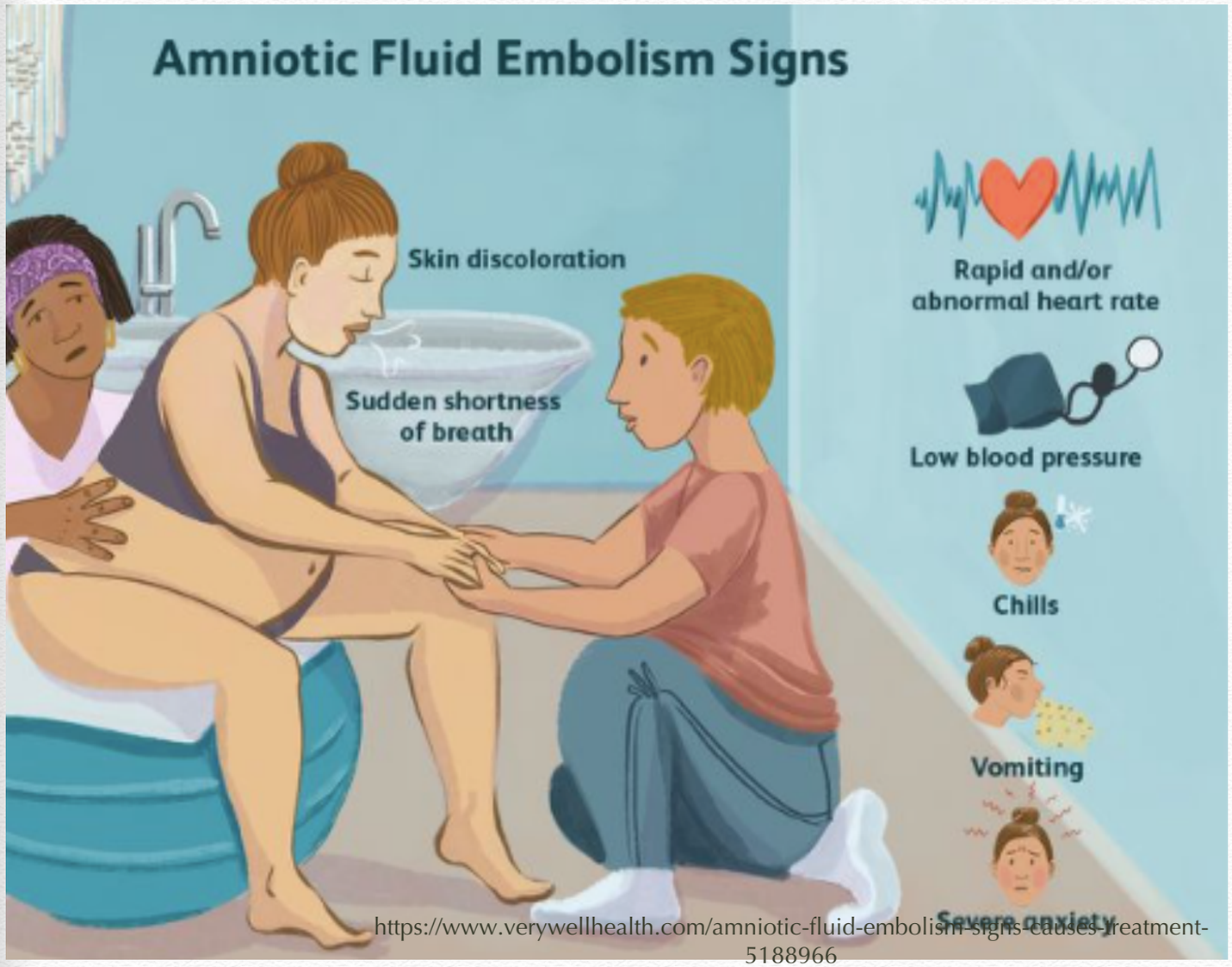
- in healthy women
- during labor
- during cesarean section
- after abnormal vaginal delivery
- during 2nd trimester
- up to 48 hours postpartum.
- during abortion
- after abdominal trauma
- during amnio-infusion

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4874066/



Amniotic Fluid Embolism

Signs & Symptoms



https://www.verywellhealth.com/amniotic-fluid-embolism-signs-causes-treatment-5188966

- Rapid dyspnea
- Hypoxia
- Unconsciousness
- HOTN
- Cardiovascular collapse
- Progresses to DIC

Amniotic Fluid Embolism

DIAGNOSIS BY EXCLUSION

RULE OUT:

- Total spinal
- L.A.S.T
- Eclampsia/seizures
- Pulmonary Embolism
- Anaphylaxis
- Septic shock

Amniotic Fluid Embolism

Since AFE is considered an **ANAPHYLACTIOD** reaction which implies massive **histamine release**: it is prudent to give **ANAPHYLAXIS** meds as well:

- **Epi:** 10-100 mcg doses to effect, up to 1 mg for CV collapse
- **Hydrocortisone:** 100-200mg
- **Diphenhydramine (H_1):** 100mg
- **Famotidine (H_2):** 20mg
- **Bronchodilators:** albuterol, sevo, epi, terbutaline, ketamine
- Pressors: epi/levo/neo gtt.
    - If vasoplegia: vasopressin --> methylene blue.

- **Ketamine** useful in induction/GA as it is a cardiac-stable, potent broncho dilator. Benefit of bronchodilation here outweighs the potential PVR increases (which aren't reliably shown to be consequential)
- **Etomidate** will also maintain CV stability for RSI & airway securement.

Amniotic Fluid Embolism

A-OK Therapy for AFE

- **A**tropine 0.2-1mg
- **O**ndansetron 8mg
- **K**etorolac 30mg

Limited evidence:

- Unable to test A-OK in RCT due to unpredictability and unknown cause of anaphylactic/anaphylactoid reaction AFE.
- AFE is diagnosis of exclusion with no known diagnostic serum markers/labs.
- Only available literature is retrospective

HOWEVER:

- Multiple case studies show presumed efficacy of AOK therapy.
- AOK medications block pathways contributing to cardiovascular collapse in AFE

Amniotic Fluid Embolism

Atropine 1mg

- Blocks ACh
  - inhibits vagal reflex
  - inhibits ACh-mediated pulmonary artery vasoconstriction/spasm

Ondansetron 8mg

- Blocks 5HT(serotonin)
  - 5HT causes pulmonary vasculature vasoconstriction & PLT entrapment

Ketorolac 30mg

- Blocks Thromboxane A2
  - Thromboxane A2 activates PLT to recruit more PLT in pulmonary vasculature, worsens pulmonary HTN and RV strain

AOK-for-AFE-HANDOUTS-no-references
Sohn_Jackie
Amniotic fluid embolism

Amniotic Fluid Embolism

AOK for AFE Case Report 1

Case Report | Open Access
Volume 2017 | Article ID 8458375 | https://doi.org/10.1155/2017/8458375

Show citation

Atypical Amniotic Fluid Embolism Managed with a Novel Therapeutic Regimen

Shadi Rezai,[1] Alexander C. Hughes,[2] Tracy B. Larsen,[3] Paul N. Fuller,[1] and **Cassandra E. Henderson**[4]

- A 26 y/o hispanic G2P1001, at 38wks 1day. PMH: obesity (BMI 41) & GDM2. Pt arrived to ER c/o of shortness of breath x 8 hrs.
- Maternal BP WNL, HR 144, RR 24, mild fever. FHR tachycardia with non-reactive tracing.
- Emergent C/S under GA with propofol/succs RSI induction (BP dropped to 80/40, treated with vasopressors)

After placenta delivery:

- **HR 140s, SpO2 72%, ETCO2 dropped from 32 to 0.** Vent/circuit checked-OK.
- **A-OK: 0.2 mg Atropine, 8 mg Ondansetron, and 15 mg Ketorolac given IV**
- **Within 2-3 min, SpO2 recovered to 97% and BP increased to 138/68 mm Hg, CO2 returned to 32 mm Hg,** but tachycardia remained with a HR of approximately 140 BPM
- 2L EBL PPH: uterotonics, blood products
- Once stabilized, remained intubated to ICU

Amniotic Fluid Embolism

DIAGNOSIS BY EXCLUSION

AOK for AFE Case Report 1

RULED OUT:

- **PE:** CT scan with & without contrast:
  - only showed bibasilar atelectasis with no evidence of definite consolidation and/or pneumonia.

