



Bradycardia and Sinus Pause with Pressure Support Ventilation and Weaning from Mechanical Ventilation

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Abstract

Background: Bradycardia during weaning from Mechanical Ventilation (MV) and Pressure Support Ventilation (PSV) is a rare occurrence, with only one such case report found in the literature. We present the case of a 23-year-old transgender (male to female) Abdominal Gunshot Wound (GSW) victim who developed significant bradycardia and sinus pause during PSV trial.

Case Presentation: The patient presented to the Emergency Department with multiple GSWs to the abdomen. She was intubated for airway protection. Exploratory laparotomy was performed and patient underwent partial liver resection for liver lacerations and right hemidiaphragm injury. She remained intubated in the Surgical Intensive Care Unit (SICU) post-operatively and had multiple planned returns to the Operating Room (OR). On hospital day 9 the patient was on minimal MV settings, 0.8 mcg/kg/h of Dexmedetomidine, and 6 mg/h of Morphine. Continuous Positive Airway Pressure (CPAP) and PSV trial was initiated, pressure Support (PSupp) 7 cm H₂O and Positive End Expiratory Pressure (PEEP) 5 cm H₂O. Immediately, the patient became bradycardic and had a sinus pause for approximately 15 sec, at which point the ventilator mode was switched back to Pressure Regulated Volume Control (PRVC), and the patient instantly converted back to Normal Sinus Rhythm (NSR) 70 to 90 beats per minute. The following day, another CPAP trial was performed after stopping all sedation, which again resulted in episode of bradycardia and pause, reverting to NSR after being placed back on PRVC. The patient self-extubated ahead of the third CPAP trial, on 20 mcg/kg/h of Propofol and 1.2 mcg/kg/h of Dexmedetomidine. She became bradycardic to 30 to 40 beats per minute for approximately 10 sec, and spontaneously returned to NSR 70 to 80 beats per minute.

Conclusion: MV setting alterations during weaning procedures have direct effects on patient hemodynamics, due to effect on intrathoracic pressure and the autonomic nervous system. The decreased airway pressure during CPAP and PSV trials or immediately after extubating produces an increase in cardiac output due to decreased intra-thoracic pressure and thus, increased venous return to the right atrium. This, in turn, causes an increase in parasympathetic output, which may cause profound bradycardia or sinus pause.

Keywords: Bradycardia; Weaning; Ventilator; Critical care

Abbreviations

CMV: Controlled Mechanical Ventilation; CPAP: Continuous Positive Airway Pressure; ED: Emergency Department; GSW: Gunshot Wound; HAART: Highly Active Anti-Retroviral Therapy; HIV: Human Immunodeficiency Virus; ICU: Intensive Care Unit; MV: Mechanical Ventilation; NSR: Normal Sinus Rhythm; OR: Operating Room; PEEP: Positive End Expiratory Pressure; PSV: Pressure Support Ventilation; PRVC: Pressure Regulated Volume Control; PSupp: Pressure Support; SICU: Surgical Intensive Care Unit; VAP: Ventilator Associated Pneumonia

Introduction

Mechanical ventilation is a ubiquitous supportive treatment modality in Intensive Care Units (ICUs). It is the most common support intervention in critical care units [1]. Prolonged duration of mechanical ventilation is associated with various adverse outcomes, including Ventilator Associated Pneumonia (VAP) and ventilator-associated lung injury [2]. Thus, there is a clear benefit to weaning patients from mechanical ventilators as early as possible. Decreased duration of mechanical ventilation decreases morbidity and mortality. Ventilator setting alterations during weaning procedures have a direct effect on hemodynamics of the patient, due to its effect on the autonomic nervous system and variations in heart rate [3]. Despite this, there is a scarce supply of

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Received Date: 14 Sep 2020

Accepted Date: 05 Oct 2020

Published Date: 08 Oct 2020

Citation:

Qasawa R, Munie S, Killu K.
Bradycardia and Sinus Pause with
Pressure Support Ventilation and
Weaning from Mechanical Ventilation.
Ann Clin Case Rep. 2020; 5: 1881.

ISSN: 2474-1655

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research and data on the topic of hemodynamic changes that take place during weaning from mechanical ventilation.

It has been proposed that the decrease in Positive End-Expiratory Pressure (PEEP) during weaning produces an increased cardiac output due to decreased intra-thoracic pressure and thus, increased venous return to the right atrium. This, in turn, causes an increase in parasympathetic output, which may cause profound bradycardia or sinus pause [4]. This is the case of a 23-year-old Gunshot-Wound (GSW) victim who underwent multiple abdominal surgeries who developed sinus pause during Continuous Positive Airway Pressure (CPAP) trial after 8 days of mechanical ventilation.

