CARDIOGENIC OSCILLATION AND VENTILATOR AUTOTRIGGERING IN BRAIN-DEAD PATIENTS: A CASE SERIES

By Richard Arbour, RN, MSN, CCRN, CNRN, CCNS

Abstract

Brain death is manifested by a flaccid, areflexic patient on assessment of brain function with fixed and dilated pupils at midpoint, loss of consciousness, no response to stimulation, loss of brainstem reflexes, and apnea. A lesion or clinical state responsible for the loss of consciousness must be found. An integral part of clinical evaluation of brain death is apnea testing, which indicates complete loss of brainstem function and respiratory drive. Ventilator triggering or overbreathing the ventilator’s set rate may be considered consistent with intrinsic respiratory drive consequent to residual brainstem function. Ventilator autotriggering, however, may potentially occur in a brain-dead patient as a result of interaction between the hyperdynamic cardiovascular system and compliant lung tissue altering airway pressure and flow patterns. Also, chest wall and precordial movements may mimic intrinsic respiratory drive. Ventilator autotriggering may delay determination of brain death, prolong the intensive care unit experience for patients and their families, increase costs, risk loss of donor organs, and confuse staff and family members. A detailed literature review and 3 cases of cardiogenic ventilator autotriggering are presented as examples of this phenomenon and highlight the value of close multidisciplinary clinical evaluation and examination of ventilator pressure and flow waveforms. (American Journal of Critical Care. 2009;18:496,488-495)

Brain death is complete and irreversible loss of function in all areas of the brain, including the brainstem, that causes total, irreversible loss of consciousness, unresponsiveness, lack of brainstem reflexes, and apnea.¹ Conditions ultimately culminating in brain death include brain trauma, brain infarction, brain hemorrhage, prolonged cardiopulmonary arrest, intracranial tumors, meningitis/encephalitis, subarachnoid hemorrhage, exposure to agents such as cocaine, and heavy-metal intoxication.¹ Secondary brain injury may lead to catastrophic elevations of intracranial pressure (ICP) and brainstem herniation.¹,²

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Cardiopulmonary Consequences of Brain Death

Physiological changes associated with brain death include a hyperdynamic cardiovascular state and significant alterations in vasomotor tone. A hyperdynamic cardiovascular state may produce a hyperdynamic precordium, which can be mistaken for respiratory movements. This hyperdynamic cardiovascular state may also displace sufficient intrathoracic volume and lung tissue to cause cardiogenic oscillation in pressure and flow waveforms beyond set trigger sensitivities and trigger ventilator breaths in the absence of an intrinsic respiratory drive.

Cardiogenic oscillations are fluctuations in ventilator airway pressure, flow, and potentially volume waveforms in phase with the cardiac cycle. When cardiogenic oscillations in flow or pressure waveforms reach or exceed the ventilator’s trigger sensitivity threshold, a ventilator breath may be delivered in the complete absence of an intrinsic respiratory drive. The purpose of this article is to review brain death physiology and testing, and to illustrate the potential delay or confusion in determination of brain death when ventilator breaths are triggered by cardiogenic oscillation in ventilator airway pressure and baseline flow.

After documenting the absence of cranial nerve function and after pupils are fixed and dilated at midpoint in a flaccid, areflexic, unresponsive patient, loss of intrinsic respiratory function is determined by apnea testing. Apnea testing is required for formal documentation of intrinsic respiratory drive loss and carbon dioxide reactivity at the level of the brainstem. A patient’s triggering of ventilator breaths or “overbreathing” the ventilator set rate may be misinterpreted as residual intrinsic respiratory drive. A more comprehensive review of apnea testing and formal brain death examination may be found in existing practice parameters and other excellent references available on the topic.

During assist/control mode ventilation, the patient’s inspiratory effort is used to trigger the ventilator for breaths above the set respiratory rate. The 2 more common types of ventilator triggering modes are flow triggering and pressure triggering.