- **Acute pulmonary pathology**: CXR negative

- **DVT:** Lower extremity Doppler ultrasound:
  - negative results for deep venous thrombosis

- **Labs: (DIC? LAST?)**
  - negative for DIC (normal Pt/PTT/INR)
  - Blood culture, urine culture, and sputum cultures negative.

- **Abnormal placental pathology: Abruption? Chorioamnionitis?**: [2L EBL PPH]
  - Placental pathology negative

Pt extubated the day after c/s.
Remained afebrile, asymptomatic with stable vital signs.
The patient and newborn had uneventful recovery, discharged 2 days later.

Amniotic Fluid Embolism

AOK for AFE Case Report 2

Ochsner J. 2022 Fall; 22(3): 253–257. PMCID: PMC9477128
Published online Fall 2022. doi: 10.31486/toj.21.0107 PMID: 36189093

Atropine, Ondansetron, and Ketorolac: Supplemental Management of Amniotic Fluid Embolism

Miranda Long, MD, [1] Jane Martin, MD, [1,2] and Joseph Biggio, MD, MS [1,2]

Author information Copyright and License information PMC Disclaimer

- A 34y/o caucasian G4P2 had repeat c/s 39 wks.
- After placenta delivery, hypoxia & hypotension developed, followed by cardiac arrest.
- Protocols for management of maternal cardiac arrest were followed. (CPR, AED, ACLS, LUD)
- Echocardiogram demonstrated right ventricular dilation and hypokinesis.
- **AOK was administrated during prolonged cardiac arrest, and spontaneous circulation returned**.
- The patient extubated 3 days later and discharged one week later **without neurologic deficits.**

Amniotic Fluid Embolism

AOK for AFE Case Report 2

- Normal CS under spinal anesthesia.
- ~15 min after delivery, during closure, patient c/o *feeling of impending doom*.
- HR decreased to 40/min, SpO2 44% on room air, & decorticate posturing.
- Immediate conversion to GA: RSI & ETT
- PEA noted.
    - ACLS/Maternal CPR initiated
    - Atropine (1 mg IV) & epinephrine (1 mg IV) administered with subsequent return of spontaneous circulation.
        - Bedside echo: RV failure - Milrinone infusion.
- A 2nd episode of PEA:
    - **Atropine (1 mg IV), ondansetron (8 mg IV), and ketorolac (30 mg IV) were administered.** Second dose of epinephrine (1 mg IV) was given, and return of spontaneous circulation was achieved.
- 9 mins later, a 3rd episode of PEA - CPR resumed:
    - Dobutamine infusion and 3rd dose of epi (1 mg IV), with resulting return of spontaneous circulation.

Amniotic Fluid Embolism

ANESTHETIC MANAGEMENT

CONSCIOUS:

- CALL FOR HELP: May progress to LOC, STAT C/S
- AOK therapy, ANAPHYLAXIS therapy, supportive care, O2, usually stat c/s from fetal distress
- If STAT C/S: GA with ETT: **ketamine/etomidate*** & Roc/Succs RSI induction +/- vasopressors: cardiac stable induction**

UNCONSCIOUS:

- CALL FOR HELP - OBGYN FOR **STAT C/S**
- CPR, AED, A-OK THERAPY
- Continue CPR to circulate medications: AOK + EPINEPHRINE
- INTUBATE - NO MEDS (unconscious) – ketamine, versed & fent to maintain GA: sevo < ½ MAC ok: ^^ sevo relaxes uterus (anticipating DIC/PPH in AFE) and N_2O may increase PVR – skip and opt for 100% O_2
- BLOOD PRESSURE SUPPORT, DIC/PPH support. – **MAY NEED V-A ECMO**

*IVs in Upper extremities - Lower extremity flow impaired by gravid uterus

Amniotic Fluid Embolism

DIAGNOSIS BY EXCLUSION

RULE OUT:

- Total spinal
- L.A.S.T
- Eclampsia/seizures
- Pulmonary Embolism/pathology
- Anaphylaxis
- Septic shock

Amniotic Fluid Embolism

Since AFE is a diagnosis by exclusion – you'll likely be throwing the kitchen sink at these pts wondering WHAT is causing collapse.

- ACLS protocols – myocardial infarction?
- It could be anaphylaxis: you give anaphylaxis meds.
- It could be LAST (even at sub-toxic doses): you give intralipid
- (which a few case reports have shown intralipid to achieve ROSC in suspected AFE due to intrinsic inotropic and pulm vasodilating properties of intralipids completely apart from the "lipid-sink" indication*)

- You Rule out Eclamptic Seizure → MAG?
- You Question a PE → ECMO/TTE/septic emboli → PE/Stroke?

- **Just to explain that AOK is not the only TX these pts will receive… but can be added to this cloudy clinical picture in hopes of ROSC.**

ECLAMPSIA

ECLAMPSIA

ANESTHESIA MANAGEMENT

[Prevent aspiration, break seizure, & control HTN]

PREVENT ASPIRATION - suction, positioning

4g MAGNESUIM SULFATE IV bolus, then 1-2mg/hr gtt

Benzodiazepines to break seizure.

If airway compromise - **EXPECT ++AIRWAY EDEMA.**
Intubation: RSI with succs & propofol.
Propofol push alone may break seizure, but the risk of apnea/aspiration in pregnant airway requires intubation
Considered full stomach up to 72h post-partum

BLOOD PRESSURE CONTROL *if > 160 SBP or 110 DBP*.

ECLAMPSIA

ANESTHESIA MANAGEMENT

- **Differential dx:**
  - epilepsy, cerebral infarction/hemorrhage, subarachnoid hemorrhage, cerebral edema, malignant HTN, benign & malignant cerebral tumors, cerebral abscess, viral, bacterial, parasitic infestations
  - hyponatremia, hypocalcemia, hypoglycemia, & hyperglycemia.

- **BP control:**
    - IV Labetalol (20, 40, 80 mg dose, max 220mg total)
    - IV Nicardipine
    - IV Hydralazine
    - PO Nifedipine
  - *Avoid Labetalol is asthmatics.* *(B2 blockade = airway smooth muscle construction)*
  - Avoid ACE-I, Spironolactone in pregnancy.

    - Expect exaggerated HTN response to intubation.
      - IV anti-HTN meds: esmolol, metoprolol, hydralazine, labetalol
      - ++ propofol

ECLAMPSIA

ANESTHESIA MANAGEMENT

- Magnesium will potentiate NMBDs
- DO NOT FLUID OVERLOAD – Needs **fluid restriction**.
- LABS:
  - CHEM, LFTs, CBC
  - COAGS: regardless of PLT count: +/- TEG.
    - Liver dysfunction highly possible, and coag factor dysfunction possible

ECLAMPSIA

Magnesium Sulfate

- **MgSO4- is NOT for BP control. It's for seizure prevention.**

- **Adverse effects**: muscle weakness leading to respiratory paralysis and failure (careful emerging in GA, Mag causes extra muscle relaxation) then progresses to cardiac arrest as hypoxia worsens and myocardial conduction fails.
- Toxicity: 6-9mg/dL is therapeutic. If DTRs intact, resp failure is not from Mag — seek another cause.

Mag Toxicity Reversal:

- (1g calcium gluconate **(10 mL of 10%)** IV over 1-2 min (slowly) to avoid HOTN +/- bradycardia)
  - Ca++ competitively inhibits Mag at NMJ, but can wear off if Mag level stays high
- Furosemide may help increase urinary excretion of magnesium
- Ca++ gluconate = 1/3 potency of CaCl.

