

Daily anesthesia challenge: DAC

Question: Tuesday, July 31

What is the highest possible alveolar partial pressure of O₂ when a normal adult breaths 100% O₂? (Assume at sea level, respiratory quotient of 1)

Answer:

The alveolar air equation allows for the calculation of the partial pressures of the alveolar gas components. When breathing pure O₂, it will be:

$$P(\text{alv}) \text{ O}_2 = P_{\text{atm}} - (P_{\text{alv}} \text{ H}_2\text{O} + P_{\text{alv}} \text{ CO}_2) = 760 - (47 + 40) = 673 \text{ mmHg}$$

Question: Monday, July 30

Your patient with a pacemaker has serum potassium of 2.9 mEq/l. His only medication is Furosemide, which he has been taking for 2 years. Should he receive supplemental KCl before receiving an anesthetic?

Answer:

There is controversy to proceed with an elective case in the presence of chronic hypokalemia. Isolated serum potassium concentration should not be considered the only factor in anesthesia decision making. The clinical context (acute vs. chronic, acid base status, expected intraoperative hyperventilation, fluid shifts, associated electrolytes imbalances, etc) is more important than just a single extracellular K concentration.

Acute changes in extracellular potassium are of concern in asymptomatic patients with pacemaker. Chronic imbalances of potassium trigger compensatory mechanisms which restore resting membrane potential. (The normal ratio intracellular to extracellular ratio of K is 39:1, which is equivalent to -90 mV resting membrane potential). By giving potassium IV to this patient will increase extracellular K, however it will be ineffective in replacing chronic total body potassium loss (total body potassium deficits exceed 500 mEq). In addition, it may create a less negative resting membrane potential. Consequently, the action potential threshold is thus lowered and myocardium becomes more sensitive to electrical depolarization; therefore this may precipitate arrhythmias. The available clinical data do not support the practice of routine potassium replacement in asymptomatic patients with chronic hypokalemia (2.6 to 3.5 mEq/l) undergoing elective surgery.

References:

1. Hirsh et al. The overstated risk of preoperative hypokalemia. *Anesth Anal* 1988;67:133-136
2. Stoelting RK, Dierdorf SF. Electrolyte and Acid-base disturbances. In *Anesthesia and co-existing disease*. 4th ed. 2002

Question: Friday, July 27

Upon emergence from general anesthesia of a 90 yo man you noticed that the patient's end expiratory concentration of volatile anesthetic is 2% of Desflurane. (MAC awake = 0.3 MAC) The patient should respond to "open your eyes" command, however the patient is not responsive. Why?

Answer:

It has been found through meta-analysis that MAC of potent volatile anesthetics decrease by 6% per decade of age for inhaled anesthetics (in patients older than 40 years of age – the largest body of data). At an extreme age, the MAC is reduced approximately 50%. It is remarkable that the MAC-awake decreases with increasing age and it does so in a manner parallel to the effect of age in MAC itself. That is, the ratio of MAC-awake to MAC does not change with increasing age. (NB: If a patient reaches 135 years of age, he has 50% chance to stay still after surgical incision is made with no anesthesia on board – MAC approaches zero, at that age)

Reference:

Eger EI. Age, minimum alveolar anesthetic concentration, and minimum alveolar anesthetic concentration awake. *Anest Anal* 2001; 93:947-53

Question: Thursday, July 26

In what West zone of the lung must the pulmonary artery catheter (PAC) rest to have pulmonary artery wedge pressure (PAWP) reflecting accurate left atrial pressure (LAP)?

Answer:

The PAC must be in the West zone 3. In lung zones 1 and 2, alveolar pressure can exceed pulmonary venous pressure (zone 2) or both pulmonary arterial and pulmonary venous pressure (zone 1). When this occurs, the pressure sensed by the PAC can be influenced by the alveolar pressure and have a little relationship to pulmonary venous pressure, LAP or LVEDP; therefore alveolar or airway pressures are monitored rather than the LAP. Fortunately, the supine position favors the creation of zone 3. However, patients in lateral or semi-sitting position may have considerable portions of their lungs behave like zone 2.

