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Andhra Hospitals

The peoples pulse

E journal of Paediatrics

Issue. 1

Vol3

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Foreword

Greetings from the Andhra Hospitals!

For the past two decades, we are delivering excellence in terms of quality health care in the field of Paediatrics including Paediatric and Neonatal intensive care, Paediatric cardiac intensive care and other allied Paediatric sub-specialities. We take pride in introducing ECMO in pediatric specialty for the first time in the state of Andhra Pradesh. In this context, we convey our heartfelt thanks to you for your continued support and encouragement which played a pivotal role in our success.

As a token of our commitment to contribute to continuous medical education, we are introducing this monthly E-journal to showcase important clinical guidelines, recent advances in paediatric sub-specialties, interesting case reports, image quiz, OSCE scenarios etc, gathered from our patient database. We hope this endeavor would prove to be useful to practicing paediatricians, intensivists, neonatologists and post-graduate students.

Please send your valuable feedback and suggestions to brevanth99@gmail.com

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Introduction

The most effective method for assessing the patient's gas exchange is by measuring the values of oxygen (O_2), carbon dioxide (CO_2) and pH of the arterial blood. An ABG analysis evaluates how effectively the lungs are delivering oxygen to the blood and how efficiently they are eliminating carbon dioxide from it. The test also indicates how well the lungs and kidneys are interacting to maintain normal blood pH (acid-base balance). Blood gas studies are usually done to assess respiratory disease and other conditions that may affect the lungs, and to manage patients receiving oxygen therapy. In addition, the acid-base component of the test provides information on renal function.

Defining terms

pH: A reflection of the blood hydrogen ion (H+) concentration. Normal pH =7.4 (Range 7.35 – 7.45). pH is calculated on a logarithmic scale.

PaO_2: partial pressure of oxygen dissolved in the blood

$PaCO_2$: partial pressure of dissolved CO_2 in the blood. Normal 35 to 45 mmHg.

Bicarbonate (HCO_3^-): Bicarbonate is regulated by the lungs (through CO_2 removal) and by the kidneys (through H^+ ion and HCO_3^- excretions or reabsorbtion). Normal HCO_3^- is 22 – 26 mEq/ml

Base deficit: a calculated number (by the gas machine) that represents the theoretical deficit (of a base) or excess (of an acid) present in the sample. A base deficit indicates metabolic acidosis. (< -2)

Base excess: A positive value represents the theoretical excess of a base and this indicates metabolic alkalosis. (> +2).

Normal base deficit/excess = -2 to +2

Acidosis: Too much acid in the blood – pH < 7.35

Alkalosis: Too much alkali (or base) in the blood – pH > 7.45

Hypoxaemia: Low level of oxygen in the blood, generally considered to be PaO_2< 75 mmHg when the patient is breathing room air (21% oxygen) at sea level.

Hypercarbia/hypercapnia: High level of CO_2 in the blood with a $PaCO_2$> 45 mmHg

The Method.

The blood gas results should be considered in parts. The result below shows what each part represents.

RESULT	PART	REPRESENTS
pH	Acid-base	pH scale – acidity to alkalinity
$PaCO_2$	Respiratory indicator	Potential cause of the pH problem.
HCO_3^-std	Metabolic indicator	Potential cause of the pH problem.

The pH result presents us with a potential acid-base problem. The $PaCO_2$ and the HCO_3^- present us with possible causes for the problem. The first thing to do is to identify the problem and then determine what has caused it.

Step 1 : Identify the source.

The first thing to do is to identify whether the blood sample has been obtained from a capillary, a venous (central line) or an arterial source. It will affect the interpretation and applicability of the blood gas results.

1. Arterial samples provide the most accurate results.
2. Significant changes occur to the oxygencontent of the blood (PaO_2) as it passes from the artery through the capillary bed.
    - A wide range of oxygen values is possible when the source comes from a capillary stab.
    - The degree of squeezing that went into providing sufficient capillary blood for the sample also affects the oxygen result.
    - Venous blood is usually about 70% saturated and has a PaO_2 of about 40 mmHg but the value does not correlate well with arterial oxygen content
3. **The pH of venous or capillary blood is usually about 0.033 lower than in arterial blood.** It has slightly greater acidity. This difference is not big enough to make a difference to decisions on patient care.

4. **The carbon dioxide level ($PaCO_2$) of capillary or venous blood is usually about 4.5 mmHg higher than in arterial blood**. This difference is big enough to make a difference to blood gas interpretation. The effect of this difference on the $PaCO_2$ interpretation will be discussed in the relevant section.

Step 2: Identify the pH part of the gas

Normal limits	
pH	7.35 – 7.45

- How severe is the pH problem?
- It is vital that the blood pH is maintained within this narrow range. A pH of 7.2 or as high as 7.6 is tolerable but still needs correction in most cases. Cell destruction commences outside these ranges and patients will die if their pH falls and remains below this level. Once the severity of the pH problem is established, two categories of severity have been suggested. The first is 'severe' and the second is 'life-threatening'.

When assessing the pH, three questions need to be asked.

1. Is the pH within normal limits?
2. If the pH is not within normal limits, is it showing an acidosis or an alkalosis?
3. If there is an alkalosis or an acidosis, how severe is the problem?

If the pH is not within normal limits, is it showing an acidosis or an alkalosis?

The term acidotic is used for any blood pH less than 7.35

- The term alkalotic is used for any pH value greater than 7.45.

You should now be able to identify whether the pH is within normal limits and whether any pH problem is 'acidosis' or 'alkalosis'. If you don't feel able to do this, read back over the previous section.