ACOG Eclampsia Checklist

https://www.scdhhs.gov/sites/default/files/ACOG-District-II-Checklist-Eclampsia.pdf

Eclampsia Checklist

- ☐ Call for assistance **(Hospital should identify a Rapid Response Team)** to location of the event
- ☐ Check in:
  - ○ OB Attendings/ Fellows/Residents
  - ○ Three RNs
  - ○ Anesthesia
  - ○ Neonatology (if indicated)
- ☐ Appoint a leader
- ☐ Appoint a recorder
- ☐ Appoint a primary RN and secondary personnel
- ☐ Protect airway
- ☐ Secure patient in bed, rails up on bed, padding
- ☐ Lateral decubitus position
- ☐ Maternal pulse oximetry
- ☐ IV access/PEC labs
- ☐ Supplement oxygen (100% non-rebreather)
- ☐ Bag-mask ventilation on the unit
- ☐ Suction available
- ☐ Continuous fetal monitoring (if appropriate)

Initial Medications

- ☐ Load 4-6 grams 10% magnesium sulfate in 100 ml solution IV over 20 minutes
- ☐ Magnesium sulfate on infusion pump
- ☐ Magnesium sulfate and pump labeled
- ☐ Magnesium sulfate 10 grams of 50% solution IM (5 grams in each buttock) if no IV access
- ☐ Magnesium sulfate maintenance 1-2 grams/hour continuous infusion

Contraindications: pulmonary edema, renal failure, myasthenia gravis

Anticonvulsant Medications

(For recurrent seizures or when magnesium sulfate is contraindicated):

- **Lorazepam** (2-4 mg IV x 1, may repeat x 1 after 10-15 minutes)
- **Diazepam** (5-10 mg IV every 5-10 minutes to maximum dose 30 mg)
- **Phenytoin** (15-20 mg/kg IV x 1, may repeat 10 mg/kg IV after 20 minutes if no response); avoid with hypotension, may cause cardiac arrhythmias
- **Keppra** (500 mg IV or orally, may repeat in 12 hours); dose adjustment needed if renal impairment

Persistent Seizure

- ☐ Neuromuscular block and intubate
- ☐ Obtain radiographic imaging
- ☐ ICU admission

Antihypertensive medications **SBP ≥ 160** or **DBP ≥ 110**

- **Labetalol** (20, 40, 80, 80 mg IV* over 2 minutes, escalating doses, repeat every 10 minutes or 200 mg orally if no IV access); avoid in asthma or heart failure, can cause neonatal bradycardia
- **Hydralazine** (5-10 mg IV* over 2 minutes, repeat in 20 minutes until target blood pressure is reached)
- Repeat BP every 10 minutes during administration

*** Maximum cumulative IV administered doses should not exceed 25 mg hydralazine; 220 mg labetalol in 24 hours.**

After Seizure

- ☐ Assess neurologic status every 15 minutes
- ☐ PEC labs: CBC, Chem 7, LFT, Uric Acid, LDH, T&S, PT/PTT, Fibrinogen, Magnesium
- ☐ Foley catheter (Hourly I&O. Report output < 30 ml/hour)

Strict I&O (no less than every 2 hours). Report urine output to the clinician if < 30 ml/hr. Foley catheter should be placed if urine output is borderline or strict I&O cannot be maintained. Urometer should be utilized if the urine output is borderline or < 30 ml/hr.

Delivery Plan

- ☐ Ensure that there is an appropriate plan for delivery

Magnesium Toxicity

- ☐ Stop magnesium maintenance
- ☐ Calcium gluconate 1 gram (10 ml of 10% solution) IV over 1-2 minutes

Postpartum

- ☐ Oral antihypertensive medication postpartum if > 150/100.
- ☐ Blood pressure monitoring is recommended 72 hours after delivery and/or outpatient surveillance (e.g., visiting nurse evaluation) within 3 days and again 7-10 days after delivery or earlier if persistent symptoms.

Debrief

- ☐ Debrief with the whole obstetric care team and document following the debrief

ACOG

Safe Motherhood Initiative

District II

HELLP

Preeclampsia vs HELLP

- **PreE comes in mild-severe cases, whereas HELLP is ALWAYS severe, ALWAYS an emergency.**
- Is preE —> HELLP a continuum? Or separate etiologies? Unclear.
    - HELLP has long been considered a severe form of preE but HTN isn't necessarily part of the diagnosis.
    - PreE & Eclampsia however do retain HTN in diagnosis.
- **HELLP has 20/30% risk of DIC**
- **HELLP is an EMERGENT condition.**

***H*: Hemolysis**

***EL*: Elevated Liver (enzymes)**

***LP*: Low Platelets**

HELLP

***H*: Hemolysis**

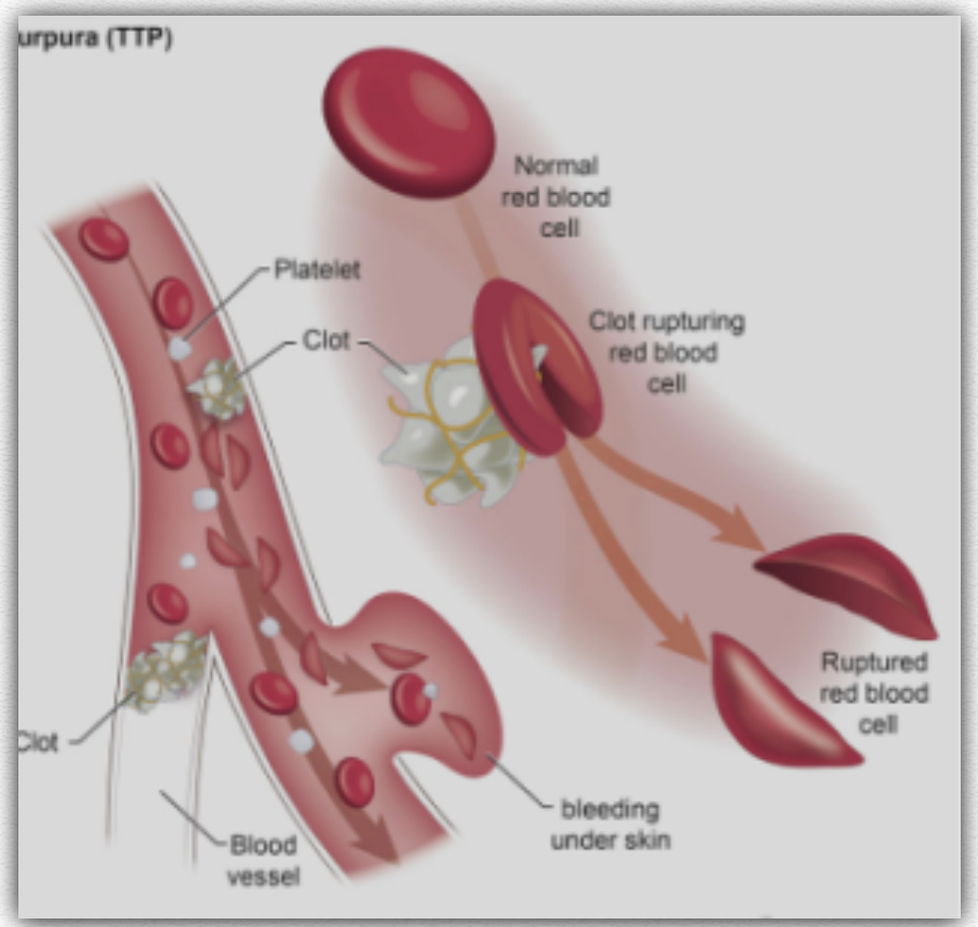
***EL*: Elevated Liver (enzymes)**

***LP*: Low Platelets**

HELLP

H: Hemolysis

- As systemic inflammation of microvasculature activates PLTs—> activates coagulation cascade —> forms PLT plugs and tiny fibrin clots along the microvascular endothelium
- RBCs floating through these micro vessels get damaged/sheared from scraping along these hardened fibrin clots (**SCHISTOCYTES**)



https://cancer.osu.edu/for-patients-and-caregivers/learn-about-cancers-and-treatments/cancers-conditions-and-treatment/benign-blood-diseases/thrombotic-thrombocytopenic-purpura

