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Acute respiratory distress syndrome - the ultimate non-cardiogenic pulmonary oedema

Johan P Schoeman BVSc, MMedVet, PhD, DSAM, Dipl.ECVIM
SAVC, RCVS and European Specialist in Veterinary Internal Medicine
Professor: Department of Companion Animal Clinical studies
Faculty of Veterinary Science, University of Pretoria, Onderstepoort, South Africa

Abstract

Acute respiratory distress syndrome (**ARDS**) is a type of respiratory failure characterised by the rapid onset of diffuse inflammation in the lungs.

Clinical signs include dyspnoea, tachypnoea and cyanosis. Current consensus holds that "acute respiratory distress syndrome" is the best term for this condition, instead of the former "adult respiratory distress syndrome" because ARDS can affect people of all ages.

Globally, ARDS affects more than 3 million people a year and an, as yet, an unknown number of dogs and cats. As far as South African veterinarians are concerned, ARDS most frequently occurs in cases of complicated canine babesiosis and, in those with aspiration pneumonia. Moreover, ARDS is the leading cause of death in people affected by Covid-19 disease - the worldwide pandemic caused by the SARS CoV-2 virus.

Other causes of ARDS include pancreatitis, trauma, bacterial-, viral-, protozoal- or all causes of pneumonia as well as sepsis. The underlying mechanism involves diffuse injury to the cells lining the alveoli, surfactant dysfunction, inappropriate activation of the immune system leading to systemic inflammatory response syndrome (SIRS) and a concomitant coagulopathy.

Ultimately, ARDS impairs the lungs' ability to exchange oxygen and carbon dioxide and is the most fatal form of non-cardiogenic pulmonary oedema.Diagnosis is based on a PaO_2/FiO_2 ratio (ratio of partial pressure arterial oxygen and fraction of inspired oxygen) of less than 300 mm Hg (normal is 400-500 mmHg), despite a positive-end-expiratory pressure (PEEP) during ventilation of more than 5 cm H_2O. There are modified diagnostic criteria for children and areas of the world with fewer resources.

Importantly, cardiac-related pulmonary oedema must be excluded before this diagnosis can convincingly be made.

The primary treatment involves mechanical ventilation together with treatments directed at the underlying cause.

Low volume, low-pressure mechanical ventilation is the current modality of choice. If oxygenation remains insufficient, certain lung recruitment manoeuvres and neuromuscular blockers may be used.

If these also prove insufficient, extracorporeal membrane oxygenation (ECMO) may be an option, but this is unfortunately only available at certain centres in first world countries. Despite these treatment options, the syndrome is associated with a death rate between 35 and 50 % in people and between 84% and 100% in dogs and cats.

Brief historical background

Early reports from World War II described findings consistent with the modern definition of ARDS, although it was not recognised as a clinical entity yet. Specifically, many previously healthy young soldiers, who had been successfully resuscitated in the field, continued to develop multiple organ failure days to weeks later.

Acute respiratory distress syndrome was first described in 1967 *(*Ashbaugh *et al*., 1967*)*. Initially, there was no clearly established definition, which resulted in controversy regarding the incidence of ARDS. Later, in 1988, an expanded definition was proposed, which quantified physiological respiratory impairment.

Then, In 1994, a new definition was recommended by the American-European Consensus Conference Committee which recognised the variability in the severity of pulmonary injury (Bernard et al., 1994).

This definition required that the following criteria be met:

- acute onset, persistent dyspnoea
- bilateral infiltrates on chest radiograph consistent with pulmonary oedema
- hypoxemia, defined as $PaO_2:FiO_2 < 200$ mmHg
- absence of left atrial (LA) hypertension

If $PaO_2:FiO_2$ was < 300 mmHg, this definition recommended the classification of "acute lung injury" (ALI). Note that according to these criteria, arterial blood gas analysis and chest radiographs were required for a diagnosis.

However, in 2012, the Berlin Definition of ARDS was devised by the European Society of Intensive Care Medicine and endorsed by the other relevant societies.

These recommendations were an effort to improve clinical usefulness and to clarify terminology.

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Notably, the Berlin guidelines discourage the use of the term "acute lung injury", because the term was commonly being misused to characterise a less severe degree of lung injury.

Instead, ARDS was classified as mild, moderate or severe according to arterial oxygen saturation (Ranieri *et al.*, 2012). As a result, the Berlin definition (see the section on diagnosis) represent the current international consensus guidelines for both the clinical and research classifications of ARDS in humans.