1. "Severe" is used for
    - any acidosis, where the pH is less than 7.2 and greater than 7.0

- any alkalosis where the pH is greater than 7.6 and less than 7.8.

2. "Life-threatening"is used for
    - any acidosis, where the pH is less than 7.0
    - any alkalosis where the pH is greater than 7.8.

If the pH result shows a "severe" problem, action needs to be taken promptly. If the pH result shows a "life-threatening" problem, action needs to be taken urgently to try to prevent an arrest.

The rest of the gas result will show the cause of the acidosis or alkalosis. Once the cause has been identified, appropriate corrective action can be taken below 7.0 or above 7.8.

Step 3 : Assess the respiratory part of the gas : the $PaCO_2$

The respiratory part of the blood gas comprises two results:

- The carbon dioxide level,
    - $PaCO_2$ (partial pressure of carbon dioxide)
- The oxygen level,
    - PaO_2 (partial pressure of oxygen).

There are two questions that need to be asked about the carbon dioxide level for blood gas interpretation.

1. Is the carbon dioxide level ($PaCO_2$) within normal limits?
2. Would this result cause acidosis or alkalosis?

CO_2 reacts with water in the body to form carbonic acid, which then breaks down to form bicarbonate and hydrogen ions. The higher the level of CO_2, the more acidic the patient will become

$H_2O + CO_2 \rightleftarrows H_2CO_3$ (carbonic acid) $\rightleftarrows HCO_3^- + H^+$

Is the carbon dioxide level ($PaCO_2$) within normal limits?

Normal arterial limits	**Normal capillary/venous limits**

35 – 45 mmHg	**40 – 50 mmHg**

If the sample is from a central line (venous) or capillary source, the pCO_2 is normally 4.5mmHg higher than the arterial sample

Would the $PaCo_2$ result cause acidosis or alkalosis?

$PaCO_2$	**pH**	**Result**
High	Acidosis.	Respiratory acidosis.
Low	Alkalosis.	Respiratory alkalosis.

A high carbon dioxide level means greater than average concentrations of carbonic acid in the blood.

- If the pH of the blood is acidotic and the carbon dioxide level is high, the carbonic acid would be the cause of the problem. This would be a 'respiratory acidosis'.
- Hypo-ventilation problems are the cause of 'respiratory' acidosis. The patient's ventilator settings will need adjusting. If the patient is not ventilated, he/she may need intubation and ventilation.
- Respiratory acidosis is the most common problem seen on general PICU.

A low carbon dioxide level indicates less than the usual amount of carbonic acid circulating.

- If the pH of the blood is alkalotic and the carbon dioxide is low, insufficient carbon dioxide would be the cause. This would be a 'respiratory alkalosis'.
- Hyper-ventilation is the cause of 'respiratory' alkalosis. The patient's ventilator settings will need adjusting. Hyperventilation is rare in non-intubated patients, but can occur as a response to stress and anxiety.

If the $PaCO_2$ is normal, then we need to look for some other cause of the deranged pH.

Even if the $PaCO_2$ value has not caused the pH problem, the identification of a deranged PaCO2 made by the physician will be helpful when more complex situations need to be interpreted (Mixed causes and compensations.)

Step 4 : Assessing the metabolic part of the gas : the Standard Bicarbonate.

The standard bicarbonate (HCO_3^-std) and the base excess represent the 'metabolic' part of the gas result.

Bicarbonate is an important buffer which mops up carbonic acid from respiratory acidosis (i.e. it attempts to neutralise the acid). There are two bicarbonate results recorded on the blood gas – standard and actual. Standard bicarbonate means the bicarbonate level when it is measured under standard conditions, so only the effect of metabolic acids will be detected. Therefore, this is the one we record and interpret. In the blood gas machine, standard conditions for this measurement are:

PaCO2 -40 mmHg Temp- 37°C

The above conditions mimic normal healthy respiratory status, with a pCO_2 of 40 mmHg and normal patient body temperature.

When considering the HCO_3^-std, two questions need to be asked.

1. ***Is the standard bicarbonate level (HCO_3^-std) within normal limits?***
2. ***Would this result cause acidosis or alkalosis?***

Is the standard bicarbonate level (HCO_3^-std) within normal limits?

Normal limits.	
Standard Bicarbonate - HCO_3^-std	22-26 mmol/L

Would this result cause acidosis or alkalosis…….?

If there is an excess of bicarbonate, there will not be much acid. It would cause an alkalosis: metabolic alkalosis. If there is a deficiency of bicarbonate, there is more acid than the bicarbonate can deal with. It would cause an acidosis: metabolic acidosis.

HCO_3^-std	pH	Result
Low	Acidosis	Metabolic acidosis.
High	Alkalosis	Metabolic alkalosis.

The following table shows all the blood gas interpretations which you have learnt to make so far.

Step 5 : The Base Excess or Base Deficit.

Normal limits.	
Base excess	-2 to +2 mmol/L

It expresses the difference between the **patient's** standard bicarbonate level and the **normal** standard bicarbonate (24 mmol/L).

- If the HCO_3^-std is 30 mmol/L, that is 6 mmol/L more than 24 mmol/L. The patient has a base excess of +6 mmol/L.
- If the HCO_3^-std is 20 mmol/L, that is 4 mmol/L less than 24 mmol/L. The patient has a base excess of -4 mmol/L or a base deficit of 4 mmol/L.

