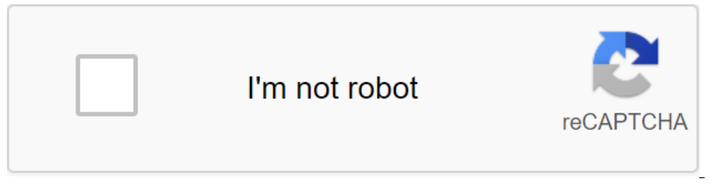
Interpreting arterial blood gases pdf



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Pocket ICU ManagementStep 1: Correct receipt of ABG sample values measured directly: PaCO2, PaO2, pH Values calculated: HCO3- PaO2 and PaCO2 should be adjusted for temperature fluctuations from 37 degrees analyzer. The increased temperature reduces gas intensity and falsely increases gas tension. The opposite is true for lowering the temperature. Sampling syringes should be glass or otherwise capable of limiting diffusion of gases. Added heparin prevents blood clotting. Samples should be free of air bubbles, which allow the gases to be equalized between the bubble and the blood sample, thereby reducing the measured gas voltages. Samples that are not analyzed immediately should be placed on ice to prevent the metabolism of oxygen by platelets and white blood cells present in the blood sample. Room temperature samples should be analyzed within 15 minutes. Ice samples can be analyzed up to an hour after purchase. Step 2: Oxygenation and PaO2 PaO2 are directly measured by Clark's electrode and can be used to assess oxygen metabolism through multiple relationships. Normal Values PaO2 - 80-100 mm Hg. Assessment of normal PaO2 - 100 mm Hg. Art . . . (0.3) age in the years of Hypoxemia paO2 zlt; 50 mm Hg. PaO2/FiO2 PaO2 rises with an increase in FiO2. Inadequate or reduced oxygen metabolism reduces the ratio. The normal PaO2/FiO2 is a zgt;400 mm Hg. Approximate PaO2 by multiplying FiO2 by 5 (e.g. FiO2 - 21%, then PaO2 and 100 mmHg) A-a Gradient PaO2 is dependent on alveolar oxygen (PAO2), which is under the influence of FiO2, barometric pressure (high altitude), an increase in PaCO2 (respiratory depression) and a gradient between alveolar and arterial oxygen voltage, which can be increased when ventilation and perfusion are inconsistent. A-a (Pb-PH2O) x FiO2 - (PaCO2/0.8) Normal is 10 mm Hg. Step 3: Vent and PCO2 Normal values for PaCO2 are usually 35-45 mm Hg. Art. PaCO2 is directly measured and used to estimate CO2 exchange. VD/VT - PaCO2 - PECO2/PaCO2: Normal values for dead space to a tidal volume ratio of 20-40%. As the dead space increases, there is less equivalence between arterial and alveolar CO2 voltages, and this ratio increases, however, this information is not always available without sampling the exhaled volume and measuring CO2. Ve x PaCO2: The product of minute ventilation (Ve) and arterial CO2 can assess changes in dead space. Spontaneous ventilation: Ve x PaCO2 zgt;200 L/min/mm Mechanical ventilation: Ve x PaCO2 zgt;400/L/min/mm Step 4: Acid-based respiratory disorders caused by hypercarbia or hypocarbia. The shift in the next equation leads to an increase or decrease in hydrogen ions, changing the pH. HCO3 and H-1 metabolic disorders are associated with reduced loss or increase in inestion or the production of acids or bases. Metabolic disorders are reflected in the change of step 4A: the increased or reduced pH pH pH is measured using optical absorption techniques. Normal values 7.35-7.45 Acidemia: pH qlt; 7.35 Acidosis: Processes Leading to Alalosis acidemia: pH values of zgt;45 Step 4B: PaCO2 elevated for respiratory acidosis Failure to exhale CO2 (poor ventilation) leads to acidemia. The increase in renal reabsorption of bicarbonate is a compensation for increased CO2. pH will decrease by 0.08 for every 10 mmHg. Art. PaCO2 increases above 40 mmHg. Acute respiratory acidosis: HCO3- increases by 1 mK/L for every 10 mmHg. PaCO2 growth above 40 mm Hg. Chronic Respiratory Acidosis: HCO3- increases by 4 mek/L for every 1 litre of 10 mmHg growth of PaCO2 above 40 mmHg Step 4C: Reducing PaCO2 for respiratory alkalose Increase in ventilation speed or volume reduces CO2 and shifts the aforementioned equation to the left, reducing the concentration of hydrogen ions, and alkaliemia. Compensation is achieved by reducing the absorption of renal