

Cardiogenic oscillation induced ventilator autotriggering

Narender Kalaria, Akhilesh Gupta, Ajay Goila, Rajesh Sood

Abstract

Cardiogenic oscillation during mechanical ventilation can auto-trigger the ventilator resembling patient initiated breath. This gives a false sense of intact respiratory drive and determination brain death, even if other tests are positive, is not appropriate in such a situation. It will prolong the ICU stay and confound the brain-death determination. In this case report, we describe a 35 year old man who was brought to the hospital after many hours of critical delay following multiple gun shot injuries. The patient suffered a cardiac arrest while on the way from another hospital. After an emergency laparotomy, patient was shifted to Intensive Care Unit (ICU) with Glasgow Coma Scale (GCS) score of E1V1M1 and was mechanically ventilated. Despite absence of brainstem reflexes, the ventilator continued to be triggered on continuous positive airway pressure (CPAP) mode and the patient maintained normal oxygen saturation and acceptable levels of carbon dioxide. An apnoea test confirmed absent respiratory drive. Ventilatory waveform graph analysis, revealed cardiogenic oscillation as the cause for autotriggering.

Key words: Autotriggering, cardiogenic oscillation, ventilator

Spurious auto-triggering of ventilator can create confusion in mechanical ventilatory management of patients and confounds the diagnosis of brain death.

We describe a 35-year-old man with multiple gunshot injuries to abdomen, cervical spine and thigh was brought to hospital after many hours of critical delay. The patient had a cardiac arrest while on the way from another hospital. On arrival in emergency department, trachea was intubated and immediate fluid resuscitation measures were taken. Focused Assessment with Sonography in Trauma (FAST) ultrasound was positive for blood; emergency laparotomy was done.

Patient was shifted to Intensive Care Unit (ICU) with Glasgow Coma Score (GCS) of E₁V₁M₁, pupils

mid-dilated sluggishly reacting to light. The patient was ventilated using Detex Ohmeda – Engstrom Care station on Synchronized Intermittent Mandatory Ventilation (SIMV) mode with a tidal volume of 550 ml, positive end-expiratory pressure (PEEP) of 6 cm H₂O, pressure trigger sensitivity of -2 cm H₂O, fraction of inspired concentration of oxygen (FiO₂) at 0.5, ventilatory rate of 14 breaths/min and pressure support of 15 cm H₂O.

Once the patient was stabilized, we decided to put him on continuous positive airway pressure (CPAP) mode with a pressure support of 15 cms of H₂O, trigger of - 0.25 cm H₂O, FiO₂ of 0.5 and PEEP of 5 cm H₂O on Day 3. Patient was maintaining gases on this mode with respiratory rate of 25/min. GCS of patient was still E₁V₁M₁, pupils became fixed, dilated, coughing and gag reflex were absent but haemodynamic parameters were stable. But with no brain stem reflexes, it was confounding as to how he could maintain his respiratory drive? Then, apnoea test was performed that was negative for respiratory drive. Ventilation was reinitiated on CPAP mode with trigger of - 0.25 cm of H₂O that again started triggering spontaneous breaths [Figure 1]. The

Access this article online	
Quick Response Code:	Website: www.jnaccjournal.org
	DOI: 10.4103/2348-0548.154239

Department of Anesthesia, Dr. Ram Manohar Lohia Hospital and Post Graduate Institute of Medical Education and Research, New Delhi, India

Address for correspondence:

Dr. Narender Kalaria, Department of Anesthesia, Post Graduate Institute of Medical Education and Research, Chandigarh - 160 012, India.
E-mail: naren.keli@gmail.com

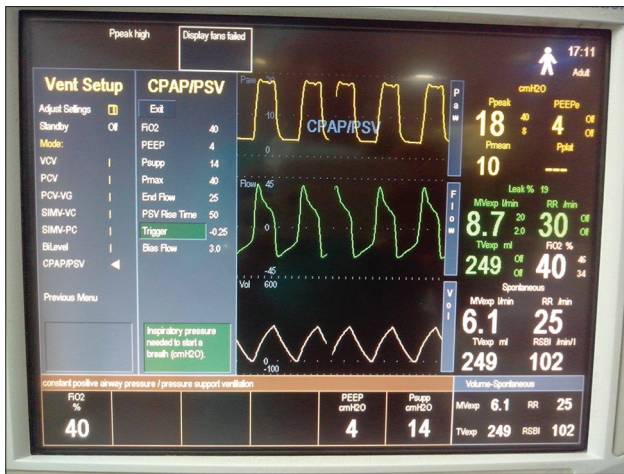


Figure 1: The ventilator being triggered at a trigger sensitivity of -0.25 cms of H₂O. All the breaths are being sensed as patients own spontaneous efforts

trigger was increased to -1 cm H₂O from -0.25 cms H₂O. We observed that there was no triggering from the patient, only cardiogenic oscillations were seen on airway pressure and flow waveform [Figure 2]. So, it was inferred that the ventilator was being auto-triggered due to cardiogenic oscillations that was being misinterpreted by us as patient's spontaneous effort.

