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# Intra-Aortic Balloon Pump Deflation Generated Suction-like Forward Aortic Blood Flow and Auto-Triggering of a Flow-Triggered Mechanical Ventilator

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# Intra-Aortic Balloon Pump Deflation Generated Suction-like Forward Aortic Blood Flow and Auto-Triggering of a Flow-Triggered Mechanical Ventilator

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## Abstract

Auto-triggering with the mechanical ventilators can be a problematic nuisance; however, it can be catastrophic too if the resultant respiratory alkalosis is not recognized early and treated with technical changes within the ventilatory mode and/or trigger settings of mechanical ventilation. We hereby present a postoperative case of a patient in which intra-aortic balloon pump (IABP) inflation-deflation cycle was triggering the mechanical ventilator. Intra-aortic balloon pump deflation generated suction-like forward aortic blood flow related auto-triggering of a flow-triggered mechanical ventilator should always be crossed off the checklist to correct the unexplained respiratory alkalosis in intensive care unit patients with cardiogenic shock who are either medically paralyzed or are on drug-induced paralysis (neuromuscular blockade).

## Introduction

Auto-triggering with the mechanical ventilators can be a problematic nuisance; however, it can be catastrophic too if the resultant respiratory alkalosis is not recognized early and treated with technical changes within the ventilatory mode and/or trigger settings of mechanical ventilation. We hereby present a postoperative case of a patient in which intra-aortic balloon pump (IABP) inflation-deflation cycle was triggering the mechanical ventilator.

## Case Report(s)

A 56 year-old-female was admitted to the surgical intensive care unit with open chest after aortic valve replacement for severe aortic stenosis and IABP in-situ for cardiogenic shock with severe pulmonary hypertension. Mechanical ventilation was maintained with assist/control mode at set respiratory rate of 14 breaths per minute with flow trigger at 2 liters/min.

Neuromuscular blockade was maintained with continuous infusion of cisatracurium and titrated to the train-of-four twitches 1/4. On post-operative day 2, it was noticed that breath frequency has increased to 26 breaths per minute. Considering that patient might be breathing over the ventilator, the patient received a loading dose of 5-10 mg of cisatracurium to intensify the neuromuscular block to the train-of-four twitches 0/4. Arterial blood gases were drawn that showed respiratory alkalosis with pH 7.56. However, despite the additional dose of paralytic agent, the breath frequency did not improve. After re-consultation and re-evaluation with respiratory therapist who had observed similar case scenarios wherein patients with IABP in-situ and adequate neuromuscular blockade tended to hyperventilate over the set-ventilatory rate secondary to possible auto-triggering by the IABP, it was decided to change the ventilatory trigger to pressure trigger of 2 cm of water that resolved the hyperventilation and potentially catastrophic rising blood pH.

## Discussion

The underlying mechanism for this auto-triggering was theorized to be secondary false positive flow trigger response to the forward blood flow in the thoracic descending aorta that happens due to the suction-like mechanism occurring due to the deflation of the IABP balloon at the onset of the systole to reduce the afterload. The suction-like phenomenon decompresses/empties the cardiac blood volume and increases the displaced pulmonary volume that eventually drops the expiratory flow that auto-cycles the flow-triggered mechanical ventilator to deliver non-initiated inspired breath to the patient. However, as the intrathoracic and pulmonary displacement changes is only limited to the flow, the auto-triggering is not initiated secondary to the pressure trigger that is dependent on generation of the negative intrathoracic pressure.

IABPs are commonly used to help increase myocardial perfusion in patients experiencing cardiogenic shock,

intractable angina, low cardiac output after cardiopulmonary bypass, and as a bridge to further therapy in patients with MI, heart failure and arrhythmias to name a few (1). The pump is timed to inflate immediately after aortic valve closure thereby forcing blood into the coronary arteries, which branch off the proximal aorta, leading to increased myocardial perfusion. The IABP then deflates immediately before aortic valve opening, thus allowing forward flow of blood during systole (1). A search of the literature found a case report of autocycling in a patient with both IABP and LVAD in use; however, the authors attributed the cause of the autocycling to the intrathoracic pressure gradients caused by the LVAD and not the IABP (2). The authors of that case reported that their patient's hyperventilation was managed by turning off the trigger sensitivity and switching to SIMV. No documented case of isolated IABP-associated autocycling with flow trigger could be found. In another study, autocycling was shown to occur in 50% of patients with total artificial hearts (TAH's) when using the flow-trigger setting (3). This was attributed to the significant pulmonary volume displacement caused by TAH oscillations while employing PEEP, which will work to maintain end-expiratory pressure at the expense of altered flow and, subsequently, may result in autocycling of the ventilator (3). We believe it is possible that the IABP device in our case caused the same pulmonary volume displacement as has been seen with TAH devices (4) and, therefore, lead to the autocycling that we observed.

## Conclusion

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In summary, intra-aortic balloon pump deflation generated suction-like forward aortic blood flow related auto-triggering of a flow-triggered mechanical ventilator should always be crossed off the checklist to correct the unexplained respiratory alkalosis in intensive care unit patients with cardiogenic shock who are either medically or surgically paralyzed or are on drug-induced paralysis (neuromuscular blockade).

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