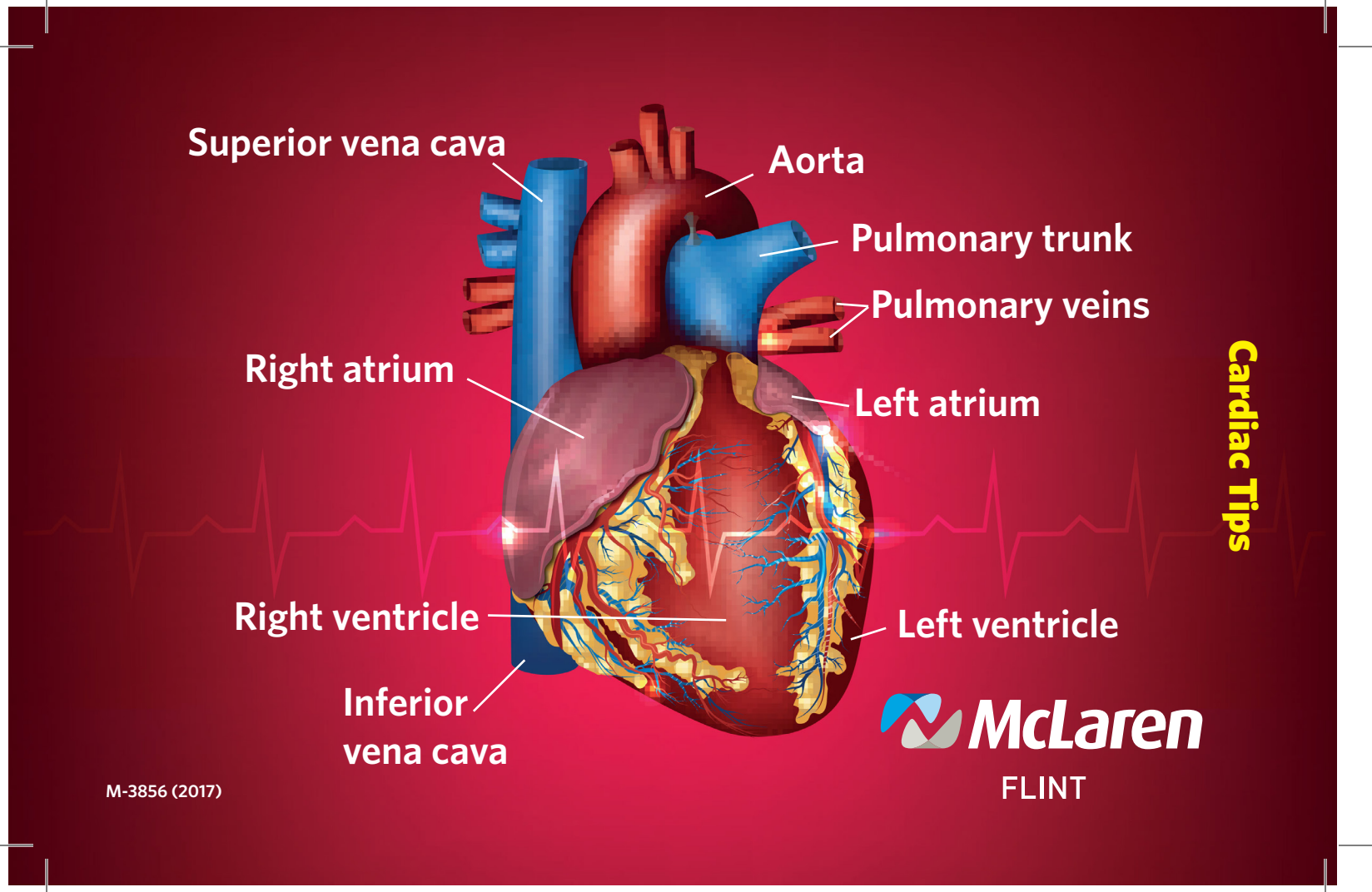


Cardiac Tips

McLaren FLINT

M-3856 (2017)

