Episode 24: The Evils of Sodium Bicarbonate

On this episode: Dr. Jed Wolpaw

Please take a look at the VANCS study on the use of vasopressin as a sole agent in the treatment of vasoplegic shock. This episode is a review of the reasons **never** to use sodium bicarbonate in lactic acidosis.

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What is the VANCS study?

Summary
Prospective, single center, randomized trial
Vasopressin (up to 0.06 U/min)[1] vs **N**orepinephrine (up to 60 µg/min) for patients with vasoplegic[2] shock after **C**ardiac **S**urgery
Vasopressin group • No ↑ adverse events • Lower occurrence of atrial fibrillation • ↓ need for dialysis • ↓ length of ICU or hospital stay

[1] One might worry about ischemia in doses up to 0.06 U/min

[2] Defined as mean arterial pressure < 65 mmHg resistant to fluid challenge and cardiac index > 2.2

Background on bicarb review

- Sodium bicarbonate was taken off the algorithm for cardiac arrest but still commonly used in code situations. It was thought to improve right heart function and response to catecholamines and vasopressors. Four common arguments might be made to support the use of sodium bicarbonate in acidosis:[3]
    1. Low pH decreases cardiac function, and thus is bad
    2. Bicarb can increase pH
    3. This will improve cardiac function
    4. Any adverse events outweighed by benefits

Low pH is bad

- Lots correlated, but not causation. Acidotic people seem to die at higher rate than those who are not, but this is not necessarily from acidosis. If they are sick enough to have pH of 7.0, then they're probably in bad shape. Their counterpart with pH of 7.3 is probably in better shape. It's not that you'll die from acidosis, but you'll die. The question is not whether pH is an indicator of "badness" but whether giving bicarb is helpful or harmful.
- Is pH in itself bad? Finding the measured blood pH to be low doesn't tell us anything about intracellular pH or mitochondrial pH. Bonventre and Cheung's study looked at this and found that mitochondrial pH stayed normal even when extracellular (blood) pH was lowered from 7.4 to 6.9. This tells us that acidosis in blood does not necessarily cause intracellular/mitochondrial acidosis, and thus not that big of a deal.
- There are interesting models where we are allowing people to get hypercapnic (ARDS or severe status asthmaticus). In ARDS, tidal volumes are kept low to protect lung, but become hypercapnic → acidotic. 1996 study showed that when these people have pH 7.4 decreased rapidly to ~7.25 (<1hr), systemic vascular resistance ↓ and cardiac output ↑. Pulm vascular resistance didn't change. Why is this significant? People say acidosis ↑ pulmonary pressure, like in pulmonary hypertension. 7.15 pH did not have ↑ arrhythmia or ↓ cardiac output. Some patients went down to 7.0 but still didn't have these complications.
- This calls into question if pH in and of itself is a huge problem. Some models show that acidosis can be a good thing. Bonventre and Cheung's study exposed hepatocytes to anoxic environment, then split into acidic and non-acidic environments. The ones in acidotic environment survived longer and thus acidity was protective. Similarly, acidosis of reperfusion of heart **limits** infarct size. Lactate itself can ↓ cardiac contractility in animal models so when buffered[4] to pH to 7.4, you'll see ↓ cardiac contractility. So what? Sodium bicarb ↑ production of lactate → cardiac contractility ↓, even IF it's changing pH to become less acidotic.

Bicarbonate can raise pH

- Yes, of the blood. However, most studies and models that looked at this show that intracellular pH falls. Produced CO_2 diffuse into cell and get trapped, causing intracellular acidosis.

[3] This episode largely follows Forsythe and Schmidt's article. Also check out Sabatini and Kurtzman!

[4] In other words, ↑ lactate without causing acidosis

Does increasing pH help?

- Studies show no difference in hemodynamic variables, including cardiac output, when giving bicarb vs saline. Sure, you can see blood pressure go up, but as the same amount as saline. Transient benefit of bicarb is from giving bolus (~50 CC) of fluid. A bolus of bicarb is 1000 mEq /L of sodium vs a bag of normal saline (154 mEq/L). A bolus of fluid to heart is beneficial, but not directly from the bicarb itself. After administering bicarb, no difference in pulmonary output, cardiac output, nor response to vasopressors. This persisted for people with pH of 6.9.
- Acidosis can ↓ expression of beta receptors, thus decreasing response to catecholamines. However, BICARB doesn't fix it. Something about acidotic state is causing that issue.
- People also mention bicarb helps with resuscitation from ventricular fibrillation. Code team still give thinking it will help. This comes from one canine study from 1995, but subsequent dog and human studies showed that bicarb didn't help with resuscitation. In fact, bicarb made it worse – it's associated with WORSE mortality, so don't give bicarb!!

Negative effects of bicarb?

- Hypervolemia, hyperosmolarity, hypernatremia, transient rise in intracranial pressure.
- Lowers PAO_2, by worsening intrapulmonary shunt, from 5 to 15 mmHg. Humans were made acidemic which was then corrected with bicarb. The resulting left shift in the oxygen curve was found to last up to 8 hours even after resolution of alkalosis, suggesting the effects aren't just from increase in pH. The effect on 2,3-BPG lasts this long as well, making it harder to deliver oxygen to tissues for up to 8 hours! This may also be why bicarb ↑ lactate production.
- Serum ionized Ca is also reduced, thus maybe why ↓ cardiac contractility! Ca is given with bicarb but hard to keep up if keep giving lots of it.

Summary

- People argue for giving bicarb because postop cardiac patients have stiff heart. Anything to ease work would be good. Acidosis makes it worse so bicarb will make heart happy. This is NOT necessarily TRUE! Yes, studies have not been done on specifically cardiac postop patients, but bicarb
    - Does NOT lower pulm pressure
    - Does NOT increase cardiac output
    - Worsens oxygen delivery
    - Decreases intracellular pH
- This is specific to LACTIC acidosis, not from bicarb-losing pathology like diarrhea, enterocutaneous fistula, or renal tubular acidosis. These are totally reasonable to replace with bicarb.
- But in lactic acidosis, body has not lost bicarb. When liver starts clearing lactate, it turns lactate back into bicarb. Giving bicarb would not be the correct treatment of this problem; It doesn't accomplish what people want. Bicarb
    - Does NOT improve response to epinephrine
    - Does NOT improve cardiac output or lower pulmonary resistance
- No evidence based reason to give bicarb in lactic acidosis, but many reasons to avoid it. These articles stated no threshold in support of giving bicarb. Please withhold giving bicarb for lactic acidosis until solid evidence from studies like RCT exist.

How do you treat vasoplegic shock? Do you use vasopressin as first line? Do you give bicarb for lactic acidosis?

Review questions

Link jumps to appropriate section

1. VANCS study compared what two treatments for vasoplegic shock? Why is this significant?
2. How do the studies on ARDS and hepatocytes demonstrate that pH itself isn't a major concern?
3. How was it shown that bicarb doesn't improve cardiac output or pulmonary status?
4. Why doesn't bicarb necessarily improve response to catecholamine?
5. How does bicarb decrease oxygen delivery?
6. Will you give sodium bicarb for lactic acidosis?