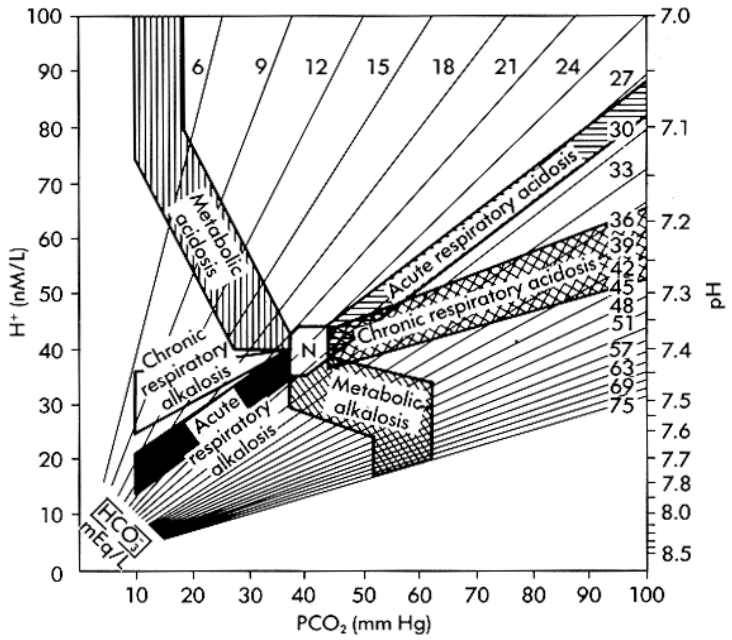

Clinical Cases in Acid-Base Interpretation

Revision 2.0

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CASE 1 – A Serious Drug Error

A 23-year-old man was found to be apneic and unresponsive in the surgery ward following reconstructive knee surgery. About 30 minutes earlier he received 25 mg of intravenous (IV) morphine for pain relief. While he is being assessed and resuscitated, an arterial blood gas sample was taken, revealing the following:

pH	7.08
PCO_2	80
HCO3	23

Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as acidemic. (Recall that the "normal range" for arterial blood pH is 7.35 to 7.45).

Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is respiratory, with the PCO2 being severely elevated. Since the bicarbonate is normal, there is no primary metabolic disturbance. (Recall that that the "normal range" for arterial PCO2 is 35- 45 mm Hg).

Step 3: For a respiratory disturbance, is it acute or chronic?

Recall that for acute respiratory disturbances (where renal compensation does not have much time to occur) each arterial PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.08, while for chronic respiratory disturbances (where renal compensation has time to occur) each PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.03.

In our case an arterial PCO2 shift of 40 mm Hg is accompanied by a pH shift of 0.32 units, or a 0.08 pH shift for each PCO2 shift of 10 mm. This means that the respiratory disturbance is acute. (If it were chronic the pH shift would be 0.003 x 40 = 0.12, for a resulting pH of 7.4 – 0.12 = 7.28).

Steps 4-7: Not applicable in this case.

DIAGNOSIS: Acute Respiratory Acidosis

CLINICAL NOTES

Morphine and other opiate analgesics are respiratory depressants and in large doses will stop breathing. This effect is mediated via μ (mu) opioid receptors in the medulla, the part of the brainstem that regulates breathing and other autonomic functions.

When examined, the patient was cyanotic (blue), with small pinpoint pupils, but he still had a pulse. He was given 100% oxygen by positive-pressure ventilation and 0.4 mg of IV naloxone (Narcan), an opiate antagonist. The patient quickly improved. Upon review of the incident it was noted that the analgesic order was actually for 25 mg of IV meperidine (Demerol), an amount roughly equal to 2.5 mg of morphine.

Drug errors of this kind are frightfully common. The Institute of Medicine estimates that as many as 44,000 to 98,000 people die in U.S. hospitals each year as the result of medical errors. This means that more people die from medical errors than from motor vehicle accidents, breast cancer, or AIDS.

