Acidosis respiratoria pdf 2020

By random looking at some of the physiotherapy journals of our work center, we find an article that tells about this issue, published in the year-long issue of the magazine Kinesitarapy Scientifique. Since respiratory physiotherapy is one of the subjects of our profession that has always caught our attention, and more evaluation, we read it with interest. And we dare to give the reader a small summary. The Manual Respiratory Motion Assessment (MARM) method, or Manual Respiratory Motion Assessment (EMR) in our free translation, aims to systematically review the movements in the ventilation according to the position and voluntary actions of the subject. Consider the relative distribution of ventilation movements in the upper, lower and abdominal segments. Incorporating the movement of the flex-expansion of the spine is considered as the third degree of freedom, so the form of expansion facilitates vertical inspiration and the flexion of horizontal inspiration with diaphragmatic elongation. EMR is about creating a mental image of movements manually perceived by a physiotherapist. This image is represented by a semicircle. Three degrees of freedom are analyzed: the lifting of the sternum and the upper part of the chest and the lateral opening of the lower part of the chest. The creators of the technique assume that optimal ventilation is in the balance between the thoracic and abdominal segments, in proportion to 50% of the total ventilator. The technique is done, with his eyes closed, putting his hands on the back and bottom of the chest, with the thumbs aligned parallel to the sides of the spine, pointing to the head, in the dorsolumbar charnela, and the rest of the fingers wrapping the lower ribs to perceive abdominal movement. The palpation is presented, as we have already said, in a semicircle with a horizontal radius, dividing it into two parts to represent the movement of the chest in the upper half and the abdomen in the lower half. Thus, the inspiring volume is represented by the angles of two radii, one upper A and one lower B in relation to the horizontal radius C. The balance is represented by the difference between the angles of the AC and the CB. The speed of chest mobility is represented by the AC/CB factor, expressed in percentage. In addition, the score is also qualitative (lightness, effort and asymmetry). EMR is performed during movements at rest, in chest and abdominal ventilation, and in different positions. The technique has been tested for its reproducibility and sensitivity compared to two physical methods. Though reasonable, given its subjectivity, it may be a way to enrich evaluations in respiratory patients. Reference: Selleron B. L'Valuation Manuel de mouvements ventilatoires par la matod MARM (Manual assessment of breathing movements). Kinesham University, No. 549, December 2013. READ MORE PHYSIOTHERAPY. Dear users: Medycyna Praktyczna uses cookies and other related technologies on its services. We use cookies to adapt our services to your needs, as well as for analytical and advertising purposes. We use our own and third-party cookies, our business partners. Cookies can be controlled with browser settings. Information about the settings, as well as details of the technologies we use, are included in our Privacy Policy. Using our services without a browser setting, you agree with cookies and related technologies described in the Privacy Policy. Your consent is free, but your absence can affect the user experience and the comfort of using our services. This consent may be withdrawn at any time, but the processing of the data carried out up to that time will remain lawful. By clicking OK, they agree to use the technologies listed above and confirm that your browser settings are adjusted according to your preferences. Page 2 Acute Respiratory Failure Chronic Respiratory Failure REASIT AND ETIOPATOGENIAArriba Respiratory Failure is a condition, in which changes in the function of the respiratory system impair the exchange of gas at the pulmonary level, leads to hypoxemia (reduction of partial oxygen pressure in the blood (PaO2) (PaO2) (60 mmHg (8.0 kPa) or hypercapnia (increased partial carbon dioxide pressure (PaCO2) ≥45 mmHg (6.0 kP). that the patient is at rest, breathes the surrounding air and at sea level. They differ: hypoxemic respiratory failure, also referred to as partial or type 1 (without hypercapnia) and hypoxymic-hypercapical, global or type 2. Hypoxemia 1. Mechanisms of hypoxemia development. 1) Lack of regulation of alveolar ventilation on pulmonary blood infusion (a) Reducing alveolar ventilation (e.g. due to altelektase or filling of alveoli fluid) with pulmonary infusion maintained or slightly reduced → reduction of partial oxygen pressure in the alveolar air → worse oxygenation of the blood, come out of the alveolar pulmonary → oxygenated blood is mixed in the pulmonary veins with the worst oxygen blood, coming from poorly ventilated areas of the → , in the lungs left ventricle and arteries of the cerebral circulation. b) Reducing pulmonary infusion with preserved or increased ventilation, especially due to pulmonary embolism or shock. 