Flow Triggering Versus Pressure Triggering

In flow triggering, a controlled ventilation is initiated when a change in the baseline gas flow within the ventilator circuit, such as that produced by a patient’s effort, exceeds a preset threshold of typically 1 to 5 L/min. With flow triggering, the inspiratory valve is open, allowing bias flow into the ventilator circuit. More sensitive flow-triggering thresholds such as 1 to 2 L/min require less inspiratory effort for the ventilator breath to be delivered.

In pressure triggering, the inspiratory effort must generate a sufficient reduction in airway pressure below the set trigger threshold for the ventilator breath to be delivered. Thresholds may range from -0.5 cm H₂O to -2.0 cm H₂O or from -1.0 to -2.0 cm H₂O negative pressure. With a more sensitive pressure-triggering threshold such as -0.5 to -1.0 cm H₂O, the patient must generate less inspiratory effort in order for a ventilator breath to be delivered, increasing the likelihood of patient-ventilator dysynchrony and autotriggering.

Ventilator Autotriggering

Ventilator autotriggering is the triggering of a ventilator breath in the absence of inspiratory muscle activity. This phenomenon may occur with excessive ventilator trigger sensitivity and in all triggering modes. A select listing of potential intrinsic and extrinsic causes of ventilator autotriggering is presented in the Table.

A proposed mechanism for cardiogenic ventilator autotriggering is outlined here. During systole and cardiac contraction, the volume reduction of the heart and the volume reduction within the chest with stroke volume can cause cyclical displacement (compression/expansion) of compliant lung tissue. This distortion (compression/expansion) of lung tissue in response to the cyclical volume changes of the heart and volume loss from the chest with

<table>
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<th>Table: Potential causes of ventilator autotriggering*</th>
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* Based on data from Nilsestuen and Hargett, Hess, Kondili et al, Campbell et al, Sager et al, and Al-Khafaji and Manning.

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triggering threshold above the flow or pressure displacement produced by the artifact. Autotriggering produced by a persistent leak in a chest tube may respond to an increase in the bias flow (in flow triggering) greater than the leak rate through the chest tube.

Literature Review: Cardiogenic Oscillation, Waveforms, and Ventilator Management

Multiple reports of ventilator autotriggering consequent to significant cardiogenic oscillation in ventilator airway pressure and flow waveforms have been published. Cardiogenic autotriggering has occurred in both patients who were not brain dead and patients who were brain dead.

Cardiogenic oscillations in airway pressure or flow waveforms indicate transmission of cardiac artifact through the chest into the ventilator circuit. Interventions for cardiogenic ventilator autotriggering include increasing the pressure and/or flow triggering threshold above the flow or pressure displacement produced by the artifact. Autotriggering produced by a persistent leak in a chest tube may respond to an increase in the bias flow (in flow triggering) greater than the leak rate through the chest tube.

Abbreviations: O2 conc., oxygen concentration; PEEP, positive end-expiratory pressure; P high, high pressure; Ppeak, peak airway pressure; Pmean, mean airway pressure; RR, respiratory rate; O2 (%), oxygen concentration; Ti/Ttot, time of inspiration/total breath duration ratio; MVe (l/min), exhaled minute volume; Vti, inspired tidal volume; Vte, exhaled tidal volume; cm H2O, centimeters of water pressure.

Figure 1 Illustration of cardiogenic oscillations reflected in pressure and flow waveforms. Deflections in airway pressure and flow waveforms in phase with the cardiac cycle were identified by waveform analysis and correlating frequency of waveform oscillations with electrocardiography (ECG)/heart rate, pulse palpation, auditory ECG signal (beep volume), and hyperdynamic precordium all in phase with cardiac cycle. Pressure waveform displacement of 2 to 3 cm H2O pressure and flow waveform displacement of 5.0 to 6.5 L/min is noted in this ventilator graphic. Ventilator graphic file was downloaded from the Servo-I ventilator; Maquet, Inc, Bridgewater, New Jersey.