Top: Apparatus to deliver oxygen by positive pressure ventilation.
Bottom: Apparatus in clinical use.
(Image credit: www.matrxmedical.com)

CASE 2 – Pyloric Stenosis

A 4-week-old baby boy is admitted to hospital with history of projectile vomiting of several days duration. The following blood gases are obtained:

pH	7.50
PCO2	49 mmHg
HCO3	37 mEq/L

Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as alkalemic. (Recall that the "normal range" for arterial blood pH is 7.35 to 7.45).

Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is metabolic, with the HCO3 being elevated. Since the PCO2 is raised in the face of an alkalemia, there is not a primary respiratory disturbance – the raised PCO2 merely indicates that respiratory compensation has occurred.

Step 3: Not applicable in this case.

Step 4: The expected PCO2 in metabolic alkalosis is 0.7 x HCO3 + 20 mm Hg = [0.7 x 37] + 20 = 46 mm Hg. Since the actual PCO2 (49) and the expected PCO2 (46) are approximately the same, this suggests that respiratory compensation is appropriate.

Steps 5-7: Not applicable in this case.

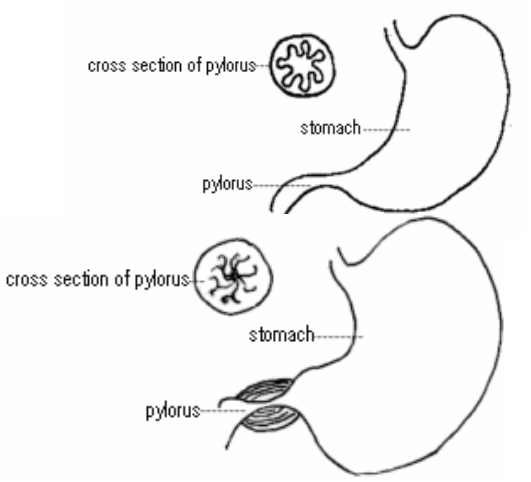
DIAGNOSIS: Metabolic Alkalosis from Persistent Vomiting due to Pyloric Stenosis

CLINICAL NOTES

Pyloric stenosis is a narrowing of part of the stomach (the pylorus) that leads to the small intestines. This occurs when the muscle around the pylorus has grown too large (hypertrophied). Most babies with pyloric stenosis begin to vomit during the second to third week of life. These babies begin with "spitting up" that later becomes projectile vomiting after feeding. The loss of gastric acid is the basis for the metabolic alkalosis in this setting.

The diagnosis of pyloric stenosis is often suspected on clinical grounds and confirmed by ultrasonic imaging or a barium swallow study. Also known as infantile hypertrophic pyloric stenosis or gastric outlet obstruction, pyloric stenosis is relatively common, especially in firstborn male infants.

Pyloric stenosis is fixed with an operation called a pyloromyotomy, where the surgeon spreads open the muscle around the pyloric valve. Some scientists believe that babies with pyloric stenosis lack receptors in the pyloric muscle to detect nitric oxide, an important chemical messenger that relaxes smooth muscle. As a result, the muscle is in a state of almost continual contraction, which causes it to hypertrophy over time. This process may take some time, which is why pyloric stenosis usually appears in babies a few weeks after birth rather than immediately. (Incidentally, do not confuse nitric oxide (NO) with nitrous oxide (N_20), the "laughing gas" often used in anesthesia.)



Top: Diagram of normal stomach and pylorus. Note the cross-section showing normal pyloric opening. **Bottom:** Diagram of stomach with pyloric stenosis. Note the cross-section showing how the pyloric opening is very narrowed. *(From www.pedisurg.com)*

CASE 3 – Woman with Panic Attacks

A 26-year-old woman is undergoing treatment for frequent panic attacks. The attacks are accompanied by hyper-ventilation, a racing heartbeat (tachycardia), dizziness, feelings of "unreality" and tingling in the hands. In one particularly severe attack, when taken to the emergency department, an arterial blood-gas sample was taken, which revealed the following:

pH	7.52
PCO2	26 mm Hg
HCO3	22 mEq/L

Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as alkalemic. (Recall that the "normal range" for arterial blood pH is 7.35 to 7.45).

Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is respiratory, with the PCO2 being lowered primarily.

Step 3: For a respiratory disturbance, is it acute or chronic?

Recall that for acute respiratory disturbances (where renal compensation does not have much time to occur) each arterial PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.08, while for chronic respiratory disturbances (where renal compensation has time to occur) each PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.03.

In our case an arterial PCO2 shifted down by 14 mm Hg is accompanied by a pH shift up of 0.12 units, or a 0.086 pH shift for each PCO2 shift of 10 mm. This means that the respiratory disturbance is acute, the 0.086 pH shift for each PCO2 shift of 10 mm being close enough to the 0.08 expected. (If it were chronic the pH shift would be about 0.003 x 14 = 0.042, for a resulting pH of 7.4 + 0.042 = 7.44).

Steps 4-7: Not applicable in this case.

DIAGNOSIS: Acute Respiratory Alkalosis from Hyperventilation due to Panic Attack

Could You Have Panic Disorder?

(From http://www.nimh.nih.gov/anxiety/getpd.cfm)

Do you experience sudden episodes of intense and overwhelming fear that seem to come on for no apparent reason?

During these episodes, do you also experience several of the following:

- Racing, pounding, or skipping heartbeat
- Chest pain, pressure, or discomfort
- Difficulty catching your breath
- Choking sensation or lump in your throat
- Excessive sweating
- Lightheadedness or dizziness
- Nausea or stomach problems
- Tingling or numbness in parts of your body
- Chills or hot flashes
- Shaking or trembling
- Feelings of unreality, or being detached from your body

During these episodes, do you have the urge to flee, or the feeling that you need to escape?

During these episodes, do you think something terrible might happen—that you might die, have a heart attack, suffocate, lose control, or embarrass yourself?

Do you worry a lot about these episodes or fear that they will happen again? And does this fear cause you to avoid places or situations that you think might have triggered the attack?

If you answered yes to most of these questions, chances are you are suffering from panic disorder. If so, you are not alone.

Panic disorder is very different from everyday anxiety. More than 3 million American adults have, or will have, panic disorder at some time in their lives. Most frequently, it starts in young adulthood. Usually, it does not go away by itself. But with proper treatment, people with panic disorder can be helped.

CASE 4 –Man with Hodgkin's Disease

A 52-year-old man with Hodgkin's disease is treated with ABVD[1], a combination chemotherapy regimen. Unfortunately, despite treatment with antiemetics, he suffers from severe, persistent vomiting[2]. When seen by his physician, he is dehydrated and has shallow respirations. Blood gas data is as follows:

pH	7.56
PCO2	54 mm Hg
BUN	52 mg/dl (NL=7 – 18)
Creatinine	1.8 mg/dl (NL=0.7 – 1.2)
K+	2.8
HCO3	45 mmol/L
BUN/creatinine ratio[3]	29:1 (NL=12:1 - 20:1)

Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as alkalemic. (Recall that the "normal range" for arterial blood pH is 7.35 to 7.45).

Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is metabolic, with the HCO3 being elevated. Since the PCO2 is raised in the face of an alkalemia, there is obviously not a primary respiratory disturbance – the raised PCO2 merely indicates that respiratory compensation has occurred.

Step 3: Not applicable in this case.

Step 4: The expected PCO2 in metabolic alkalosis is 0.7 x HCO3 + 20 mmHg = [0.7 x 45] + 20 = 52 mm Hg. Since the actual PCO2 (54) and the expected PCO2 (52) are approximately the same, this suggests that respiratory compensation is appropriate.

Steps 5-7: Not applicable in this case.

DIAGNOSIS: Metabolic Alkalosis from Persistent Vomiting due to Chemotherapy.

CLINICAL NOTES

[1] The following are the drugs used in the ABVD regimen.