In dogs and cats, we are still left with an old 2007 definition, in which ALI is seen as a clinical entity (Wilkins *et al.*, 2007).

Pathophysiology

The **pathophysiology of acute respiratory distress syndrome** involves the accumulation of fluid in the lungs not readily explained by heart failure – in other words, non-cardiogenic pulmonary oedema.

It is typically provoked by an acute injury to the lungs that results in vascular leakage, alveolar epithelial damage and consequent flooding. The triggering insult to the lung tissue results in a release of cytokines and other inflammatory mediators secreted by local epithelial and endothelial cells. After this, inflammatory cells such as neutrophils and T-lymphocytes migrate into the inflamed lung tissue and contribute to the amplification of the phenomenon.

The above changes are indicative of the initial exudative phase of ARDS. Typical histological findings involve diffuse alveolar damage and hyaline membrane formation in alveolar walls. This hyaline membrane formation progress and the alveolar lumens fill with fibrin and cell debris. Fibroplastic proliferation, initially in the pulmonary interstitium and later in the alveolar walls, leads to narrowing and collapse of airspaces and characterises the second proliferative phase of ARDS. Thirdly, the fibrotic phase is the final morphologic stage of lung injury in ARDS before recovery. This fibrosis, in fact, begins much earlier in the syndrome. Although the triggering mechanisms are not completely understood, recent research has examined the role of, *inter alia*, inflammation and mechanical stress.

Inflammation, following systemic sepsis, causes pulmonary microvascular endothelial cell dysfunction, with resultant fluid leakage from capillaries and impaired drainage of fluid from the lungs – the so-called exudative phase. Incidentally, it is worth keeping in mind that the lungs are the "first stop" for inflammatory products from anywhere in the body. In the secondary phase, endothelial cell dysfunction causes cells and inflammatory exudate to enter and accumulate in the alveoli.

This non-cardiogenic pulmonary oedema increases the thickness of the interstitial layer separating the blood in the capillary from the air in the alveolar space, which increases the distance that the oxygen must diffuse to reach the blood. The ensuing impaired gas exchange leads to hypoxia, increased work of breathing and induces inexorable alveolar scarring, which culminates in proliferative and fibrotic changes within the lungs, as mentioned above.

Fluid accumulation in the lungs and decreased surfactant production by the damaged type II pneumocytes may cause alveoli to collapse or become completely saturated with fluid. A brief revision of the normal fluid mechanics in the lung is necessary at this stage to understand ARDS better. Fluid normally accumulates in the lung interstitium as a result of an imbalance between an increase in hydrostatic pressure, a decreased oncotic pressure, as well as impaired lymphatic drainage or increased vascular permeability. Hence, classic cardiogenic pulmonary oedema is a consequence of mitral valve insufficiency, secondary to an increase in pulmonary vascular hydrostatic pressure. This oedema fluid contains little protein and readily responds to diuretic therapy, mainly because capillary endothelial permeability is unchanged.

Conversely, in most forms of non-cardiogenic pulmonary oedema and especially in ARDS, vascular permeability is increased as a result of direct and indirect lung injury. Therefore, in contrast to cardiogenic oedema, non-cardiogenic oedema fluid is high in protein, which modifies oncotic pressure gradients, further altering fluid flux.

The resultant accumulation of interstitial fluid decreases lung compliance, compresses smaller airways and floods alveoli with fluid – all of which ultimately increase pulmonary vascular resistance, which further exacerbates pulmonary vascular hydrostatic pressure leading to pulmonary hypertension. Moreover, this progressive loss of aeration contributes to the "right to left" shunting of blood in ARDS. A traditional right-to-left shunt refers to blood passing from the right side to the left side of the heart without travelling to the capillaries of the lung (e.g., as seen in reverse patent ductus arteriosus).

Yet, in ARDS, right-to-left shunting occurs within the lungs, because blood from the pulmonary artery will eventually enter capillaries unable to exchange gas with the damaged, fluid- and debris-filled alveoli.

Consequently, the blood flowing through the alveolar capillaries become progressively less oxygenated, resulting in massive shunting within the lung. As alluded to, the collapse of the alveoli and the terminal bronchioles further interferes with the process of normal gas exchange. As a result, it is common to see patients with a PaO_2 of 60 mmHg, despite being mechanically ventilated with 100% inspired oxygen.