A base excess between……

- -2 mmol/L (representing a HCO_3^-std of 22 mmol/L) and
- +2 mmol/L (representing a HCO_3^-std of 26 mmol/L)

…….is considered to be within the normal range.

Step 6 : Check for compensation.

What is compensation?

In the earlier section on pH, categories of pH result were established. The categories were 'life-threatening' or 'severe'. In the same way that nurses and medical staff would wish to correct these problems promptly, so also does the body. It tries to establish a normal pH again. This is

compensation. (N.B. before compensation mechanisms take place gases are considered uncompensated)

There are four ways the body can do this:

1. If the problem is acidosis, it can:
    - get rid of more carbon dioxide (take deeper breaths or breathe faster)
    - retain/produce more bicarbonate to neutralise the acid (via the renal system)
2. If the problem is alkalosis, the body can:
    - Retain more carbon dioxide (take shallower breaths or breathe out less often).
    - Lose more bicarbonate (via the renal system).
    - (Remember that alkalosis is a much less common problem than acidosis, so you will not see many alkalotic problems on the ICU.)

Obviously if one of the respiratory or renal systems is not functioning properly because of illness or injury, the body will be more dependent on the other method for excreting waste acids.

There are two types of compensation.

1. Partially compensated, where the pH, pCO_2 and HCO_3^- are all abnormal but either the $PaCO_2$ or Bicarbonate are trying to compensate (normalise) the pH
2. Compensated – where the pH is within normal limits, but the pCO_2 and HCO_3^-std are abnormal

	Gas Result	**Notes.**
pH	7.29	*Acidotic*
$PaCO_2$	52 mmHg	*High $PaCO_2$. Would cause acidosis*
HCO_3^-std	27 mmol/L	*High HCO_3^- std. Would cause alkalosis*
Base excess	+3 mmol/L	

Consider the example gas again.

1. A respiratory acidosis can be identified.

2. There are, however, three abnormal results and the $PaCO_2$ and HCO_3^-std would cause different problems.
    - this is a 'partially compensated respiratory acidosis'.

As in the previous example, what would you write for the gas below?

	Gas Result	Notes
pH	7.36	
$PaCO_2$	62 mmHg	
HCO_3^-std	28 mmol/L	
Base excess	+4 mmol/L	

This gas shows compensated respiratory acidosis

The pH is normal, so on PICU we would refer to the blood gas as normal. However, in compensated problems, there has been an acidosis or alkalosis to which the body has reacted to achieve a pH of 7.35 – 7.45.

How can you tell which problem came first?

- Did the acidosis come first and then the body compensated to bring the pH back to normal?
- Did the alkalosis come first and then the body compensated to bring the pH back to normal.

The key to working out which problem came first, is to re-examine the pH value. The body never overcompensates.

- If a fully compensated 'acidosis' exists, then the pH will be normal but less than 7.4.
- If a fully compensated 'alkalosis' exists then the pH will be normal but greater than 7.4.

	Gas Result.	Notes.

pH	7.36	*normal*
$PaCO_2$	62 mmHg	*High pCO_2. Would cause acidosis.*
HCO_3^-std	28 mmol/L	*High HCO_3^- std. Would cause alkalosis.*
Base excess	+4 mmol/L	

Look at this blood gas which you have just worked on again and follow through the reasoning.

- The pH is normal.
- The $PaCO_2$ would cause acidosis.
- The HCO_3^-std would cause alkalosis.
- The acidosis came first because the pH is less than 7.4.
- This is a compensated respiratory acidosis.

Acid Base disturbance (Respiratory)	**PH**	**$PaCO_2$**	**Bicarb HCO_3^-**
Acute Respiratory Acidosis (uncompensated)	< 7.35 ↓	↑>45	Normal
Respiratory Acidosis (Partial compensation)	< 7.35 ↓	↑>45	↑>26
Chronic Respiratory Acidosis (compensated)	Normal	↑	↑
Acute Respiratory Alkalosis (uncompensated)	↑	↓	Normal
Respiratory Alkalosis (Partial compensation)	↑	↓	↓
Chronic Respiratory Alkalosis (compensated)	Normal	↓	↓

Acid Base disturbance (Metabolic)	**PH**	**$PaCO_2$**	**Bicarb HCO_3^-**
Acute Metabolic Acidosis (uncompensated)	< 7.35 ↓	Normal	↓<22
Metabolic Acidosis (Partial compensation)	< 7.35 ↓	↓<35	↓<22
Chronic Metabolic Acidosis (compensated)	Normal	↓<35	↓<22
Acute Metabolic Alkalosis (uncompensated)	↑	Normal	↑>26
Metabolic Alkalosis (Partial compensation)	↑	↑>45	↑>26
Chronic Metabolic Alkalosis (compensated)	Normal	↑>45	↑>26

To look for mixed problems:

Mixed acid–base disorders are diagnosed when the secondary response differs from that which would be expected.

