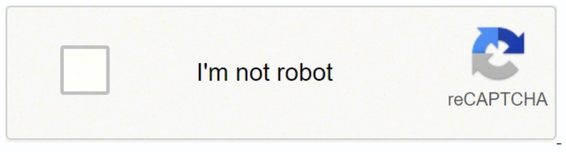

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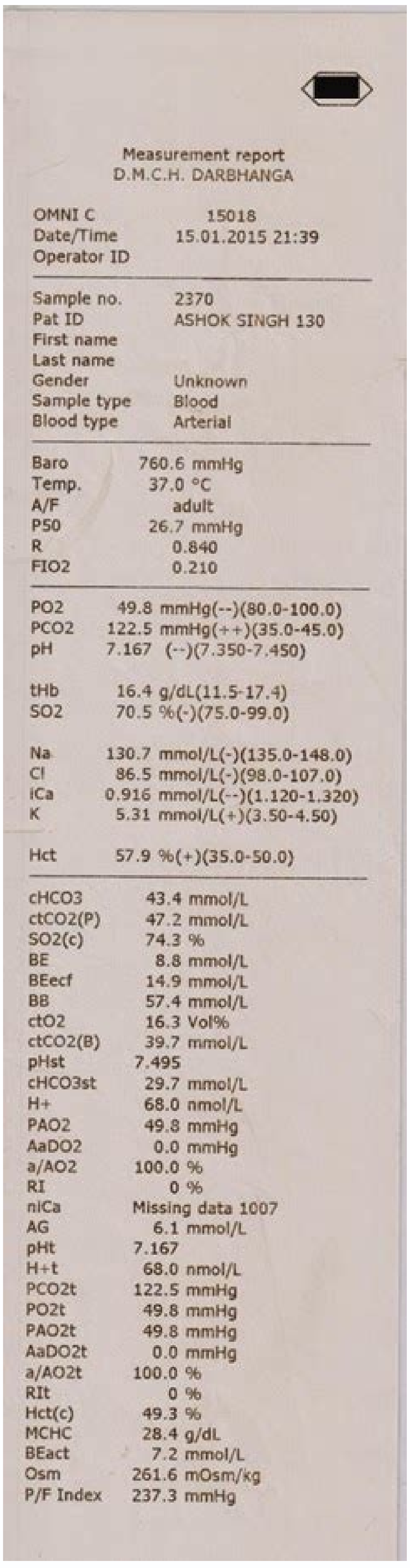
Abg report interpretation



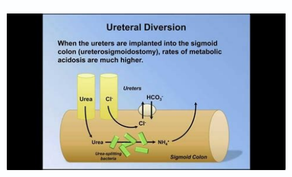
Measurement report
D.M.C.H. DARBHANGA

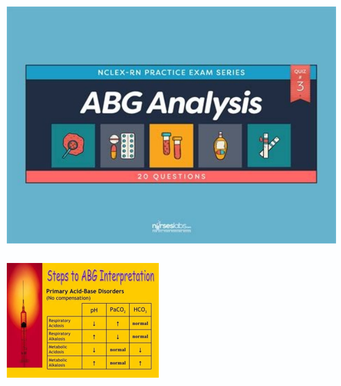
OMNI C	15018
Date/Time	15.01.2015 21:39
Operator ID	
Sample no.	2370
Pat ID	ASHOK SINGH 130
First name	
Last name	
Gender	Unknown
Sample type	Blood
Blood type	Arterial
Baro	760.6 mmHg
Temp.	37.0 °C
A/F	adult
P50	26.7 mmHg
R	0.840
FIO2	0.210
PO2	49.8 mmHg(--)(80.0-100.0)
PCO2	122.5 mmHg(++)(35.0-45.0)
pH	7.167 (--)(7.350-7.450)
tHb	16.4 g/dL(11.5-17.4)
SO2	70.5 %(-)(75.0-99.0)
Na	130.7 mmol/L(-)(135.0-148.0)
Cl	86.5 mmol/L(-)(98.0-107.0)
iCa	0.916 mmol/L(--)(1.120-1.320)
K	5.31 mmol/L(+)(3.50-4.50)
Hct	57.9 %(+)(35.0-50.0)
cHCO3	43.4 mmol/L
ctCO2(P)	47.2 mmol/L
SO2(c)	74.3 %
BE	8.8 mmol/L
BEecf	14.9 mmol/L
BB	57.4 mmol/L
ctO2	16.3 Vol%
ctCO2(B)	39.7 mmol/L
pHst	7.495
cHCO3st	29.7 mmol/L
H+	68.0 nmol/L
PAO2	49.8 mmHg
AaDO2	0.0 mmHg
a/AO2	100.0 %
RI	0 %
nICa	Missing data 1007
AG	6.1 mmol/L
pHt	7.167
H+t	68.0 nmol/L
PCO2t	122.5 mmHg
PO2t	49.8 mmHg
PAO2t	49.8 mmHg
AaDO2t	0.0 mmHg
a/AO2t	100.0 %
RIt	0 %
Hct(c)	49.3 %
MCHC	28.4 g/dL
BEact	7.2 mmol/L
Osm	261.6 mOsm/kg
P/F Index	237.3 mmHg