This relentless loss of aeration may follow different patterns, depending upon the nature of the underlying disease and other factors (see Table 1 for factors predisposing to ARDS in dogs). In sepsis or trauma-induced ARDS, the lung infiltrates are usually more patchy. The dorsal and caudal lung segments tend to be worse

affected and the distribution is less homogeneous than in, for example, aspiration-induced ARDS, which commonly involves the ventral lung lobes. Loss of aeration also causes important changes in mechanical stress that are fundamental to the amplification of inflammation and the progression to ARDS in mechanically ventilated patients.

Mechanical stress

As the loss of aeration and the underlying disease progress, the end-tidal volume (Vt) increases to a level incompatible with life. Thus, mechanical ventilation has to be initiated to relieve the respiratory muscles of their work and to protect the affected patient's airway.

However, mechanical ventilation, *per se*, constitutes a risk factor for the development (or the worsening) of ARDS – as is currently found in the COVID 19 patient that is ventilated too early in the course of the disease. Aside from the infectious complications that potentially arise from endotracheal intubation, the positive-pressure ventilation also directly alters lung mechanics during ARDS.

This form of mechanical stress in the alveolus is thought to be caused by the pulmonary pressure gradient (P_l) generated by the ventilator or, better still, its cyclical variations. The better outcome obtained in individuals ventilated with a lower volume is thought to result from the beneficial effect of the lower P_l. The generated P_l applied on the alveolar surface determines the shear stress to which alveoli are exposed (colleagues interested in the pathophysiology of myxomatous mitral valve degeneration will also be familiar with the role of shear stress in this condition). Moreover, ARDS is characterised by a heterogeneous reduction of the airspace, thus a tendency towards higher P_l for the same V_t, and ultimately towards higher stress on fewer lung units.

Additionally, not all alveoli are similarly affected and this heterogeneity of alveoli at different stages of the disease is further exacerbated by the gravitational gradient to which they are exposed and the different perfusion pressure at which blood flows through them. Alveoli are "kept open" using PEEP, a feature of modern ventilators which maintains a positive airway pressure throughout the whole respiratory cycle.

Although a higher mean pressure throughout the whole respiratory cycle slows the collapse of diseased alveoli, this benefit must be weighed against the corresponding elevation in plateau pressure. Consequently, newer ventilatory approaches attempt to maximize mean airway pressure for its ability to "recruit" collapsed alveoli, while minimizing the shear stress caused by the frequent opening and closing of aerated units.

Diagnosis

Diagnostic criteria for ARDS have changed over time as understanding of pathophysiology has evolved.

The international consensus criteria for ARDS were most recently updated in 2012 and are known as the "Berlin definition"(Ranieri *et al*., 2012).

They broadened the diagnostic thresholds, discouraged the term "acute lung injury" and defined grades of ARDS severity according to the degree of hypoxia.

According to the 2012 Berlin definition, ARDS is characterised by the following:

- lung injury of acute onset, within 1 week of an apparent clinical insult and with the progression of respiratory symptoms
- bilateral opacities on chest imaging (chest radiographs or CT scans) not readily explained by other lung pathologies such as intraparenchymal bleeding secondary to rodenticide poisoning, effusions, lobar/lung collapse or nodules
- respiratory failure not explained by heart failure or volume overload
- decreased PaO_2/FiO_2 ratio that indicates reduced arterial oxygenation from the available inhaled gas
- Classification into mild, moderate or severe ARDS

    - *mild ARDS*: PaO_2/FiO_2 ratio = 201 – 300 mmHg
    - *moderate ARDS*: PaO_2/FiO_2 ratio = 101 – 200 mmHg
    - *severe ARDS*: PaO_2/FiO_2 ratio ≤ 100 mmHg

    º Note that the Berlin definition requires a minimum PEEP of 5 cmH_2O for consideration of the PaO_2/FiO_2 ratio. This degree of PEEP may be delivered noninvasively with continuous positive airway pressure (CPAP) to diagnose mild ARDS.

Notably, equivalent criteria have not been validated for veterinary patients. In addition, a recent study in dogs and cats demonstrated marked discrepancies in clinical and pathologic criteria, which indicates that there is still a lot of refinement required in this arena (Boiron *et al*., 2019).

Diagnostic imaging

Radiologic imaging has been a longstanding criterion for the diagnosis of ARDS. Original definitions of ARDS specified that correlative chest radiographic findings were required for diagnosis.

However, the diagnostic criteria have been expanded over time to accept CT and ultrasound findings as equally contributory.

