Respiratory function – Dead space

Definition

Dead space is the volume of a breath that does not participate in gas exchange. It is ventilation without perfusion. Physiologic or total dead space is the sum of anatomic dead space and alveolar dead space. Anatomic dead space is the volume of gas within the conducting zone (as opposed to the transitional and respiratory zones) and includes the trachea, bronchus, bronchioles, and terminal bronchioles; it is approximately 2 mL/kg in the upright position. Alveolar dead space is the volume of gas within unperfused alveoli (and thus not participating in gas exchange either); it is usually negligible in the healthy, awake patient. The ratio of physiologic dead space to tidal volume is usually about 1/3.

Factors that increase dead space:

- General anesthesia – multifactorial, including loss of skeletal muscle tone and bronchoconstrictor tone
- Anesthesia apparatus/circuit
- Artificial airway
- Neck extension and jaw protrusion (can increase it twofold)
- Positive pressure ventilation (i.e. increased airway pressure)
- Upright posture as opposed to supine (because of decreased perfusion to the uppermost alveoli)
- Pulmonary embolus, PA thrombosis, hemorrhage, hypotension, surgical manipulation of pulmonary artery tree – anything that decreases perfusion to well-ventilated alveoli
- Emphysema (blebs, loss of alveolar septa and vasculature)
- Age
- Anticholinergic drugs

To calculate the physiologic dead space, the following equation can be used:

$$V_D = V_T \times (PaCO_2 - PexpCO_2) / PaCO_2$$

V_D = physiological dead space (volume)
V_T = tidal volume
$PexpCO_2$ = expired PCO_2
$PaCO_2$ = arterial PCO_2

Sources

1. Anesthesiology review, 3rd ed. / Faust, Ronald J. Churchill Livingston, 2002.
2. Butterworth, John F. *Morgan & Mikhail's Clinical Anesthesiology,* 5th ed., McGraw-Hill, 2013.

Shunt and Venous Admixture

Definition

- **Shunt** is the blood which enters the systemic arterial circulation without participating in gas exchange
- **Venous admixture** is that amount of mixed venous blood which would have to be added to ideal pulmonary end-capillary blood to explain the observed difference between pulmonary end-capillary PO_2 and arterial PO_2
- **Shunt fraction** is the calculated ratio of venous admixture to total cardiac output
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- **The shunt equation,** otherwise known as the Berggren equation, is used to calculate the shunt fraction:

- $Qs/Qt = (Cc_{O2} - Ca_{O2}) / (Cc_{O2} - Cv_{O2})$
  where
  Qs/Qt = shunt fraction (shunt flow divided by total cardiac output)
  Cc_{O2} = pulmonary end-capillary O_2 content, same as alveolar O_2 content
  Ca_{O2} = arterial O_2 content
  Cv_{O2} = mixed venous O_2 content

Blood passing through areas of absolute shunt receives no oxygen (V/Q = 0), therefore, arterial hypoxemia resulting from intrapulmonary shunt is minimally responsive to supplement oxygen. Shunt effect (venous admixture or low V/Q) is the more common clinical scenario in which areas of lung have poor alveolar ventilation compared to the degree of alveolar perfusion ($0 < V/Q < 1$).

Because these areas still participate in gas exchange, albeit small, arterial hypoxemia improves with supplemental oxygen. Normally, a small percentage of venous blood from the pleural, bronchiolar and thebesian veins bypasses the right ventricle and empty into the left atrium; this represents a true anatomic shunt, which is 2-5% of cardiac output.

The arterial hypoxemia that arises from hepatopulmonary syndrome also represents an intrapulmonary anatomic shunt secondary to pulmonary AV malformations. Other disease states that represent an absolute shunt include acute lobar atelectasis, ARDS, advanced pulmonary edema and consolidated pneumonia.

Disease states that create venous admixture or low V/Q states include mild pulmonary edema, post-op atelectasis, and COPD. All these disease states are normally mitigated by hypoxic pulmonary vasoconstriction, which refers to the attempt of normal lungs to optimally match ventilation and perfusion. This response constricts vessels in poorly ventilated regions of the lung and directs pulmonary blood flow to well-ventilated alveoli. Distinguishing between true shunt and reduced V/Q can also be performed noninvasively by simultaneously plotting SaO_2 versus FiO_2. A true shunt shifts the curve downward, whereas venous admixture (low V/Q) shifts the curve rightward.

- Physiological shunt:
  - Anatomical shunt
    - Bronchial veins
    - Thebesian veins
  - Functional shunt
    - V/Q scatter
- Pathological shunt:
  - Intracardiac shunt
  - Pulmonary AVM
  - Intrapulmonary shunt (true shunt)

Sources

1. Miller, RD et al. Miller's Anesthesia, 7th edition, Churchill Livingstone: p 2026-33. 2009
2. Barash, PG. Clinical Anesthesia, 6th ed. (Philadelphia), p. 240, 2009

Chest wall compliance: Calculation

Definition

Chest wall compliance is the opposite of elasticity, and elasticity is the tendency of lung tissue to return to its original (or relaxed) position after an applied force has been removed. Compliance is essentially the ability of the lung tissue to “absorb” the same applied force, which generally results from a change in intrathoracic pressure.

Chest wall compliance can be calculated by this formula:

Compliance = Δ V / Δ P

Where change in volume is in liters and change in pressure is in cm H_20.

There are two different types of compliance: static and dynamic. **Static compliance is a measured during plateau (rather than peak inspiratory) pressure.** PEEP should be subtracted from plateau inspiratory pressure in making this calculation. Because the pressure-volume curve (see below) is not linear during inspiration and expiration due to changes in the lung tissue, **Dynamic compliance varies and is a calculated with a measurement of tidal volume at a given intrathoracic pressure during which there is airflow through the lungs at any point during inspiration or expiration.**

Lungs with low compliance are stiff lungs and will require much greater pressure to reach a given volume compared to lungs that have high compliance. There are several factors that affect lung compliance including alterations in the ribs (ie. fractures), ossification of the costal cartilage, obesity, muscular or neural changes to intercostal muscles (ie. paralysis or strain/pain), position (prone/supine), structural abnormalities (ie. kyphosis or scoliosis), increased intraabdominal pressure, and age.

Normal chest wall compliance in adults is approximately 100-200ml/cm H_2O. However, children have far lower chest wall compliance at 2.5-5.0ml/cm H_2O.

Another way to determine chest wall compliance is graphically using a pressure volume curve. The slope of the line in the is equal to the lung compliance.

Subspecialty

General

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Keyword history

- 59%/2015
- 11%/2012

Similar Keyword:

Lung compliance: Measurement

Sources

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5. MECHANICS OF VENTILATION