References:

1. West JB et al. Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. *J Appl Physiol* 1964; 19:713-724.

2. O'Quin R, et al. Pulmonary artery occlusion pressure: clinical physiology, measurement and interpretation. *Am Rev Respir Dis* 1983; 128:310-326.
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Question: Wednesday, July 25

What are the ventricular function changes that occur with aging?

Answer:

With aging, we observe an increase of left ventricular tension wall with thickening of the left ventricle secondary to changes in blood pressure and vascular changes with little changes in cardiac index. Heart response to stress is blunted; plasma noradrenaline is increased. Basal sympathetic output is increased with desensitization of beta-adrenoceptors. With aging, diastolic function is decreased, with impaired relaxation.

References:

1. Ergina P, Gold S, Meakins J. Perioperative care of the elderly patient. *World J Surgery* 1993; 17: 192-8
 2. Lakatta EG. Deficient neuroendocrine regulation of the cardiovascular system with advancing age in healthy humans. *Circulation* 1993; 83: 631-6
 3. Priebe H-J. The aged cardiovascular risk patient. *Br J Anaesth* 2000; 85:763-78
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Question: Tuesday, July 24

A 7-year-old, 30-kg, asymptomatic female developed laryngo-spasm at the end of general endotracheal anesthesia for tonsillectomy and adenoidectomy with sevoflurane anesthesia without muscle relaxant. Succinylcholine (6 mg) was administered, and ventricular fibrillation ensued. What would be your initial therapy?

Answer:

It appears that the patient had hyperkalemia induced by succinylcholine with subsequent ventricular fibrillation. (Ventricular fibrillation, ventricular tachycardia, wide QRS complexes, and/or peaked T waves can persist as long as potassium levels are high. Defibrillation is ineffective in the presence of high potassium levels.) Calcium (chloride or gluconate) should be administered intravenously immediately. Calcium directly antagonizes hyperkalemia-induced depolarization of resting membrane potential. Calcium, among other electrophysiologic effects, increases the threshold potential, thereby restoring the gap between the resting membrane potential and threshold potential in the heart, and preventing spontaneous depolarization. Because calcium chloride is more likely to cause tissue necrosis with extravasation, calcium gluconate is increasingly used.

Question: Monday, July 23

You are planning a TIVA for a 90 years old patient with Remifentanyl and Propofol. Would you use the same dose of Remifentanyl in this patient, as you would use for a 40 years old? Explain your choice.

Answer:

Unique features of remifentanil are its rapid clearance and rapid k_{e0} , resulting in a rapid onset and offset of drug effect. It is tempting to speculate that these characteristics will make remifentanil an easy drug to titrate, and that clinicians will not need to consider patient covariates (age, co-morbidities etc) when choosing a dosing regimen. (However, the rapid onset of drug effect may be accompanied by rapid onset of adverse events such as apnea and muscle rigidity. The rapid offset of drug effect can cause severe patient pain when the anesthesiologist is ill equipped to deal with the problem, such as when the patient is being transported to the recovery room.)

Age is inversely correlated with the central volume distribution, clearance and potency of Remifentanyl. These combined pharmacokinetic and pharmacodynamic changes would make an elderly person more sensitive to opioids, including Remifentanyl, therefore the dosage need to be reduced.

References:

1. Egan TD, Lemmens HJ, Fiset P, Hermann DJ, Muir KT, Stanski DR, Shafer SL: The pharmacokinetics of the new short-acting opioid remifentanil (GI87084B) in healthy adult male volunteers. *Anesthesiology* 1993; 79:881-92.
 2. Minto CF. Schnider TW. Egan TD. Youngs E. Lemmens HJ. Gambus PL. Billard V. Hoke JF. Moore KH. Hermann DJ. Muir KT. Mandema JW. Shafer SL. Influence of age and gender on the pharmacokinetics and pharmacodynamics of remifentanil. I. Model development. *Anesthesiology*. 86(1):10-23, 1997.
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Question: Friday, July 20

A baby boy was just born 5 minutes ago and an arterial blood gas (room air) shows: pH 7.23 PaCO₂ 48 mmHg, PaO₂ 50 mmHg, BE -8. Is this ABG normal?