Generally, radiographic findings of fluid accumulation (pulmonary oedema) affecting both lungs, which is unrelated to increased cardiopulmonary vascular pressure (such as in left heart failure), may be suggestive of ARDS. Ultrasound findings suggestive of ARDS include the following:

- Sub-pleural consolidations
- Absence or reduction of lung sliding

- "Spared areas" of normal parenchyma
- Pleural line abnormalities (irregular thickened and fragmented pleural line)
- Non-homogeneous distribution of B-lines or lung rockets (a characteristic ultrasound finding suggestive of fluid accumulation in the lungs) (Volpicelli *et al*., 2012)

Importantly, echocardiography is used to rule out mitral valve disease as a cause of lung disease and to document right heart involvement secondary to pulmonary hypertension.

Treatment

Acute respiratory distress syndrome is usually treated with mechanical ventilation in the intensive care unit (ICU). Mechanical ventilation is usually delivered through an endotracheal tube or by tracheostomy when prolonged ventilation (≥2 weeks) is necessary. The role of non-invasive ventilation (NIV) is limited to the early period of the disease or to prevent worsening respiratory distress in patients with atypical pneumonia, lung bruising or in patients undergoing major surgery, who are at risk of developing ARDS.

Treatment of the underlying cause is crucial. Appropriate antibiotic therapy is started as soon as culture results are available or if an infection is suspected, partly based on serum CRP levels. Where possible, the origin of the infection is removed. When sepsis is diagnosed, appropriate local protocols are followed.

Mechanical ventilation

The overall goal of mechanical ventilation is to maintain acceptable gas exchange to meet the body's metabolic demands and to minimise adverse effects in its application. Previously, mechanical ventilation aimed to achieve relatively high tidal volumes (V_t) of 12–15 ml/kg (where the weight is ideal body weight rather than actual oedematous weight). Yet, recent studies have shown that high tidal volumes can overstretch alveoli, resulting in volume trauma with secondary lung injury.

The ARDS Clinical Network completed a clinical trial that showed improved mortality when people with ARDS were ventilated with a low tidal volume of 6 ml/kg compared to the traditional 12 ml/kg. Low tidal volumes (V_t) may however cause a rise in blood carbon dioxide concentrations (permissive hypercarbia) and alveolar collapse, because of their inherent tendency to increase shunting within the lung. Low tidal volume ventilation turned out to be the primary independent variable associated with reduced mortality in the ARDSnet trial (Brower *et al*., 2000). A patient with ARDS, on average, spends between 8 and 11 days on a mechanical ventilator;

Positive end-expiratory pressure

Positive end-expiratory pressure (PEEP) is used in mechanically ventilated people with ARDS to improve oxygenation. In ARDS, three populations of alveoli can be distinguished.

There are normal alveoli that are always inflated and engaged in gas exchange, flooded alveoli that can never, under any ventilatory regime, be used for gas exchange and atelectatic or partially flooded alveoli that can be "recruited" to participate in gas exchange under certain ventilatory regimens.

The recruitable alveoli, in turn, represent a continuous population, some of which can be recruited with minimal PEEP and others that can only be recruited with high levels of PEEP.

An additional complication is that some alveoli can only be opened with higher airway pressures than are needed to keep them open, hence the justification for manoeuvres where PEEP is increased to very high levels for seconds to minutes before subsequently dropping the PEEP to a lower level. PEEP can be harmful; high PEEP necessarily increases mean airway pressure and alveolar pressure, which can damage normal alveoli by overdistension. As a result, a compromise between the beneficial and adverse effects of PEEP is necessary.

Extracorporeal membrane oxygenation

Extracorporeal membrane oxygenation (ECMO) is mechanically applied to prolonged cardiopulmonary support. This form of mechanical ventilation is applied when the alveoli are damaged to such an extent that oxygenation has to take place by the machine outside the body.

There are two types of ECMO: venovenous which provides respiratory support and venoarterial which provides respiratory and hemodynamic support. People with ARDS, who do not require cardiac support, typically undergo venovenous ECMO.

Prone position

The position of lung infiltrates in acute respiratory distress syndrome is non-uniform. Repositioning into the prone position (face down) in people might improve oxygenation by relieving atelectasis and improving perfusion. If this is done early in the treatment of severe ARDS, it confers a mortality benefit of 26% compared to supine ventilation (Sud *et al*., 2014).

Fluid management

Several studies have shown that pulmonary function and outcome are better in people with ARDS who lost weight or whose pulmonary wedge pressure was lowered by diuresis or fluid restriction.