So, this case highlights the role of cardiogenic oscillation in auto-triggering the ventilator and mimicking spontaneous breaths. Airway pressure and flow waveforms identified this auto-triggering. Raising the trigger sensitivity abolished this auto-triggering and was confirmed by apnoea test.

The most probable mechanism of cardiogenic oscillation induced auto-triggering is change in volume of beating heart causing an intra-thoracic pressure change that moves air in and out of the lungs. The movement of the heart may cause displacement with consequent compression or expansion of the adjacent lung, resulting in movement of gas at the airway.^[1] The magnitude of the cardiogenic oscillation increases significantly with high cardiac output, high filling pressures, large cardiothoracic ratio and low resistance of the respiratory system.^[2]

There are many clinical implications of auto-triggering caused by cardiogenic oscillation in ICU. Patients appear to breath spontaneously that possibly leads to a mistaken decision to wean them from mechanical ventilation, raising false hope of recovery among patient's family members. The determination of brain death may not be appropriate. It will prolong the ICU stay and confound

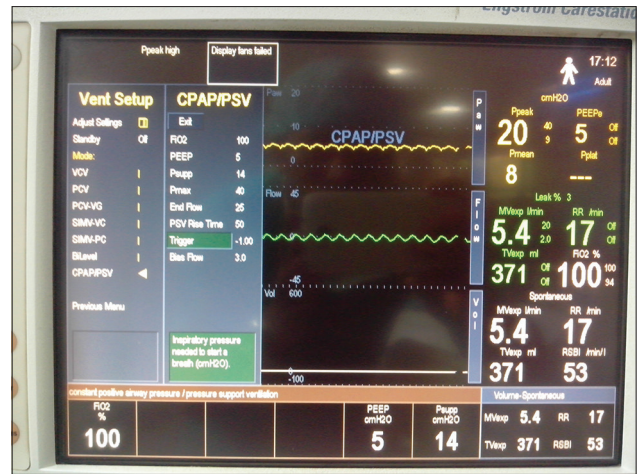


Figure 2: The ventilator not being triggered at a trigger sensitivity of -1.0 cms of H₂O. Only cardiac oscillatory waves are being shown on airway pressure and flow waveforms. Spontaneous Minute ventilation being shown is from the previous respiratory cycle

the brain-death determination, adding to the financial costs of ICU care and delay in organ donation or may even abort this opportunity.^[3]

In conclusion, auto-triggering caused by cardiogenic oscillation must be anticipated. Spontaneous breaths can be distinguish from auto-triggering by adjusting trigger sensitivity, careful monitoring of the graphs on the ventilator display. Spontaneous breaths should be eliminated by temporal hyperventilation or by test administration of sedatives/muscular relaxants. Auto-triggering can be prevented by triggering sensitivity adjustment, use of pressure triggering because it is usually less prone to noise than flow triggering and changing to intermittent mandatory ventilation (IMV) mode.

REFERENCES

1. West JB, Hugh-Jones P: Pulsatile gas flow in bronchi caused by the heartbeat. *J Appl Physiol.* 1961; 16: 697-702
2. Imanaka H, Nishimura M, Takeuchi M, Kimball WR, Yahagi N, Kumon K. Autotriggering caused by cardiogenic oscillation during flow-triggered mechanical ventilation. *Crit Care Med.* 2000; 28(2): 402-7.
3. Arbour R. Cardiogenic oscillation and ventilator autotriggering in brain-dead patients: a case series. *Am J Crit Care.* 2009; 18: 488-96.

How to cite this article: Kalaria N, Gupta A, Goila A, Sood R. Cardiogenic oscillation induced ventilator autotriggering. *J Neuroanaesthesiol Crit Care* 2015;2:134-5.

Source of Support: Nil, **Conflict of Interest:** None declared.