Answer:

Normal neonates are born with mild respiratory and metabolic acidosis and low arterial oxygen tension in arterial blood.

Question: Thursday, July 19

The gradient of CO₂ between arterial partial tension and alveolar tension (approximated by ET_{CO₂}) is used to evaluate the alveolar dead space (PaCO₂– ET_{CO₂}). In what physiological circumstances do we encounter negative values of the above gradient, (PaCO₂ < ET_{CO₂}) therefore making it less useful for dead space evaluation?

Answer:

1. Because of mixing in the heart and syringe, PaCO₂ sampled at the radial artery is the spatial and temporal mean of alveolar PCO₂ and therefore it is quite possible for ETCO₂ to exceed the sampled PaCO₂.
2. The increased cardiac output and increased O₂ production, reduced FRC and low compliance associated with pregnancy may result in greater cyclical variations in alveolar PCO₂ during a respiratory cycle and also in more alveoli with long time constants.
3. The PCO₂ of most alveolar gas is less than PaCO₂ but, in the terminal part of the expiration, PCO₂ rises rapidly and may exceed PaCO₂. The combined effect of these two mechanisms increases the slope of phase III and the likelihood of sampling a PETCO₂ greater than PaCO₂.
4. The presence of a wide range of V/Q mismatching and reduced FRC may result in negative P(a-ET)CO₂ values in patients after cardiopulmonary bypass.
5. Increased CO₂ production and reduced FRC may be responsible for the negative P(a-ET)CO₂ values observed in infants.

References:

1. Fletcher R. Dead-space and the single breath test for carbon dioxide during anaesthesia and artificial ventilation. *Br J Anaesth* 1984;56:109-19.
2. Shankar KB, et al. Arterial to end-tidal carbon dioxide tension difference during cesarean section anaesthesia. *Anaesthesia* 1986;41:698-702.
3. Russell GB, et al. Stability of arterial to end-tidal carbon dioxide gradients during postoperative cardiorespiratory support. *Can J Anaesth* 1990;37:560-6.
4. Rich GF et al. Continuous end-tidal CO₂ difference sampling within the proximal endotracheal tube estimates arterial CO₂ tension in infants. *Can J Anaesth* 1991;38:201-3.

Question: Wednesday, July 18

What happens if you put the wrong volatile anesthetic in a vaporizer calibrated for another volatile anesthetic? (For example: Isoflurane is put in a Halothane vaporizer)

Answer:

The incorrect agent in a agent specific vaporizer will deliver either an overdose or under dose. The most important factor in determining the direction of error is the VAPOR PRESSURE. If an agent with high vapor pressure is put into a vaporizer meant for a less vapor pressure volatile agent, the output will be excessive. If an agent with a lower vapor pressure than the agent intended for the vaporizer is accidentally used, the anesthetic output will be lower than anticipated. Halothane and Isoflurane have similar vapor pressure, however if they are interchanged, the outputs will still not be accurate. The use of Isoflurane in a Halothane vaporizer will give 25-50% more anesthetic output than the dial setting.

Reference:

1. Dorsh JA Understanding anesthesia equipment. 1994; 91-148.
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Question: Tuesday, July 17

What are the changes of FRC following induction of general anesthesia with Sodium Thiopental? What if Ketamine is used instead?

Answer:

The FRC is reduced by 20% during general anesthesia induced with Thiopental and maintained with volatile anesthetics and narcotics, irrespective of whether breathing is controlled or spontaneous. An initial decrease of 200 ml occurs with loss of consciousness induced by barbiturates. With Ketamine, however, no change occurs in either adults or children.

References:

1. Wahba RWM: Perioperative functional residual capacity. Can J Anesth 1991; 38:3, 384-400.
 2. Shulman et al. The effect of ketamine on FRC in young children. Anesthesiology 1988; 62 551-556.
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Question: Monday, July 16

A patient with moderate mitral stenosis, chronic pulmonary hypertension undergoes emergent exploratory laparotomy for bowel obstruction under general anesthesia with Isoflurane in O₂ and air, Fentanyl and Cisatracurium. Her vital signs are: BP 85/50 mmHg, HR 110/min, sinus tachycardia and RBBB, CVP 19, PA 55/30 mmHg, PCWP 20 mm Hg. What is your estimation for her LV preload? Explain.