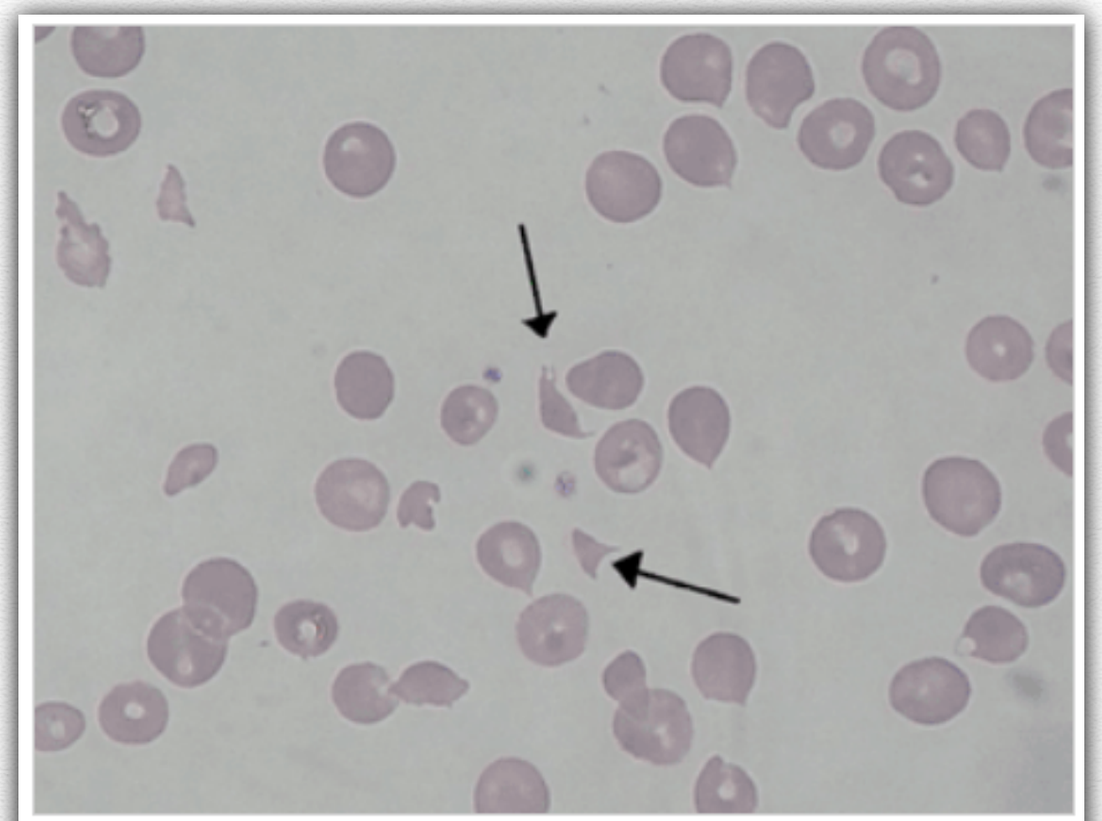


Image: Schistocytes in blood smear (Photo courtesy of Dr. Paulo H.O. Mourao).

https://www.labmedica.com/hematology/articles/294741378/fragmented-red-blood-cell-measurement-automated.html

HELLP

H: Hemolysis

LAB FINDINGS in HEMOLYSIS:

- **Schistocytes:**
  - **Indicates profound widespread microangiopathic thrombosis**
  - **Considered a hematologic emergency. (NO NEURAXIAL)**
    - (HELLP, hemolytic anemia, TTP, HUS)

- **Elevated Billirubin** 2/2 increased hemoglobin catabolism
  - **> 1.2mg**

- **Elevated Lactate dehydrogenase**. LDH released from RBC breakdown
  - (normal ~140-280 u/L, **hemolysis dx > 600 u/L**)

- **Elevated ALT/AST**
  - (normal ~8-50 u/L, **elevated > 70 IU/L)**

- **Thrombocytopenia**
  - **< 100K from consumption**

- **Decreased Serum haptoglobin.** SH binds free hemoglobin from lysed RBCs – rapidly consumed in hemolytic states.
  - (normal 40-200, **< 40 mg/dL**)

HELLP

EL: Elevated Liver

- *Only* alk phos is normally elevated in pregnancy
- PreE pt w/ elevated liver transaminases (AST, ALT) lactate dehydrogenase (LDH), & uric acid might be progressing to HELLP.
  - High AST & ALT, bilirubin, & LDH
  - Liver transaminases typically >500 U/L in HELLP. (Much higher in HELLP than preE. PreE may see only a 2-3x increase in severe cases)

Preeclampsia-induced Liver Dysfunction, HELLP syndrome, and acute fatty liver of pregnancy

HELLP

EL: Elevated Liver

HELLP Fatal Liver complication:

Glisson's Capsule *Rupture*

Glisson's capsule/liver capsule hematoma:

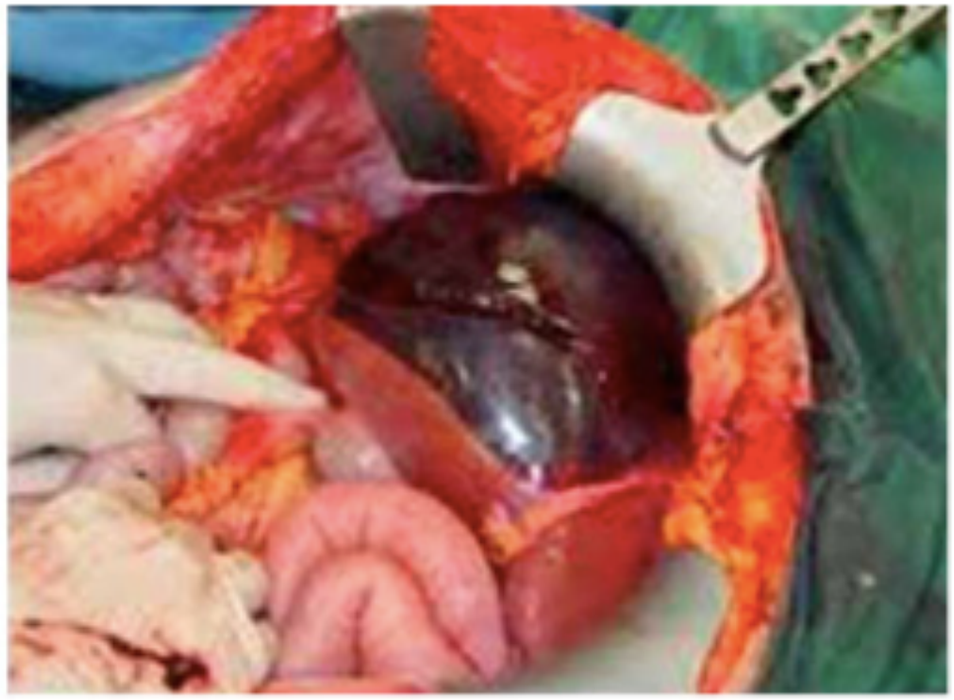


Fig.2: Intraoperative finding of subcapsular hematoma.

http://www.casereports.in/articles/6/1/Rupture-of-Subcapsular-Hematoma-of-Liver-in-Severe-Preeclampsia-with-HELLP-syndrome.html