· Adriamycin · Bleomycin
· Vinblastine · Dacarbazine

[2] "The control of vomiting consists of two anatomically and functionally separate units, a vomiting center and a chemoreceptor trigger zone. The vomiting center is in the reticular formation of the medulla and is excited directly by visceral afferent impulses (sympathetic and vagal) arising from the gastrointestinal tract and other peripheral trigger areas. These trigger areas are found in the pharynx, cardiac vessels, peritoneum, bile ducts, cortex and stomach. The chemoreceptor trigger zone is on the floor of the fourth ventricle, on the blood side of the blood-brain barrier. The chemoreceptor trigger zone is unable to cause vomiting without an intact vomiting center." (http://gastroresource.com). Chemotherapy causes the release of serotonin (5-HT), and other chemicals in the small intestine, which through the mechanism outlined above stimulates vomiting (emesis). Antiemetics block one or more of these signals. Those that block 5-HT3 receptors are particularly effective; these include ondansetron, dolasetron and granisetron. Their antiemetic effects are postulated to stem from blockade of 5-HT3 receptors located on the nerve terminals of the vagus nerve in the periphery and centrally in the chemoreceptor trigger zone of the area postrema.

[3] "The ratio of BUN: creatinine is normally 10:1. With dehydration, the ratio can increase to 20:1 or even higher. An increased BUN: creatinine ratio may also be due to certain types of kidney disease, breakdown of blood in the intestinal tract, increased dietary protein, or any clinical circumstance in which insufficient blood is flowing through the blood vessels to the kidneys (such as heart failure or kidney artery disease)." (From http://www.prlnet.com)

"Remember that BUN that is filtered at the glomerulus is reabsorbed in the renal tubules in a process that is dependent on the rate of flow of fluid through the tubules (lower flow rate = more reabsorption = higher serum BUN). Creatinine is not reabsorbed in this way. Thus an elevated BUN/creatinine ratio (>20) suggests that low flow of fluid through the nephrons is the basis for the elevation in BUN and, to a lesser extent, creatinine." (http://www.mcl.tulane.edu).

CASE 5 – Diabetic Ketoacidosis

A 31 year old man presents with lethargy, weakness, labored respiration, and confusion. He has had diabetes for 15 years, and has been suffering from the "intestinal flu" for a day or so, for which he has been avoiding food to help prevent further vomiting and "make his stomach ache go away". Since he stopped eating, he thought that it would be a good idea to stop taking his insulin. When seen in the emergency department his urine dipped positive for both glucose and ketones and his breath had a strange sweet, fruity smell. The following arterial blood gas data was obtained:

pH	7.27
PCO_2	23 mm Hg
Na^+	132 mEq/L
Cl^-	83 mEq/L
K^+	4.9 mEq/L
HCO3	10 mEq/L
Glucose	345 mg/dL

Step 1. The pH is 7.27, which is considerably less than normal (7.35-7.45), so the patient is acidemic.

Step 2: The PCO2 is low, so the respiratory system is not causing the acidosis; rather, the drop in PCO2 must be a compensatory process. The bicarbonate is low, which indicates that a metabolic acidosis is present.

Step 3: Not applicable in this case.

Step 4: According to "Winter's formula" the expected PCO_2 in metabolic acidosis is [1.5 x HCO3] + 8 = [1.5 x 10] + 8 = 23 mm Hg. Since the actual and expected PCO2 are the same, this suggests that the respiratory compensation is appropriate.

Step 5: The anion gap = $[Na^+]$ - $[Cl^-]$ - [HCO3] = [132] - [83] - [10] = 39. The anion gap is obviously elevated. This means that the metabolic acidosis is of the elevated anion gap type.

Step 6: Remember that corrected HCO3 = measured HCO3 + (anion gap - 12). In this case the **c**orrected HCO3 = 10 + (39 – 12) = 37. The corrected HCO3 is much higher than a normal HCO3, suggesting there is a concurrent metabolic alkalosis, likely as a result of persistent vomiting.

Step 7: Not applicable in this case.