Answer:

Preload is the force (i.e. load) acting to stretch the resting myofibril. Preload is commonly estimated as the LV end-diastolic pressure, diameter, or volume. This force, which acts to stretch the myocardium, is resisted by the stiffness of the LV muscle.

Major clinical errors would result if the high absolute values of PADP (pulmonary diastolic pressure) or PCWP were presumed to indicate increased preload in this patient. A patient with mitral stenosis resulting in pulmonary hypertension who develops tachycardia with RBBB has 4 reasons for PADP to overestimate LVEDP, which is used as a "surrogate" to estimate preload of the patient.

It has been shown there is a poor correlation between PAD and LVED volume measured by radionuclide angiography and between PAD and LVED area measured by TEE. Dr.

Thys demonstrated a close relationship between LVED volume and cardiac index. This relationship did not hold for cardiac index and PCWP. There are several reports of TEE being helpful in diagnosing hypovolemia when systemic hypotension has been accompanied by high filling pressures. In addition, compared with measurements of CVP, PAD and PCWP, TEE was much more sensitive in detecting hypovolemia.

References:

1. Leung JM, et al. TEEE: prediction of intraoperative hypovolemia. *Anesth Anal* 1990; 70:S236
 2. Clements FM, Harpole: Estimation of LV volume and EF by 2D TEE: comparison of short axis imaging and simultaneous radionuclide angiography. *Br J Anaesth* 1990; 64:331
 3. Mark JB: Predicting left ventricular end-diastolic pressure. In *Atlas of cardiovascular monitoring*. 1998; 60-65.
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Question: Friday, July 13

Which area of the kidney is most at risk for ischemic necrosis and why?

Answer:

The external medullary region of the kidney receives the lowest rate of the blood flow, about 40% of the rate received by the renal cortex. The medullary area is comprised mostly of the tubules of the thick ascending loop of Henle. These tubules create the hyperosmotic medullary gradient necessary for the countercurrent exchange mechanism of urinary concentration. This is done via Na⁺/ K⁺ ATP-ase pump responsible for 70-90% of renal O₂ consumption. Therefore the medullary area has high O₂ consumption and low O₂ delivery, being on the verge of hypoxia even in normal kidney.

References:

1. Brezis M, Rosen S. Hypoxia of the renal medulla: its implications for disease. *New Engl J Med* 1995;332: 647-655.
 2. Zimmerhackl B et al. The medullary microcirculation. *Kidney Int* 1987; 31:641-647.
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Question: Thursday, July 12

What are the differences of blood pressure recordings between the aortic root and radial artery?

Answer:

As the arterial blood pressure wave travels from the central aorta to the periphery, several characteristic changes occur.

Compared with central aortic pressure, peripheral arterial waveforms have higher systolic pressure, lower diastolic pressure, and thus wider pulse pressure.

Furthermore there is a delay in the arrival of the pressure pulse at the peripheral sites, so that the systolic pressure upstroke begins approximately 60 msec later in the radial artery than in the aorta. This temporal must be borne in mind when timing cardiac mechanical events from a peripheral arterial waveform.

Finally, despite the morphologic and temporal differences between peripheral and central arterial waveforms, the MAP in the aorta is just slightly greater than in the radial artery.

References:

1. Abrams JH, et al. Cardiopulmonary monitoring. In Wilmore DW, Brennan MF, HArken AH at al. Care of the surgical patient. Critical Care. 1989:1-27
 2. O'rourke MF, Yaginuma T. Wave reflections and the arterial pulse. Arch Intern Med 1984; 144:366-71.
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Question: Wednesday, July 11

In the operating room, we encounter the apnoeic oxygenation everyday (rapid sequence induction, difficult ventilation/ difficult intubation, thoracic surgery, laryngeal surgery, etc). What are the changes of blood gases within first minute of apnea? What are changes at 15 minutes?