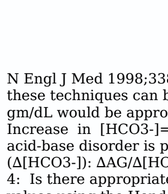
Measurement report

16.02.17 09:10
Serial number : 4500
Instrument ID : QF4500
Operator ID : DR.ABG
CCU Local District Hospital

Pat. ID	047328	
Patient Name	Mr. White	
Date of birth	03.05.72	
Remark	Room Air	
FIO_2	0.21	
Temperature	37.0 °C	
pH	7.390	[7.350 – 7.450]
pCO_2	4.99 kPa	[4.67 – 6.00]
pO_2	12.25 kPa	[10.67 – 13.33]
$cHCO_3^-$	24.0 mmol/L	[22 – 26]
BE	0.7 mmol/L	[-2.0 – +2.0]
Na^+	140.0 mmol/L	[135.0 – 148.0]
K^+	3.90 mmol/L	[3.50 – 4.50]
Cl^-	100.0 mmol/L	[98.0 – 107.0]
Ca^{2+}	1.200 mmol/L	[1.120 – 1.320]
Hct	40.0 %	[35.0 – 50.0]
tHb	130.0 g/L	[120.0 – 150.0]
COHb	1.0 %	[0.5 – 2.5]
O_2Hb	97.0 %	[95.0 – 99.0]
MetHb	1.0 %	[0.4 – 1.5]
SO_2	99.0 %	[75.0 – 99.0]
Glu	4.0 mmol/L	[3.3 – 6.1]
Lac	1.0 mmol/L	[0.4 – 2.2]







How to interpret abg. How to interpret abg when ph is normal. What is abg report.