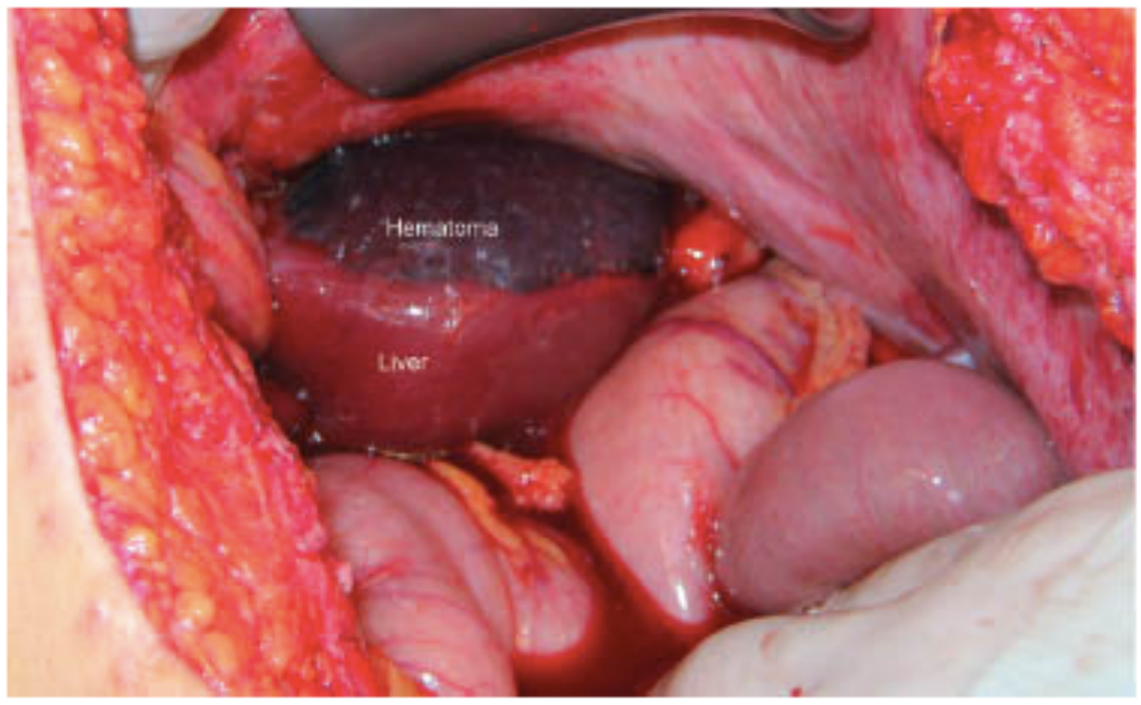


Figure 1

https://www.degruyter.com/document/doi/10.1515/crpm-2013-0083/html

HELLP

EL: Elevated Liver

- HELLP & PreE can have liver subcapsular hematoma.
  - Fatal if ruptured.
  - **Can also occur postpartum**
  - **Any HELLP pt c/ RUQ pain needs workup.**

- Patients with subcapsular hepatic hematoma typically present with:
  - sudden onset severe pain:
    - epigastric & RUQ, radiating to back & right shoulder.
  - nausea & vomiting
  - shortness of breath.
  - anemia
  - hypotension

  - Diagnostic Imaging: U/S, CT scan, or MRI

Hepatic Rupture Complicating HELLP Syndrome in Pregnancy

HELLP

LP: Low PLT = THROMBOCYTOPENIA

- Thrombocytopenia from PLT activation & consumption secondary to inflammation activating coagulation cascade throughout entire body.
- Often cannot have neuraxial (PLT < 70,000/mm^3)
- ***GA usually only safe method for cesarean***
  - **Airway may be VERY swollen**
    - more prone to mucosal bleeding & swelling.
  - **CAREFUL intubation and** CAREFUL extubation**.
    - Extubate wide awake

HELLP Syndrome – StatPearls – NCBI Bookshelf

HELLP

NEURAXIAL CONSIDERATIONS

Usually not candidates for Neuraxial Anesthesia —> GA

- **Liver dysfunction & Thrombocytopenia continue to worsen until delivery.**
- **RAPID, continuous drop in PLT.**
    - Minimum Q6h labs, or as close to epidural/neuraxial placement as possible.
- Liver dysfunction can increase coagulopathy
    - High risk of DIC (20-30%)
        - Q6h PLT & **coags/TEG** if bleeding concerns
        - C/S usually GA if no epidural in place prior to diagnosis
            - **Watch for increased airway swelling!**
            - **POSITIONING/RAMPING, PREOXYGENATE**
            - **Glidescope to decrease instrumentation and increase 1st pass success.**

HELLP

***H*: Hemolysis**

***EL*: Elevated Liver (enzymes)**

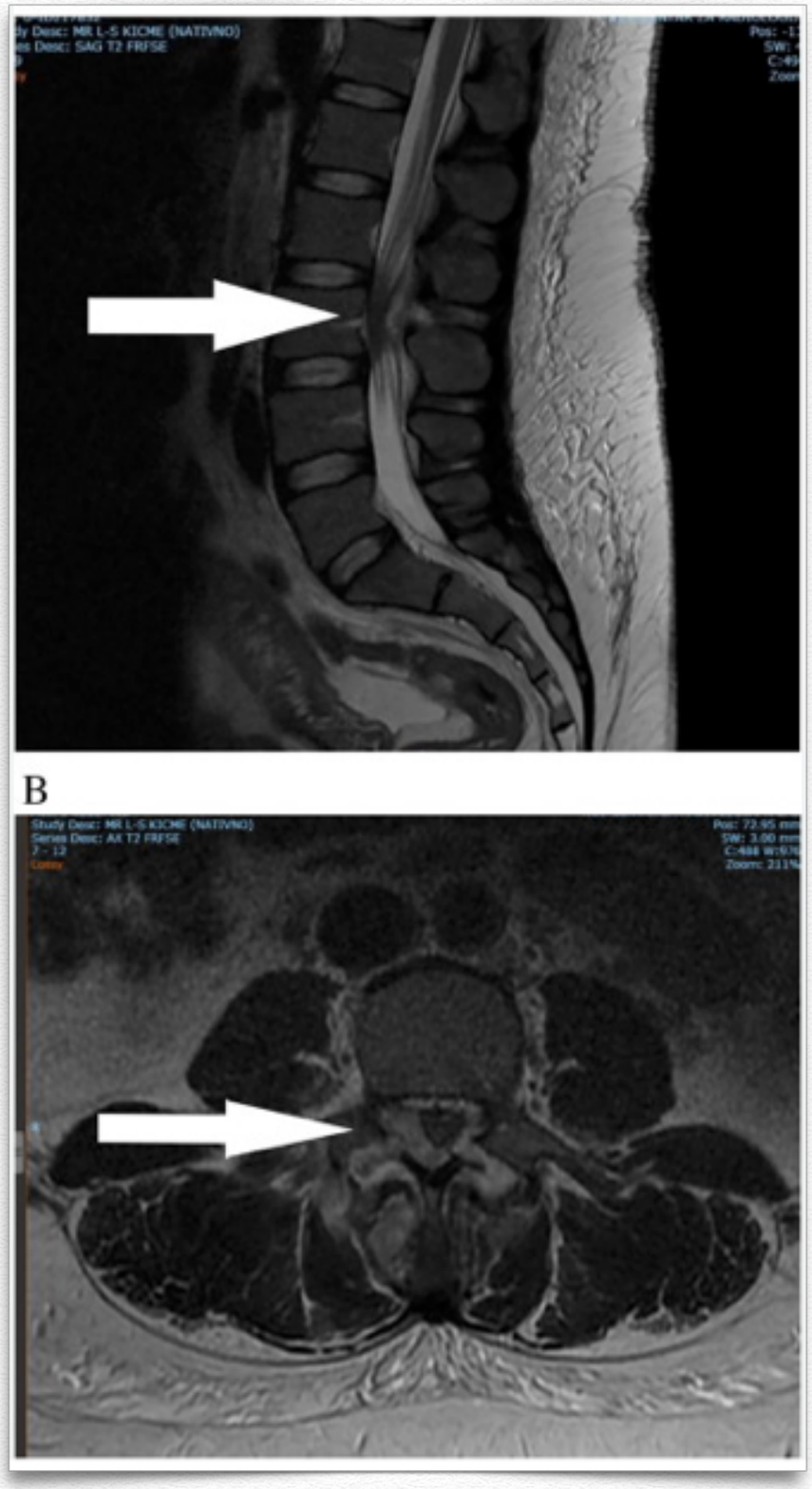
***LP*: Low Platelets**

DIC Risk = Hematoma Risk

- Labs usually improve by 72 hrs postpartum, but some values, especially LFTs, may take several weeks to recover.