Answer:

In adults, during apneic oxygenation, almost all CO₂ produced within the body is retained and the PaCO₂ rises approximately 6 mmHg in the first minute because of the wash-in of venous blood into the arterial compartment (venous blood has a CO₂ tension 6 mmHg higher than that of arterial blood) and then 3-4 mmHg each minute thereafter because of normal CO₂ production. This can differ if the patient has been hyperventilated prior apnea. The rate of increase in CO₂ tension in adults is reduced by 1/3 when apnea is preceded by hyperventilation to an initial CO₂ tension of 14 mmHg.

The end result is respiratory acidosis with acidemia. The reduction of pH in adults represents an increase of in H⁺ ion of 224% in 15 minutes.

The rate of decrease in O₂ tension in adults was reported at 9 mmHg per minute. In pediatric population the rate of decrease in O₂ tension is 3 times more than adults: approximately 31 mmHg. The difference between adults and pediatric patient in the rate of decrease in O₂ tension is not surprising. Pediatric patients have a higher metabolic rate and as a result, the O₂ reserve in the lungs (FRC) is exhausted more rapidly.

References:

1. Eger EI, Severinghaus. The rate of rise of PaCO₂ in apneic anesthetized man. *Anesthesiology* 1961;22:419
 2. Fraioli RL, Sheffer LA et al. Pulmonary and cardiovascular effects of apneic oxygenation in man. *Anesthesiology* 1973;39:588-596
 3. Frumin MJ, Epstein R et al. Apneic oxygenation in men. *Anesthesiology* 1959; 20 789-798.
 4. Cook TM, Wolf AR, Henderson. Changes in blood gas tensions during apnoeic oxygenation in paediatric patients. *British Journal of Anesthesia* 1998; 81: 338-342.
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Question: Tuesday, July 10

Does the duration of seizure affect the therapeutic efficacy?

Answer:

Yes. In current practice, a series of 10-15 treatments are administered at a rate of 2-3 per week. It has been reported that seizures less than 30 seconds in duration are not clinically effective. Cumulative seizure time over several treatments is also important: less than 210 seconds is without any benefit; cumulative seizure duration of more than 1000 seconds did not show additional improvement in symptoms.

References:

1. Maletsky BM. Seizure duration and clinical effect in electroconvulsive therapy. *Compr Psychiatry* 1978; 19:541-550
 2. Gaines Gy, reed DI. Electroconvulsive therapy and anesthetic considerations. *Anesth Anal.* 1986; 65:1345-1346
 3. Dew RE. Kimball JN. Rosenquist PB. McCall WV. Seizure length and clinical outcome in electroconvulsive therapy using methohexital or thiopental. *Journal of ECT.* 21(1):16-8, 2005 Mar
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Question: Monday, July 09

A pregnant woman is to undergo an appendectomy at 21 weeks gestation. The surgeon asks you if fetal monitoring should be used during the case. What are the pros and cons for FHR monitor? What would you do?

Answer:

PROs:

1. There are many factors that can influence uteroplacental blood flow and therefore fetal well-being. Neither the anesthesiologist nor the surgeon may be aware of fetal compromise if only maternal vital signs are being monitored. For example, surgical

retractors or other surgical manipulations could reduce blood flow. An otherwise acceptable maternal blood pressure may be compromising uteroplacental flow. Uterine activity may be periodically reducing uteroplacental flow and require tocolytic therapy.

2. While intraoperative electronic monitoring may difficult to interpret, sustained bradycardia in the fetal heart rate (FHR) tracing almost always indicates compromise of fetal well being.
3. Medico legally, in some jurisdictions even the nonviable fetus is protected by law as a "person" and therefore may be legally entitled to monitoring.
4. Documentation that fetal compromise has *not* occurred is reassuring to the patient, the obstetrician, the surgeon, and the anesthesiologist.

CONS

1. FHR monitoring is imprecise and has many false positives and negatives, even in the term (unanesthetized) parturient.
2. The imprecision of monitoring is even greater in very premature infants and when the mother is under general anesthesia. For example, FHR variability frequently disappears after the mother is given barbiturates for induction.
3. In preivable infants, the medical team may be placed in a difficult management dilemma by the diagnosis of fetal distress (e.g., sustained bradycardia): delivery will be fatal, but the diagnosis of distress would seem to require intervention.
4. FHR monitoring may be practically difficult during abdominal surgery, may complicate the surgical procedure, and has not been shown to improve fetal outcome.
5. Anesthesiologists and surgeons should already employ every known technique to maximize uteroplacental blood flow (careful surgical technique, maintenance of maternal blood pressure, left uterine displacement, etc.) anyway, so monitoring can only cause anxiety and medicolegal complications, not improve outcome.

