Case report

Patient-ventilator interaction: Unusual ventilator graphics and management using Positive End Expiratory Pressure – A case report

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Abstract

Approximately 25% of patients receiving mechanical ventilation have significant patient ventilator asynchrony which is associated with prolonged duration of mechanical ventilation. Here we report an unusual presentation of ventilator graphics "saw tooth" appearance in both pressure-time and flow-time graphs. An increase in the applied PEEP level resolved the saw tooth pattern in the graphics. The possible explanation for the graphics displayed is heart-lung interaction which is cardiogenic oscillation. If this is true, then this pattern can be used as a clinical tool to apply lung inflation manoeuvres.

Keywords: Cardiogenic oscillation, dysynchrony, patient-ventilator interaction, positive end expiratory pressure, ventilator graphics.

Introduction

The interaction and influence of mechanical ventilator on the human cardiovascular system is complex. Cardiac pulse related pressure oscillation appearing at airway opening is called cardiogenic oscillation. This signal in an intubated patient can signify a decrease in the compliance of respiratory system[1] and autotriggering.[2] These dys-synchronous (uncoupling of mechanical breath and neural breath) interactions cause discomfort for the patient, thereby compelling administration of sedation. However, if synchrony is improved in one phase of the breath it can facilitate in the improvement of synchrony for the remaining areas of the breath.[3]

This case report describes unusual ventilator graphics, probably due to cardiogenic oscillations.

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Case report

A 76 year old male was brought to emergency department with complaints of dyspnoea, wheezing and cough with expectoration. Patient had a history of adrenal insufficiency, old cerebrovascular accident and hypertension. On examination he had facial puffiness, abdominal distension but no new neurological findings. On auscultation, he had bilateral rhonchi. He was diagnosed with acute exacerbation of bronchial asthma and pneumonia. He was admitted to the high dependency unit and treated with oxygen and nebulised bronchodilators. An echocardiography ruled out cardiac cause of dyspnoea. On the 8th day of hospital stay, patient had episodes of vomiting followed by hypotension (90/60 mm Hg) and hypoxia (SpO_2 70%). This was managed successfully by fluid resuscitation, endotracheal intubation and mechanical ventilation. The ventilator settings were pressure control mode (P-SIMV), inspiratory pressure (PIP) of 12 cm H_2O (achieving an exhaled tidal volume (VT_E of 655 mL), inspired oxygen concentration (FiO_2) of 1.0, positive end expiratory pressure (PEEP) of 5 cm

How to cite this article: Babu A, Unnikrishnan R. Patient-ventilator interaction: Unusual ventilator graphics and management using Positive End Expiratory Pressure – A case report. Ind J Resp Care 2014; 3(2):509-11.

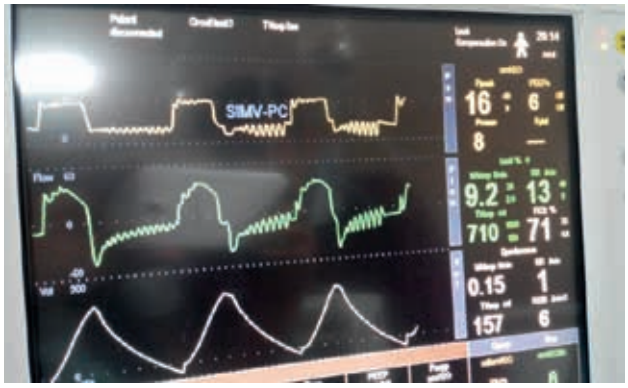

Figure 1: Oscillations seen on both the pressure-time and flow-time graphs

Figure 2: Disappearance of the oscillations with increase in PEEP

H_2O and pressure support (PS) of 10 cm H_2O, with a set frequency of 12 breaths/minute. On the next day, the respiratory therapist noted an abnormal pattern in both pressure-time and flow-time scalar *(Figure 1)*.