Table 1. Primary Acid–Base Disturbances with a Secondary ("Compensatory") Response.*

Metabolic acidosis
pH <7.38 and bicarbonate [HCO_3^-] <22 mmol per liter
Secondary (respiratory) response: $Paco_2 = 1.5 \times [HCO_3^-] + 8 \pm 2$ mm Hg† or $[HCO_3^-] + 15$ mm Hg‡
Complete secondary adaptive response within 12–24 hr
Superimposed respiratory acidosis or alkalosis may be diagnosed if the calculated $Paco_2$ is greater or less than predicted
Metabolic alkalosis
pH >7.42 and [HCO_3^-] >26 mmol per liter
Secondary (respiratory) response: $Paco_2 = 0.7 \times ([HCO_3^-] - 24) + 40 \pm 2$ mm Hg or $[HCO_3^-] + 15$ mm Hg‡ or $0.7 \times [HCO_3^-] + 20$ mm Hg§
Complete secondary adaptive response within 24–36 hr
Superimposed respiratory acidosis or alkalosis may be diagnosed if the calculated $Paco_2$ is greater or less than predicted
Respiratory acidosis
pH <7.38 and $Paco_2$ >42 mm Hg
Secondary (metabolic) response
Acute: [HCO_3^-] is increased by 1 mmol/liter for each $Paco_2$ increase of 10 mm Hg above 40 mm Hg
Chronic: generally [HCO_3^-] is increased by 4–5 mmol/liter for each $Paco_2$ increase of 10 mm Hg above 40 mm Hg
Complete secondary adaptive response within 2–5 days
Superimposed metabolic alkalosis or acidosis may be diagnosed if the calculated [HCO_3^-] is greater or less than predicted
Respiratory alkalosis
pH >7.42 and $Paco_2$ <38 mm Hg
Secondary (metabolic) response
Acute: [HCO_3^-] is decreased by 2 mmol/liter for each $Paco_2$ decrease of 10 mm Hg below 40 mm Hg
Chronic: [HCO_3^-] is decreased by 4–5 mmol/liter for each $Paco_2$ decrease of 10 mm Hg below 40 mm Hg
Complete secondary adaptive response in 2–5 days
Superimposed metabolic alkalosis or acidosis may be diagnosed if the calculated [HCO_3^-] is greater or less than predicted

Evaluation of the Metabolic Component of an Acid–Base Disorder:

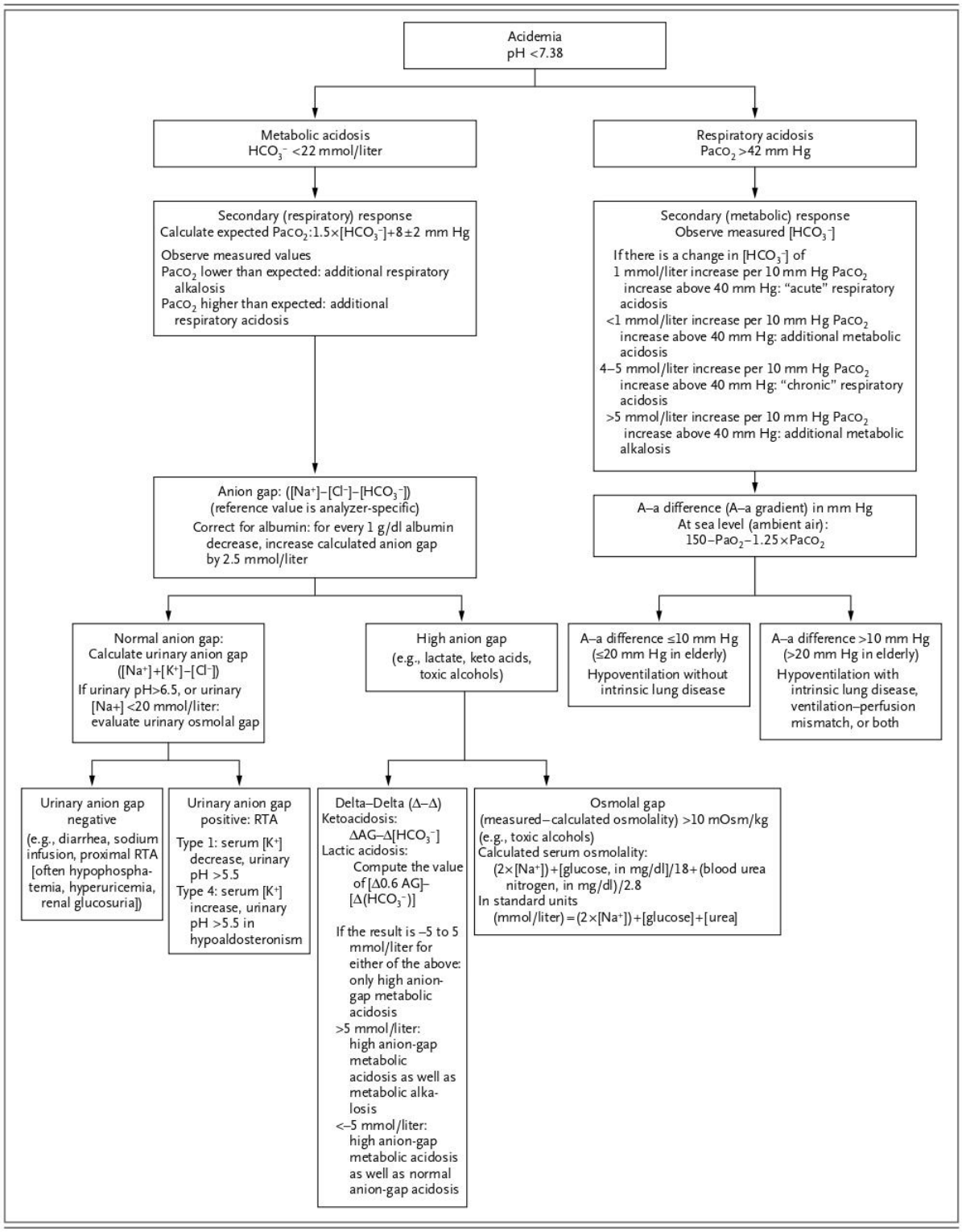

Table 2. The Anion Gap in Relation to Common Medical Conditions with Metabolic Acidosis.*