Case Presentation

A 23-year-old transgender (male to female) patient presented to the emergency room after sustaining a GSW to the abdomen. She has a past medical history significant for HIV on intermittent HAART. She presented to the Emergency Department (ED) ambulatory and hemodynamically stable. She was found to have a bullet hole on the right flank as well as a bullet hole at the anterior aspect of the left costal margin. She was sedated and intubated for airway protection upon arrival to the trauma bay. She was taken to the Operating Room (OR) where a right chest tube was placed and exploratory laparotomy was performed. A liver exploration was performed and patient was found to have a liver laceration separating segments VI and VII as well as right hemidiaphragm injury. Segments VI and VII of the liver were resected along with the gallbladder. An Abthera wound VAC was placed. She was admitted to the Surgical ICU (SICU) postoperatively. She remained intubated and had three planned returns to the OR. Additionally, patient required chemical and physical restraints due to agitation and attempts to self-extubate. On hospital day 3, she underwent exploratory laparotomy, partial liver resection of necrotic segments V and VIII, omental flap to liver, and drain placement. On hospital day 5, she underwent exploratory laparotomy, open G-tube placement, and wound VAC exchange. Finally, on hospital day 8, she underwent exploratory laparotomy, abdominal washout, and primary closure of fascia with wound VAC placement. The following day, patient's ventilator settings were V_T 400, RR 16, FiO_2 30%, PEEP 5. Patient was on 5 mcg/kg/h of Propofol, 0.8 mcg/kg/h of Dexmedetomidine, and 6 mg/h of Morphine. CPAP trial was performed, at which point patient was placed on PSupp 7 cm H_2O and PEEP 5 cm H_2O . Patient immediately became bradycardic and had a sinus pause, which lasted approximately 15 sec, at which point she was immediately converted back to Controlled Mechanical Ventilation (CMV) on her previous settings. She then converted back to her average heart rate, ranging from 70 to 90 beats per minute.

The following day, another CPAP trial was planned with patient off sedation. However, patient self-extubated ahead of the trial while on CMV 400/16/30/5. At that time, patient was on 20 mcg/kg/h of Propofol and 1.2 mcg/kg/h of Dexmedetomidine. Patient was immediately placed on non-rebreather at 50% FiO_2 and 15 liters/min flow rate. She again became bradycardic; her heart rate decreased to low 30s to 40s for approximately 10 sec, after which she spontaneously returned to her normal heart rate of 70 to 80 beats per minute. Within 15 min, patient was switched to nasal cannula at 5 liters/min with non-labored breathing at respiratory rate of 31 breaths per minute. She was continuously weaned to room air over the next 24 h, which she tolerated well. She remained hemodynamically stable; however, she was tachypnic with respiratory rate reaching 41 breaths per minute. She did not appear to be in respiratory distress, maintaining

SaO_2 100% and stated she was comfortable.

Discussion and Conclusion

Bradycardia during ventilator weaning is a rare occurrence, with only one such case report found in the literature. It is not uncommon for patients to experience hemodynamic changes while on mechanical ventilation. With regards to heart rate, tachycardia is more common than bradycardia [3]. Because preload has a direct impact on stroke volume, changes in intrathoracic pressure caused by changes in PEEP have a direct effect on cardiac output. Intrathoracic pressure during mechanical ventilation is positive [1]. As intrathoracic pressure and right atrium pressure increase during positive pressure inspiration, venous return decreases [5]. Therefore, increasing PEEP (which increases intrathoracic pressure) decreases venous return to the right atrium; thus, preload is decreased which leads to decreased cardiac output. Conversely, decreasing PEEP decreases intrathoracic pressure and increased blood return to the right atrium and thus, increases preload; this then increases cardiac output.

In response to the above-mentioned changes, the autonomic nervous system is activated and subsequently causes physiologic alterations aimed at maintaining cardiac output [3]. A patient who experiences decreased venous return to the right heart will experience increase in heart rate to accommodate this and maintain cardiac output. Therefore, a patient with decreased preload due to higher PEEP may have increased heart rate. Contrary to this is a patient with decreased PEEP, who will have increased preload and a resulting increase in parasympathetic activity and may lead to a vagal response; this would ultimately cause a decrease in heart rate, as occurred in this case report.

An association between intrathoracic pressure and left ventricular after load was explained by Buda et al., stating negative intrathoracic pressure affects left ventricular function by increasing left ventricular transmural pressures and thus, after load. Stated another way, decreasing PEEP also increases left ventricular after load [4]. This would cause a decrease in cardiac output in a patient with impaired myocardial function; however, in a patient without such impairment, the increased cardiac output gained by decreased intrathoracic pressure will outweigh the decrease in cardiac output as a result of increased left ventricular after load [4]. An understanding of the mechanics that occur in a patient on a ventilator is exceedingly important to manage these patients safely as well as to recognize the hemodynamic changes taking place [6].

Availability of Data and Materials

All data generated or analyzed during this study are included in this published article, including details obtained directly from the patient's medical chart.

Authors' Contribution

RQ obtained all information from the medical chart, researched available data, and drafted the manuscript. SM and KK confirmed all obtained information and data were relevant and made appropriate adjustments to the manuscript. All authors read and approved the final manuscript.

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