The respiratory therapist immediately performed a ventilator circuit check, presence of intrinsic PEEP (PEEPi) and an endotracheal (ET) suctioning to rule out moving secretions in the airway. There was no water in the circuit and moderate amount of yellow secretions were obtained by ET suctioning. There was no PEEPi and the ventilator graphics remained the same. The inspiratory flow was increased to rule out possibility of flow inadequacy [Airway pressure (P_{aw}) "sucked down"] but the oscillatory wave pattern did not change. However, the pulse rate of the patient and the appearance of abnormality in the graphics matched well leading us to conclude that this must be cardiac oscillation. A possibility of lung collapse or consolidation was also considered due to the primary diagnosis of pneumonia. The respiratory therapist then altered the PEEP from 5 to 10 cm H_20 *(Figure 2)*. This strategy proved successful as the graphics became normal.

Discussion

The ventilator control centre in humans is present in the brain stem and is regulated with the signals from the chemoreceptors [receptors that sense oxygen tension (PO_2), carbon dioxide tension (PCO_2) and hydrogen ion concentration (pH)] located in the great vessels and the fourth ventricle of the brain and from the mechanoreceptors (stretch and irritant receptors) located in the thorax and ventilator muscles.[4] The brain cortex can also influence the ventilator drive.[4] With this complex array of interaction the respiratory centre in the brain stem achieves the goal, which is to always provide adequate gas exchange (physiologic pH and PO_2) with least amount of ventilator muscle loading and air trapping.[5]

Mechanical ventilation can modulate or have a profound effect on the respiratory centre.[4] The interaction between the mechanical ventilator and the respiratory centre is of considerable importance and is called patient-ventilator interaction. The assist/control mode is the most common mode of invasive mechanical ventilation employed in intensive care units because it reduces the use of sedatives which in turn facilitates ventilator muscle recovery.[6] The benefit of this mode is achieved only when there is synchrony between the mechanical ventilator and the patient's spontaneous breathing in all phases of breathing (trigger, limit, cycle and baseline) and this is called synchronous support.

The controlled mechanical breaths is known to suppress spontaneous ventilation.[4] The mechanical breath elicited by a spontaneous breath is called entrainment.[7] The entrainment breath can cause an increase in tidal volume (V_T) or trigger a second breath depending on the phase of the control breath. The breath cycle criteria can also influence the respiratory centre.[4] When the mechanical breath Ti (inspiratory time) terminates before the neural breath Ti, it can lead to respiratory muscle activation beyond the machine flow delivery phase, excessive

muscle loading, triggering of an additional breath and excessively high volumes. If the mechanical breath Ti exceeds neural breath Ti, then the patient experiences dyspnoea and the expiratory muscle may be recruited to terminate the breath.

Issues such as accurately anticipating and adapting to the breath pattern (loading pattern) and support pattern will be difficult to achieve in situations where the ventilator mode provides more than one breath type.[3]

Delayed or missed triggers are uncomfortable for patients and leads to increase in the intensity of effort and causes increased dyspnoea.[1] Overventilation and overdistension is sensed by the mechanoreceptors which causes the shortening of neural inspiratory drive and activation of expiratory muscles.[8]

The beneficial effects of assisted modes of mechanical ventilation are appreciated only when the mode interacts with the patient demands during all the phases of a breath (trigger, limit, cycle). Synchronous interactions are always beneficial and this is determined by both patient and ventilator factors. It is common to find at least 25% of patients receiving mechanical ventilation to have patient ventilator asynchrony.[9]

For the successful management of patient-ventilator asynchrony, the clinician must understand the pathophysiology, interpretation of flow-time graphics and types of patient-ventilator asynchrony. This will improve the differential diagnosis of asynchrony. An algorithm is also proposed to help clinicians at bedside to decrease the incidence of dys-synchrony but it remains to be tested prospectively.[10]

In this case, the patient was successfully treated by increasing PEEP but the need for such treatment and safety needs to identified. It is important to highlight the importance of treating each situation discretely.

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