- PLTs usually normalize within 24-48h postpartum

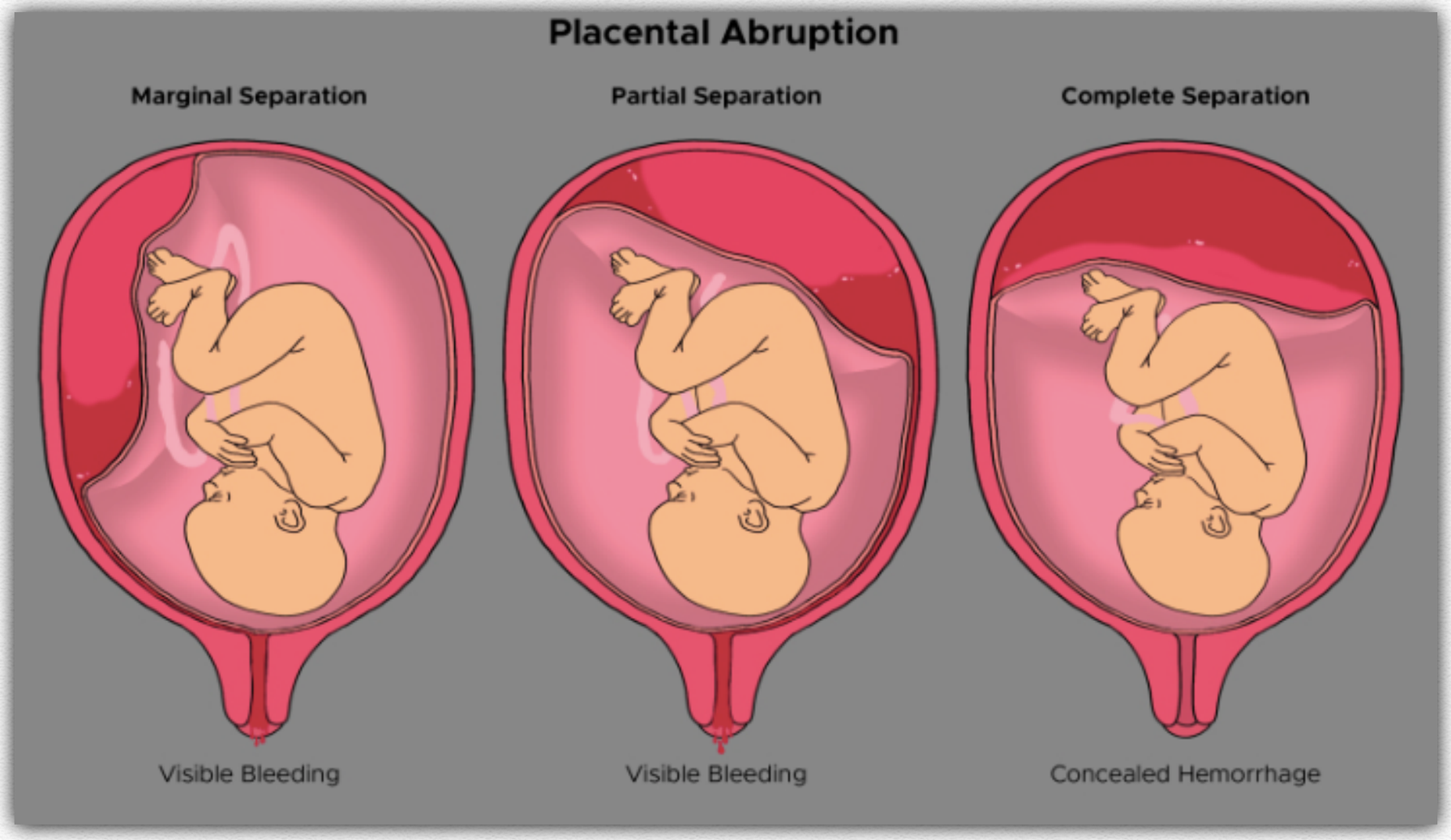
- If epidural catheter is in place waiting to be pulled – recheck CBC/PLT/Coags/fibrinogen/TEG postpartum prior to removal.



PLACENTAL ABRUPTION

PLACENTAL ABRUPTION

- The placenta detaches from the uterine wall
- Impaired fetal bloodflow/O2 delivery
- Maternal blood loss from hemorrhage into retroplacental space.
- Hemorrhage can be visible or concealed.

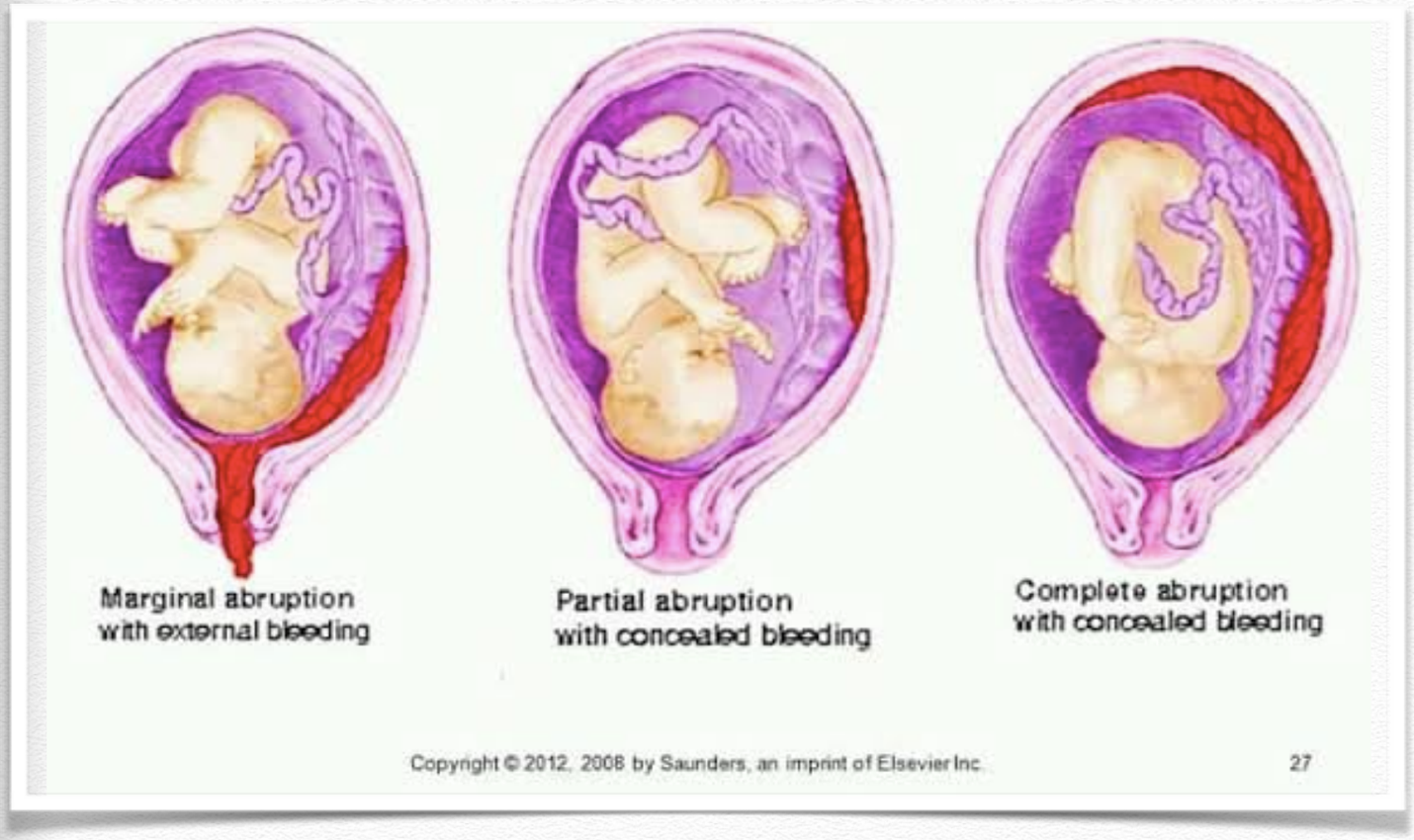


https://www.ncbi.nlm.nih.gov/books/NBK482335/figure/article-17041.image.f1/

PLACENTAL ABRUPTION

PAINFUL DARK RED **vaginal bleeding is cardinal sign.**

- Maternal instability, and usually FHR abnormal as fetal O2 delivery impaired
- EMERGENT C/S.
- HIGH D.I.C RISK. USUALLY GA if no labor epidural in place.

