Objective information characterizing ventilation in anesthetized cattle is very limited. However, clinical experience with cattle supports the premise that hypoventilation usually accompanies the management of general anesthesia in this species. Accordingly, review of considerations for and techniques of the management of respiratory depression in cattle is appropriate. However, anatomical and physiological characteristics of cattle differ in some important ways from many other commonly anesthetized species. Such knowledge implies that direct application of principles of ventilatory support used with other, especially smaller, species may not be appropriate. Once again, nearly non-existent published results of focused studies of cattle hamper decision making in this regard. In this chapter, we will briefly review the mechanics of ventilation in cattle and the pathophysiology of intermittent positive pressure ventilation (IPPV). We will conclude with some personal opinion regarding the use of IPPV in anesthetized cattle. Discussion will focus on conditions with adult cattle. In calves less than 3 months of age, the development of the compartmented stomach is incomplete. As a result and given our present limited species-specific knowledge, we apply the same principles and techniques of mechanical ventilation to calves that is used with monogastric species.

Physiological Considerations

Cattle are large animals, and as such, many physiological considerations applicable to horses should also apply to them [1]. For example, the effects of gravity on distribution of pulmonary blood flow and ventilation are expected to be important as are the modifications observed with body positional changes from the standing posture. Large animal species tend to develop a large imbalance in the distribution of ventilation relative to pulmonary blood flow. This results in a large alveolar to arterial oxygen tension difference (PA-aO2). In conscious cows positioned in supine (dorsal) position, PaO2 significantly decreases [1,2].

On the other hand although of comparable size and weight, some mechanics of ventilation of cattle and horses are notably different. Some of these differences are likely related to differences in lung structure [3]. The bovine lung is smaller, is divided into separate lobes, each with distinct lobules separated by complete septa. Unanesthetized cows have a higher resting respiratory rate compared to horses [4,5]. The ratio of tidal volume to lung volume is higher in horses than in cattle. The maximum change in pleural pressure and the non-elastic work of breathing are higher in cattle, whereas the lung's dynamic compliance is greater in horses [5]. The difference in compliance likely accounts for the greater respiratory rate and lower tidal volume observed in cattle, as this may minimize the work of breathing. The diaphragm is more vertical and flatter in cattle than in horses, and most of the bovine lung is cranial to the abdomen. A flatter conformation results in decreased efficiency [6]. The monophasic inspiratory and expiratory gas flow of cattle also differs from that of the horse but resembles that of humans and dogs [4]. During awake, resting conditions, alveolar ventilation and oxygen consumption in cattle is greater than that of horses [7].

The ruminant stomach is immediately caudal to the diaphragm and occupies nearly three quarters of the abdomen [8]. With its high capacity of 115 - 150 L in adult cattle, or 250 - 300 g/kg of body weight [9,10] it can markedly interfere with normal ventilation, especially when the animal is positioned in dorsal recumbency. The rumen is a large fermentation chamber, prone to distension. The association of this large stomach with a compartmentalized chest wall results in the most cranial insertion of the diaphragm among mammals [8]. Therefore, increased intragastric pressure represents an additional unique pathophysiological condition for the diaphragm [11]. At moderate intragastric pressure respiratory rate increases, while at high pressure, a pattern of decreased respiratory rate, end-expired lung volume and breath-holding at end inspiration is observed [11,12]. Increased gastric pressure, gastric distension and cephalad displacement of the diaphragm results in a significant decrease in lung dynamic compliance, tidal volume, minute ventilation. As a result PaCO2 increases and PaO2 decreases [11,12].

Cattle seem particularly sensitive to respiratory tract disease. Both chronic obstructive disease and pneumonia are common.
Chronic respiratory disease causes the functional residual capacity to increase and this is associated with an abnormal distribution of ventilation. The distribution of respiratory system time constants is increased, a phenomenon likely accentuated by the limited interdependence between lobules and the lack of collateral ventilation. Pneumonia causes PaO₂ to decrease and PaCO₂, and the work of breathing to increase [13,14]. Lung dynamic compliance also decreases and pulmonary vascular resistance increases resulting in pulmonary hypertension [15]. Hypoxic pulmonary vasoconstriction is known to be intense in the bovine species. It results in pulmonary hypertension, which when severe, may lead to heart failure [16]. Cattle seem to have limited cardiovascular reserve, probably accounting for their limited athletic performance. The cardio-pulmonary response of cattle to exercise is qualitatively similar but quantitatively different than in other species [17]. The increase in cardiac index, stroke index, heart rate and cardiac power output induced by dobutamine was noted low in comparison with responses reported in humans, dogs and horses [18]. During exercise, oxygen consumption is reported to increase 4.5 - 8 fold in the bovine compared to 15 and 30 fold in dogs and horses, respectively [17]. Moreover hemoglobin concentration increases less in exercising cattle (1.1 - 3.5 g/dl) than in horses (7.5 g/dl), limiting the O₂ carrying capacity of blood. Thus, the depressant effects of anesthesia and the unwanted changes induced by IPPV can be expected to be of greater disadvantage to the bovine species.