N Engl J Med 1998;338:26-34. Can J Anesth 2009;56:247-256. Interpretation of ABGs Interpretation of Arterial Blood Gases (ABGs) David A. Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart and base excess methods. If ΔAG/Δ[HCO3-] > 2.0, then a concurrent metabolic alkalosis is likely to be present. A summary of these techniques can be found in some of the suggested articles. In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the “normal” anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 gm/dL would be approximately 7 meq/L.) If the anion gap is elevated, consider calculating the osmolal gap in compatible clinical situations. Disorder Expected compensation Correction factor Metabolic acidosis PaCO2 = (1.5 x [HCO3-]) +8 ± 2 Acute respiratory acidosis Increase in [HCO3-]= Δ PaCO2/10 ± 3 Chronic respiratory acidosis (3-5 days) Increase in [HCO3-]= 3.5(Δ PaCO2/10) Metabolic alkalosis Increase in PaCO2 = 40 + 0.6(ΔHCO3-) Acute respiratory alkalosis Decrease in [HCO3-]= 2(Δ PaCO2/10) Chronic respiratory alkalosis Decrease in [HCO3-] = 5(Δ PaCO2/10) to 7(Δ PaCO2/10) If the observed compensation is not the expected compensation, it is likely that more than one acid-base disorder is present. Adrogué, H.J. and N.E. Madias. Clinical physiology of acid-base and electrolyte disorders, 5th ed. This discussion does not include some methods, such as analysis of base excess or Stewart’s strong ion difference. Fidkowski, C And J. Assess the ratio of the change in the anion gap (ΔAG) to the change in [HCO3-] (Δ[HCO3-]): ΔAG/Δ[HCO3-] This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present. Helstrom. Suggested additional reading: Rose, B.D. and T.W. Post. Acidosis Respiratory pH ↓ PaCO2 ↑ Acidosis Metabolic& pH ↓ PaCO2 ↓ Alkalosis Respiratory pH ↑ PaCO2 ↓ Alkalosis Metabolic pH ↑ PaCO2 ↑ Step 4: Is there appropriate compensation for the primary disturbance? Usually, compensation does not return the pH to normal (7.35 – 7.45). It is unclear whether these alternate methods offer clinically important advantages over the presented approach, which is based on the “anion gap.” 6-step approach: Step 1: Assess the internal consistency of the values using the Henderseon-Hasselbach equation: [H+] = 24(PaCO2) [HCO3-] If the pH and the [H+] are inconsistent, the ABG is probably not valid. In primary respiratory disorders, the pH and PaCO2 change in opposite directions; in metabolic disorders the pH and PaCO2 change in the same direction. Kaufman, MD Chief, Section of Pulmonary, Critical Care & Sleep Medicine Bridgeport Hospital-Yale New Haven Health Assistant Clinical Professor, Yale University School of Medicine (Section of Pulmonary & Critical Care Medicine) Introduction: Interpreting an arterial blood gas (ABG) is a crucial skill for physicians, nurses, respiratory therapists, and other health care personnel. pH < 7.35 acidemia pH > 7.45 alkalemia This is usually the primary disorder Remember: an acidosis or alkalosis may be present even if the pH is in the normal range (7.35 – 7.45) You will need to check the PaCO2, HCO3- and anion gap Step 3: Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the PaCO2? N Engl J Med 1998;338:107-111. Management of life-threatening acid-base disorders—second of two parts. The following six-step process helps ensure a complete interpretation of every ABG. If this ratio falls outside of this range, then another metabolic disorder is present: If ΔAG/Δ[HCO3-] < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present. Many methods exist to guide the interpretation of the ABG. Step 5: Calculate the anion gap (if a metabolic acidosis exists): AG= [Na+]-([Cl-] + [HCO3-])-12 ± 2 A normal anion gap is approximately 12 meq/L. Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure Toxic ingestion is suspected OSM gap = measured OSM – (2[Na+] - glucose/18 – BUN/2.8 The OSM gap should be < 10 Step 6: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in [HCO3-]. ABG interpretation is especially important in critically ill patients. pH Approximate [H+] (nmol/L) 7.00 100 7.05 89 7.10 79 7.15 71 7.20 63 7.25 56 7.30 50 7.35 45 7.40 40 7.45 35 7.50 32 7.55 28 7.60 25 7.65 22 Step 2: Is there alkalemia or acidemia present? New York: McGraw Hill Medical Publishing Division, c2001. It is important to remember what the expected “normal” anion gap for your patient should be, by adjusting for hypoalbuminemia (see Step 5, above.) Table 1: Characteristics of acid-base disturbances Disorder pH Primary problem Compensation Metabolic acidosis ↓ ↓ in HCO3- ↓ in PaCO2 Metabolic alkalosis ↑ ↑ in HCO3- ↑ in PaCO2 Respiratory acidosis ↓ ↑ in PaCO2 ↑ in [HCO3-] Respiratory alkalosis ↑ ↓ in PaCO2 ↓ in [HCO3-] Table 2: Selected etiologies of respiratory acidosis Airway obstruction- Upper - Lower COPD asthma other obstructive lung disease CNS depression Sleep disordered breathing (OSA or OHS) Increased CO2 production: shivering, rigors, seizures, malignant hyperthermia, hypermetabolism, increased intake of carbohydrates Incorrect mechanical ventilation settings Table 3: Selected etiologies of respiratory alkalosis CNS stimulation: fever, pain, fear, anxiety, CVA, cerebral edema, brain trauma, brain tumor, CNS infection Hypoxemia or hypoxia: lung disease, profound anemia, low FiO2 Stimulation of chest receptors: pulmonary edema, pleural effusion, pneumonia, pneumothorax, pulmonary embolus Drugs, hormones: salicylates, catecholamines, medroxyprogesterone, progestins Pregnancy, liver disease, sepsis, hyperthyroidism Incorrect mechanical ventilation settings Table 4: Selected causes of metabolic alkalosis Hypovolemia with Cl- depletion GI loss of H+ Vomiting, gastric suction, villous adenoma, diarrhea with chloride-rich fluid Renal loss H+ Loop and thiazide diuretics, post-hypercapnia (especially after institution of mechanical ventilation) Hypervolemia, Cl- expansion Renal loss of H+: edematous states (heart failure, cirrhosis, nephrotic syndrome), hyperaldosteronism, hypercortisolism, excess ACTH, exogenous steroids, hyperreninemia, severe hypokalemia, renal artery stenosis, bicarbonate administration Table 5: Selected etiologies of metabolic acidosis Elevated anion gap: Methanol intoxication Uremia Diabetic ketoacidosisa, alcoholic ketoacidosis, starvation ketoacidosis Paraldehyde toxicity Isoniazid Lactic acidosisa Type A: tissue ischemia Type B: Altered cellular metabolism Ethanolb or ethylene glycolb intoxication Salicylate intoxication a Most common causes of metabolic acidosis with an elevated anion gap b Frequently associated with an osmolal gap Normal anion gap: will have increase in [Cl-] GI loss of HCO3- Diarrhea, ileostomy, proximal colostomy, ureteral diversion Renal loss of HCO3- proximal RTA carbonic anhydrase inhibitor (acetazolamide) Renal tubular disease ATN Chronic renal disease Distal RTA Aldosterone inhibitors or absence NaCl infusion, TPN, NH4+ administration Table 6: Selected mixed and complex acid-base disturbances Disorder Characteristics Selected situations Respiratory acidosis with metabolic acidosis ↓in pH ↓ in HCO3 ↑ in PaCO2 Cardiac arrest Intoxications Multi-organ failure Respiratory alkalosis with metabolic alkalosis ↑in pH ↑ in HCO3- ↓ in PaCO2 Cirrhosis with diuretics Pregnancy with vomiting Over ventilation of COPD Respiratory acidosis with metabolic alkalosis pH in normal range ↑ in PaCO2, ↑ in HCO3- COPD with diuretics, vomiting, NG suction Severe hypokalemia Respiratory alkalosis with metabolic acidosis pH in normal range ↓ in PaCO2 ↓ in HCO3 Sepsis Salicylate toxicity Renal failure with CHF or pneumonia Advanced liver disease Metabolic acidosis with metabolic alkalosis pH in normal range HCO3- normal Uremia or ketoacidosis with vomiting, NG suction, diuretics, etc. In addition, you will find tables that list commonly encountered acid-base disorders. Management of life-threatening acid-base disorders—first of two parts.

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