

Machine Problems (Yikes!) in a patient with a BT Shunt

Moderators: Kristin L. Richards, M.D., Laura Hastings, M.D.

Institution: Children's Hospital Los Angeles/University of Southern California

Objectives:

1. Develop an approach to intraoperative hypercarbia
2. Describe the effects of hypercarbia on a BT shunt
3. Recognize the anesthesia machine malfunctions that would result in hypercarbia
4. Review the criteria for a diagnosis of MH
5. Develop an approach on how to approach the parents after an intraoperative complication

Case History:

A 4 month old patient, with a BT Shunt presents for pre-Glenn cardiac catheterization. No recent URI symptoms, per the parents. Baseline oxygen saturation is 85%. No prior anesthetic records are available.

Questions:

What is a BT Shunt? What are the indications for placement of a BT Shunt? What are the physiologic goals of management of patients with BT Shunts? What are the concerns regarding hypercarbia in relation to the shunt? What is a "normal" oxygen saturation for a BT Shunt patient? What should be the next step in the management of this patient?

Case history (continued)

This 4 month old female, born at 38 weeks gestation, birth weight 2780 grams, to a G2P2 mother who had prenatal care. The initial APGAR scores were 9/9 and the patient was transferred to the newborn care unit. She was soon noted to be cyanotic, oxygen saturation level 76%. She was then intubated and found to have a hypoplastic RV, near tricuspid atresia and pulmonary atresia. She was initially maintained on PGE until undergoing a BT Shunt (3.5), ligation of PDA, and atrial septectomy procedure at 4 days of life.

She has exhibited poor weight gain and her current weight is 4.37 kg.

The parents are Mandarin Chinese speaking only and an interpreter is present. There is no family history of anesthetic problems. On physical exam, sleeping infant, slightly cyanotic, sucking on a pacifier. No distress. No syndromic features noted. Chest is symmetrical and without wheezes or rales. Shunt murmur is present. Capillary refill < 2 seconds. Temperature is 36.5, HR: 135, RR 30, BP 85/54, SpO₂ 85% on room air.

Current Medications: Aspirin, Enalapril, Lasix, Prevacid

Current Labs: Hematocrit 36, Platelets 220, K⁺ 3.1, HCO₃ 22

Echocardiogram (1 mos prior) hypoplastic RV, severe tricuspid valve stenosis, pulmonary atresia, BTS not well visualized. Normal cardiac function.

EKG: normal sinus rhythm, left axis deviation, left ventricular hypertrophy with strain pattern, right atrial enlargement

What are the possible anesthetic complications? What instructions did you give for the home medications and NPO? Is the aspirin important? What do you think of the shunt size and the patients' weight? Should this patient have a type and cross or a type and screen sent pre-procedure? What is your anesthetic plan? If the HCO_3^- had been 30 pre-operatively would you delay the case? What would cause an elevated bicarbonate level in this patient? Is it acceptable for this to be the 3rd case of the day?

Intraoperative Care

She undergoes a mask induction with sevoflurane and intubation without difficulty. After confirmation of the endotracheal tube, the end-tidal CO_2 is in the 70's with tidal volumes 10 cc/kg and RR: 30. HR: unchanged from baseline.

Questions:

What is your first step in management of this situation? What else would you like to know? What is your differential diagnosis? What would your concerns be if the end-tidal CO_2 was low? Could this be secondary to endotracheal tube position?

Intraoperative Care (continued)

The ETT is confirmed in position by bilateral breath sounds as well as by fluoroscopy. The ETT is suctioned and the patient is hand-ventilated. An arterial blood gas is drawn and the CO_2 correlates with the EtCO_2 . The CO_2 absorber is changed, no improvement. The patient is placed on an ambu-bag and the hypercarbia improves. Once placed back on the ventilator the end-tidal returns to 70's. The temperature is 37.6

Questions:

What machine issues would result in hypercarbia? Inspiratory or expiratory valve malfunction? Would you consider changing to a new machine or an ICU ventilator? What is the differential diagnosis for intraoperative hypercarbia? What if the end tidal CO_2 and PaCO_2 didn't correlate, does that change the management or differential? What is the typical medical management strategy for BT Shunt patients? What is the goal of a pre-Glenn cardiac catheterization?

What if her oxygen saturation is now 65%

What other evaluation would be necessary? Are you concerned about a hypoxic gas mixture? What features help to prevent delivery of a hypoxic gas mixture? Would an echocardiogram be of any value at this time? Should this case continue? What do you tell the parents?

Intraoperative care (continued)

After the patient desaturates to 65%, she is hand ventilated with an ambu bag until her oxygen saturations improve. At this time an arterial line is inserted, her blood pressure remains stable and subsequent arterial blood gases are drawn with the patient back on the ventilator. The decision is then made to place the patient on an ICU ventilator. Her hypercarbia then resolves and her arterial blood gas normalizes. She is extubated to room air upon completion of the procedure.

Postoperative Care

The cardiac catheterization demonstrated that there is good flow through the BTS, branch PA's fill well and are confluent. Normal R SVC with an innominate vein and no Left SVC. RV is small chamber with sinusoids that fill branches of both the right and left coronary arteries.

What is the significance of the sinusoids? Would knowing any of this information changed your management of this patient?

The patient is monitored for six hours in the post anesthesia recovery unit. She remains hemodynamically stable, tolerates room air without any issue and appears comfortable. Would you discuss the intra-operative issues with the parents? Would you replace the anesthesia machine in the cardiac catheterization lab?