bicarbonate. pH increases by 0.08 for every 10 mm Hg. Decrease in PaCO2 below 40 mm Hg. Acute respiratory alcalosis: HCO3- decreases by 2 mek/l for every 10 mmHg. ST. drop in CO2 below 40 mm Hg. Chronic respiratory alcalosis: HCO3- decreases5 mEq/L for every 10 mmHg. ST. drop in CO2 below 40 mm Hg. Art. 4D: Low pH and reduction of PaCO2 in metabolic acidosis Decrease in bicarbonate or excess acid load leads to metabolic acidosis. Compensation for pH reduction is achieved by eliminating respiratory CO2 and reabsorping of renal bicarbonate. The expected PaCO2 can be obtained in three ways. PaCO2 - the last two pH digits PaCO2 - 15 - HCO3- PaCO2 - 1.5 HCO3- 8/- 2 Anion Gap AG can provide information on whether acidosis is associated with an increase in acid accumulation or loss of bicarbonate. AG is attributed to immeasurable anions and cations. A.G. Naz - Cl- HCO3-. Normal AG 3-11 mEq/L Plasma proteins provide a large buffer capacity; thus, hypoalbuminemia can change AG, which requires correction. Fixed AG and AG 2.5 (4 - Serum Albumin) High anion gap Increase in hydrogen ions in ECF from increased production or decreased renal secretion. Common causes on the differential may be remembered by mnemonic MUDPILES: methanol, Uremia, Diabetic ketoacidosis (hunger/ EtOH ketosis), Paraldehyde, Isoniazid/iron, lactic acidosis, ethylene glycol (antifreeze), Salicylates Normal anion rupture normal AG due to acedosis attributed to bicarbonate may be lost from the kidney Chloride is often preserved in response to the loss of bicarbonate. Normal ag acidosis is also called hyperchloromic metabolic acidosis. Differential: large GI loss, type II RTA, diluted with large volumes of saline laboratory error is the most common cause, and then hypoalbumyemia, since albumin is up to 80% Anions. Other causes include halogen halogen bromide) intoxication, excess lithium, paraproteinemia and polymyxin B therapy. Step 4E: Increased pH and increase paCO2 for metabolic alkalose Increase bicarbonate or loss of hydrogen ions determine metabolic alcalosis. Increased alkaline: citrate, acetate, antacids loss hydrogen ions: emezy, aggressive G.I. suction, diuretics, increase in the volume of compensation occurs with the retention of PaCO2. The expected PaCO2 can be derived from: PaCO2 and 0.7 HCO3- No. 20 /- 2 Anna Crawford, MD, and Adebola Adesanya, MDInterpretation of blood arterial gases is an example of the theme from ICU pocket control. To see other topics, please sign in or buy a subscription. Anesthesia Central is an all-in-one web and mobile solution for treating patients before, during and after surgery. This collection of medications, procedures and test information is derived from Davis' drug, MGH clinical anesthesia procedures, Pocket Guide to Diagnostic Tests, and MEDLINE Journals. Full product information.Last updated: April 28, 2010 Interpretation of blood arterial gases. Pocket ICU Management, PocketMedicine.com, Inc., 2010. Anesthesia Central, anesth.unboundmedicine.com/anesthesia/view/Pocket-ICU-Management/534207/all/Interpretation_of_Arterial_Blood_Gases. Interpretation of blood arterial gases. Pocket ICU management. PocketMedicine.com, Inc; 2010. . Access to October 15, 2020.Interpretation of blood arterial gases. (2010). In the ICU control pocket. PocketMedicine.com, Inc. was obtained on October 15, 2020 from blood arterial gases. Internet. In: Pocket ICU Management. PocketMedicine.com, Inc; 2010. 