Perioperative Considerations for Infants with Single Ventricle Physiology Undergoing Laparoscopic Surgical Procedures

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**Objectives:**
1) Review the anatomy and consequential physiology of staged single ventricle repairs.
2) Discuss the additional peri-operative concerns and the pre-operative preparation for this patient population.
3) Review the anesthetic considerations pertinent to laparoscopic procedures.
4) Understand the utility of invasive arterial pressure and cerebral oximetric monitoring during laparoscopic surgical procedures in this patient population.
5) Develop a peri-operative anesthetic plan for a child with palliated single ventricle physiology undergoing a laparoscopic procedure.

**Background Questions:**
What are some common examples of single ventricle anatomy that require palliation in the early neonatal period? What is their significance on neonatal physiology? Are there other diseases or syndromes that are commonly associated with these types of congenital heart disease? If so, what are they?

What is the initial palliation for 1) hypoplastic left heart syndrome (HLHS), 2) tricuspid atresia, and 3) unbalanced complete atrioventricular septal defect? What is the Norwood procedure? What are a Blalock-Taussig shunt (BTS) and a RV-PA (Sano) conduit? What is the difference between a BTS and a modified BTS? What are the physiologic concerns for a patient with a BTS or a RV-PA conduit?

**Case:**
8-week old with a history of hypoplastic left heart syndrome (HLHS) s/p emergent balloon atrial septostomy and stage I Norwood procedure with a 3.5 mm right modified BTS is scheduled for laparoscopic Nissen fundoplication and gastrostomy tube placement.

**Pre-operative Questions:**
What pre-operative laboratory results and imaging data do you want to know and why? What medications do you expect this patient to be receiving preoperatively? Do any of these medications impact your anesthetic management or evoke concerns? What is this patient’s expected systemic $O_2$ saturation and why? Are there expected pertinent physical exam findings? Do you have any other pre-operative concerns or items that need to be addressed? Is a
laparoscopic technique contraindicated in this patient? What is the significance of a prolonged NPO time in this type of patient that lacks IV access?

**Intra-operative Questions:**
How are you going to induce and maintain anesthesia in this patient (i.e. sedation, analgesia, neuromuscular blockade, Fio2, etc.)? What type of vascular access and monitoring is needed for this procedure? Do you need invasive arterial monitoring? If so, where should you place your arterial catheter? Where will you place your pulse oximetry probe? Does this patient need to receive antibiotics for SBE prophylaxis?

What is cerebral oximetry (NIRS)? What physiologic parameters affect NIRS? Besides NIRS, what other physiologic parameters are you going to follow? What is the importance of the diastolic blood pressure in this patient? Does this apply to the same patient with a RV-PA conduit instead of a BTS? Which components of blood gas analysis or other lab studies are valuable for the management of this patient? Why? What are your target values?

**Scenario:**
What physiologic changes do you expect with pneumoperitoneum? How will these affect the variables you are monitoring? How might you respond to such changes?

**Discussion:**
Potential respiratory consequences of CO2 pneumoperitoneum include increased intra-abdominal pressure which may impair diaphragmatic motion, decreased functional residual capacity and pulmonary compliance, increased airway resistance, and decreased tidal volume and minute ventilation. These changes may lead to intrapulmonary shunting, an increased alveolar-to-arterial oxygen gradient and possible hypoxemia. Hypercarbia may result from the absorption of CO2 across the peritoneum and from induced alterations in respiratory mechanics. Keep in mind that patient positioning and intra-abdominal insufflation may lead to changes in pulmonary and cardiovascular mechanics, as well as bronchospasm, increased airway secretions, atelectasis, pneumothorax and even CO2 embolism. In single ventricle physiology, the cardiac output is divided between systemic and pulmonary circulations, depending in a large extent on the relative resistances of the two circuits (SVR vs. PVR). Neonates and infants are more susceptible to large swings in PVR than older children. Anesthetic techniques, surgical techniques and choice of ventilation mode all affect the ratio of SVR:PVR, in a summary fashion.

**Scenario (continued):**
Shortly after insufflation and pneumoperitoneum (12 mmHg CO2), the NIRS increases with no apparent change in any other parameter. Why might this occur in this patient? Is it reassuring? What else might help decide if this is good or bad?

About 15 minutes after pneumoperitoneum is initiated there is a gradual, but obvious decrease in the O2 saturation and EtCO2. What is your differential diagnosis and how will you pinpoint the problem? What are you going to do if you no longer hear a BTS murmur?
Post-operative care
Should this patient be taken to the PACU or the ICU for postoperative care? Do you want to extubate this patient in the OR? If not, how long might you anticipate the need for ventilator support?

Additional Discussion Items
More single ventricle pathologies than HLHS, TA and unbalanced CAVSD
HLHS s/p Norwood frequently have feeding difficulty and GI pathology
Goals: Maintain ventricular function and balance systemic versus pulmonary blood flow
How to monitor systemic perfusion/oxygen supply?
How to monitor pulmonary perfusion?
NIRS, lactate, SvO2

Pre-operative Knowledge Topics:
Shunt materials
Shunt murmur
Intracardiac mixing
Medications (ASA, afterload-reducers)
PBF and pulmonary vasculature
Pulmonary and systemic vascular resistance and Qp:Qs
NPO
Anticoagulation
Arterial line placement
Vasoactive drugs
Diastolic BP and coronary anatomy in HLHS
Blood products and target hematocrit (40-45)
  15 mL/kg pRBC (Leukoreduced and irradiated?)
ABG, base deficit and lactate
  How often to sample
  Treating worsening base deficit
Target O2 sat and F\textsubscript{2}O\textsubscript{2}
NIRS
  What is cerebral oximetry?

References:


