Acute Hypercarbia: Causes / Effects

Anesthetic Pearls: Anesthetic Implications and Management of Hypercarbia

Hypercarbia is a $PaCO_2$ >45 mmHg

Increased $PaCO_2$ is secondary to inadequate ventilation and / or increased CO_2 production.

Decreased Ventilation / Elimination	Increased CO_2 Production
CNS Depression	Exogenous CO_2 (laproscopy)
Neuromuscular depression	Malignant hyperthermia
Rebreathing (stuck circuit valve, absorbant exhausted)	Reperfusion (limb after tourniquet, AAA unclamp)
Inadequate minute ventilation	Sepsis
Increased airway resistance	Fever
COPD	TPN
V/Q mismatch (PE)	Shivering
	Bicarbonate administration

Intra-Op Signs: Flushed skin, cardiac dysrythmias (PVC / PAC, tachycardia), HTN, depressed respirations, coma

CNS: ↑ CBF, narcosis if $PaCO_2$>90, mild ↑ $PaCO_2$ leads to ↑ EEG frequency, ↑↑↑ CO_2 leads to ↓ frequency & amplitude of the EEG (CNS depression, coma)

Cardiovascular: $C0O_2$ is direct depressant of CV system but activates sympathetic nervous system. Moderate ↑ $PaCO_2$ leads to ↑ BP & ↑ CO (cardiac output) while ↑↑↑ $PaCO_2$ leads to ↓ CO / BP / HR. Arrhythmias occur with hypercarbia (especially if combined with halothane anesthetic).

Respiratory: ↑ respiratory response (↑ MV) until $PaCO_2$ approximately 100, at that point any further increase in $PaCO_2$ leads to ventilatory depression. Hypercarbia leads to ↑ PVR (pulmonary vascular resistance), Right-shift of the O_2 dissociation curve (less affinity of hemoglobin for oxygen).

GI: Direct effect is an increase of hepatic and portal venous blood flow. But, if the sympathetic nervous system is not completely blocked during general anesthesia, increased $PaCO_2$ leads to splanchnic nerve stimulation with vasoconstriction and decreased hepatic blood flow.

Metabolic: ↑ $PaCO_2$ leads to ↑ norepinephrine & epinephrine. ↑ $PaCO_2$ leads to ↓ pH which causes ↑ K+.

MAC: No change with $PaCO_2$ between 20-100. Very high $PaCO_2$ (> 100) causes anesthesia / narcosis & decreases MAC ($PaCO_2$ of ~ 250=1 MAC).

Local Anesthetics: Weak bases that exist in equilibrium [BH^+ <=> ($B + H^+$)]. If hypercarbia present, ↓ pH and more exists in the BH+ (ionized) form that does NOT easily cross membranes therefore causing decreased activity of the local anesthetic.

Management: First, the arterial pH must be evaluated to determine if the elevation in $PaCO_2$ is acute or chronic. A normal pH indicates a chronic condition (COPD), while a pH < 7.35 suggests an acute process with concomitant respiratory acidosis. The $EtCO_2$ can be used roughly to follow $PaCO_2$. Discrepancies arise due to V/Q mismatching. If physiologic dead space increases (extremely high V/Q ratio, areas ventilated but not perfused), the $EtCO_2$ will be much lower than the $PaC0_2$ ($EtCO_2$-$PaCO_2$ gradient). This situation occurs with pulmonary embolism (air / amniotic fluid / fat embolism) and cardiac arrest.

1. Ensure adequate oxygenation (Sat, PaO_2)
2. Ensure adequate ventilation
   - A. Spontaneously ventilating patient: Check patent airway, ? intubate & control ventilation
   - B. Mechanically ventilated patient: Increase MV, check equipment (valves, absorber, vent)
   - C. Check the inspired CO_2 level: More than 1-2 mm Hg inspired CO_2 indicates rebreathing of CO_2 due to incompetent valve in breathing circuit, exhausted soda lime (increase the fresh gas flow to convert the circle system to a semiopen system, the inspired CO_2 should drop markedly), or administration of exogenous CO_2.
3. Obtain ABG to confirm hypercarbia
4. Look for causes of increased CO_2 production (fever, sepsis, MH)
5. If hypercarbic in PACU with extubated patient: Check residual neuromuscular block, consider Naloxone +/- Flumazenil to reverse opioid / benzodiazepine effects. ? re-intubate & control ventilation