High anion gap
Overproduction of acid
Ketoacidosis (diabetic ketoacidosis, alcoholic ketoacidosis, starvation)
Lactic acidosis
L-Lactic acidosis
Type A — hypoxic (septic shock, mesenteric ischemia, hypoxemia, hypovolemic shock, carbon monoxide poisoning, cyanide)
Type B — nonhypoxic (thiamine deficiency, seizure, medications [nonnucleoside reverse-transcriptase inhibitors, metformin, propofol, niacin, isoniazid, iron], intoxication [salicylate, ethylene glycol, propylene glycol, methanol, toluene ingestion (early), paraldehyde])
D-Lactic acidosis in the short-bowel syndrome
Underexcretion of acid (advanced renal failure)†
Impaired lactate clearance in liver failure (also type B acidosis)
Cell lysis (massive rhabdomyolysis)
Use of penicillin-derived antibiotics
Pyroglutamic acid (5-oxoproline)[32]
Normal anion gap
Loss of bicarbonate
Gastrointestinal conditions (diarrhea, ureteral diversions, biliary or pancreatic fistulas)
Renal conditions (type 2 [proximal] renal tubular acidosis, toluene ingestion [late in the process of toluene intoxication], conditions associated with medications [ifosfamide, tenofovir, topiramate, carbonic anhydrase inhibitors such as acetazolamide])[3,41]
Decreased renal acid excretion
Early uremic acidosis
Type 1 renal tubular acidosis (e.g., due to amphotericin, lithium, Sjögren's syndrome)[3]
Type 4 renal tubular acidosis (hypoaldosteronism or pseudohypoaldosteronism)
Other causes: fluid resuscitation with saline, hyperalimentation (lysine, histidine, or arginine hydrochloride), administration of hydrochloride, ammonium chloride, cholestyramine, hippuric acid, sulfuric acid

* An anion gap of more than 10 mmol per liter above the upper limit of the reference value is highly suggestive of organic acidosis. A minor increase in the anion gap is less helpful in diagnosing metabolic acidosis.
† Advanced renal failure is indicated by a glomerular filtration rate below 20 ml per minute.

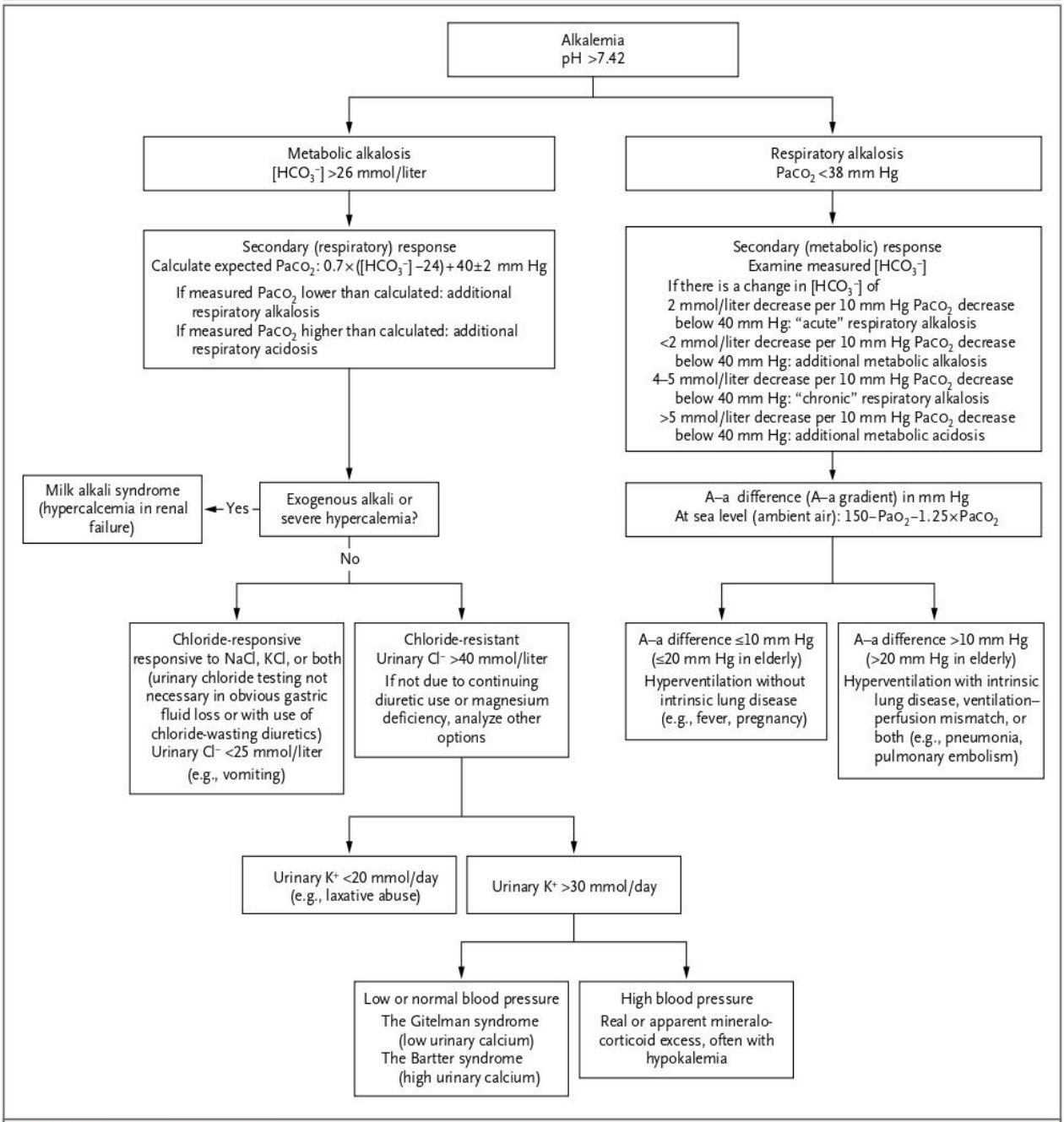

Figure 2. Assessment of Alkalosis.