Breed differences in respiratory and cardiovascular physiology and pathophysiology have been reported. For example, several western European bovine breeds have been commercially selected for their hereditary muscular hypertrophy phenotype. However these "double muscled" breeds seem more sensitive to respiratory disease, have less ability to compensate for changes in ventilation and have a poorer cardiac reserve [16-22]. Double muscled bovine have less aerobic metabolic capacity than conventional cattle, and are therefore more sensitive to stress and present higher morbidity and mortality rates [18]. They have lower lung weight to body weight and heart weight to body weight ratios than conventional breeds; pulmonary resistance is higher [17,18]. The distribution of pulmonary ventilation and blood flow may be different in doubled muscled breeds compared to conventional breeds [19]. Under basal physiological conditions, double muscled cattle may have a lower PaO₂ than dairy cattle and in respiratory disorders, they develop a more precocious and more severe hypoxemia [16].

**Intermittent Positive Pressure Ventilation**

IPPV is commonly used for ventilatory support of anesthetized animals. Mechanical ventilation is accomplished by generating a gas pressure in the airway that is greater than ambient and thus "forces" gas into the lungs. Inspiratory gas flow continues until the apparatus delivers a specific preset/predetermined inspiratory pressure, volume, or flow or until a specific time of gas flow is reached. With most ventilators used to facilitate anesthetic management of veterinary patients, inspiration is "limited" on the basis of either pressure or time. The distribution of ventilation within the lung is determined by inflation pressure and time and regional lung time constants. Expiration is usually accomplished by quickly returning the upper airway to ambient pressure and permitting the gas to flow out of the lungs, i.e., the pressure gradient is thus reversed favoring a passive lung deflation. Because airway pressure returns to ambient at the conclusion of expiration (i.e., air flow ceases when there is no longer a pressure gradient between the lungs and ambient), the term intermittent positive pressure ventilation defines this type of artificial or mechanical ventilation. If the airway pressure is not allowed to return to ambient pressure, then this situation is denoted by the term PEEP, i.e., positive end-expiratory pressure. PEEP is sometimes used with smaller species and humans to minimize collapse of small airways and alveoli and improve compliance of dependent regions of lung. Its effectiveness in cattle remains to be verified, but any benefit is expected at best to be minimal.

There are some undesirable side effects of IPPV. Most prominent and consistent among these is the depression of circulatory system function. The increased airway pressure causes increased intra-thoracic pressure. This event causes an unfavorable pressure gradient between peripheral veins and the right atrium so that ventricular filling decreases, which in turn results in a decrease in stroke volume, cardiac output and aortic blood pressure. These changes are in proportion to the increase in intrathoracic pressure. Though not specifically studied in anesthetized cattle to date results of studies of anesthetized horses [23-25] indicate that the hemodynamic depression frequently accompanying IPPV is most severe in magnitude of change in larger species such as horses (and presumably cattle) compared to usual clinical and laboratory experiences with smaller, monogastric animals such as dogs and cats. The application of PEEP adds further depression in this regard [26]. In addition to the mechanical effects imposed by elevated intra-thoracic pressure it is likely hemodynamics are also influenced by the magnitude of reduction in PaCO₂ accompanying effective IPPV. In animals with an intact sympathetic nervous system, CO₂ acts as a sympathetic stimulant with effects to increase cardiac output and blood pressure. Reducing PaCO₂ tends to remove this hemodynamic "brace".

Other considerations of IPPV are related to respiratory system function. For example, during spontaneous ventilation, variations in respiratory frequency and tidal volume are commonly used to clinically assess the depth of general anesthesia; a decrease in either (or both) suggesting an increasing depth of anesthesia. These signs are no longer available during controlled conditions of IPPV.

Properly conducted IPPV improves alveolar ventilation, which in turn increases the delivery and rate of rise of inhalation
anesthetic concentration. As a consequence and presuming other factors constant changes in anesthetic depth occur much more rapidly. The accelerated rate of change in anesthetic depth must be anticipated to prevent anesthetic overdose. The most dramatic adverse effect of IPPV on the respiratory system is ventilator induced lung injury (VILI). VILI is probably a rare incident in cattle with normal lungs and a peak airway pressure during IPPV of 30 cm H2O or less. On the other hand, use of high airway pressures and/or large tidal volumes may result in lung damage especially in the presence of lung disease. Two different mechanisms can induce lung injury. Barotrauma probably starts as disruption of the alveolar membrane and is usually accompanied by subcutaneous emphysema, pneumomediastinum and pneumothorax. The second mechanism, volutrauma causes VILI by overdistension of the lungs. It has been reported that high airway pressures in the absence of overdistention did not induce lung damage, suggesting that alveolar size rather than pressure was responsible for the injury [27-31]. With this form of VILI, interstitial or alveolar pulmonary edema is observed; alveolar distention inducing permeability pulmonary edema. Moreover, airway trauma may occur due to the repeated closure and reopening of small airways with each breath. Finally, VILI causes the release of inflammatory mediators from activated neutrophils; these mediators may contribute to permeability pulmonary edema and loss of surfactant function [32].