Hence, furosemide is often used as low dose intermittent boluses of 1 mg/kg IV q 6 – 12 hrs or as a CRI of 0.2 mg/kg/hr, whilst guarding against dehydration and reduction of blood pressure and consequent effects on vital organ function.

Colloids are not indicated, because the altered permeability of the capillary-alveolar membrane causes the fluid to accumulate in the interstitial space.

Medications

Currently, it is still uncertain whether or not treatment with corticosteroids improves overall survival. However, corticosteroids may increase the number of ventilator-free days during the first 28 days of hospitalisation (Lewis *et al.*, 2019).

Inhaled nitric oxide (NO) selectively widens the lung's arteries which allows for more blood flow to the opened alveoli and evidence of increased oxygenation status, Yet, despite this, no evidence exists that inhaled nitric oxide decreases morbidity and mortality in people with ARDS (Adhikari *et al*., 2007).

Furthermore, nitric oxide may cause kidney damage and is no longer recommended as therapy for ARDS regardless of severity (Adhikari *et al*., 2014). In addition, there is no evidence that treatments with exogenous surfactants, statins, beta-blockers or n-acetylcysteine decrease early mortality, late all-cause mortality, duration of mechanical ventilation or number of ventilator-free days (Lewis *et al*., 2019).

There is ongoing research on the treatment of ARDS by interferon (IFN) beta-1a to aid in preventing leakage of vascular beds.

Traumakine (FP-1201-lyo) is a recombinant human IFN beta-1a drug, which is undergoing international phase-III clinical trials after an open-label, early-phase trial showed an 81% reduction-in-odds of 28-day mortality in ICU patients with ARDS (Bellingan *et al.*, 2014).

The drug is known to function by enhancing lung CD73 expression and increasing the production of anti-inflammatory adenosine, such that vascular leaking and escalation of inflammation are reduced (Kiss *et al.*, 2007).

Moreover, aspirin has been studied in those who are at high risk and was unfortunately not found to be useful (Fan *et al.*, 2018).

Lastly, prednisolone (0.5 – 1 mg/kg q 24 hr) is used to delay/prevent pulmonary fibrosis. However, this treatment is purely empirical and has no evidence base in the literature.

Table 1: Predisposing factors for the development of non-cardiogenic pulmonary oedema in dogs.

Neurogenic pulmonary oedema	Seizures Electrocution Head trauma
Post-obstructive pulmonary oedema	Strangulation Laryngeal paralysis Pulmonary re-expansion
Systemic diseases	Envenomation, esp. bee stings Gastric/Splenic/Mesenteric torsion Paraquat poisoning Parvoviral enteritis Sepsis Shock Severe pancreatitis Severe trauma Virulent babesiosis Uraemia
Direct pulmonary injury	Acute interstitial pneumonia Aspiration pneumonia Bacterial pneumonia Hyperoxia Lung lobe torsion Near-drowning Parasitic pneumonitis Pulmonary contusion Smoke inhalation
Profound hypoalbuminemia	Protein-losing nephropathy Lymphangiectasia Liver failure
Impaired lymphatic drainage	Lymphangitis Lymphatic neoplasia
Miscellaneous causes	Vasculitis Drowning High altitude Air embolism Phaeochromocytoma

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QUESTION 1

Which ONE of the following statements regarding ARDS is WRONG?

a. Young World War II soldiers who developed multiple organ failure days after the initial injury, did in retrospect, succumb to ARDS.
b. Causes of ARDS include pancreatitis, trauma, bacterial-, viral-, protozoal pneumonia as well as sepsis.
c. Adult respiratory distress syndrome is the correct term because ARDS affects adults.
d. The underlying mechanism involves diffuse injury to the cells lining the alveoli and surfactant dysfunction.
e. The mortality rate of ARDS in people is around 35 – 50%.

QUESTION 2

Which ONE of the following statements regarding the pathophysiology of ARDS is WRONG?

a. The exudative phase is the initial phase of ARDS.
b. The proliferative phase is characterised by progressive hyaline membrane formation.
c. During the proliferative phase, there is fibroblast proliferation in the pulmonary interstitium.
d. The fibrotic phase is the final morphologic stage of ARDS.
e. Fortunately, fibrosis is a late phenomenon and does not begin early in ARDS

QUESTION 3

Which ONE of the following statements regarding the pathophysiology of ARDS is WRONG?

a. Endothelial cell dysfunction causes cells and inflammatory exudate to enter and accumulate in the alveoli.
b. Non-cardiogenic pulmonary oedema increases the thickness of the interstitial layer, which increases the distance that the oxygen must diffuse to reach the blood.
c. The ensuing impaired gas exchange leads to hypoxia, increased work of breathing and induces alveolar scarring.
d. This scarring culminates in proliferative and fibrotic changes within the lungs.
e. Fluid accumulation in the lungs and increased surfactant production by the damaged type I pneumocytes may cause alveoli to collapse.