DIAGNOSIS: Elevated Anion Gap Metabolic Acidosis with concurrent Metabolic Alkalosis.

CLINICAL NOTE: Diabetic ketoacidosis (DKA) is a condition of insulin insufficiency associated with hyperglycemia, dehydration, and an acidosis-producing metabolic derangement (ketonemia). Infections, cessation of insulin treatment, and new onset of diabetes are frequently responsible. Urinary tract infections (UTIs) are the single most common responsible infection. DKA is usually characterized by hyperglycemia over 300 mg/dL, low bicarbonate (<15 mEq/L), and acidemia (pH <7.30) with urine testing showing ketonuria and glycosuria. Classic symptoms of hyperglycemia (thirst, polyuria, nocturia and polydipsia) are common. Labored respiration, with increased depth and rate of breathing, occurs because the patient is hyperventilating to lower the PCO2. This is known as "Kussmaul breathing", after Adolph Kussmaul, the 19th century German doctor who first noted it.

The treatment goals of the patient with DKA are: (1) aggressively correct hypovolemia and dehydration using 0.9% or 0.45% saline, (2) decrease the serum glucose and reverse ketonemia / acidemia with insulin, (3) correct electrolytes (esp. K^+), and (4) treat the underlying cause (such as a UTI or other infection). Therapy involves an initial intravenous (IV) bolus of 0.1 to 0.2 U/kg regular insulin followed by a continuous infusion of 0.1 U/kg/hour. Try to lower the serum glucose by 75-100 mg/dL/hour, monitoring glucose frequently.

Finally, note that potassium deficits can be large in DKA even with paradoxically high K^+ due to the acidemia, which shifts H^+ into cells and K^+ out of cells into blood.

CASE 6 – Man with a Flail Chest

A 22-year-old man was severely injured in the chest from a motor vehicle accident. A large flail rib segment in his thorax is compromising his breathing. A blood gas sample was taken, revealing the following:

pH	7.21
PCO_2	65
HCO3	25

Step 1: Acidemic, alkalemic, or normal?

The pH of the arterial blood gas identifies it as acidemic. (Recall that the "normal range" for arterial blood pH is 7.35 to 7.45).

Step 2: Is the primary disturbance respiratory or metabolic?

The primary disturbance is respiratory, with the PCO2 being significantly elevated. Since the bicarbonate is normal, there is no primary metabolic disturbance. (Recall that that the "normal range" for arterial PCO2 is 35- 45 mm Hg).

Step 3: For a respiratory disturbance, is it acute or chronic?

Recall that for acute respiratory disturbances (where renal compensation does not have much time to occur) each arterial PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.08, while for chronic respiratory disturbances (where renal compensation has time to occur) each PCO2 shift of 10 mm Hg is accompanied by a pH shift of 0.03.

In our case an arterial PCO2 shift of 25 mm Hg is accompanied by a pH shift of 0.19 units, or about a 0.08 pH shift for each PCO2 shift of 10 mm. This means that the respiratory disturbance is acute. (If it were chronic the pH shift would be 0.003 x 25 = 0.075, for a resulting pH of 7.4 – 0.12 = 7.33).

Steps 4-7: Not applicable in this case.

DIAGNOSIS: Acute Respiratory Acidosis from Hypoventilation Secondary to Flail Chest Injury

CLINICAL NOTE

Flail chest is the paradoxical movement of a segment of chest wall caused by anterior and posterior fractures of 3 or more ribs. It requires a significant force over a large area to create multiple anterior and posterior rib fractures, although if the ribs are weakened (as in osteoporosis or multiple myeloma), then a much lower force may suffice. The diagnosis is made on clinical grounds (inspection, palpation etc.) and confirmed by x-ray studies.

Of interest, respiratory insufficiency in flail chest patients is just as likely to result from underlying pulmonary contusion effects and ventilation perfusion mismatching than from the actual chest wall defect itself.