The last potential adverse effect of IPPV to be considered is the associated rise in intra-abdominal pressure. Ruminants have a preponderance for regurgitation and especially during anesthesia, risk lung aspiration of regurgitated material. The raised intra-thoracic pressure accompanying IPPV accounts for an increase of varying magnitude in intra-abdominal pressure. The increase in intra-abdominal pressure adds risk for regurgitation and lung aspiration in cattle.

**Decision: to Apply or not to Apply IPPV to Anesthetized Cattle**

The decision to apply IPPV is obvious in two circumstances: conditions of apnea and at times when drugs that inhibit the function of the respiratory muscles (e.g., neuromuscular blocking drugs such as atracurium) are used to facilitate anesthetic management. Although arterial oxygenation can be maintained for some time during apnea, the duration of apnea without harm depends upon a multitude of factors, including FIO2 prior to and during apnea, and the rate of rise of CO2. As a result, the duration of apnea before hypoxemia develops is unpredictable.

IPPV is used to correct unacceptable conditions of hypercapnia. Three major determinants of the degree of hypercapnia in anesthetized horses and presumably similarly managed cattle are anesthetic agent, anesthetic dose and duration of anesthesia. For example, some anesthetics are more potent depressants of ventilation than others (e.g., isoflurane > halothane). Thus, choice of anesthetic agent is an important consideration in the development/management of hypercapnia. Inhalation anesthetics depress ventilation in a dose-related manner, i.e., the greater the anesthetic dose the greater the magnitude of PaCO2. Management of anesthesia at a level just sufficient for necessary surgical conditions reduces the need for IPPV. Duration of anesthesia influences the efficiency of ventilation and gas exchange at least in horses (data for cattle are not available). Hence, the need for IPPV to maintain acceptable PaCO2 is greater for long vs short anesthesia times. Regardless, moderate hypercapnia is well tolerated by human and horse patients and the use of "permissive hypercapnia" is widely reported. There is no evidence to suggest that anesthetized healthy cattle react differently in this regard. Moreover, as previously noted, hypercapnia stimulates sympathetic nervous system response which contributes to maintenance of adequate hemodynamics. Consequently, in the absence of an oxygen deficit, and as long as pHa is near normal, IPPV and associated cardiovascular depression can be usually avoided when PaCO2 is 55 - 60 mmHg or less.

IPPV may be used to improve arterial oxygenation. However, because one of the most important roles of the cardiovascular and respiratory functions is O2 delivery, one must consider the benefits and disadvantages of the use of IPPV on both O2 content and blood flow. As previously noted, IPPV can reduce cardiac output. Thus an improvement in O2 content in face of a significant decrease in blood flow may actually decrease overall O2 delivery.

In summary, because IPPV has both life-saving and life-threatening influences, its merits and direct application to bovine patients requires individualized consideration.

**Techniques of IPPV for Cattle**

Internationally, there are many different ventilators available for use with adult cattle. The specifics of application will obviously depend on the specific ventilator and individual patient factors.

In general, peak inspiratory pressure should be maintained less than 25 - 30 cmH2O. This peak inspiratory pressure will usually be associated with an adequate tidal volume (i.e., 8 - 12 ml/kg). However, abdominal distension will reduce respiratory system compliance and higher pressures may be necessary. In such cases, appropriate consideration of benefits and risks of elevated peak inspiratory pressure is weighed in hopes of improved individualized therapy. Presuming an adequate tidal volume, respiratory rate is commonly adjusted within the range of 10 - 15 breaths/min. With most contemporary large animal ventilators adequate inspiratory time is about 2 - 3 sec./breath.

The adequacy of IPPV should be monitored. If end-tidal CO2 is measured, a value of 35 - 45 mmHg is appropriate with considerations for some Pa-ACO2 difference. If blood gas analysis is available a PaCO2 of 40 - 55 mmHg is satisfactory. Arterial blood gas analysis also gives valuable information on the magnitude of oxygenation and acid base status. Ideally, the
inspired O₂ should be also measured and maintained high. Because of the effects of IPPV on cardiovascular performance it is especially important to monitor for this when mechanical ventilation is used. Heart rate and rhythm (ECG) and pulse quality should be observed. It is our opinion that IPPV should not regularly be conducted in the absence of arterial blood pressure monitoring. Indirect (e.g., Doppler, oscillometric) methods may be adequate but direct measurement (e.g., auricular artery) is usually preferred. Direct measurement also provides ready access for obtaining arterial blood for blood gas analysis.

References


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