Cardiogenic oscillations may be seen in greater amplitude in patients who have high stroke volumes/cardiac output and a more hyperdynamic cardiovascular system, as well as in patients with a larger heart size relative to thoracic volume and low resistance within the respiratory system. Recognition of autotriggering is facilitated by close physical assessment and examination of the ventilator circuit as well as ventilator waveform analysis. Cardiogenic oscillations in airway pressure or flow waveforms indicate transmission of cardiac artifact through the chest into the ventilator circuit.
Cardiogenic Oscillation and Autotriggering in Patients Who Are Not Brain Dead

High-amplitude cardiogenic oscillation presents significant implications for ventilator management. Imanaka and colleagues\textsuperscript{15} studied autotriggering and cardiogenic oscillation during flow-triggered ventilation. The investigators reported that cardiogenic autotriggering was more common during flow triggering and in patients with a hyperdynamic cardiovascular state, low-resistance pulmonary physiology, and a larger cardiothoracic ratio. In this study, the mean (SD) deflection in airway pressure in phase with the cardiac cycle was 1.37 (0.28) cm H$_2$O. The mean (SD) deflection in inspiratory flow in phase with the cardiac cycle was 4.67 (1.26) L/min in the group that experienced autotriggering. When trigger sensitivities were increased beyond the amplitude of cardiogenic oscillations, autotriggering stopped.

In a case report, Op’t Holt\textsuperscript{16} noticed cardiogenic triggering in a patient with acute respiratory distress syndrome during flow-triggered ventilation concurrent with sedation and neuromuscular blockade, eliminating the possibility of intrinsic inspiratory effort. Clinical assessment revealed movement of the chest wall and oscillation within ventilator waveforms in phase with the cardiac cycle. Flow triggering was changed to pressure triggering with a sensitivity of -2.0 cm H$_2$O pressure, which eliminated autotriggering. The patient had a hyperdynamic, low-resistance hemodynamic profile that contributed to the autotriggering.

Cardiogenic Oscillation and Autotriggering in Brain-Dead Patients

Willatts and Drummond\textsuperscript{17} reported a 64-year-old patient who became hemodynamically unstable after catastrophic progression of cerebral infarction. Results of 2 clinical neurological examinations and apnea testing met all criteria for brain death. Family members consented for organ donation. During ventilator management, flow triggering was used with a sensitivity of 1 L/min. The bedside nurse noticed the patient was intermittently assisting the ventilator above the set rate. With high levels of pressure support ventilation, the patient generated a measured respiratory rate of 23 breaths/min. On continuous positive airway pressure with no assist, no respiratory efforts were apparent. The transplant coordinator and bedside staff were concerned about the determination of brain death. The patient was extubated with no respiratory drive and became asystolic. An opportunity for organ donation was lost.

Ng and Tan\textsuperscript{18} reported a 22-year-old patient who met all clinical criteria for brain death after severe head trauma. Organ recovery for transplantation was planned and became the focus of management in the intensive care unit (ICU). Immediately before transfer to the operating room for organ recovery, the patient’s family members noted that the patient “appeared” to take a spontaneous breath. Importantly, ventilator management included a pressure triggering sensitivity of -1 cm H$_2$O. The family members questioned the determination of brain death and requested further neurological evaluation, delaying organ recovery. Additional clinical evaluation was performed and results were consistent with brain death. Organ recovery proceeded after a delay of 3 hours.

Cole\textsuperscript{19} reported a 27-year-old patient with fulminant hepatic failure. When the patient’s level of consciousness critically decreased, he was intubated and received controlled ventilation. Computed tomography of the head showed significant brain edema, and neurological deterioration continued until findings on an initial clinical examination (areflexic, flaccid, pupils fixed and dilated) were consistent with brain death. Immediately before the initial apnea testing, apparent ventilation efforts were noted on the ventilator flow waveforms, causing cancellation of apnea testing and delay in determination of brain death. The next day, apnea testing was attempted again. Close examination of ventilator waveforms revealed oscillations in phase with the patient’s heart rate in the absence of movement of the chest and abdominal wall. Apnea testing then confirmed brain death. Cardiogenic oscillations were initially misinterpreted as inspiratory efforts and delayed apnea testing.