Whatever decision is made, it should not be a unilateral one by any one practitioner. The surgeon, the anesthesiologist, and the obstetrician and other obstetrical personnel must jointly arrive at a consensus after careful counseling of the mother.

References:

1. Kendrick JM, Woodard CB, Cross SB. Surveyed use of fetal and uterine monitoring during maternal surgery. AORN J 1995; 62:386-92.
 2. Liu PL, Warren TM, Ostheimer GW et al. Foetal monitoring in parturients undergoing surgery unrelated to pregnancy. Can Anaesth Soc J 1985; 32:525-32.
 3. Authors: Stephen B. Corn, M.D. and B. Scott Segal, M.D. Department of Anesthesia, Harvard Medical School
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Question: Friday, July 06

What is the flow path of the fresh gas from the common gas outlet during expiration?

Answer:

During expiration, the inspiratory valve is closed. Gas flow from the flowmeter is continuous and has to flow somewhere. Gas flow follows the path of least resistance: via absorber and to the bellows and to the scavenging system (if the FGF is excessive).

Question: Thursday, July 05

What is the exit for the gas during the inspiratory phase of mechanical ventilation?

Answer:

Trick question! There is none. The ventilator relief valve is the outlet for excess gas during mechanical ventilation but it only relieves gas during exhalation. It is pressurized shut by the drive gas during mechanical inspiration. This has clinical consequences if, for example, an O₂ flush is activated during mechanical inspiration.

Question: Tuesday, July 3

You are ventilating a pediatric patient with the following settings: TV 50 ml, RR 20/min, I: E 1:1 using low flow technique (FGF 0/5 L/min). The surgical procedure has concluded. The operation ended earlier than expected. You turn off the vaporizer and turn up the fresh gas flow (FGF) to the maximum O₂ settings to wash out the volatile anesthetic while leaving the ventilatory parameters unmodified. Does this last maneuver (increasing FGF) affect the tidal volume?

Answer:

It depends on what anesthesia machine you are using. With technology, comes responsibility: know your anesthesia machine!

YES. In conventional anesthesia machine, FGF continues to flow into the breathing circuit, including during mechanical inspiration. The product: $FGF \times \text{inspiratory time} =$ augmentation of the tidal volume. $VT (\text{delivered}) = VT (\text{set}) + (FGF \times \text{inspiratory time})$. Furthermore the spirometer will falsely report the tidal volume because it includes the breathing hose compliance. During volume controlled ventilation of neonates and children, these discrepancies may constitute an overwhelming percentage of desired tidal volume.

NO. The new anesthesia machines are equipped with fresh gas decoupling system (FGD) that simply delivers the set tidal volume to the patient independently of fresh gas flow settings. FGD is accomplished in a variety of ways, all of which have increased the complexity of the circuits, valves, feedback and control mechanisms.

Question: Monday, July 2

During an anesthetic, an ASA-I healthy patient suddenly begins to desaturate. You check the oxygen analyzer which shows falling concentrations of O₂. You suspect that the gas supplied by the O₂ pipeline may not be O₂. You open the reserve O₂ cylinder while O₂ pipeline is still connected. Will SpO₂ of the patient improve?

Answer:

In the United States, the O₂ pipeline pressure is 45-55 psig whereas pressurized O₂ in the cylinder (2200 psig – full cylinder) is down regulated to 40 psig by the O₂ cylinder pressure regulator. Therefore the pipeline's higher pressure will prevent flow from the down regulated O₂ cylinder. If the pipeline pressure falls below the down regulated cylinder pressure, then O₂ will flow from the opened cylinder.

In the above scenario the SpO₂ will not improve because the pipeline will supply the unknown (non-O₂) gas to the anesthesia machine and the patient.

The pipeline must be disconnected or at a supply pressure lower than the down-regulated cylinder pressure for O₂ to flow from an open O₂ cylinder.