Hemodynamic Parameters:

Central venous pressure (CVP) -2-6mmHg

Right ventricular pressure (RVP) – 15-30/2-8mmHg

Pulmonary artery pressure (PAP) – 15-30\8-15 mmHg

Pulmonary artery wedge pressure (PAWP) 5-12 mmHg

Stroke volume (SV) – 60-100 ml\beat

Cardiac Output (CO= SV x heart rate) – 4-8 L\min

Cardiac index (CI) – 2.5- 4 L\min\m2

Stroke volume index (SVI) – 33-47 ml\beat\m2

Pulmonary vascular resistance (PVR) – <250 dynes\sec\cm5

Systemic vascular resistance (SVR) – 800-1200dynes\sec\cm5

Right ventricular stroke work (RVSW) -10-15 g-m\beat

Left ventricular stroke work (LVSW) – 60-80 g-m\beat

Mixed venous oxygen saturation (SVO2) – 60-80%

McLAREN FLINT TITRATABLE DRIP PARAMETERS

Alteplase:

Acute STEMI: 15 mg IV Bolus then 0.75 mg/kg (up to 50 mg) over the next 30 minutes, then 0.5 mg/kg (up to 35 mg) over the next 60 minutes

Stroke: 0.09 mg/kg IV loading dose (up to 9 mg), then 0.81 mg/kg (up to 81 mg) IV over 1 hour (MUST start within 3 hours of onset)

Pulmonary Embolism: 100 mg IV over 2 hours OR 10 mg IV bolus, then 90 mg over 2 hours

Max, including bolus: 90 mg for Stroke, 100 mg for PE, 85 mg Acute STEMI

Amiodarone (Cordarone)

Bolus: 150 mg/100 ml D5W

1 mg/min x 6 hours: 33.3 ml/hour = 1 mg/min

0.5 mg/min x 18 hours: 10 ml/hour = 0.5 mg/min

Measure QT interval every 8 hours; Preferable to give via central line; use in-line filter; Many drug-drug interactions (check Lexicom) **Max daily dose:** 2.1 grams

Aminocaproic Acid (Amicar) 4 to 5 gram Loading dose during first hour, followed by 1 g/hr. for 8 hours or until bleeding controlled. **Max daily dose:** 30 grams.

For Subarachnoid Hemorrhage, may continue to 72 hours; discontinue infusion 4 hours prior to angiography or 2 hours prior to endovascular ablation.

Cisatricurium: 0.2 mg/kg IV bolus, then 1-2 mcg/kg/min IV continuous infusion, titrate to patient condition with Train of Four 2-3 of 4. **Max rate:** 2 mcg/kg/min

Clevidipine (Cleviprex): 1 mg/hr. continuous infusion. Double dose every 90 seconds until approaching SBP goal of < 160, then increase by 1 mg/hr. every 5 minutes. **Max rate:** 21 mg/hr.

DexMEDetomidine (Precedex): 0.2 mcg/kg/hr. continuous infusion. Increase by 0.1 mcg/kg/hr. every 15 minutes until desired response of RASS -1 to 0. **Max rate:** 1.5 mcg/kg/hr.

Diltiazem (Cardizem) bolus 0.25 mg/kg actual body weight over 2 minutes. **Max 25 mg bolus.**

Diltiazem (Cardizem) 5 mg/hr. continuous infusion. Increase rate every 5 mg/hr. every 15 minutes to desired heart rate; **Max rate:** 15 mg/hr. Hold for SBP < 90.

Desired Heart Rate: _____

DOBUTamine 2.5 mcg/kg/min continuous infusion. Increase by 2.5 mcg/kg/min every 15 minutes until desired response is achieved: CI ≥ 2, HR < 110, MAP >65, or SBP > 90. **Max:** 20 mcg/kg/min.