QUESTION 4

Which ONE of the following statements regarding the pathophysiology of ARDS is WRONG?

a. In most forms of non-cardiogenic pulmonary oedema, vascular permeability is increased as a result of direct and indirect lung injury.
b. In contrast to cardiogenic oedema, non-cardiogenic oedema fluid is low in protein.
c. The accumulation of interstitial fluid causes decreased lung compliance and compress smaller airways.
d. Increase pulmonary vascular resistance further exacerbates pulmonary vascular hydrostatic pressure leading to pulmonary hypertension.
e. Progressive loss of aeration contributes to the "right to left" shunting of blood in ARDS.

QUESTION 5

Which ONE of the following statements regarding ventilation in ARDS is WRONG?

a. Mechanical stress in the alveolus is thought to be caused by the cyclical variations in pulmonary pressure gradient (Pl) generated by the ventilator.
b. The better outcome obtained in individuals ventilated with a higher tidal volume is thought to results from the beneficial effect of the higher PI.
c. ARDS is characterised by a heterogeneous reduction of the airspace, thus a tendency towards higher Pl for the same Vt, and ultimately towards higher stress on fewer lung units.
d. All the alveoli are not similarly affected and this is exacerbated by the gravitational gradient to which they are exposed.
e. Alveoli are "kept open" using PEEP, a feature of modern ventilators which maintains a positive airway pressure throughout the whole respiratory cycle.

QUESTION 6

Which one of the following statements regarding the diagnostic criteria for ARDS is WRONG?

a. Lung injury of acute onset, within 1 week of an apparent clinical insult
b. Progression of respiratory symptoms
c. Bilateral opacities on chest imaging are not readily explained by other lung pathologies such as effusions, lobar/lung collapse or nodules
d. Respiratory failure caused by heart failure or volume overload
e. Decreased PaO2/FiO2 ratio that indicates reduced arterial oxygenation

QUESTION 7

Which ONE of the following statements regarding the ultrasonographic features of ARDS is WRONG?

a. Sub-pleural consolidations
b. Lung sliding
c. Spared areas of normal parenchyma
d. Pleural line abnormalities (irregular thickened fragmented pleural line)
e. Non-homogeneous distribution of B-lines or lung rockets

QUESTION 8

Which ONE of the following statements regarding the treatment of ARDS is WRONG?

a. It is usually treated with mechanical ventilation in the intensive care unit (ICU).
b. Mechanical ventilation is usually delivered through an endotracheal tube or by tracheostomy when prolonged ventilation (≥2 weeks) is necessary.
c. The role of non-invasive ventilation (NIV) is limited to the late period of the disease.
d. Treatment of the underlying cause is crucial.
e. Appropriate antibiotic therapy is started as soon as culture results are available or if an infection is suspected.

QUESTION 9

Which ONE of the following statements regarding the treatment of ARDS is WRONG?

a. Positive end-expiratory pressure (PEEP) is used in mechanically ventilated people with ARDS to improve oxygenation.
b. Three populations of alveoli can be distinguished – normal, flooded and recruitable.
c. The recruitable alveoli represent a continuous population, some of which can be recruited with minimal PEEP and others with high levels of PEEP.
d. There is no justification for manoeuvres where PEEP is increased to very high levels for seconds to minutes.
e. High PEEP increases mean airway pressure and alveolar pressure, which can damage normal alveoli by overdistension.

QUESTION 10

Which ONE of the following statements regarding the treatment of ARDS is WRONG?

a. Extracorporeal membrane oxygenation (ECMO) is mechanically applied for prolonged cardiopulmonary support.
b. Repositioning into the prone position (face down) in people might improve oxygenation by relieving atelectasis and improving perfusion.
c. Pulmonary function and outcome are better in people with ARDS who lost weight or whose pulmonary wedge pressure was lowered by diuresis or fluid restriction.
d. Treatment with corticosteroids improves overall survival and ventilator-free days.
e. Recombinant IFN enhances lung CD73 expression and increases the production of anti-inflammatory adenosine, such that vascular leaking and escalation of inflammation are reduced.

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