Reference values for the alveolar–arterial (A–a) oxygen tension difference are less than 10 mm Hg in young persons and less than 20 mm Hg in the elderly. $Paco_2$ denotes partial pressure of arterial carbon dioxide (mm Hg), and Pao_2 partial pressure of arterial oxygen (mm Hg). To convert the values for $Paco_2$, Pao_2, and the alveolar–arterial difference to kilopascals, multiply by 0.1333.

Evaluation of the Respiratory Component of an Acid–Base Disorder:

Table 3. Common Medical Conditions Characterized by Respiratory Acidosis and Alkalosis.*

Type of Acidosis	Common Medical Conditions
Respiratory acidosis	
Acute	
Normal alveolar–arterial O_2 difference	Depression of the central respiratory center by cerebral disease (encephalitis or trauma) or drugs (narcotics, barbiturates, or benzodiazepines)
High alveolar–arterial O_2 difference†	Airway obstruction related to acute exacerbations of asthma or pneumonia
Chronic	
Normal alveolar–arterial O_2 difference	Neuromuscular disease (e.g., myasthenia gravis, amyotrophic lateral sclerosis, Guillain–Barré syndrome, or muscular dystrophy), kyphoscoliosis
High alveolar–arterial O_2 difference†	Chronic obstructive pulmonary disease
Respiratory alkalosis	
Acute	
Normal alveolar–arterial O_2 difference	Pain, anxiety, fever, stroke, meningitis, trauma, severe anemia, salicylate toxicity
High alveolar–arterial O_2 difference†	Pneumonia, pulmonary edema, pulmonary embolism, aspiration, congestive heart failure, sepsis
Chronic	
Normal alveolar–arterial O_2 difference	Pregnancy, hyperthyroidism, hepatic failure
High alveolar–arterial O_2 difference†	Pulmonary embolism in pregnancy, liver failure with aspiration pneumonia

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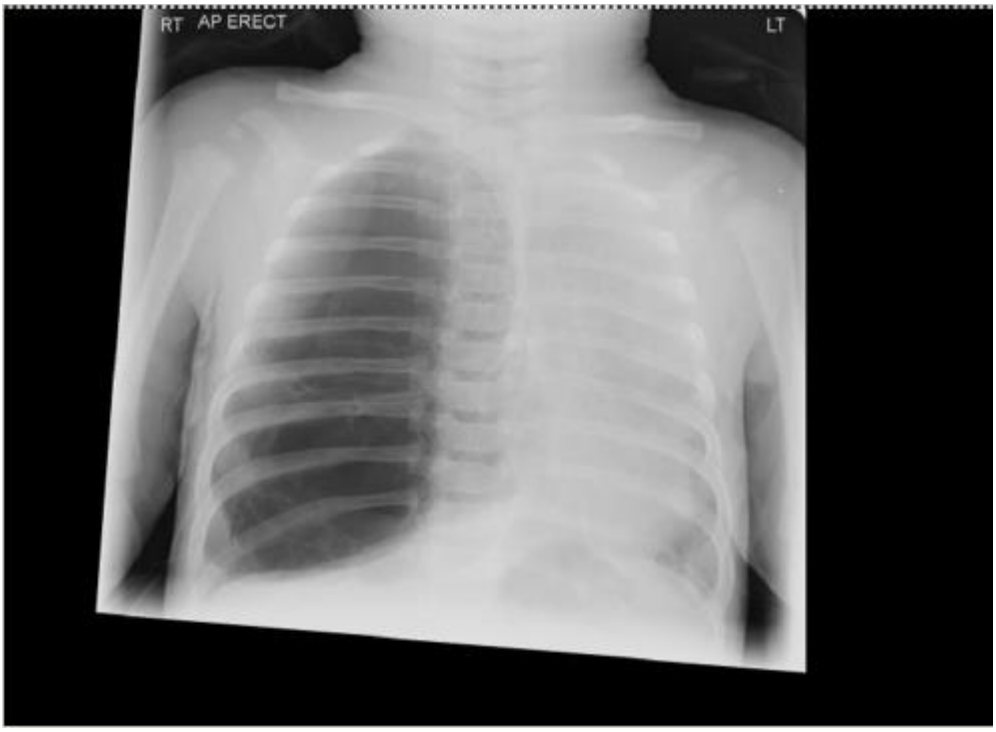
A 3 month old baby was admitted to the paediatric unit with a history of poor weight gain and tachypnoea. She was born at 39+1 weeks gestation by normal vaginal delivery following an unremarkable pregnancy. Her birth weight plotted on the 75th centile, but had gradually drifted down to the 9th centile where it plotted on the day of admission. Her mother had concerns that her work of breathing had increased over the previous few days, she struggled to feed and was only able to take 40 mls of milk per feed. She had not been coryzal or pyrexial. On examination she was pink and well perfused with normal heart sounds and normal femoral pulses. There was moderate sub-costal and intercostal recession and head bobbing. Auscultation of the chest revealed reduced air entry on the right with no added sounds. Heart sounds were normal. Her abdomen was soft and a 1e2 cm liver edge was palpable.A chest X-ray was performed and is shown below



