Intern Emerg Med (2009) 4:331–332
DOI 10.1007/s11739-009-0268-x

IM - PHYSICAL EXAMINATION

Pulse oximeter, the fifth vital sign: a safety belt or a prison of the mind?

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Received: 11 May 2009 / Accepted: 20 May 2009 / Published online: 13 June 2009

Case report

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A 48-year-old woman hospitalized in the hematologic clinic because of chronic myeloid leukemia, during the night required the attention of the on-call physician for dyspnea associated with tachycardia. General physical examination revealed pallor and rhythmic tachycardia (145 beats/min). Her temperature was 98.6°F (37°C), respiratory rate was 32 breaths/min, blood pressure was 104/54 mmHg, and oxygen saturation was 95%. A complete blood count performed in the afternoon demonstrated an Hb of 9 g/dL, but the patient reported she had hematemesis in the late evening. "Did you give oxygen?", asked the physician to the nurse. "No doctor, oxygen saturation is 95%. She called me for tachycardia, should I ask for the cardiologist?" Blood withdrawn was performed again, showing a further decrease in the Hb level (6 g/dL). Blood was requested and in the meantime colloid infusion started. The reluctance of the nurse was finally overcome and oxygen was added at 10 L/min. Tachycardia and dyspnea gradually relapsed.

Comment

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Oxygen saturation (SaO_2), together with measurements of heart rate, blood pressure, respiratory rate and temperature, is now included among the essential vital sign assessment in patients care [1]. The routine monitoring of this parameter has increased the recognition of hypoxic episodes occurring in different clinical settings or during various procedures. However, while a low SaO_2 (<90–92%) always alarm care givers, there is also the risk of a false assumption, that is "normal" saturation ($SaO_2 \geq 94$) means no hypoxia. When speaking with students, a misconception about the relationship between arterial oxygen partial pressure (PaO_2) and hemoglobin saturation is not rarely encountered [2] because oxygen delivered (DO_2) to tissue is perceived normal only on the basis of SaO_2 and PaO_2 [3]. Although the primary factor that determines how much oxygen is actually bound to hemoglobin is the partial pressure of oxygen (pO_2) in the hemoglobin solution, SaO_2 and PaO_2 cannot be considered alone without considering blood hemoglobin content nor cardiac output (CO) because $DO_2 = CO \times CaO_2$ where CaO_2 is the oxygen content of blood. An anemic patient (Hb = 7 g/dL) may have an O_2 saturation of Hb of 100% with a pO_2 of 95 mmHg, thus looking deceptively non-hypoxic, although the total DO_2 of arterial blood is reduced by an half. With very low Hb values, DO_2 may even become inadequate to meet the tissue oxygen requirements (VO_2) for aerobic metabolism, a failure defining circulatory shock. Clinicians challenged to support DO_2 to avoid tissue hypoxia have to rapidly consider all the different components because CaO_2 is derived from hemoglobin content (Hb), SaO_2, and a constant K (the coefficient for hemoglobin–oxygen binding capacity) ($CaO_2 = Hb \times SaO_2 \times K$) [4]. Respiratory failure and inadequate DO_2 can thus result from malfunction of any aspect of the "ventilatory apparatus", oxygen uptake in the lung via inspiration, passive oxygen diffusion into arterial blood, hemoglobin content and dissociation kinetics or from low CO. Each of these parameters

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taken alone can fail to detect the presence of tissue hypoxia [4].

Marked dyspnea of BC, with the increased ventilatory rate and depth, was mainly due to the hypoxic stimulus of chemoceptors. Carbon dioxide is the main stimulus to ventilation. However, an arterial PaO_2 of <60 mmHg also acts as an important stimulus. In particular carotid body senses soluble O_2 and signals the respiratory center to increase respiratory rate and depth (hypoxix ventilator response) [5]. Although O_2 solubility in blood is very low (0.003 mL/dL per mmHg, a constant independent of prevailing pO_2), O_2 content in plasma is a variable dependent on prevailing pO_2 (0.3 mL O_2/dL). In conclusion, a FiO_2 increase cannot further enhance the oxygen Hb transport so that DO_2 cannot be significantly enhanced; however, soluble O_2 in plasma may increase thus reducing the stimulus at the chemoceptors level. Oxygen administration might thus reduce the hypoxic ventilator response even if only rapid plasma expansion with consequent CO increase may have increased DO_2.

Conflict of interest statement The authors declare that they have no conflict of interest related to the publication of this manuscript.

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