Effective analgesia is essential to allow the deep-breathing exercises (incentive spirometry) necessary to prevent respiratory deterioration from atelectasis and retained secretions. Patients whose pain cannot be managed with oral or IV / IM narcotics may benefit from local anesthetics delivered via a thoracic epidural catheter.

In some cases, patients require endotracheal intubation with positive pressure mechanical ventilation. Some of these patients also need surgical (operative) repair, especially when there is severe underling lung injury that makes ventilator weaning difficult.

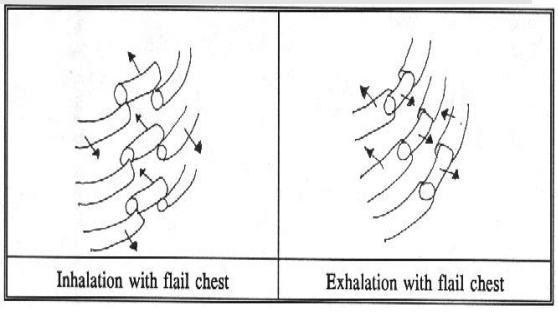


Figure: A flail chest segment moves paradoxically (in an opposite direction from the rest of the ribs) and impairs ventilation as a result. The result is hypoventilation.
Image Credit: tooldoc.wncc.nevada.edu /breath8.JPG

CASE 7 – Woman with a NSAID-Associated Nephropathy

A 39-year-old woman had severe chronic back pain, which she treated aggressively with a variety of OTC Non Steroidal Anti-Inflammatory Drugs (NSAIDs) for a number of years. At a routine clinical visit her blood pressure is found to be elevated at 155/95. Her urine dips 2+ positive for protein, and microscopic examination of her urine reveals 4-5 white blood cells per high-power field (4-5 WBC / hpf) with a specific gravity of 1.01 and a pH of 5.0. An arterial blood gas sample is as follows:

pH	7.30	
PCO2	32	mm Hg
HCO3	15	mEq/L
Na	138	mEq/L
K	5.1	mEq/L
Cl	111	mEq/L

Step 1: Acidemic, alkalemic, or normal?

The pH is 7.30, which is far less than normal (7.35-7.45), so the patient is acidemic.

Step 2: Is the primary disturbance respiratory or metabolic?

The PCO2 is low, so the respiratory system is not causing the acidosis; rather, the drop in PCO2 must be a compensatory process. The bicarbonate is low, which indicates that a metabolic acidosis is present.

Step 3: Not applicable in this case.

Step 4: According to "Winter's formula" the expected PCO_2 in metabolic acidosis is [1.5 x HCO3] + 8 = [1.5 x 15] + 8 = 31 mm Hg. Since the actual and expected PCO2 are approximately the same, this suggests that the respiratory compensation is appropriate.

Step 5: The anion gap = $[Na^+]$ - $[Cl^-]$ - [HCO3] = [138] - [111] - [15] = 12. The anion gap is not elevated. This means that the metabolic acidosis is of normal anion gap type.

Step 6: Not applicable in this case.

Step 7: Urinary electrolytes are needed to determine the urinary anion gap, but are unavailable here. On clinical grounds one would expect a renal rather than GI cause for her normal anion gap metabolic acidosis.

DIAGNOSIS: Nonanion gap metabolic acidosis (hyperchloremic metabolic acidosis) with appropriate respiratory compensation.

CLINICAL NOTE

This is a case of analgesic nephropathy resulting from chronic NSAID use. The kidneys have been damaged, as evidenced by proteinuria and the fact that they are unable to secrete a normal H+ load.

The renal toxicity of NSAIDs results from the blocking of prostaglandin formation, which can impair glomerular filtration, especially in hypovolemic patients. Specifically, NSAIDs inhibit the enzyme cyclooxygenase (COX), which converts arachidonic acid to a variety of prostaglandins, some of which dilate the renal microvasculature, an effect especially important in volume-depleted states, in which high circulating levels of norepinephrine and angiotensin II cause systemic and renal vasoconstriction.

Possible manifestations of NSAID associated nephropathy include acute interstitial nephritis, renal papillary necrosis, nephrotic syndrome, hypertension, and electrolyte disturbances.

