Chapter 26
Acid/Base Disturbances

Joseph P. Cravero

You are asked to anesthetize an 11-month-old who was born with tetralogy of Fallot characterized by severe pulmonary outflow tract obstruction. He underwent a repair as a newborn. He is scheduled for a hypospadias repair. He has clear lung fields but left ventricular function is depressed, and he is maintained on chronic Lasix treatment as well as a salt-restricted diet. ABG reveals a pH = 7.44, $paCO_2$ = 64, paO_2 = 62, and HCO_3- = 40, on room air.

J.P. Cravero, MD, FAAP
Department of Anesthesiology, Perioperative, and Pain Medicine, Boston Children's Hospital, Boston, MA, USA

Harvard Medical School, Boston, MA, USA
e-mail: joseph.cravero@childrens.harvard.edu

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Preoperative Evaluation

Questions

1. Interpret the ABG. How might you explain the findings? Is this patient alkalotic? Is the patient acidotic? Based solely on the $PaCO_2$ – what would you expect the pH to be in this case? Explain why this patient's acid/base profile is the way it is. What does chloride loss have to do with the development of alkalosis? Why would the fact that there is a salt-restricted diet add to the problem?

2. Is he ready for the OR? Does his acidosis/alkalosis need to be corrected prior to the OR? Where should you attempt to keep his CO_2 during the operation? Is there a problem with normalizing his CO_2? How much oxygen should he be on?

3. What is a "normal" bicarbonate level? How does the concentration of HCO_3^- relate to pH?

4. What is the most common cause of alkalosis in a child coming for surgery? If a child with severe vomiting or an NG tube to suction is given H_2 receptor blockade – does that stop the development of alkalosis? Could that treatment cause them to become acidotic?

5. Why does K^+ depletion cause alkalosis?

Preoperative Evaluation

Answers

1. The patient's blood pH is in the normal range between 7.35 and 7.45. He has a significant metabolic alkalosis that is largely compensated by a respiratory acidosis. There are many possible explanations for these findings. Most probably the ABG is due to the fact that this patient has poor cardiac function that has necessitated him being on long-term diuretics to avoid congestive heart failure. This has led to chronic chloride and sodium loss. Since the kidney must maintain electrical neutrality, for every positive ion excreted, a negative ion needs to be absorbed. When chloride is deficient, bicarbonate is resorbed to maintain electrical neutrality – leading to accumulation of bicarbonate in the bloodstream and the resulting alkalosis. On the other hand, when carbon dioxide levels in the blood increase, there is a rise in carbonic acid. A 1 mm Hg change in the $PaCO_2$ above or below 40 mmHg results in a 0.008 unit change in the pH in the opposite direction. If there was no alkalosis, we would expect the pH to be $7.4 - 24(0.008) = 7.2$ (approximately). The entire problem might be avoided if the patient was not on a salt-restricted diet (and inadequate Cl in the diet). GI absorption would make up for losses, and alkalosis would be minimal since the kidneys would be able to continue to resorb chloride instead of bicarbonate.

2. Strictly in terms of his acid/base status, the patient is ready for the OR for this procedure. Attempting to correct his metabolic alkalosis prior to the operating room would take a long time and would almost certainly result in adverse effects (likely worsening of his cardiopulmonary status because of volume overload). During the case it would be wise to allow his CO_2 to remain moderately elevated to the degree it is at his baseline. Respiratory drive in a child like this is primarily based on the pH of the CSF in the midbrain around the respiratory centers. Normalization of the respiratory acidosis would leave this child very alkalotic and would almost certainly lead to respiratory depression.

3. Bicarbonate is normally about 24. Bicarbonate is related to pH through the Henderson-Hasselbalch equation which in this case would be $pH = 6.1 + \log([HCO_3-]/.03 \times PaCO_2)$. A change in the HCO_3- concentration of 10 meq/L will result in a change in pH of approximately 0.15 pH units in the same direction.

4. The most common cause of alkalosis is acid loss through vomiting and/or NG suction. H_2 blockers can decrease H^+ secretion in the stomach and stop the process since the fluid lost would lack significant H^+ ions. It is unlikely that the patient would become acidotic unless the fluid loss was so great it led to significant hypovolemia and poor perfusion.

