Cardiogenic oscillation induced ventilator autotriggering

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Abstract

Cardiogenic oscillation during mechanical ventilation can auto-trigger the ventilator resembling patient initiated breadth. 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 E1VT1M1 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 E1VT1M1, 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 E1VT1M1, 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

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trigger was increased to -1 cm H\textsubscript{2}O from -0.25 cm H\textsubscript{2}O. 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.\textsuperscript{[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.\textsuperscript{[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 the brain-death determination, adding to the financial costs of ICU care and delay in organ donation or may even abort this opportunity.\textsuperscript{[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**


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