Wijdicks et al\textsuperscript{20} followed a total of 83 patients in a neurological/neurosurgical unit who met all clinical criteria for brain death including apnea testing. Of this total, 4 patients (4.8%) occasionally triggered a ventilator breath even with results of repeated apnea testing consistent with brain death. Autotriggering was eliminated by upward adjustment of ventilator trigger sensitivities (less sensitive trigger) or by changing the triggering mode from flow to pressure triggering. The investigators hypothesize that cardiogenic autotriggering in brain-dead patients may be more common than is currently known and may go unrecognized.

Case Series: Cardiogenic Oscillation Causing Ventilator Triggering

This case series presents 3 patients with devastating neurological injuries in whom cardiogenic...
oscillation caused ventilator autotrigering. All 3 patients were flaccid and areflexic on neurological examination of brain function, with absent brainstem reflexes, fixed/dilated pupils, and no respiratory drive. Each patient was at times triggering ventilator breaths in the absence of an intrinsic respiratory drive. Autotrigering was identified by clinical evaluation and examination of ventilator pressure and flow waveforms.

Case 1
A 50-year-old man with a history of hypertension, diabetes mellitus, and congestive heart failure was admitted to the ICU through the emergency department. While being evaluated in the emergency department, the patient became obtunded and displayed decerebrate posturing. He was intubated and received controlled ventilation. Computed tomography of the head revealed a large left-sided intraparenchymal hemorrhage for which he was not a surgical candidate. His neurological condition deteriorated further. The patient’s score on the Glasgow Coma Scale was 3/15, his pupils were fixed and dilated at midpoint, he was flaccid and areflexic on neurological examination, and he had no cough, gag, or corneal reflexes.

At 9:25 AM, findings on the initial clinical examination were consistent with no brain activity. His PaCO₂ was normalized, and apnea testing and the second clinical examination were planned for later that evening after a 12-hour interval had passed. At 7:30 PM, the ventilator set rate was 9 breaths per minute and the measured respiratory rate was 12 breaths per minute, potentially creating confusion among clinicians and family members about the determination of brain death.

The patient was again assessed and found to be flaccid and areflexic on neurological examination, with bilateral fixed and dilated pupils and no intrinsic inspiratory effort. Ventilator waveform analysis revealed regular oscillations in phase with the cardiac cycle. Recordings were made of airway pressure and flow tracings that documented cardiogenic oscillations in flow waveforms with no other deflections indicating intrinsic respiratory drive. Ventilator set rate was decreased (for a duration of 15-20 seconds) to assess ventilator waveforms for deflections indicating inspiratory effort. The only waveform deflections evident in addition to backup ventilation were those in flow waveforms and in phase with the cardiac cycle. The cardiogenic deflections in flow waveforms corresponded to a hyperdynamic precordium. Waveform deflections were found to be coordinated with the cardiac cycle by increasing the volume of the QRS tone (beep) on the bedside monitor, which also matched the pattern of precordial motion, pulse palpation, and flow waveform deflections. Cardiogenic triggering was eliminated by increasing the pressure trigger threshold. Airway pressure and flow tracings illustrating ventilator flow waveform deflections in phase with the cardiac cycle and no waveform deflections indicating any intrinsic inspiratory effort are shown in Figure 2.

After the cardiogenic autotrigering had been resolved, the second clinical examination and the apnea testing were completed at approximately 10:30 PM. Clinical examination showed no brain activity. Results of apnea testing were consistent with brain death (no inspiratory effort/PaCO₂ elevated to 70 mm Hg). The patient was pronounced dead on the basis of neurological criteria, the patient’s family consented to organ donation, and the patient’s organs were recovered the following day.

Case 2
A 78-year-old woman was found unresponsive in a long-term care facility and transported to the emergency department by paramedics. Her medical history was significant for hypertension, diabetes mellitus, acute renal failure, coronary artery disease, breast cancer (untreated), and dementia. Her initial score on the Glasgow Coma Scale was 5. She was intubated and received positive pressure ventilation. Initial computed tomography of the head showed a massive left-sided intraparenchymal/intraventricular hemorrhage with significant mass effect and herniation. She was not a surgical candidate. She was admitted to the ICU for medical management. Neurological status continued to decline, with bilateral fixed and dilated pupils and no cough, gag, or corneal reflexes.