DOPamine 5 mcg/kg/min continuous infusion. Increase by 2.5 mcg/kg/min every 15 minutes until desired response is achieved: MAP ≥ 65 or SBP > 90. **Max rate:** 20 mcg/kg/min

EPINEPHrine 0.05 mcg/kg/min continuous infusion. Increase by 0.05 mcg/kg/min every 10 minutes until MAP ≥ 65 or SBP > 90 bpm. **Max dose not defined.**

Esmolol (Brevibloc) 50 mcg/kg/min continuous infusion. Increase by 25 mcg/kg/min every 5 minutes until HR < 100. Hold if SBP < 90. **Max rate** 200 mcg/kg/min.

FentaNYL 1 mcg/kg/hr. continuous infusion. Increase by 0.5 mcg/kg/hr. every 15 minutes until desired pain score 0-3. OR RASS -1 to 0. **Contact physician for further orders if unable to achieve goal or if a rate of 300 mcg/hr. is achieved.**

HYDROmorphone 0.5 mg/hr. continuous infusion. Increase rate by 0.25 mg/hr. every 30 minutes until desired pain score 0-3. **Contact physician for further orders if unable to achieve goal or if a rate of 5 mg/hr. is achieved.**

Labetalol 2 mg/minute continuous infusion. Increase by 1 mg/min every 10 minutes until SBP < 160 or HR < 100. **Max rate:** 8 mg/min. Discontinue after patient has received cumulative dose of 300 mg.

SvO2

What is SvO2?

Mixed venous oxygen saturation (SvO2) is the percentage of oxygen bound to hemoglobin in blood returning to the right side of the heart. This reflects the amount of oxygen "left over" after the tissues remove what they need. SvO2 is used to help us recognize when a patient's body is extracting more oxygen than normal. An increase in extraction is the bodies way to meet tissue oxygen needs when the amount of oxygen reaching the tissues is less than required.

What does it tell us?

Mixed venous oxygen saturation (SvO2) can help to determine whether the cardiac output and oxygen delivery is high enough to meet a patient's needs. It can be very useful if measured before and after changes are made to cardiac medications or mechanical ventilation, particularly in unstable patients.

What are the normal values? Normal SvO2 60-80%

How to use SVO2 clinically!

If the amount of oxygen being received by the tissues falls below the amount of oxygen required (because of an increased need or decreased supply); the body attempts to compensate as follows:

First Compensation: Cardiac Output increases
The cardiac output is increased in an effort to increase the amount of oxygen being delivered to the tissues as shown below. Oxygen delivery is the amount of oxygen being sent to the tissues, and is determined by the following:

Oxygen Delivery (DO2) =
Cardiac Output (HR X Stroke Volume)
X
Oxygen Content (Hgb X SaO2)

If this is not sufficient to meet tissue energy needs, we move to our second compensation.

Second Compensation: Tissue oxygen extraction increases.
Tissues begin to remove or extract a higher percentage of oxygen from the arterial blood. This results in a reduced amount of oxygen remaining in the blood as it returns to the right side of the heart (decreased SvO2).
If this is not sufficient to meet tissue energy needs, we move to our third compensation.

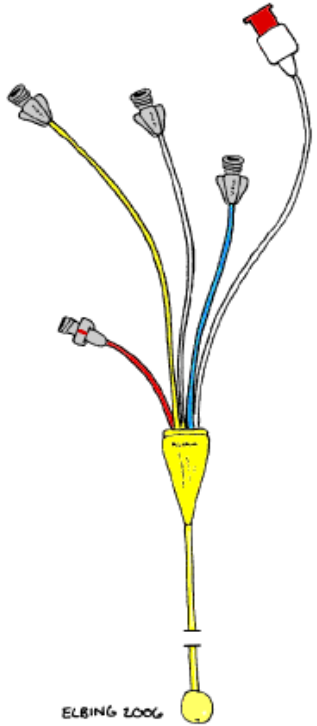
Third Compensation: Anaerobic Metabolism Increases

If the tissues fail to receive an adequate supply of oxygen, anaerobic metabolism becomes the only mechanism to produce tissue ATP. Anaerobic metabolism is inefficient, producing a large amount of metabolic waste (e.g. lactic acid) that is difficult for the body to eliminate quickly. It also produces a relatively poor supply of ATP. Prolonged anaerobic metabolism leads to energy depletion and metabolic acidosis.