Discussion:

On November 29, 1944 Drs. Blalock and Taussig directly anastomosed the subclavian artery to the pulmonary artery in a cyanotic child to increase pulmonary blood flow. The initial child treated with this had tetralogy of fallot (4). Today, this method is seldom used and connection between the two arteries is created with a gore-tex® shunt and is placed in children not only with TOF but also as part of stage I palliation for single ventricle lesions to provide consistent pulmonary blood flow.

Since it is an arterial shunt, it is useful as the pulmonary vascular resistance falls over the first several months of life. The resistance determines the flow across the shunt. The mean arterial blood pressure, the diameter of the shunt, and the pulmonary vascular resistance are directly related to the resistance, while the length of the shunt is inversely related to the resistance.

Cardiac catheterization remains the standard of care to evaluate congenital heart patients between staged procedures, most commonly single ventricle variants. The catheterization measures pressures and delineates anatomy to determine the suitability for the next surgery and optimizes the approach. General anesthesia is generally used due to the young age of the patients.

Generally, single ventricle patients that have BT shunts come to the catheterization lab at 4-6 months of age for their pre-Glenn catheterization. Since they have grown, their shunts tend to be relatively small, increasing the risk of obstructing the shunt. If there is limited blood flow, with cyanosis, the blood viscosity would be higher due to a compensatory increase in hematocrit.

Although, an intra-operative machine complication is problematic during any anesthetic it can be especially detrimental to a patient who is dependent on an arterial shunt for pulmonary blood flow and is significantly affected by hypoxia and hypercarbia. Additionally, hypercarbia can result in hypertension, tachycardia, and/or arrhythmias.

A variety of machine malfunctions can occur. For example, inspiratory or expiratory valve issues, delivery of a hypoxic gas mixture, ventilator failure, scavenging system issue, or electrical power failure are all possibilities in addition to many others.

The anesthetic circle system depends on the integrity of both the inspiratory and expiratory unidirectional valves. These valves help to ensure that the inspiratory gas will not be contaminated by the expired CO₂. (2)

As discussed in the 2008 ASA recommendations for machine checkout, an inspiratory valve malfunction may not be indicated by an elevation of the inspired CO₂ baseline. If the delivered tidal volume is greater than the volume of gas in the inspiratory limb containing CO₂, rebreathing will appear on the capnogram as a gradual, not sharp, downstroke. An expiratory valve malfunction is evident by an elevated CO₂ baseline, as there is a large volume of exhaled gas containing CO₂ that can return to the patient. (1)

Additionally, the circle system relies on the absorbent to remove CO₂ from the rebreathed gas. Exhausted absorbent, as indicated by color change, should be replaced. It is possible for absorbent material to lose the ability to absorb CO₂ yet the characteristic color change may be not be present. (1)

Possible causes of intraoperative hypercarbia include increased production, decreased elimination, or iatrogenic causes. Malignant hyperthermia, pheochromocytoma, thyroid storm, fever, total parenteral nutrition, tourniquet release, or any hypermetabolic state is reason to experience increased production of carbon dioxide. Iatrogenic causes include the administration of bicarbonate or CO₂, and depletion of the CO₂ absorbent granules. Decreased elimination could be secondary to inadequate ventilation, partial airway obstruction, or altered respiratory mechanics such as decreased compliance.

If the FiCO₂ raised, check the valves to ensure that the expiratory valve is not stuck open, confirm that the soda lime is not exhausted, and ensure adequate fresh gas flow.

In conclusion, the management of intraoperative hypercarbia includes ensuring adequate oxygenation, ventilation, and sufficient inspiratory flows, confirming FiO₂, switching to hand ventilation, checking an arterial blood gas to provide confirmation, considering secondary causes requiring specific treatment, considering changing machines or ventilators, and treating the complications of hypercapnia which can cause significant hemodynamic instability especially in a patient with a BT Shunt.

References:

1. ASA Sub-Committee on Equipment and Facilities. Recommendations for pre-anesthesia checkout procedures (2008). Available at www.asahq.org/clinical/FINALCheckoutDesignguidelines02-08-2008.pdf.
2. Han SR, Ho CS, Jin CH, Liu CC. Unexpected Intraoperative Hypercapnia Due to Undetected Expiratory Valve Dysfunction--A Case Report. *Acta Anaesthesiol Sin*. 2003 Dec; 41(4): 215-8
3. Ehrenwerth, Eisenkraft, Berry. *Anesthesia Equipment: Principles and Applications, 2nd Edition*. 01 Apr 2013. Saunders. Philadelphia, PA.
4. Blalock A, Taussig HB. The surgical treatment of malformations of the heart in which there is pulmonary stenosis or pulmonary atresia. *JAMA*. 1945; 128:189-192
5. de Leval MR, McKay R, Jones M, et al. Modified Blalock-Taussig shunt. Use of subclavian artery orifice as flow regulator in prosthetic systemic-pulmonary artery shunts. *J Thorac Cardiovasc Surg*. Jan 1981; 81(1): 112-9.
6. Fenton KN, Siewers RD, Rebovich B, Pigula FA. Interim mortality in infants with systemic-to-pulmonary artery shunts. *Ann Thorac Surg*. Jul 2003; 76(1): 152-6; discussion 156-7
7. Alkhulaifi AM, Lacour-Gayet F, Serraf A, et al. Systemic pulmonary shunts in neonates: early clinical outcome and choice of surgical approach. *Ann Thorac Surg*. May 2000; 69(5): 1499-50