The above notwithstanding, the anti-inflammatory, analgesic, and anti-pyretic properties of NSAIDs make them very useful in treating a great variety of musculoskeletal disorders, particularly when an inflammatory component is present. During the last few decades a plethora of NSAIDs have been introduced on the market, attesting to their value in the treatment of pain and inflammation.

Note that the effect of a particular NSAID can vary enormously between individuals, so two patients may experience very different benefits and side effects. The most common side effect associated with long term NSAID use is ulcers and gastro-intestinal bleeding.

CASE 8 – Man with Type I Renal Tubular Acidosis

A 49-year-old man with a history of kidney stones is being investigated for hypertension (150 / 95). His urine has a pH of 6.0 and the urinary anion gap is 10 mEq/l. An arterial blood gas sample provides the following:

pH	7.30	
PCO2	28	mm Hg
HCO3	14	mEq/L
Na	135	mEq/L
K	3.3	mEq/L
Cl	111	mEq/L

Step 1: Acidemic, alkalemic, or normal?

The pH is 7.30, which is far less than normal (7.35-7.45), so the patient is acidemic.

Step 2: Is the primary disturbance respiratory or metabolic?

The PCO2 is low, so the respiratory system is not causing the acidosis; rather, the drop in PCO2 must be a compensatory process. The bicarbonate is low, which indicates that a metabolic acidosis is present.

Step 3: Not applicable in this case.

Step 4: According to "Winter's formula" the expected PCO_2 in metabolic acidosis is [1.5 x HCO3] + 8 = [1.5 x 14] + 8 = 29 mm Hg. Since the actual and expected PCO2 are approximately the same, this suggests that the respiratory compensation is appropriate.

Step 5: The anion gap = $[Na^+]$ - $[Cl^-]$ - [HCO3] = [135] - [111] - [14] = 10. The anion gap is not elevated. This means that the metabolic acidosis is of normal anion gap type.

Step 6: Not applicable in this case, since the anion gap is not elevated.

Step 7: Since the urine anion gap (UAG) is positive in a setting of a normal anion metabolic acidosis, we should consider that the cause is likely to be renal rather than GI in origin.

Discussion

Now that we suspect a renal cause of this man's acidosis, it is helpful to consider some of the possible causes. The information below (from www.anaesthesiamcq.com) provides a synopsis of some of the key points in differentiating the types of renal tubular acidosis (RTA). Incomplete forms of RTA also occur. The arterial pH is normal here, and acidosis develops only when an acid load is present. Note also that "Type 3 RTA" is now considered to be a subtype of Type 1 where there is a proximal bicarbonate leak in addition to a distal acidification defect. The term Type 3 is thus no longer used. For more information consult the following article: *Rodriguez Soriano, J. Renal tubular acidosis: the clinical entity. J Am Soc Nephrol 2002; 13:2160.*

Comparison of Major Types of RTA

	Type 1 (distal)	Type 2 (proximal)	Type 4 (Hypoaldosteronism)
Hyperchloremic acidosis	Yes	Yes	Yes
Minimum Urine pH	>5.5	<5.5 once acidosis is established	<5.5
Plasma potassium	Low-normal	Low-normal	High
Renal stones	Yes	No	No
Defect	Reduced H^+ excretion in distal tubule	Impaired HCO3 reabsorption in proximal tubule	Impaired cation exchange in distal tubule

NOTE: The normal renal response to acidemia is to reabsorb bicarbonate and to increase hydrogen ion excretion through increased urinary excretion of ammonium ions. Each secreted hydrogen ion results in the regeneration of a bicarbonate ion in plasma. Renal tubular acidosis (RTA) refers to a defect in the ability of the renal tubules to perform these functions, resulting in the development of a normal anion gap metabolic acidosis with a positive urinary anion gap. Finally, note that some experts advocate measurement of urinary bicarbonate, which spills into the urine in the "proximal" form of RTA.

NOTES