5. K^+ depletion – which occurs with mineralocorticoid excess – increases bicarbonate reabsorption from the proximal and distal tubule resulting in alkalosis. Serum aldosterone release from the renal cortex would be suppressed by hypokalemia.

Intraoperative

Questions

1. You would like to perform a caudal for postoperative analgesia. What are your specific considerations for this patient? Does this patient need a volume load prior to a neuraxial block? What is your choice of IV fluid? If he has a pre-existing right bundle branch block from his cardiac surgery, would bupivacaine be as good a choice as ropivacaine? 2,3 chloroprocaine? Would the pH of the patient affect the onset of local anesthetic? Why?

Intraoperative

Answers

1. Hypospadias repair involves penile (sacral) dermatomes that are well covered by a caudal block. In general, caudal block performed after induction of anesthesia would decrease the MAC requirement during surgery by decreasing sensory input from the surgical intervention. The caudal would also supply an effective analgesic for the immediate postoperative time frame. A single-shot caudal or a caudal catheter with continuous infusion could be used depending on the extent of the surgery and the postoperative plans for the patient. In this particular patient, we would like to know the current hemodynamic status of the patient. If the repair is completed, is there any residual shunt? Appropriately administered caudal anesthesia does not usually have much effect on the overall hemodynamic profile of patients in the infant and toddler age groups. These blocks have been safely applied for congenital heart surgery patients prior to and after repair. The volume status of this patient should be carefully considered. Prolonged NPO times could lead to volume depletion; however this patient could be sensitive to volume overload. I would consider the NPO duration and administer a volume of fluid that would replace fluid deficit, but it would not be necessary to administer additional fluids to a child in this age group to prepare for the caudal block. If the patient was on any anticoagulants, this could impact the regional anesthesia plan. Regional anesthesia is considered safe in patients taking nonsteroidal anti-inflammatory drugs. On the other hand, if the patient was on other anticoagulants such as low molecular weight heparin or specific antiplatelet agents, the recommended time frames for interruption of these medications prior to regional anesthesia must be observed. Implications of an intravascular injection could be significant in this patient, and extra care should be taken to administer a test dose and fractionate the administration of a bolus dose. Local anesthetic toxicity is related to blockade of sodium channels. Bupivacaine is a stronger blocker of these channels than lidocaine or ropivacaine. If the patient has a pre-existing conduction abnormality such as a right bundle branch block, induction of dysrhythmias with high levels of local anesthetic could be an issue. As a result, ropivacaine or 2,3 chloroprocaine would be considered a better choice in a patient with compromised cardiac performance. Local anesthetics are weak bases. The pKa of each local anesthetic (the pH at which the drug exists 50 % ionized and 50 % unionized) will determine the concentration of the cationic and base forms that exist at any pH. This is expressed by the Henderson-Hasselbalch equation as $pH = pKa + \log([\text{base}]/[\text{cation}])$. Since it is the unionized base form of local anesthetics that determines the onset of the drug, the more acidic the patient is, the slower the onset will be.

2. What type of fluid would be most appropriate for this patient? Do you think lactated Ringer's should be avoided? Why? What would happen if you mistakenly infused an IV fluid containing bicarbonate into this child? Would he/she become dangerously alkalotic? For how long? How is bicarbonate excreted? Why would bicarbonate containing solutions exist? Is there any reason to administer such a solution on purpose?

Postoperative

Quesitons

1. On emergence your patient has developed a significant acidosis. What is the effect of acidosis on the major body systems? What would be the effect of alkalosis on these systems?

2. Normal saline contains 154 mM of Na^+ and Cl^-. It has a pH of 5 and an osmolarity of 308 mOsm/L. Lactated Ringer's solution has an average pH of 6.5, is hypo-osmolar (272 mOsm/L), and has similar electrolytes to plasma. The administration of large amounts of NS has been associated with the development of hyperchloremic acidosis. There is some theoretical concern that lactate accumulation could lead to difficulties with high volume LR administration, but this has not been clinically shown to be an issue. LR is not approved for administration with blood products. In this case, I would choose LR for fluid administration as long as blood products were not co-administered (extremely unlikely in this case). In the kidney, the distal tubule will resorb 100 % of HCO_3- until the blood level reaches 24 meq/L – at that point it will excrete almost all of HCO_3- above that level. An inadvertent bicarbonate infusion will increase the HCO_3- level significantly which will, in turn, lead to spilling of massive amounts of HCO_3- and alkalinize the urine. This is done purposely in some cases to encourage the excretion of toxins or chemotherapeutic agents. Systemic alkalosis would be very short-lived as the kidney is extremely effective at excreting excess bicarbonate.