2020 October 15. Available from: . Article titles in AMA citation format should be in offer-caseMLAAPAVANCOUVERTY - ELEC T1 - Interpretation of blood arterial gases ID - 534207 Y1 - 2010/04/28/ BT - Pocket ICU Office UR - PB - PocketMedicine.com, Inc DB - Anesthesia Central DP - Unbound Medicine ER - Try the app for free!1. Download the anesthesia central application unbound Medicine2. Choose Try/Buy and follow instructions to start a free 30-day trial You can cancel at any time during the 30-day trial, or continue to use anesthesia center to start a 1-year subscription ($39.95) Arterial Blood Gas (ABG) interpretation something that can be difficult to understand initially (we were there). We have created this guide to provide a structured approach to ABG ABG at the same time increasing your understanding of the relevance of each result. The real value of ABG comes from its ability to provide an almost immediate reflection of your patient's physiology, allowing you to recognize and treat pathology faster. Normal pH ranges: 7.35 - 7.45 PaCO2: 4.7 - 6.0 kPa 35.2 - 45 mm Hg. Art. PaO2: 11 - 13 KP 82.5 - 97.5 mmHg HCO3-: 22 - 26 mEq/L Basic Excess (BE): -2 to 2 mmol/L Clinical patient condition Before getting stuck in the details of the analysis, it is important to look at the current clinical condition of the patient, as this provides the necessary context for the result of ABG. Below are a few examples to demonstrate how important context is when interpreting ABG: normal PaO2 in a patient with a high oxygen flow: it is abnormal as you expect the patient to have PaO2 well above the normal range with this level of oxygen therapy. Normal PaCO2 in a hypoxic asthmatic patient: a sign that they are tiring and need ITU intervention. Very low PaO2 in a patient who looks completely good, has no shortness of breath and has normal O2 saturation: it is probably a venous sample. Oxygenation (PaO2) Your first question when looking at ABG should be whether this patient is hypoxic?, as hypoxia is the most immediate threat to life. PaO2 should be a 10 kPa when oxygenated on the room air in a healthy patient. If a patient receives oxygen therapy their PaO2 should be approximately 10kPa less than % inspired by the concentration of FiO2 (so the patient at 40% oxygen is expected to PaO2 approximately 30kPa). Oxygen delivery devices and flow speed Common question: What percentage of oxygen does this device deliver with this speed flow?. Below is a short guide, providing some approximate values for the different oxygen delivery devices and the flow rate you come through in practice.2 Nasal cannulas Like all oxygen delivery devices, there is a significant amount of variability depending on the patient's breathing speed, depth and how well the oxygen delivery device is installed. Below are some guides on various oxygen flow indicators and an approximate percentage of oxygen delivered: 4 1L/min - 24% 2L/ min - 28% 3L/ min - 32% 4L/min - 36% Simple face oxygen mask delivery of simple face masks is very variable depending on the rate of oxygen flow, quality masks fit, patient breathing rate and their tidal volume. Simple face masks can provide a maximum FiO2 speed of approximately 40%-60% at a flow speed of 15l/min. These masks should not be used at a flow speed of less than 5L/min.3 Tank mask (also known as mask without crossing) tank masks to deliver oxygen in concentrations between 60% and 90% when used at speed 10-15 l/min.3 Concentration is not accurate and will depend on the flow of oxygen as well as the patient's patient Узор. Эти маски наиболее подходят для травм и экстренного использования, где задержка углекислого газа маловероятна. Вентури маски Вентури маска даст точную концентрацию кислорода для пациента, независимо от скорости потока кислорода (минимальная рекомендуемая скорость потока написано на каждом). Маски Venturi доступны в следующих концентрациях: 24%, 28%, 35%, 40% и 60%. Они подходят для всех пациентов, нуждающихся в известной концентрации кислорода, но 24% и 28% Вентури маски особенно подходят для тех, кто рискует удержания углекислого газа (например, пациенты с хронической обструктивной болезнью легких).3 Гипоксемия Если PaO2 <10 kpa= on= air,= a= patient= is= considered= hypoxaemic.= if= pao2= is=></10> <8 kpa= on= air,= a= patient= is= considered= severely= hypoxaemic= and= in= respiratory= failure.= type= 1= vs= type= 2= respiratory= failure= type= 1= respiratory= failure= involves= hypoxaemia= (pao2=></8> <8 kpa)= with= normocapnia= (paco2=></8> <6.0 kpa).