Episode 59: Pulmonary Hypertension

On this episode: Dr. Jed Wolpaw and Dr. Jochen Steppan

Dr. <u>Jochen Steppan</u>, an international pulmonary hypertension expert, joins the episode to discuss pulmonary hypertension and perioperative management of patients with this disease.

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What is pulmonary hypertension?

- Disease of pulmonary vasculature
- Origin not quite known, but several etiologies
 - state of inflammation
 - metabolic causes
 - endothelial dysfunction
 - smooth muscle hypercontractility
 - impaired vasodilation
 - extracellular remodeling
- Diagnosed with right heart catheter
 - Elevated mean pulmonary arterial pressure (mPAP)
 - ≥25 mmHg at rest
 - $\circ PVR = \frac{mPAP PAOP}{CO} [PAOP = pulm artery occlusion pressure]$

Classifications based on etiology

Class	Etiology
1	Pulmonary arterial hypertension
2	Left heart disease
3	Chronic lung disease and/or hypoxemia
4	Chronic thromboembolic disease
5	Multifactorial

- Clinical diagnosis
 - Echocardiogram
 - Right ventricular systolic pressure (RVSP)
 - Assumptions
 - no issues with pulmonic valve
 - RVSP measurement relies on atrial and ventricular pressure, and according to tricuspid regurge
 - Concerns when MAP $\sim 2/3$ end systolic pressure
 - RVSP 38-40 to mean pressure of 25
- Mostly pHTN binary yes/no
 - o Mild/mod/severe not well established
 - Subclass based on pulm pressure but more classified based on symptoms (close to NYHA for HF)
 - Heart function much more predictive
 - Right heart failing → can't generate high pressures → low RVSP, but could have 'severe' pHTN

What kind of symptoms?

- Dyspnea
- Decreased exercise tolerance

• Hypoxia

Are there concerning numbers from echo report?

- E.g., pulmonary pressure is 2/3 systolic pressure
- Relative pressures more concerning for pediatrics
 - 'more than half' → slightly concerned
 - \circ 'more than 2/3' \rightarrow more than slightly concerned
- Higher peak pressure, more concerns
- Single number not adequate to describe whole state of patient

Does etiology matter when you take patient into OR?

- Yes known underlying cause → manage and optimize better
- Gives some idea of how they would react in OR
- Class 1 worst because prognosis, and much more prone to pHTN crisis
 - sudden increase → worse RV strain and function
- Good to know underlying cause

Preop patient with pHTN for surgery?

- H&P
- Exercise tolerance
- 6 min walk test
- Echo mean arterial pressure, also underlying problems like left heart or valvular
- RV function compensate on higher pulm pressure?
- Right heart catheter / vasodilatory test for those on vasodilators

What if patient is on Flolan (epopostenol) pump?

- Need to be continued throughout surgery with dedicated line
- Interruption of line, such as inadvertently, can precipitate crisis, especially given shorter half-life

What is pulmonary hypertension crisis?

- Sever exacerbation, such as if Flolan runs out or is interrupted
- Sudden † in pulm pressure with associated right heart strain
 - Right side afterload ↑ → stroke volume ↓ → Left preload ↓ → ↓ BP
 → ↓ coronary perfusion pressure.
 - \circ When both ventricles strained \rightarrow ischemia \rightarrow downward spiral

Early warning signs?

- Depends on monitors
 - PA catheter ↑ PA pressure right away
 - Echo RV function struggle
 - \circ CVP line \uparrow CVP
 - ∘ None ↓ BP
 - More difficult to figure out reason for sudden hypotension

Medications encountered for pulmonary hypertension?

- Less specific
 - Oxygen
 - o CCB
 - Diuretics
 - Anticoagulants
- Severe
 - Flolan pump
- Sildenafil
 - o PO usually before IV, later possibly combined
- If on PO, and long case, may crush and put down OG to not miss dose If a patient is on sildenafil/prostacyclin or both, consider NO?
 - Keep them on what they're on.
 - Concerns that IV meds may lead to coagulopathy, so may switch to inhaled
 - Inhaled prostacyclin
 - Deliver meds to well-ventilated alveoli → vasodilate proximal vessels
 → match ventilation/perfusion → ↑ oxygenation
 - ∘ ↓ systemic side effects, short half-life → scavenged by hemoglobin

If on IV prostacyclin, concerned about vasodilatory systemic effects or hypotensive on induction?

- Depends on volume status
- Endothelial receptor antagonists, like bosentan
 - o Intranasal, but fairly rare

Monitors:

• Swan or PA catheter for real-time monitoring

How do you decide?

• Only minority can take PA catheter in. Intuitively it makes sense for pressures, but hasn't been shown to have survival benefits in large studies. Depends on comfort and being able to interpret numbers quickly.

- Patients with pHTN are at highest risk for PA rupture during PA catheter, so don't wedge, or put as little strain on pulm system as possible.
- PA catheter more about physiology and severity of surgery
 - Drastic example:
 - Dyspneic at rest with RV dysfunction. PA close to systemic range despite maximal therapy, and undergoing major surgery like abdominal/liver resection.