There are 4 fundamental causes for a drop in SvO2:

1. The cardiac output is not high enough to meet tissue oxygen needs
2. The HGb is too low
3. The SaO2 is too low
4. The oxygen consumption has increased without an increase in oxygen delivery

PA Catheter

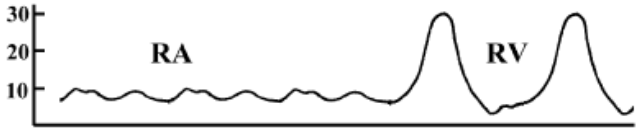


Blue Port (Proximal) – Opens in right atrium. Used for IV fluids and injecting fluid to measure Cardiac output.

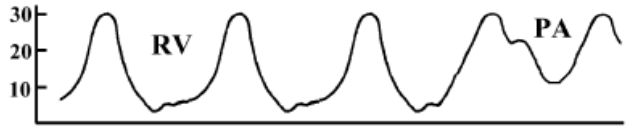
White Port (RV Paceport) - Opens in right ventricle. Used for IV infusion.

Yellow Port (Distal) – Opens at tip of catheter. Attached to pressure line for continuous measurement of PA pressures and waveforms.

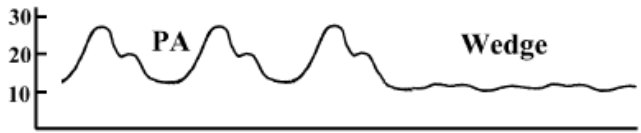
Red Port – balloon port. Up to 1.5cc air used to inflate balloon during insertion and for measuring PCWP.



RA= 0-8mmhHg



RV=15-30/0-8mmHg



PA=15-30/4-12mmHg :
Wedge=6-12mmHG

ABG's

Normal ABG values are as follows:

1.pH (acid base balance)= 7.35-7.45
2.CO2 (carbon dioxide)= 35-45
3.HcO3 (bicarbonate)= 22-26

You also must note the following:

1. CO2 greater than 45 is acidotic
2 .HcO3 less than 22 is acidotic
3. Co2 less than 35 is alkalotic
4. HcO3 greater than 26 is alkalotic

Base Excess:

1. Normal
    1. Range: -2 to +2 meq/L
2. Calculation
    1. Base Excess = (Actual_pH - Predicted_pH) * 67
    2. Calculate predicted pH based on PaCO2 (see Blood Gas)
3. Interpretation
    1. Positive (Base Excess)
        1. Metabolic Alkalosis
    2. Negative (Base Deficit)
        1. Metabolic Acidosis

1. **Metabolic Alkalosis**: If the pH is alkalotic and the Hco3 is alkalotic.
2. **Respiratory Alkalosis**: If the pH is alkalotic and the CO2 is alkalotic.
3. **Metabolic Acidosis:** If the pH is acidotic and the Hc03 is acidotic.
4. **Respiratory Acidosis:** If the pH is acidotic and the CO2 is acidotic.
5. **Compensated:** pH is anywhere inside the normal ranges (7.35-7.45)
6. **Uncompensated:** pH is anywhere outside the normal ranges (greater than 7.45 or less than 7.35, and the value (CO2 or HC03) that does not match the pH will still be in normal range.
7. **Partially compensated:** pH is anywhere outside the normal ranges, and the value that does not match the pH will be outside its normal range, indicating the body is attempting to get pH back to normal. For example, if the pH (7.20) and Co2 (50) are acidotic, the HC03 should be on the alkalotic side (27).

SYNCHRONOUS MODE:

The chamber(s) that are being paced have been adjusted so that the sensitivity is turned on. The pacemaker will only pace when it needs to.