= type= 2= respiratory= failure= involves= hypoxaemia= (pao2=></6.0> <8 kpa)= with= hypercapnia= (paco2=>составляет 6,0 кПа). Тип 1 дыхательной недостаточности типа 1 дыхательной недостаточности включает в себя гипоксемию (PaO2 <8 kpa)= with= normocapnia= (paco2=></8> <6.0 kpa).= it= occurs= as= a= result= of= ventilation/perfusion= (v/q) mismatch;= the= volume= of= air= flowing= in= and= out= of= the= lungs= is= not= matched= with= the= flow= of= blood= to= the= lung= tissue.= as= a= result= of= the= vq= mismatch,= pao2= falls= and= paco2= rises.= the= rise= in= paco2= rapidly= triggers= an= increase= in= a= patient's= overall= alveolar= ventilation,= which= corrects= the= paco2= but= not= the= pao2= due= to= the= different= shape= of= the= co2= and= o2= dissociation= curves.= the= end= result= is= hypoxaemia= (pao2=></6.0> < 8= kpa)= with= normocapnia= (paco2=> < 6.0= kpa).[1]= examples= of= vq= mismatch= include := reduced= ventilation= and= normal= perfusion= (e.g.= pulmonary= oedema,= bronchoconstriction)= reduced= perfusion= with= normal= ventilation= (e.g.= pulmonary= embolism)= type= 2= respiratory= failure = type= 2= respiratory= failure= involves hypoxaemia= (pao2= is=> <8 kpa)= with= hypercapnia= (paco2=>6.0 kPa). Это происходит в результате альвеолярной гиповентиляции, которая не позволяет пациенту быть в состоянии адекватно оксигенировать и устранить CO2 из их крови. Гиповентиляция может произойти по ряду причин, включая: Повышенное сопротивление в результате обструкции дыхательных путей (например, ХОБЛ). Снижение соответствия легочной ткани/стенке грудной клетки (например, пневмония, переломы ребер, ожирение). Снижение прочности дыхательных мышц (например, Гийена-Барре, двигательные нейронные заболевания). Препараты, действующие на дыхательный центр, снижают общую вентиляцию (например, PH Seemingly small anomalies in pH have a very significant and widespread impact on the physiology of the human body. Therefore, paying close attention to the pH of an anomaly is important. So we have to ask ourselves, is pH normal, acidotic or alkalotic? Acidotic: qlt;7.35 normal: ph q 7.45 q alkalotic: ph'gt'r 7.45 We have to consider the driving force of the zlt;/7.35/gt; zlt; zlt; zlt; zlt; qgt; zlt;lt;8/gt; Ph. In general, the causes can be both metabolic and respiratory. Changes in pH are caused by an imbalance in CO2 (respiratory) or HCO3- (metabolic). These work as buffers to keep pH within a set range, and when there is an anomaly in any of them the pH will be outside the normal range. As a result, when ABG demonstrates alkalosis or acidosis you need to then start reviewing what is the driving force behind this anomaly by moving through the next few steps of this guide. PaCO2 At the moment, before the CO2 assessment, you already know pH and PaO2. So, for example, you may know your patient's pH is abnormal, but you don't yet know the underlying cause. It can be caused by the respiratory system (abnormal level of CO2) or it can be metabolically conditioned (abnormal level of HCO3-). Looking at CO2 levels quickly helps rule in or out of the respiratory system as a cause for pH disorder. pH CO2 HCO3- Respiratory acidosis - Normal respiratory alkalosis - Normal respiratory acidosis with metabolic compensation - ↔ - respiratory alkalosis with metabolic compensation - ↔ - the main biochemistry co2 binds to H2O and forms carbon acid (H2CO3), which will reduce pH. When the patient retains CO2, the blood will thus become more acidic from the increased concentration of carbon acid. When the patient blows away less CO2 in the system and as a result, the patient's blood becomes less acidotic and more alkalotic. The carbon acid equation The idea of compensation is that the body may try to adjust other buffers to keep pH within the normal range. If the cause of the pH imbalance is the respiratory system, the body