Case:

70 y/o man is to undergo cystoscopy and transurethral resection of a bladder tumor under general anesthesia through a laryngeal mask airway. He gave a history of mild asthma and used an albuterol inhaler when necessary. Breathing room air (FIO2 = 0.21), his pulse oximeter saturation reading (Sp02) was 94%.

Discussion Questions:

Was this patient hypoxemic?

No. A hemoglobin oxygen saturation reading of 94% does not signify hypoxemia. This patient is five decades older than a 20-year-old subject; therefore, his mean Pao2 should be 5 x 5 mm Hg per decade, which is 25 mm Hg less than that (95 mm Hg) expected in a 20-year-old. 95-25=70mmHg or, using the Marshall and Whyche equation, 102 - 0.33(70) = 79 mmHg

Assuming normal adult hemoglobin, temperature, and pH, this Pao2 corresponds to a hemoglobin oxygen saturation of approximately 94% on the normal hemoglobin oxygen saturation versus P02 curve.

Although the definitions of hypoxemia must take age into consideration, from a practical point of view, hypoxemia (in the absence of anemia) is generally considered to exist when the Pao2 is less than 60 mm Hg, which is equivalent to a hemoglobin oxygen saturation of 90%.


How do methemoglobin and carboxyhemoglobin affect Sp02 readings?

In methemoglobin, iron in the heme moiety is oxidized (such as by dapsone, benzocaine, nitric oxide, prilocaine) to the Fe3+ state rather than being in the normal Fe2+ state. Methemoglobin cannot carry oxygen and creates a physiologic anemia. The absorbance spectrum for metHb shows it to have similar absorbances at 660 and 940 nm. Therefore, the more metHb that is present, the more R tends toward 1 and the Sp02 reading toward 85%. In the presence of metHb, the Sp02 reading overestimates the fractional saturation and underestimates the functional saturation.

In carbon monoxide (CO) poisoning, CO combines with Hb to create a physiologic hypoxemia. HbCO has a similar absorbance to Hb02 at 660 nm, but a very low absorbance at 940 nm. In the presence of HbCO, the Sp02 overestimates fractional saturation and underestimates functional saturation. Although blood that is poisoned with carbon monoxide appears "cherry red" in color to the naked eye, and therefore may look like it is fully saturated with oxygen, it is important to recognize that the ratio of absorbances 660/940 nm suggest blood with a hemoglobin oxygen saturation in the 90s. In a subject breathing PI02 of 1 (when functional saturation is 100%), as the level of HbCO increases, Sp02 decreases from close to 100% down to approximately 91%.

If the presence of dyshemoglobins is suspected, arterial blood must be drawn and analyzed in a hemoximeter to obtain accurate readings of saturation.

Pulse oximetry technology continues to improve. Very recently, a Company announced the introduction of a pulse oximeter that uses eight wavelengths of light, similar to the hemoximeter. This will offer the unique ability to accurately measure carbon monoxide (SpCO), methemoglobin (SpMet) and potentially other future parameters like fractional arterial oxygen saturation (Spa02) and total hemoglobin (SpHbt), all noninvasively.


**How should this patient be monitored?**

The "Standards for Basic Anesthetic Monitoring" are established by the American Society of Anesthesiologists and were last amended in October 2005. In summary, they are as follows:

- **Standard I:** Qualified anesthesia personnel shall be present in the room throughout the conduct of all general anesthetics, regional anesthetics, and monitored anesthesia care.
- **Standard II:** During all anesthetics, the patient's oxygenation, ventilation, circulation, and temperature shall be continually evaluated.

**Oxygenation**

- **Inspired gas:** During every administration of general anesthesia using an anesthesia machine, the concentration of oxygen in the patient breathing system shall be measured by an oxygen analyzer with a low oxygen concentration limit alarm in use.
- **Blood oxygenation:** During all anesthetics, a quantitative method of assessing oxygenation such as pulse oximetry shall be employed. When the pulse oximeter is utilized, the variable pitch pulse tone and the low threshold alarm shall be audible to the anesthesiologist or the anesthesia care team personnel. Adequate illumination and exposure of the patient are necessary to assess color.

**Ventilation**

- Every patient receiving general anesthesia shall have the adequacy of ventilation continually evaluated. Qualitative clinical signs such as chest excursion, observation of the reservoir breathing bag and auscultation of breath sounds are useful. Continual monitoring for the presence of expired carbon dioxide shall be performed unless invalidated by the nature of the patient, procedure or equipment.
Quantitative monitoring of the volume of expired gas is strongly encouraged.
. When an endotracheal tube or laryngeal mask is inserted, its correct positioning must be verified by clinical assessment and by identification of carbon dioxide in the expired gas. Continual end-tidal carbon dioxide analysis, in use from the time of endotracheal tubellaryngeal mask placement, until extubation/removal or initiating transfer to a postoperative care location, shall be performed using a quantitative method such as capnography, capnometry or mass spectroscopy. When capnography or capnometry is utilized, the end-tidal CO2 alarm shall be audible to the anesthesiologist or the anesthesia care team personnel.
. When ventilation is controlled by a mechanical ventilator, there shall be in continuous use a device that is capable of detecting disconnection of components of the breathing system. The device must give an audible signal when its alarm threshold is exceeded.
. During regional anesthesia and monitored anesthesia care, the adequacy of ventilation shall be evaluated by continual observation of qualitative clinical signs and/or monitoring for the presence of exhaled carbon dioxide.