***First letter** = chamber(s) being paced (a-atrium, v-vent. & d-dual)
***Second letter** = chamber(s) being sensed (a-atrium, v-vent, d-dual o-neither)
***Third letter** = how pacemaker how pacemaker operates(I-inhibited will not pace due to sensed beats. D-both inhibited and triggered response to sensed beats. O-neither triggered or inhibited.

1) DDD - both atria and ventricles are sensed and paced. Dual demand mode.
Chamber paced: Atrium & Ventricle
Chamber sensed: Atrium & Ventricle
Response to sense: Triggered & inhibited

2) AAI - atria are sensed & paced. Pacing will occur if no p wave is sensed.
Chamber paced-Atrium
Chamber sensed-Atrium
Responds to sensing

3) VVI - ventricles are sensed and pacing will occur if no QRS is sensed.
Chamber paced-Ventricle
Chamber sensed-Ventricle
Response to sensing-Inhibited

ASYNCHRONOUS:

A steady reliable form of pacing in patients with an intrinsic underlying rhythm. It can also be deleterious to the patient who has an underlying rhythm. Because there is no sensing with this mode a pacer spike could suddenly hit on a t-wave causing sustained v-tach.

1) AOO - Atrium are paced. Not sensed. Atrial pacing occurs at a fixed rate.
Chamber paced-Atrial
Chamber sensed-neither
Response to sensing-neither

2) DOO - the atria and ventricles are paced. Not sensed A|V pacing occurs at a fixed rate regardless of intrinsic rhythm.

Emergency settings for post-op CABG pt:

Rate: 80-100 Atrial MA: 20 Ventricle MA: 20 A\V interval: 150-170

Trouble shooting:

1) If there are no pacer spikes you decrease the sensitivity
2) If there are pacer spikes with no capture you increase the MA; and/or swicth polarity
3) If the pacer spikes occur in the QRS you increase sensitivity

Myocardial Stunning:

Myocardial stunning is a period of impaired contractility following a temporary period of ischemia, in which the dysfunction persists despite return of blood flow (Shavelle, 2006 Wang etal, 2003) Myocardial stunning may occur after CPB and postoperative cardiac function is often attributed to its effects (Wang etal, 2003) The impaired ventricular function related to the stunning is reported to terminate withing 2-3 days, and is not affected by preload or after load manipulation.

Myocardial hibernation is a condition of impaired LV function when the patient is at rest: it reflects a chronic reduction in blood flow. Heart function can be partially or totally normalized by improving blood flow or decreasing oxygen demand. Myocardial hibernation is a compensatory or protective mechanism during times of low blood flow.

Myocardial Ischemia and Infarction:

Myocardial ischemia, whether transient or leading to MI, may occur after cardiac surgery. Mechanism for myocardial ischemia include reperfusion injury from poor myocardial protections with cardioplegia incomplete revascularization, coronary spasm or coronary emboli. Patients with suspected MI or persistent ischemia follow the same course as uncomplicated postoperative patients, with beta blockers and IV nitroglycerin if the blood pressure allows (Khalpey et al., 2008: Lemmer et al 2003). IAPG therapy is suggested to diminish inotropic use, infarct size and myocardial oxygen demand (Khalpeyetal., 2008).

CARDIAC TAMPONADE:

Accumulation of blood in the pericardial sac that compresses the atria, restricts the venous return to the heart and ventricular filling and results in a decreased or cessation of preload, causing precipitous fall in CO. Early tamponade is usually a result of persistent mediastinal bleeding not evacuated by the chest tubes. Tamponade usually occurs within the first 12 postoperative hours.