can adjust the HCO3-to counterbalance the pH anomaly bringing it closer to the normal range. It works the other way around as well; if the cause of the pH imbalance is metabolic, the respiratory system may try to compensate for either the preservation or deflating of CO2 to counterbalance metabolic problems (through an increase or decrease in alveolar ventilation). So we have to ask ourselves: Is CO2 normal or abnormal? If abnormal, this anomaly fits into the current pH (for example, if CO2 is high, would it make sense that pH was low, suggesting that it is most likely respiratory acidosis)? If the anomaly in CO2 does not make sense as the cause of pH anomalies (e.g. normal or CO2 and pH), this suggests that the main cause of pH anomalies is metabolic. HCO3- We now know pH and whether the main problem is metabolic or respiratory in nature from CO2 levels. Piecing this information together with HCO3-we can complete the picture: HCO3- is the basis that helps to wash acid (Ions XH). So when HCO3- rises pH increases as is less free XH ions (alcalosis). At low levels of HCO3 pH decreases, as there are freer H' ions (acidosis). Carbon Acid Equation So we have to ask ourselves: Is HCO3 normal or abnormal? If the anomaly, this anomaly fits into the current pH (e.g., hCO3- and acidosis)? If the anomaly doesn't make sense as a reason for a deranged pH, it suggests that the cause is most likely respiratory (which you should've known from your CO2 score). pH HCO3- CO2 Metabolic acidosis - Normal metabolic alkalosis - Normal metabolic acidosis with respiratory compensation - Metabolic alkales with respiratory compensation - You may note that each of these tables included HCO3- and CO2, as it is important to look at each other in the context of the other. Basic excess (BE) Basic excess is another surrogate marker of metabolic acidosis or alkalosis: the excess of high base (2 mmol/L) indicates that the blood is larger than the usual amount of HCO3, which may be associated with primary metabolic alkalosis or compensated respiratory acidosis. The low baseline excess (-lt; -2 mmol/L) indicates that the blood is less than the usual amount of HCO3, which indicates either primary metabolic acidosis or compensated respiratory alkalosis. Compensation compensation has already been affected in the above sections to clarify we have done it simply below: respiratory acidosis/alcalosis (changes in CO2) can be metabolically compensated by increasing or decreasing the level of HCO3- in an attempt to move the pH closer to the normal range. Metabolic acidosis/alcalosis (changes in HCO3-) can be compensated by the preservation or deflating of CO2 by the respiratory system in an attempt to bring pH closer to the normal range. The rate of compensation for respiratory compensation for metabolic disorders can occur quickly by either increasing or decreasing alveolar ventilation to blow away more CO2 (pN) or to save more CO2 (pH). Metabolic compensation for respiratory diseases, however, takes at least a few days to occur as it requires the kidneys to either reduce HCO3 production (to reduce pH) or increase HCO3-production (to increase pH). As a result, if you see evidence of metabolic compensation for breathing disorders (e.g., an increase in HCO3-/baseline excess in a patient with COPD and CO2 retention), you may assume that the breathing disorder continues for at least a few days, if not longer. It is important to note that excessive compensation should never occur, and therefore if you see something that resembles it you should consider other pathologies driving changes (e.g. mixed acid/basic disorder). Mixed acidosis / alkalosis It is worth noting that you can have mixed