1. What is the most likely cause for the chest X-ray appearances?

The patient remained tachypnoeic with a respiratory rate of 60 breaths per minute. Her heart rate was 146 beats per minute, blood pressure 112/72, temperature 36.6 C and Saturations 97% on air. Her CXR was discussed with the radiology team and further investigations arranged

2. What further investigations would you like to perform?

3. What complications would you expect from this condition if it was left untreated?

Answers:

1. Right sided Congenital pulmonary airway malformation (CPAM)
2. ECHO, Renal Ultrasound, HRCT Chest
3. Recurrent pneumonia ,pneumothorax and Malignant transformation

Congenital pulmonary airway malformation (CPAM), formerly known as congenital cystic adenomatoid malformation (CCAM) is a congenital anomaly of the lung which results in the development of abnormal cystic pulmonary tissue, usually in one lobe of the lung. This cystic tissue is unable to function in the same way as normal lung tissue. As the malformation increases in size, it may compress surrounding structures. Definitive treatment involves surgical resection of the abnormal tissue and usually involves a lobectomy. Smaller lesions may be managed conservatively. CPAM is often seen and diagnosed on antenatal ultrasound scans. If not seen on antenatal ultrasound scans, infants may present with signs of respiratory distress or pneumonia. CT chest imaging assesses the extent of the malformation and identifies the morphology of microcystic or macrocystic appearances. High resolution CT (HRCT) chest imaging is usually performed. Renal ultrasound and echocardiography should also be performed to exclude any other congenital malformations. The abnormally functioning lung tissue leads to an increased susceptibility of recurrent pneumonia. This may be a presenting feature in a child not previously known to have CPAM. Air trapping within the cystic tissue can also lead to a spontaneous pneumothorax and should be considered in a patient with conservatively managed CPAM presenting with shortness of breath and chest pain. There is a risk of malignant transformation of the abnormal lung tissue in later childhood or adulthood. Although the risk of this is small, surgical intervention is often preferred to conservative management for this reason.

PROCEDURE PAGE

Bone Marrow Aspiration and Biopsy

Bone marrow examination provides crucial information in the diagnosis of various hematologic and oncologic conditions inchildren. Bone marrow aspiration also permits immune-phenotyping, cytogenetic analysis, and other molecular studies.

Indications:

- Pancytopenia
- Unexplained anemia, leukopenia, or thrombocytopenia (aspiration only).
- Acute or chronic leukemia (aspiration only).
- Myelodysplasia, Myeloproliferative disease.
- Non-Hodgkin or Hodgkin lymphoma.
- Childhood solid tumors (including sarcoma, Wilms tumor, neuroblastoma, germ cell tumor).
- Bone marrow failure (including acquired aplastic anemia, Fanconi anemia, Diamond-Blackfan syndrome).
- Fever of unknown origin.
- Storage disease
- Monitoring during chemotherapy or following stem cell transplantation (aspiration only).

Equipment

For Site Preparation	For Marrow Aspiration and Biopsy
10% povidone-iodine. Alcohol swabs. Sterile gloves, gown, and drape. Spinal and subcutaneous needles, 20 to 26 gauge. 1% lidocaine hydrochloride, injection. 8.4% sodium bicarbonate, injection, USP.	Sodium heparin injection Bone marrow aspiration needles (15 and 18 gauge). Bone marrow biopsy needles (11 13 gauge, 4/2 inches) Sterile syringes, 10 to 20 mL. Container with fixative for trephine biopsy specimen. sodium heparin and EDTA collecting tubes Gauze and Bandages

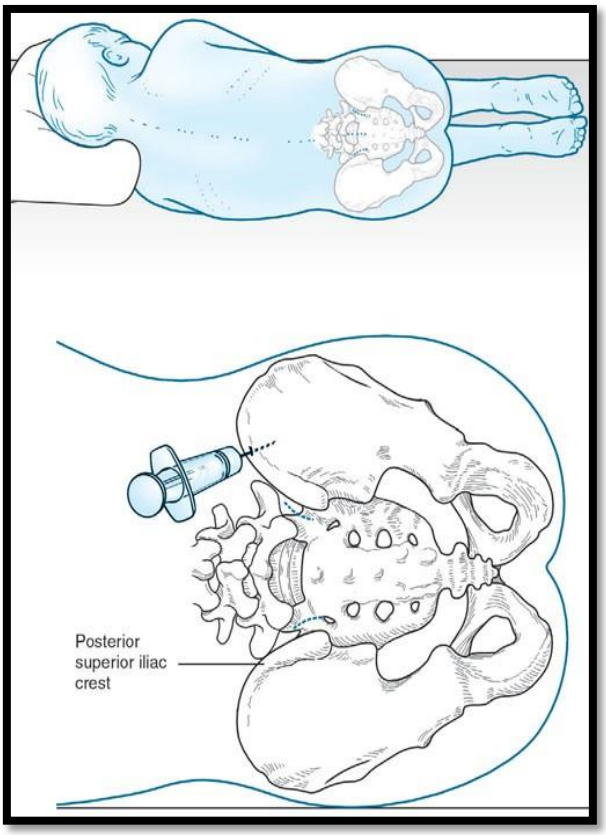
Patient preparation & Position

1. Obtain a thorough medical history and perform physical examination.

2. Obtain written informedconsent.

3. Inform patients who are not receiving general anesthesia that an unpleasant, lightening-like sensation down their lowerextremitiesmaybefeltatthetimeofsu ctionduring aspiration.