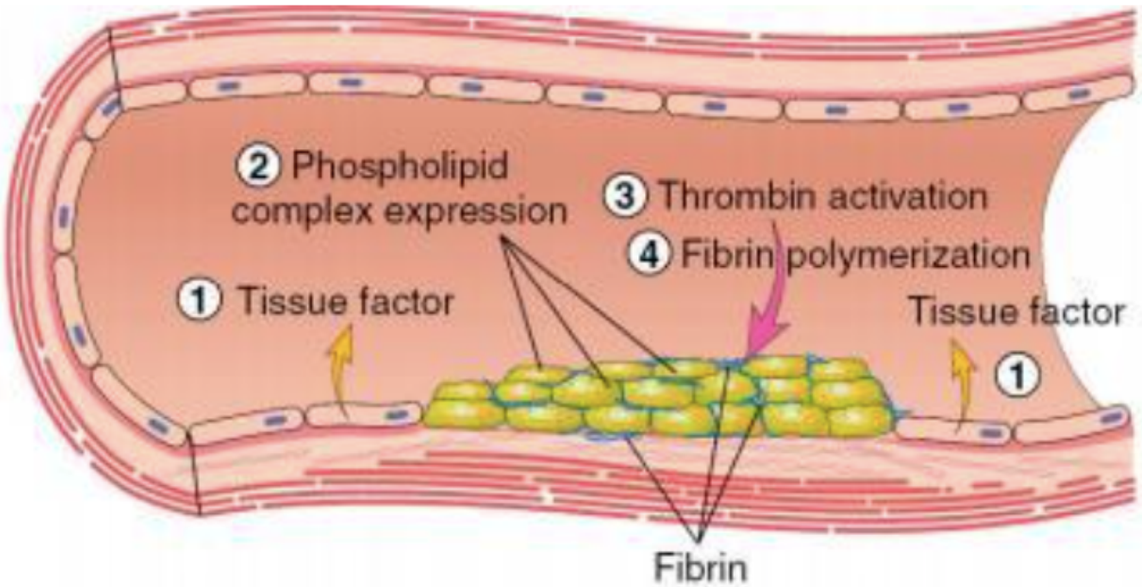


- ***CONCEALED abruption**:
- No visible bleeding as retroplacental clot continues forming.
- Pt will still report pain.
- Can be mistaken for contractions pain, except persists between cxns.
- Can have tachysystole pattern with very frequent cxns

PLACENTAL ABRUPTION

High Risk of DIC

- As placenta pulls off the uterine wall and disrupts the normal maternal-placental interface (where maternal and fetal blood normally never mix), DIC can occur if embryonic or placental/decidual Tissue Factor(thromboplastin/FIII) from retroplacental clot enters maternal circulation, causing widespread systemic activation of the clotting cascade.

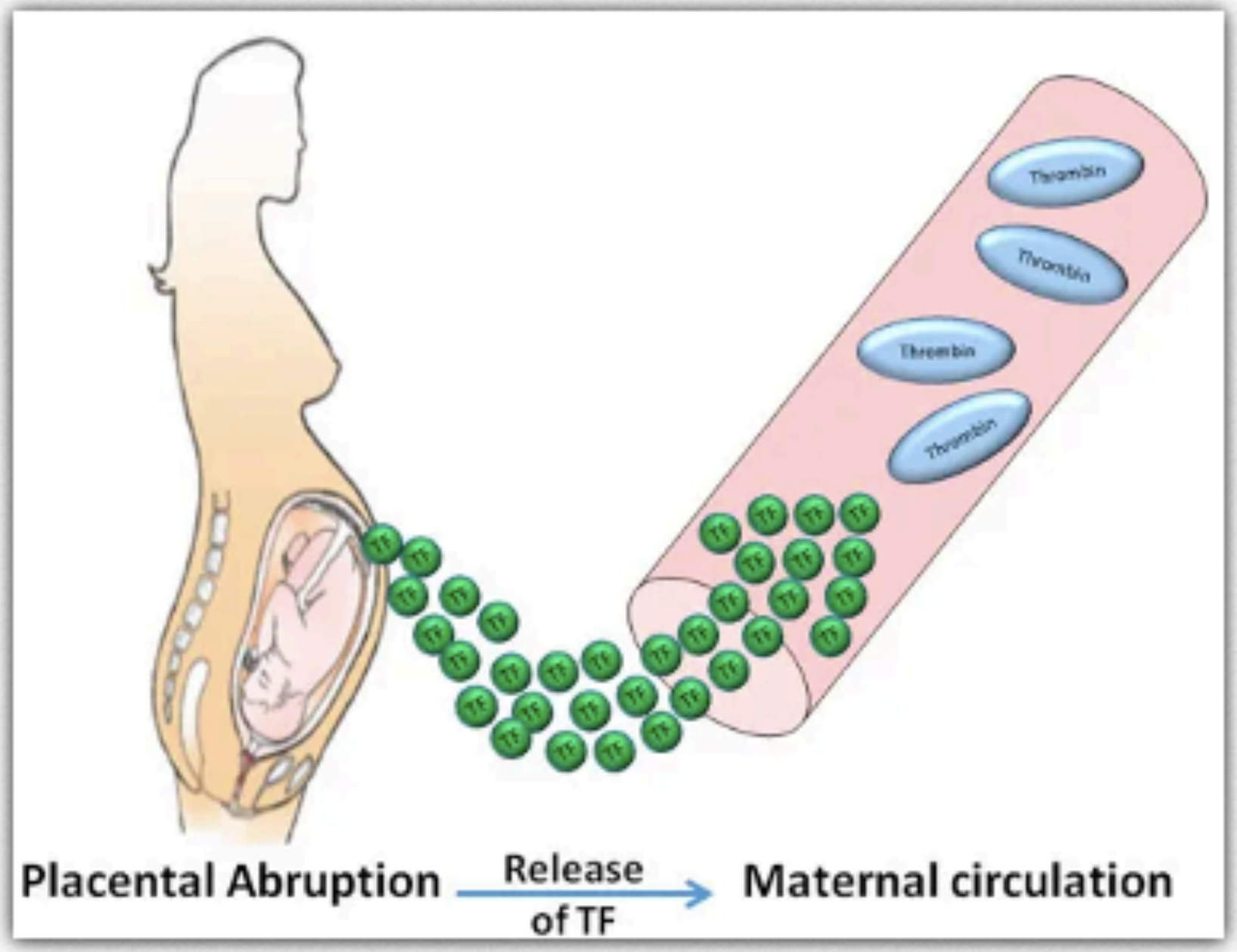


- Thromboplastin normally present on the surface of cells (macrophages, endothelial cells & monocytes) and is exposed to local, nearby circulation after local endothelial injury to initiate the clotting cascade for hemostasis.,
    - However, it does not normally float freely through circulation. Systemic bolus of TF causes clotting cascade activation through all maternal vasculature, rapidly consuming clotting factors.
        - Uncontrollable hemorrhage can occur after all clotting factors consumed.

https://www.researchgate.net/publication/320196932_Mechanism_Action_of_Platelets_and_Crucial_Blood_Coagulation_Pathways_in_Hemostasis