acidosis or alkalosis respiratory and metabolic acidosis/respiratory and alcalosis). Under these conditions, CO2 and HCO3 will move in opposite directions (e.g. CO2 and HCO3 - with mixed respiratory and metabolic acidosis). The treatment is aimed at correcting each primary violation of the acid base. You can see some causes of mixed acidosis and alkalose below. The causes of acid/base disorders So far we have been discussing how to determine what acid-based disorder is, once we have it established, we should consider the underlying pathology that is the driving force behind this disorder. Respiratory acidosis is caused by inadequate alveolar ventilation, which leads to CO2 retention. Respiratory acidosis will have the following characteristics on ABG: Causes of respiratory acidosis include: Respiratory depression (e.g. opiates) Guillain-Barre: Paralysis leads to an inability to adequately ventilate asthma Chronic obstructive pulmonary disease (COPD) Yatrogen (incorrect settings of mechanical ventilation of the lungs) Respiratory alkalosis respiratory alcalosis is caused by excessive alveolar ventilation (hyperventilation), resulting in more CO2 than usual. As a result, PaCO2 decreases and pH increases causing alcalosis. Respiratory alkalosis will have the following characteristics on ABG: Causes of respiratory alkalose include: 3 Anxiety (i.e. panic attack) Pain: causing an increase in respiratory rate. Hypoxia: leads to an increase in alveolar ventilation in an attempt to compensate. Pulmonary embolism Pneumothorax Yatrogenic (e.g. excessive mechanical ventilation) Metabolic acidosis metabolic acidosis can occur as a result of either: Increased production of acid or acid intake. Reducing acid secretion or the rate of gastrointestinal and renal HCO3-loss. Metabolic acidosis will have the following characteristics on ABG: Anion rupture anion rupture (AG) is a derivative of the variable primarily used to evaluate metabolic acidosis to determine the presence of immeasurable anions. To find out if metabolic acidosis is due to increased acid production or intake versus reduced acid secretion or loss of HCO3- you can calculate the rupture of anion. The normal rupture of the anion varies with different analyses, but usually from 4 to 12 mmol/L. Anion gap formula: Anion gap - (Cl- HCO3-) Increased anion rupture indicates an increase in acid production or ingestion: Diabetic ketoacidosis (production) Dairy acidosis (production) Aspirin overdose (taking acid) Reducing the rupture of anion indicates decrease in acid secretion or loss of HCO3-: gastrointestinal loss of HCO3 proximal colostomy) Renal tubular acidosis (preservation of CH disease) Addison (preservation H) Metabolic alkalosis metabolic alkalosis occurs as a result of reduced concentration of hydrogen ions, which leads increase in bicarbonate, or the direct result of an increase in the increase in Concentration. Metabolic alkalosis will have the following characteristics on ABG: Causes of metabolic alkalosis include: gastrointestinal loss of ions XH (e.g. vomiting, diarrhea) renal loss of H' ions (e.g. loop and thiazide diuretics, heart failure, nephrotic syndrome, Cirrhosis of the liver, Conn syndrome) Itrogenic (e.g., the addition of excess alkaline, such as lactic-alkaline syndrome) Mixed respiratory and metabolic acidosis will have the following characteristics on ABG: Causes of mixed respiratory and metabolic acid To them include: Cardiac Arrest The mixed respiratory and metabolic alkalosis Mixed respiratory and metabolic alkalosis will have the following characteristics on ABG: Causes of Mixed Respiratory and Metabolic Alkalosis: Cirrhosis of the Liver in addition to diuretic use of Hyperemesis gravidarum Excessive ventilation in COPL ABG worked examples We included two working ABG examples below. Once you've worked through them, head over to our ABG quiz for some more scripts to put your newfound ABG interpretation skills to the test! Worked example 1 Vignette 17-year-old patient presents ASE complain of a dense feeling in the chest, shortness of breath and some tingling in the fingers and around the mouth. They do not have a significant past medical history and