On ICU day 3, she was still triggering ventilator breaths with a set rate of 12 breaths per minute and the measured rate of 14 to 18 breaths per minute, delaying initiation of the brain-death protocol. Neurological assessment again revealed pupils fixed and dilated at midpoint, absence of cough, gag, and corneal reflexes, and a flaccid unresponsive state as well as absence of any inspiratory effort. Analysis of airway pressure and flow waveforms revealed no deflections consistent with intrinsic respiratory drive but did reveal deflections in phase with the cardiac cycle and ventilator breaths. Hyperdynamic precordial motion with each cardiac cycle matched the auditory tone (beep) with each QRS segment on the
cardiac monitor, pulse rate assessed by palpation, and the frequency/pattern of oscillations noted on ventilator pressure and flow waveforms. A graphic of ventilator waveforms illustrating cardiogenic autotriggering, deflections caused by ventilator breaths, and cardiogenic oscillations in flow and pressure waveforms is shown in Figure 3.

Ventilator trigger mode was changed to pressure triggering with a sensitivity of -2.0 cm H₂O, ending autotriggering. Her respiratory rate immediately matched the ventilator set rate. On data review, the patient had been triggering ventilator breaths during the night up to a respiratory rate of 16 to 18 breaths per minute, which ceased after this change in trigger mode and sensitivity. The patient, patient/ventilator interface, and flow and pressure waveforms were closely examined. Intrinsic respiratory effort was absent, and the only deflections in airway pressure and flow waveforms beyond ventilator breaths were those produced by ventilator breaths and cardiogenic oscillations. With resolution of cardiogenic autotriggering, consecutive neurological examinations and apnea testing were performed with results consistent with no brain activity. Death was pronounced on the basis of neurological criteria.

Case 3

A 68-year-old woman was found pulseless and apneic at a long-term care facility. She was resuscitated by paramedics and transported to the emergency department. On admission to the ICU she was unresponsive, areflexic, and had minimally reactive pupils. Within 4 hours of ICU admission, the patient's neurological status declined to include midpoint nonreactive pupils dilated to 6 mm and she was flaccid and areflexic on neurological examination. Further neurological evaluation and apnea testing were planned to determine brain death.

The patient was found to be triggering ventilator breaths with a set rate of 12 breaths per minute and measured respiratory rate of 20 breaths per minute. The advanced practice nurse was contacted for further clinical evaluation. Clinical data included synchrony among ventilator pressure/flow waveform...
was caused by the cardiac cycle. Flow trigger sensitivity threshold was increased in an effort to eliminate the phenomenon of cardiogenic autotriggering. A consulting neurologist confirmed severe anoxic brain injury. Refractory hemodynamic instability developed, and the patient became asystolic shortly thereafter.

Discussion

This case series illustrates cardiogenic oscillation of ventilator pressure and flow waveforms causing ventilator autotriggering in the absence of an intrinsic respiratory drive. In the first 2 cases, cardiogenic ventilator autotriggering could have delayed or canceled the determination of brain death. In these cases, the phenomenon was recognized and, in collaboration with nursing and respiratory therapy professionals, real-time changes were made in ventilator trigger sensitivities.

Cardiogenic autotriggering being mistaken for an intrinsic respiratory drive can have several implications. One implication is delay in determination of brain death and unnecessarily prolonging the oscillations, cardiac cycle, and hyperdynamic precordium, which was confirmed by direct observation of chest wall movement, auditory signal (beep volume on bedside monitor) with each QRS, and pulse palpation. The areflexic, flaccid patient with pupils fixed and dilated at midpoint raised suspicion that the ventilator triggering was caused by cardiogenic oscillations in pressure and flow waveforms. The flow trigger sensitivity was set at 5 L/min. Ventilator settings were changed for a short interval to continuous positive airway pressure of 5 cm H2O and pressure support of 0 cm H2O. Fraction of inspired oxygen was 0.50.

Flow waveform displacement in phase with the cardiac cycle was 12 to 15 L/min, significantly exceeding flow trigger sensitivity with frequency matching the patient’s heart rate. Ventilator flow, pressure, and volume waveforms are illustrated in Figure 4 and show the significant magnitudes possible in ventilator waveforms consequent to cardiogenic oscillation.