If you can only pick one?

- Arterial line
 - Usually put in awake, with lidocaine + bicarbonate [less sting]
 - Lots of guidance especially during induction phase
- CVP line
 - No survival benefits
 - No direct correlation of CVP and true fluid responsiveness, but give good idea of overall marker and volume status
 - Not many other cases you see CVP spike, so high pretest probability for right ventricular function
 - Option to administer meds, like inotropes, at higher dose than peripherally
- TEE for right heart
 - Not exactly PA catheter, but almost everything except mixed venous side
 - Can still get direct visualization over numbers

Induction

Anything prophylactically? Eg pressor drip

- Depends on severity of patient
- Severe HF + compromised RV + pHTN, then yes low dopamine drip (3-5 mic/kg/min)
 - Fentanyl/midazolam well tolerated
- Low dose Propofol judicially counteract vasodilating effects

Personal preference?

- Ketamine (~1/2 mg/kg)
- Concern of \uparrow catecholamine $\rightarrow \uparrow$ PVR
 - \circ Catechol \uparrow also \uparrow RV, can overcome \uparrow PVR
 - Maintain ventilation and not get hypercarbic/hypoxic → no ↑ PVR
- Some midazolam, opioid (fentanyl), ketamine
- Deeper → vapor
 - o Sevoflurane just in case not deep enough
 - Desflurane has theoretical concern of too fast → reflex tachycardia → contractility; also pungent

If using Propofol, counteract vasodilation?

- Usually with phenylephrine but with these patients, use vasopressin instead
- Start vasopressin drip at start, or give small boluses

Intraoperative management

- Actively changing pulm vascular resistance
- Oxygenation
 - 100% O2 to ↓ PVR
- Hyperventilate to ↓ PCO2
- Aggressive treatment of acidosis
 - Hypothermia † PVR so keep warm
- Mean pulm airway pressures
 - ∘ ↓PEEP or I:E
 - Spontaneous ventilation?
 - ∘ More pressure on vasculature → afterload for right ventricle
 - Overdistension of airway → resistance. Same with too little. U-shaped curve with FRC at lowest resistance
 - Spontaneous breathing ensures adequate ventilation, prevent hypercarbia
- Medication
 - o iNO as rescue
 - Avoid ones that actively \(\tau \) pulm vascular resistance, like phenylephrine

Patient becomes hypotensive in OR

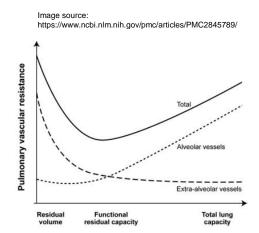
- Common causes
 - o Bleeding, too much anesthetic

What are you thinking differently?

- Avoid phenylephrine which is usually most common
- Avoid knee jerk response
 - Big bolus of cold fluids → phenylephrine --- bad!
 - \circ Bolus $\to \uparrow$ preload of right heart not well tolerated. Need to stay as close to its geometric shape
 - Cold $\rightarrow \uparrow PVR$
 - Phenylephrine α -agonist $\rightarrow \uparrow$ PVR

What medications would you use?

- Maintain coronary perfusion of RV
- Vasopressin
 - V1 receptor not in brain / pulm circulation



- Low dose like 0.2 or 0.4 units bolus ensures coronary perfusion and gets RV out of ischemic phase, then actively lower pulm resistance
- How to prepare vasopressin?
 - 0.2 per CC
 - Vial come in 20 u -- put in 100 cc bag \rightarrow 0.2 per CC
- How to prepare as IV?
 - 0.2 units/min
 - Bolus more for pinch
- Milrinone
 - Doesn't do much about pulmonary vasculature, although supporting RV
- Combine?
 - Yes especially if vasopressin drip and add milrinone on top of it
 - Not well tolerated intraoperatively become too hypotensive
- Dopamine or epinephrine
 - Low dose
 - No ideal drug but offers contractile support
- Several IVs?
 - One for specific reason
 - vasopressin for afterload
 - low dose dopamine for contractility
 - Rather than just epinephrine and one dial

Heart range goals?

- Somewhere between tachycardic and bradycardic
 - unlike aortic stenosis (brady)
- Don't want to ↑ oxygen demand or ↓ cardiac output

Ideal volume status?

- Euvolemic
- Take hypovolemic over hypervolemic
- Major goal is volume status

Postop

- Depends. Low threshold to get to ICU
- If minor operation / not much bleeding, possibly send home
- IV meds, debilitated, consider ICU
 - Maybe not surgical because not surgical issues
 - Usually medical because expertise
- If major abdominal / vascular surgery, maybe cardiac surgical ICU
- Things worried about:
 - ∘ Not well controlled pain \rightarrow ↑ catecholamines \rightarrow ↑ PVR

- o Hypoxia
- Volume status / fluid shifts

Comments? What do you do differently for pulmonary hypertension?

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Notes by Brian Park