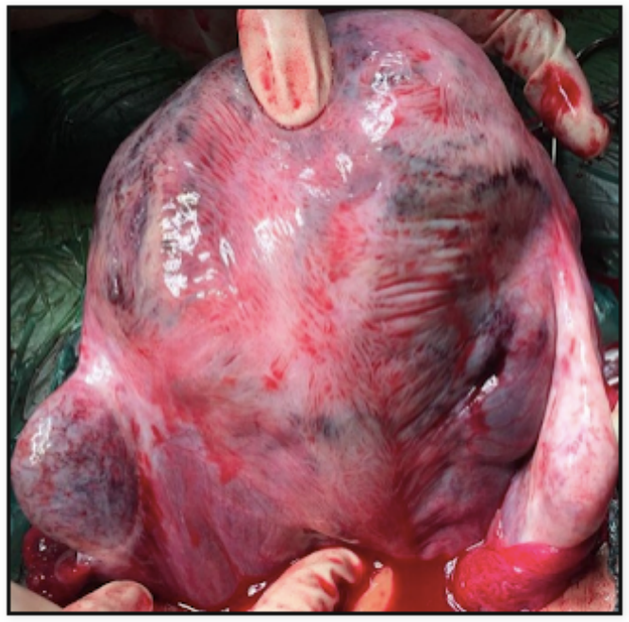
PLACENTAL ABRUPTION

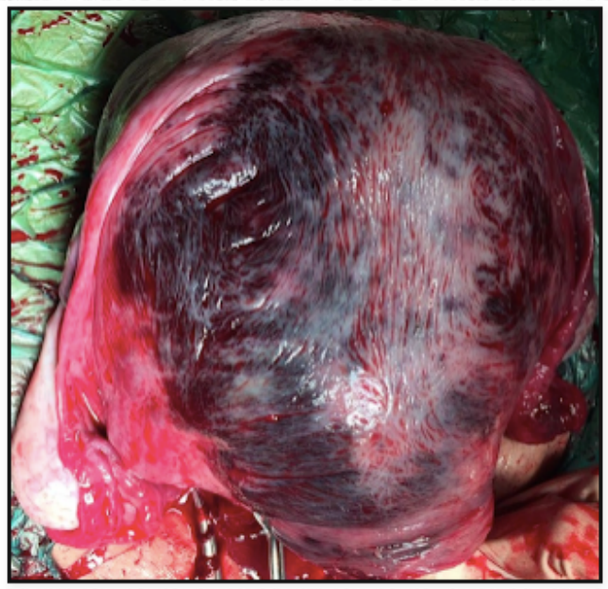
High Risk of DIC



https://obgynkey.com/disseminated-intravascular-coagulation-in-pregnancy-insights-in-pathophysiology-diagnosis-and-management/

PLACENTAL ABRUPTION





http://www.jpgo.org/2018/01/couvelaire-uterus-due-to-chronic.html

- A **couvelaire uterus** is a rare occurrence of hemorrhage into the myometrium itself, where uterine muscle swells with blood & impairs contraction. **Can be seen in concealed abruption hemorrhages.** Can cause life-threatening hemorrhage if contraction impaired. Uterus has swollen purple appearance.
- Caused by: **Abruption**, HTN, trauma, and intrauterine infection.

PLACENTAL ABRUPTION

High Risk of DIC

The risk of rapid development of DIC makes neuraxial high-risk.

- If coags/fibrinogen & PLT normal after diagnosis of abruption:
  - Spinal probably OK, and GA conversion if severe instability/long case duration etc.

- If no recent coags since diagnosis:
    - NO NEURAXIAL
    - GA for emergent C/S in stat situation where we cannot wait for coags. (Usually fetal instability in abruption—> emergent c/s)
- Expect maternal instability from concealed hemorrhage:
  - Hypovolemia from hemorrhage is relative contraindication to neuraxial sympathectomy.
    - Careful clinical judgement if difficult airway. Risk vs benefit of neuraxial.

PLACENTAL ABRUPTION

O.R. SETUP FOR ABRUPTION

All GA, Airway, and Resuscitation supplies:

- ETTs, glidescope, LMAs, induction meds
  - Etomidate/Ketamine if HD instability
- TXA, Blood, FFP, PLT in room, cryoprecipitate
- 2 large bore IVs: >/= 18g
- Fluid warmer & blood tubing
- A-line available

https://transfusionnews.com/2017/07/26/systematic-review-recommends-standard-ratios-of-blood-products-for-massive-transfusion/

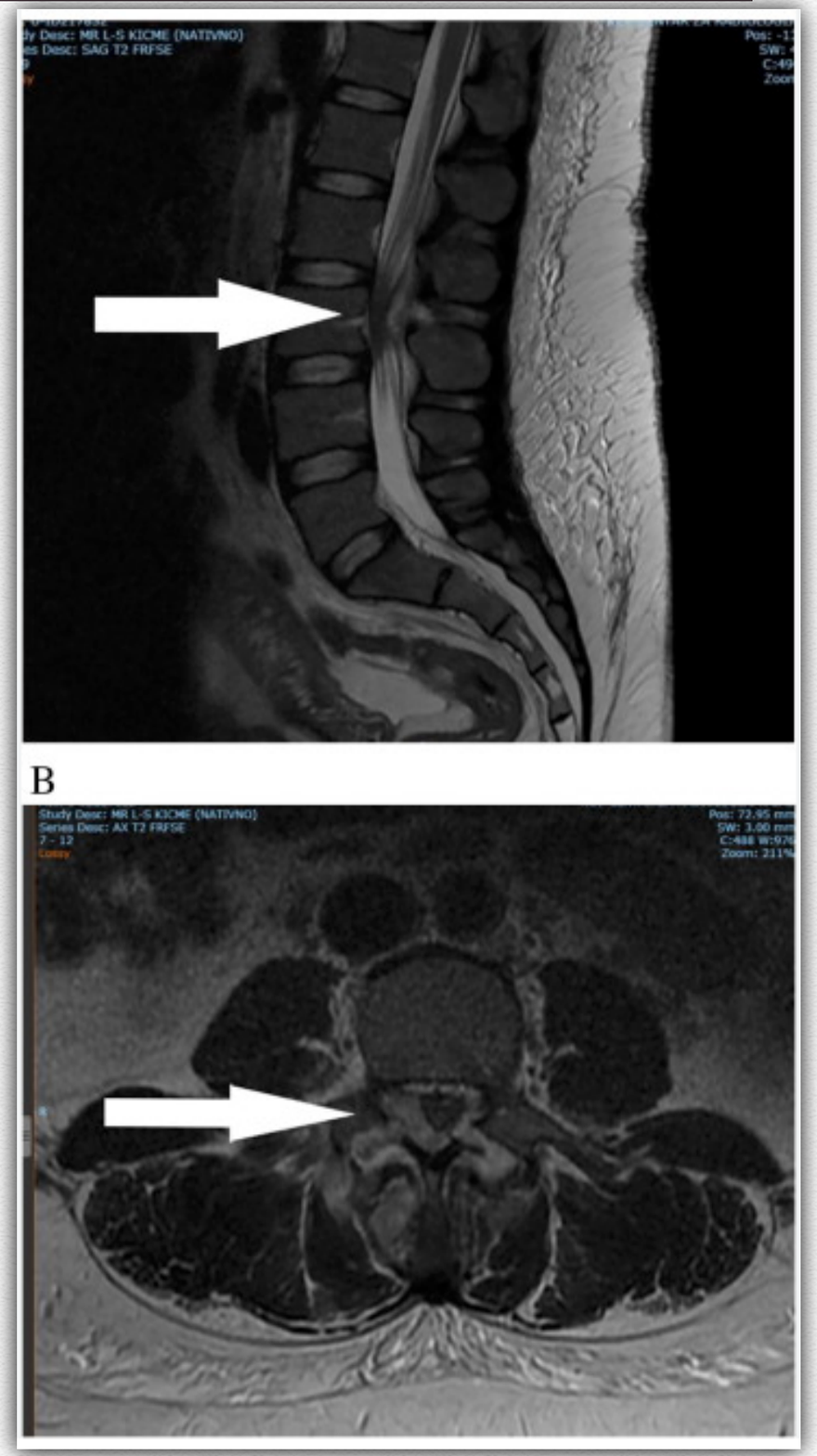
- Early and aggressive resuscitation with blood products.
- Avoid high-dose vasopressors in hypovolemic patient. —> acidosis, worsens coagulopathy
- ++ volume resuscitation with blood products.
  - RBC replaces oxygen-carrying capacity
  - FFP replaces lost coagulation factors *fibrinogen
    - cryoprecipitate
  - PLT replaces platelets for clot formation
  - Whole blood is best option, if available.

PLACENTAL ABRUPTION

DIC Risk = Hematoma Risk

- If epidural catheter in place from before abruption (for labor)
  - DO NOT REMOVE catheter until stable coags/TEG/PLT.
    - DIC risk places pt at higher risk of epidural hematoma

- OK to bolus an existing catheter for c/s
  - *watch for HD instability with sympathectomy.
- If already HD unstable –> GA for c/s as pt will not tolerate sympathectomy from neuraxial blockade.



https://www.obstetanesthesia.com/article/S0959-289X(18)30233-4/fulltext

Additional OB Anesthesia Resources

Stanford OB EMERGENCY manual

AHA Guidelines

ESC Guidelines: European Society of Cardiology

ACOG Guidelines: American College of Obstetricians and Gynecologists

SOAP: Society for Obstetric and Perinatal Anesthesia

OBG Project

OBGYN key

GLOWM.com: Global Women's Medicine

Tuohy Time: www.tuhoytime.com