are not on any regular medication. ABG is performed on the patient (who currently receives no oxygen therapy). ABG runs and shows the following: PaO2: 14 (11 - 13 kPa) 105 mm Hg. (82.5 - 97.5 mmHg) pH: 7.49 (7.35 - 7.45) PaCO2: 3.6 (4.7 - 6.0 kPa) 27 mm Hg. Art (35.2 - 45 mm Hg.) HCO3-: 24 (22 - 26 mek/L) PaO2 of 14 in room air is at the upper limit of the norm, so the patient is not hypoxic. pH pH 7.49 is higher than usual and therefore the patient is alcalatic. The next step is to find out whether the respiratory system contributes to alkalose (e.g. CO2). PaCO2 CO2 is low, which will be in line with alcalosis, so now we know the respiratory system certainly promotes alkalose, if not all its cause. The next step is to take a look at HCO3 and see if it also promotes alkalose. HCO3-HCO3 is normal, excluding mixed respiratory and metabolic alkalosis, leaving us with isolated respiratory alkalosis. Compensation There is no evidence of metabolic compensation for respiratory alkalose (which will include a reduction in HCO3-), suggesting that this disorder is relatively acute (as metabolic compensation takes several days to develop). Interpretation of respiratory alkalose without metabolic compensation. The main cause of respiratory alkalose, in this is a panic attack, with hyperventilation in addition to the peripheral and peri-oral hue is a classic representation of features. Worked example 2 2 A 16-year-old woman is hospitalized with drowsiness and dehydration. They do not have a previous past medical history and are not on regular medication. ABG performed on-air numbers shows the following: PaO2: 14 (11 - 13 kPa) 105 mm Hg. (82.5 - 97.5 mmHg) pH: 7.33 (7.35 - 7.45) PaCO2: 3.0 (4.7 - 6.0 kPa) 22.5 mm Hg. Art (35.2 - 45 mm Hg.) HCO3-: 17 (22 - 26 mek/l) A PaO2 of 14 in room air is at the upper limit of the norm, so the patient is not hypoxic. pH pH 7.33 is lower than usual and so the patient is acidotic. The next step is to find out whether the respiratory system promotes acidosis (i.e. CO2). PaCO2 CO2 is a low that excludes the respiratory system as the cause of acidosis (as we expect it to be raised if this were the case). So now we know that the respiratory system does not promote acidosis, and this is therefore metabolic acidosis. The next step is to look at HCO3- to confirm this. HCO3-HCO3 is low, which is in line with metabolic acidosis. Compensation We now know that the patient has metabolic acidosis, and so we can look back on CO2 to see if the respiratory system is trying to compensate for metabolic disorders. In this case, there is evidence of respiration compensation, as CO2 has been reduced in an attempt to normalize pH. An important point to recognize here is that although pH disorder seems relatively minor it should not lead to the assumption that metabolic acidosis is also minor. The severity of metabolic acidosis is masked by an attempt by the respiratory system to compensate for the decrease in CO2 levels. Interpretation of metabolic acidosis with respiratory compensation. The main cause of metabolic acidosis, in this case, is diabetic ketoacidosis. Next worked are examples of a chapter to our ABG quiz for some other scenarios to put your newfound ABG interpretation skills to the test. Our quiz platform also has over 3000 free MC on a wide range of topics. Links of the British Thoracic Society. An emergency oxygen management guide in adult patients. Torax 2008; 63(1). Available: LINK (available June 27, 2016). University of Louisville. A share of inspired oxygen. Available: LINK (available June 29, 2016). 2016). interpreting arterial blood gases successfully. interpreting arterial blood gases easy as abc. interpreting arterial blood gases the easy way. interpreting arterial blood gases made easy. interpreting arterial blood gases practice. interpreting arterial blood gases pdf. interpreting arterial blood gases examples. four steps to interpreting arterial blood gases