Postoperative

Answers

1. Acidosis can have multiple effects. With pH <7.22, myocardial and smooth muscle depression can result in decrease in cardiac stroke volume, cardiac output, and systemic vascular resistance. Hypotension that results can lead to poor perfusion and inadequate O_2 delivery (in spite of a rightward shift of the O_2-Hgb dissociation curve) and add to acidosis. The response to vasopressors and inotropes is impaired in this setting. Pulmonary pressures will increase secondary to increased pulmonary vascular resistance which leads to greater afterload on the right heart. An increased acidosis will lead to increasing K^+ levels which could be dangerous if K^+ was elevated to begin with. Alkalosis has several effects as well. (1) Increasing pH leads to a leftward shift of the O_2-Hgb dissociation curve. This decreases O_2 unloading which can be problematic in a patient with marginal cardiac output. (2) Calcium levels will decrease which can lead to decreased cardiac contractility – particularly in newborns and infants. (3) From the pulmonary perspective, alkalosis will lead to an increase in bronchial smooth muscle tone. (4) Respiratory alkalosis is associated with low CO_2 and will result in lower cerebral blood flow due to cerebral arteriolar constriction. The effects of pH extend to the coagulation system as well. Coagulation factors are most active at elevated pH levels (pH = 8). While it is not recommended to purposely raise the pH to these levels for bleeding prophylaxis, it is important to appreciate that significant acidosis can impair procoagulant activity.

2. Your patient is not waking up after completion of the surgery. What possible effects of acidosis could be a cause of delayed emergence?

Additional Questions

Questions

1. You are asked to provide MRI sedation for a child with a pH of 7.28. The Na is 145, the K is 4.5, the Cl is 111, and the bicarbonate is 18. How would you characterize the acidosis in this patient? High or low anion gap? What kind of problems might lead to a high gap acidosis? What kind of problems would lead to a non-anion gap acidosis?

2. You are asked to anesthetize a 5-year-old WF patient for renal transplant who is in end-stage renal failure. Her pH is 7.21. Why are renal failure patients acidotic?

2. Extremes of CO_2 retention can induce a CO_2 narcosis due to neuronal intracellular acidosis and intracranial hypertension from increased cerebral blood flow in those patients that are susceptible. In addition, monoquaternary muscle relaxants such as vecuronium and rocuronium will be potentiated and could be prolonged in their action. Finally acidosis will decrease renal and liver blood flow, thus extending the half-life of most anesthetic agents and opioids – although unionized fractions will be reduced for almost all agents at low pH.

Additional Questions

Answers

1. The anion gap is calculated by subtracting the sum of the serum anions from the sum of the serum cations as follows: $([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$. The normal range is between 3 and 11. In this case, the anion gap is elevated which indicates there is an acidosis. Possible causes include lactic acidosis and diabetic ketoacidosis. Exogenous ingestion of acid such as methanol, ethylene glycol, propylene glycol, or aspirin could account for this. Endogenous (inborn errors of metabolism) leading to an acid load can also account for this. Hyperchloremia resulting in bicarbonate loss is the most common cause of a non-anion gap acidosis. This could be due to gastrointestinal loss or rental tubular acidosis – proximal or distal.

2. If the renal damage affects glomeruli and tubules, the acidosis is a high anion gap acidosis. It is due to failure of adequate excretion of various acid anions due to the greatly reduced number of functioning nephrons. If the renal damage predominantly affects the tubules with minimal glomerular damage, a different type of acidosis may occur. This is RTA, characterized by a normal anion gap or hyperchloremic type of acidosis.

Suggested Readings

1. Kitching AJ, Edge CJ. Acid-base balance: a review of normal physiology. Br J Anaesthesia, CEPD Rev. 2(1):3–6.
2. Topulos G. Acid-base balance in anesthesia and intensive care medicine. In: Vacanti C, Sikka P, Urman R, Segal S, editors. Essential clinical anesthesia. New York: Cambridge University Press; 2011. p. 388–95.