Signs and symptoms to watch for; sudden decrease or cessation of mediastinal bleeding, dyspnea, low cardiac output with hypotension, narrow pulse pressure, tachycardia, increased cvp, sudden oliguria, anxiety\restlessness, low voltage QRS on EKG, cardiac enlargement n chest x-ray.

continuous hypotension that does not respond to fluid requires prompt intervention including bedside echo

PNEUMOTHORAX:

A pneumothorax may occur after cardiac surgery because of direct injury to the lung during surgery, central vevous cannulation or barotraumas during positive pressure ventilation. The incidence of pneumothorax afet cardiac surgery is relatively low, but the risk increases with IMA harvesting. A pneumothorax typically presents on the left side of the chest and occurs when the left parietal pleura is opened and the LIMA is dissected. This can usually be managed by placing the chest tube to suction. The incidence also increases in patients with bullious lung disease and high levels of PEEP. A tension pneumothorax can develop quickly in patients who are placed on mechanical ventilation. This complication can arise after a patient develops a right pneumothorax if the right parietal pleura is accidently cut. In this situation the patient acutely decompensates. SIS may include diminished breath sounds, although this may be heard to hear due to ventilator sounds and alarms. The patient can develop distended neck veins, hypotension, and tracheal deviation away from the collapsed lung. A CXR should be obtained and placemnt of a chest tube at the 5ht intercostal space anterior axillary line. (Weissman,2004).

SYSTOLIC DYSFUNCTION (DECREASED MYOCARDIAL CONTRACTILITY)

Contractility is the shortening of the myocardial fibers during systolic ejection (ventricular emptying). It is evaluated by the ejection fraction (EF). Cardiac contractility can be impaired postoperatively due to such factors as hypoxemia, electrolyte imbalance, narcotics, anesthesia, reprofusion injury, acidosis hypothermia, valve dysfunction, MI, and CPB. (Khalpey et a12003; masse & Antonacci,200S). Myocardial function usually' declines approximately S-6 hrs after surgery, possibly as a result of reperfusion from cardioplegia arrest and last approx 24hrs. Decreased contractility may require inotropic support with vasoactive drugs to support cardiac function. Epinephrine, however, can be associated with temporary, but significant hyperglycemia, metabolic acidosis, and increased serum lactate levels when used in the initial 6-8 post op period. These effects usually resolve in the next 24hrs (S1. Andre & Delrossi,200S). The RN must observe the patient for signs of inadequate perfusion such as low CO/CI, hypotension, mottling, end organ dysfunction and presence of metabolic acidosis. Urine output is considered a Jess reliable indictor of poor perfusion St. Andre & Delrossi, 2005).

Diastolic DYSFUNCTION:

Diastolic dysfunction may result from impaired systolic relaxation, stiffness of the left ventricle, or decreased diastolic stretching. Diastolic dysfunction commonly occurs as a result of AS, with LVH or poorly preserved intraoperative myocardial protection. Patients with decreased ventricular compliance will have diastolic dysfunction. (Salenger et al. 2003 Silvestry, 2008). This complication typically occurs for approx 2-3 hrs after surgery. If the ventricle becomes stiff during filling, and is not able to fill completely, fluid may back up into the lungs and cause heart failure.

The consequence of diastolic dysfunction is low cardiac output with a small ventricle. The hemodynamic picture is one of elevated PWP and low CO. Treatment includes fluid administration to maximize preload and vasodialators. Patients with diastolic dysfunction will require a higher PWP to maintain adequate preload. Because the ventricle has decreased compliance; the PWP may be falsely elevated despite the need for additional fluid to maximize preload. (Silvestry, 2008; St Andre & Delrossi, 2005) After maximizing fluid administration, infusion of an inotropic agent may be necessary to augment CO.

NOTES

WHEN TO CALL THE DOCTOR!!

- Chest tube bleeding greater than 200ml/hr
- Urine output less than 30ml/hr for 2 consecutive hours
- CI less than 2.2 that does not respond to fluid
- Before initiating new drug therapy
- Significant titration increases in medications already in use
- Abnormal ABG's
- Rhythm Changes
- Prior to cpap & extubation
- Before giving any blood products
- Neurological changes
- Abnormal labs that don't have replacement ordered
- Poor/inaccurate invasive line waveforms
- Oxygen desaturation
- Accidental line removal

FLINT