4. The posterior superior iliac crest is the optimal location for bone marrow aspiration and biopsy in most children (Fig.1)



5. The posterior superior iliac crest can usually be identified by a dimple in the skin located at the lateral edgeof Michaelis'rhomboid.

6. Michaelis' rhomboid is a diamond-shaped area over the posterior aspect of the pelvis formed by the posterior superior iliac spines, the gluteal muscles, and the groove at the distal end of the vertebralcolumn. Thisareacanbelocatedin

mostpatientsbypalpationwith the thumb, even if anatomic landmarks are notvisible

7. Alternative sites include the anterior iliac crest in obese patients and the tibia in infants younger than 3months.

8.The sternum, which is used in some adults, should be avoided inchildren.

9. If the posterior iliac crest is used, the patient is placed in the right or left decubitus position, with the hips flexed and the knees drawnup.

10. If the anterior iliac crest is used, the patient is placed in the supine position with the hips and kneesflexed.

11. Occasionally, thin patients who do not receive general anesthesia may be placed in the proneposition

Procedure

Bone Marrow Aspiration

1. Clean the site with povidone-iodine followed by alcohol swab.
2. Place sterile drape.
3. Inject lidocaineintradermally with a subcutaneous needle to produce a small wheal.
4. Use a larger bore needle to push through the skin and subcutaneous tissue and inject 2–3 mL (maximum 3 mg/kg/ dose) more lidocaine along the periosteum.
5. Hold the bone aspirate needle horizontally using the index finger near the tip of the needle for control.
6. Advance the needle through the skin, subcutaneous tissue, and the surface of the cortical bone with steady pressure and a twisting motion.An abrupt decrease in resistance occurs when the needle penetrates the cortex and enters the spongy marrow cavity.
7. Advance the needle 1 cm more before the stylet is removed.
8. Attach a 10-mL or 20-mL syringe to the end of the needle and pull the plunger back quickly to aspirate approximately 0.25 mL of bone marrow.
9. If an aspirate is not obtained, replace the stylet and advance or reposition the needle.
10. This first pull contains the marrow particles or spicules that should be used for preparing initial smears.
11. A heparinized, larger syringe (30 mL) may be used to obtain additional marrow for cytogenetic analysis, flow cytometry, and other studies

Bone marrow Biopsy

1. The trephine biopsy is the preferred method to evaluate cellularity and detect bone marrow metastasis in lymphoma and many childhood solid tumors.
2. Biopsy specimen is obtained through the same incision site.
3. Hold the biopsy needle in the same manner as the aspiration needle but angle it to sample a different area from the aspiration. Advance the needle with steady pressure to the periosteum and twist into the surface of the cortical bone.
4. Remove the obturator and push the needle through the cortex using a rotating, twisting motion

until decreased resistance is met. Advance the needle another 1–2 cm.

5. Reinsert the obturator until resistance is met to gauge the length of the specimen.

Rotate the needle 360 degrees vigorously several times while moving it back and forth vertically and horizontally to break the biopsy core off the surrounding bone.

6. Carefully remove the needle and insert a separate blunt obturator into the distal end of the needle to force the core out through the hub onto a glass slide.
7. Specimen should be at least 1.5 to 2 cm in length for optimal processing.
8. If the specimen is inadequate or consists mostly of cartilage or cortical bone rather than core marrow, which appears dark red with a fine, white trabecular network, attempt additional biopsies.
9. The specimen should be placed in an appropriate fixative.
10. Apply direct pressure to the site for at least 5 minutes once the procedure is

completed and the needle removed and place a pressure dressing.

Risks and complications

1. Risk of bleeding is low if adequate pressure is provided over site to achieve primary hemostasis.
2. Defects in coagulation should be corrected before the procedure.Platelet transfusion is indicated when technical difficulties are anticipated in patients, especially those who are obese, with severe thrombocytopenia.
3. Risk of infection and osteomyelitis is extremely low when procedure is performed in sterile fashion.4. Pain and discomfort are alleviated with adequate sedation and analgesics.
5. Bleeding at any site, with or without development of a hematoma, is rare if adequate pressure is applied.
6. Retroperitoneal hemorrhage, osteomyelitis, and needle breakage have also been rarely described.

Practical Points

1 Adolescents may require only local anesthesia for the procedure.

2. Conscious sedation or general anesthesia is generally necessary in young children, particularly if repeated procedures are required.

3. Adding local anesthesia in young patients also decreases postprocedural discomfort at the site.

4. Lidocaine used for local anesthesia should be buffered with sodium bicarbonate (sodium bicarbonate mixed withlidocaine in a 1:4 ratio) to reduce burning during injection.

5. Obtaining spicules (bone marrow particles rich in hematopoietic elements) on the first pull of the aspiration may be easier using a larger syringe (30 or 60 mL).

6. Aspirating more than 0.25 mL of marrow initially dilutes the sample with sinusoidal blood and interferes with morphologic studies.

7. If an aspirate is "dry" and an adequate specimen cannot be obtained, a touch imprint of the biopsy core may be helpful for cytologic examination.

8. A dry tap usually indicates myelofibrosis or a marrow cavity packed with malignant cells.

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