Waveform analysis and clinical examination at this time indicated that the ventilator autotriggering was caused by the cardiac cycle. Flow trigger sensitivity threshold was increased in an effort to eliminate the phenomenon of cardiogenic autotriggering. A consulting neurologist confirmed severe anoxic brain injury. Refractory hemodynamic instability developed, and the patient became asystolic shortly thereafter.
ICU experience for patients’ families. A second implication is possible confusion about the diagnosis of brain death on the part of patients’ family members and clinicians. Risks include raising false hope of recovery among patients’ family members and questioning by clinicians of the appropriateness of the determination of brain death and whether or not to proceed with formal brain-death protocols. A third implication is ICU resource utilization. The longer time required for ICU care and brain-death determination adds to the financial costs of ICU care. When costs associated with controlled ventilation and services of all care providers are added in, total ICU costs may exceed $5000 daily. A fourth implication involves organ donation. Longer ICU management times for potential donors increase risk of organ loss related to refractory hemodynamic instability. As reported by Willatts and Drummond, autotriggering may abort opportunities for organ donation. A fifth implication is the potential to decrease the interval between the terminal event of catastrophic brainstem injury as the final common pathway to brain death and the final declaration of death based on neurological criteria.

Brain death and its characteristic findings of a patient who is apneic, flaccid, and areflexic on neurological examination with pupils fixed and dilated at midpoint, as well as onset of potential biphasic hemodynamic consequences, occurs at the point of brainstem herniation. Formal protocols for determining brain death may take as long as 6 to 24 hours, creating a delay between occurrence of the terminal event of brainstem herniation or total loss of brain/brainstem function and pronunciation of brain death. Effective clinical surveillance to determine loss of brain and brainstem function more quickly and to identify cardiogenic autotriggering enables appropriate changes in ventilator trigger mode and sensitivity, and can facilitate more timely initiation of a brain death protocol, decreasing the interval between occurrence of brain death (terminal event) and formal declaration of brain death. In case 1, determination of brain death was facilitated by astute clinical assessment and early identification of cardiogenic autotriggering, avoiding delay in pronouncement of death and facilitating organ recovery. In case 2, identifying cardiogenic ventilator triggering eliminated delay in determination of brain death and avoided prolonging the ICU
experience for the patient’s family. In case 3, a patient who was apneic, flaccid, and areflexic on neurological examination and who had pupils fixed, dilated, and at midpoint was triggering ventilator breaths. Appropriate clinical evaluation was done, including ventilator waveform analysis in context with the neurological assessment. A hyperdynamic precordium as well as large-amplitude cardiogenic oscillations were identified in pressure, volume, and flow waveforms. Identifying this phenomenon enabled a more appropriate ventilator trigger mode and sensitivity to be set and optimized ventilator management.

Summary
Ventilator autotriggering caused by cardiogenic oscillation in brain-dead patients may be more common than is generally realized. This phenomenon can prolong the ICU experience for patients’ families, create confusion regarding the determination of brain death, and add to delay in organ recovery for transplantation.

A patient with a catastrophic brain injury who has fixed and dilated pupils at midpoint and is flaccid and areflexic on neurological examination may overbreathe the ventilator set rate because of significant cardiogenic oscillation in ventilator pressure and flow waveforms. To identify this clinical situation, clinicians must conduct a physical assessment to determine the presence or absence of intrinsic respiratory effort. An analysis of ventilator pressure and flow waveforms to assess for oscillations in phase with the cardiac cycle and to rule out waveform deflections indicating intrinsic respiratory drive is also appropriate. More detailed waveform analysis may be facilitated by decreasing the size of the scale on the respective waveform graphics display, which will enhance visualization of cardiogenic oscillations. Adjusting the ventilator trigger mode and/or sensitivity beyond the amplitude produced by the cardiogenic oscillation in the ventilator waveform can eliminate autotriggering, optimize ventilator management, and appropriately facilitate determination of brain death.

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FINANCIAL DISCLOSURES
None reported.

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