Circulation
. Every patient receiving anesthesia shall have the electrocardiogram continuously displayed from the beginning of anesthesia until preparing to leave the anesthetizing location.
. Every patient receiving anesthesia shall have arterial blood pressure and heart rate determined and evaluated at least every 5 minutes.
. Every patient receiving general anesthesia shall have, in addition to the above, circulatory function continually evaluated by at least one of the following: palpation of a pulse, auscultation of heart sounds, monitoring of a tracing of intraarterial pressure, ultrasound peripheral pulse monitoring, or pulse plethysmography or oximetry.

Body temperature
. Every patient receiving anesthesia shall have temperature monitored when clinically significant changes in body temperature are intended, anticipated or suspected.

The reader should visit the ASA website for the most recent version of the standards [http://www.asahq.org/publicationsAndServices/standards/02](http://www.asahq.org/publicationsAndServices/standards/02) . pdf

After uneventful inhalation mask induction, cystoscopy was begun and the SpO2 was noted to decrease to 81% with the patient breathing O2 at 2 L per minute and N2O at 3 L per minute. The patient developed respiratory distress. The laryngeal mask airway was easily maintained and no gross secretions were noted. What acute diagnostic and therapeutic interventions would you perform?

Immediately the patient should be examined for airway obstruction, bilateral breath sounds and the quality of the breath sounds. The FiO2, end-tidal CO2, heart rate, blood pressure, and SpO2 should also be checked. The bladder irrigation fluid intake and output, as well as the intravenous fluid intake, should be reviewed.

Therapeutically the FiO2 should be increased immediately to 1 and a change to manually assisted ventilation should be considered. If the SpO2 does not increase rapidly in response to these interventions, then tracheal intubation and initiation of positive pressure ventilation should be performed.


What is the difference between shunt, ventilation/perfusion mismatch, and dead space?

The relative quantity of perfusion and ventilation defines these terms. A normal lung has a small portion in which perfusion and ventilation are perfectly matched. Two extreme conditions may exist at either end of the spectrum of perfectly matched perfusion and ventilation; those extremes are shunt and dead space ventilation.
In a shunt, the alveolus is perfused, but it is not ventilated. The physiologic result is hypoxemia. This is characterized by a failure of the Pao2 to rise despite an increase in the FIO2. Hypoxemia caused by small amounts of shunted blood (i.e., less than 20% of cardiac output) is easily overcome by increasing the FIO2. This is because the blood that is not shunted will contain enough "extra" dissolved oxygen to make up for the deficit that the shunted blood contributes to the combined total of blood oxygen content. Once the shunt fraction approaches 0.3 (30%), increasing the FIO2 even to 1 will not be effective in preventing hypoxemia. The term venous admixture is often used synonymously for shunt.

In dead space, the alveolus is ventilated, but it is not perfused; hence, the alveolar space is essentially "dead." The physiologic result is hypercarbia. This is characterized by a failure to maintain a normal Paco2 despite an increase in minute ventilation (i.e., tidal volume x respiratory rate).

Ventilation/perfusion mismatch is the term applied to any condition that is not a perfect match, that is, shunt or dead space. The physiologic result is a variable combination of hypoxemia and hypercarbia. The resultant degree of hypoxemia and hypercarbia depends on the relative contribution of partial shunt and dead space.


What is the differential diagnosis of pulmonary edema?

Pulmonary edema is usually thought to be either cardiac or noncardiac in etiology.

Conditions that physiologically result from an increase in the pressure within the vessel that cause transudation of fluid from inside the vessel to outside the vessel are traditionally termed congestive heart failure.
(CHF). CHF will be associated with a diminution of cardiac function and an increase in the left ventricular end-diastolic pressure.

A decrease in the pressure outside of the vessel may also result in the net movement of fluid from inside to outside. The classic example of this would be negative pressure pulmonary edema, a condition seen in young patients who are inspiring against a closed glottis.

A change in the permeability of the vessel wall is the major cause of pulmonary edema seen in acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). These conditions require a history of exposure to a risk factor, the development of bilateral infiltrates on chest radiography, and documentation of the absence of heart failure either by echocardiogram or pulmonary artery catheterization. Risk factors for ALI and ARDS include aspiration, long bone fracture, massive transfusion related acute lung injury (TRALI), pancreatitis, inhalational injury, and sepsis.

In this patient, acute fluid overload from absorption of bladder irrigation fluid should be considered. Physiologically, this picture resembles CHF and will respond to treatment with diuretics.