2) Shunt (short-circuit) non-oxygenated blood a) Intralegon: if blood flow is maintained through the lung area excluded from ventilation and gas markings (e.g., due to obstruction of the airways or filling pulmonary alveoli with fluid), non-oxygenated blood from this territory enters the pulmonary veins and is mixed there with oxygen blood from the properly ventilated pulmonary alveo. The higher the amount of non-oxygenated blood in the mixture, the more hypoxemia and the worse the response to oxygen therapy without assistance positively under the pressure of pulmonary ventilation. b) Extralight: the connections between lung and systemic circulation (cyanogen and major vascular heart disease) cause hypoxemia with poor reaction to oxygen therapy. In this situation, hypoxemia was caused not by a change in the gas marking of the lungs, but by an extra-light veneer short circuit. 3) Alveolocapyl diffusion disorder caused by thickening of the alveol-capillary barrier and reduced oxygen permeability caused, in particular, by interstitial lung diseases. 4) Reducing the partial oxygen pressure in the mixture is an inspired gases that occur while staying at high altitudes due to reduced atmospheric pressure. In this situation, hypoxemia is not due to respiratory disorders, but also a decrease in PO2 in inspired air. 2. Consequence of hypoxemia: 1) tissue hypoxemia (hypoxia) → anaerobic metabolism → milk acidosis → cell death → multiorganic insufficiency → death 2) compensatory reactions (transient and disappear with mild hypoxemia): supported): tachycardia, tachycardia, high blood pressure, increased heart output fraction, hyperventilation 3) pulmonary hypertension, secondary to reflex narrowing of pulmonary arterioles and increased endurance; it is made by constantly remodeling the walls of pulmonary vessels 4) right heart failure: overload, hypertrophy and insufficiency of the right ventricle due to pulmonary hypertension of secondary hypoxemia, caused by various diseases of the respiratory system (cortex: expansion of the right ventricle increased resistance to pulmonary vessels) 5) secondary polycythemia (polyglobulia): Chronic hypoxemia stimulates renal secretion of erythropoietin and increases erythroposis 6) accropacy and hypertrophic osteotrostrophism →cap. Hypercapnia 1. Hypercapnia Mechanism: Alveolar hypoventilation plays a major role because CO2 crosses the alveolicapilary barrier 20 times faster than O2. Therefore, the thickening or decrease in permeability of this barrier, as well as the reduction of lung infusions, do not have such a significant effect on the exchange of CO2 between air and blood, as in the case of O2. 2. Causes of hypoventilation. 1) Increased respiratory system load (respiratory work). (a) Increased resistance to airflow in the airways: upper respiratory tract blockage (inordous body, oblection, loss of consciousness), blockage of the airways low contraction of the bronchi of smooth muscles and mucous membranes (COPD, bronchial asthma, bronchial blockage of bronchial discharges or swelling of the sleep). b) Reducing pulmonary distension: filling pulmonary alveoli with fluid (pulmonary swelling, intracellular hemorrhage), pneumonia, interstitial lung disease, hypoventilation, dynamic hyperinsufulation (especially COPD), pulmonary hemorrhage (extravasive blood), fluid in the pleural cavity or cavity, pneumoplex. c) Reducing breast wall extension: significant obesity, diaphragmatic height (intestinal dystencia, acytes, diaphragmatic paralysis), deformities, injuries and chest wall tumors. d) It is necessary to increase ventilation per minute (relative hypoventilation): shock, hypovolemia, sepsis, pulmonary embolism. 2) Changing the effectiveness of the respiratory muscles and nervous system. (a) Decrease in respiratory center activity: drug overdose (opioids and hypnotics) or drugs, brain stem damage, central sleep apnea, hypothyroid coma. b) Changes in neural and neuromuscular transmission: frenic nerve damage, spinal cord injury at the level of the cervix or chest, Guillain-Barre syndrome, myastenic crisis, tetanus, botulism, muscle relaxive drugs, intermittent acute porphyria, lateral amyotrophic sclerosis, multiple sclerosis. c) Weakness of respiratory muscles: overload (increased respiratory work), electrolyte changes (potassium deficiency, magnesium, phosphates), acidosis, malnutrition, hypoxia, shock, muscle diseases. 3) Increased ventilation of the physiological dead space. (a) Increase in the volume of thawed gas in the airways not involved in the exchange of gas (anatomical dead space). b) Increased alveolar dead space due to increased intra-alveolar pressure above infusion pressure and/or Alveoli. 3. Effects of hypercapnia. 1) Respiratory acidosis →cap. 19.2.2. 2) Headache and changes in the level of consciousness: confusion, pathological drowsiness and hypercapon coma (associated with the enlargement of cerebral vessels and increased intracranial pressure). 3) Hypoxymic respiratory stimulus: chronic respiratory failure with hypercaptal leads to a decrease in the sensitivity of the respiratory center located in the spinal lamp and in the lump at high partial pressure of CO2. The main stimuli that activate the respiratory center come from PaO2 sensitive than-stimulators found in sleepy and aortic organs. In this situation, too intensive oxygen therapy and paO2 too highly reduce the activity of the respiratory center and cause hypoventilation and increased hypercapnia, which can lead to a hypercapic coma. hypercapic.