Reversion of Supra-Ventricular Tachycardia Through High Positive End-Expiratory Pressure During Mechanical Ventilation in Critically Ill Patients

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ABSTRACT

The Valsalva manoeuvre is widely recognised for its effectiveness in reverting supra-ventricular tachycardia (SVT) in patients with good coordination. However, this is not applicable in sedated ventilated patients and there is a dearth of literature regarding the application of Valsalva in unconscious patients on mechanical ventilation. The authors, for the first time, present a novel non-pharmacological method to treat SVT in critically ill patients on mechanical ventilation, employing the high positive end-expiratory pressure (PEEP) technique. This method successfully reverted SVT in two patients. While the exact mechanism remains unconfirmed, it is believed to be similar to that of the Valsalva manoeuvre. Further research is needed to validate this technique and determine the optimal ventilator settings, including the PEEP threshold, for such cases.

Key Words: *Valsalva manoeuvre, Supraventricular tachycardia, Positive end-expiratory pressure, Mechanical ventilation.*

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Tachyarrhythmias are common among critically ill patients, with a high in-hospital mortality of 29% in patients with supraventricular arrhythmias.[1] The well-known postural modification to the standard Valsalva manoeuver enables promising effects for conversion from supra-ventricular tachycardia (SVT) to sinus rhythm, avoiding adverse effects caused by pharmacological cardioversion.[2] However, the success of such a procedure relies mainly on the conscious, oriented, and cooperative patient. Furthermore, in the authors' setup, pharmacological treatment for SVT is often delayed due to medication non-availability. As a first-line treatment for SVT, the ideal form of Valsalva manoeuver in patients on mechanical ventilation has never been reported previously. Thus developing non-pharmacological treatment modalities for SVT seem to be the need of the hour.

In ventilated patients, due to the closed loop developed during mechanical ventilation, it is easy to control the airway pressure by modulating the invasive ventilator's settings to efficiently influence the intrathoracic pressure and haemodynamics. This may result in stimulation of the vagus nerve, thus reversing or preventing SVT. Positive end-expiratory pressure (PEEP) has been evidenced to inhibit the phrenic motoneuronal activity in cats in dosage effect through the vagal nerve afferents.[3] In a case report by Hsu and Trott, a modified Valsalva manoeuver by adding inspiratory hold for 30 seconds terminated SVT within 30 seconds.[4] However, in both publications, the appropriate settings of PEEP without significant haemodynamic compromise, while triggering the activation of the vagal nerve are unclear.

Here, a high PEEP strategy to reverse SVT was successfully tested in two ventilated patients. The first case was a severely injured, 35-year male patient who was transferred to the emergency intensive care unit (EICU) nine days after a car accident. He remained orally intubated due to a persistently low level of consciousness following severe head trauma, with a Glasgow coma scale score of E2VTM1. Despite pulmonary contusion, he responded well to oxygen (oxygenation index 221). The primary reason for his referral was the deterioration of his condition due to new-onset sepsis-associated oliguria for two days, which was beyond the capability of the local hospital. After admission, continuous renal replacement therapy was initiated due to his deteriorating renal function. A low dose of norepinephrine (0.08 ug/kg/min) was infused to maintain a mean arterial pressure of 85 mmHg. The next day, around noon, the monitor showed a sudden tachyarrhythmia (heart rate 170 beats/minute), with decreased blood pressure (BP of 82/59 mmHg) and a drop in oxygen saturation (SpO_2 from 97 to 94%). A

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bedside electrocardiogram confirmed paroxysmal SVT. His serum potassium and core temperature were normal. Electrical conversion was not urgently considered as the haemodynamics did not seem unacceptable. The SVT persisted for 15 minutes despite bilateral alternate carotid sinus massages.

The ventilator mode was then switched from pressure-controlled to volume-controlled ventilation. The respiratory rate was set at 15 breaths/minute, tidal volume (VT) at 450 ml, inspired oxygen (FiO_2) at 0.5, inspiratory / expiratory ratio (I/E) at 1:2, and PEEP at 5cm H_2O. PEEP was gradually increased from 5 to 12 cm H_2O in increments of 2 cm H_2O. Ventilator parameters were carefully monitored to avoid a plateau pressure of 30 cm H_2O or higher. Upon adjusting PEEP to 12 cm H_2O, the heart rate immediately decreased to 113 beats/min and returned to sinus rhythm within seconds. BP and SpO_2 also returned to baseline levels (147/85mmHg and 98%, respectively) (Figure 1A and B). PEEP was then reset to 5 cm H_2O, and the SVT did not recur.

Figure 1: Reversion of SVT to sinus rhythm *via* high positive end-expiratory pressure (PEEP) during mechanical ventilation. (A) Monitoring of an SVT episode starting at 11:40 (not displayed) and reverting to sinus rhythm by 11:58. It shows changes in heart rate (HR), SpO_2, and arterial pressure. (B) Gradual increase of PEEP to manage SVT with meticulous monitoring to maintain plateau pressures of less than 30 cm H_2O.
Note: Ventilator time was uncalibrated, showing a 15-minute delay.

This method was successfully replicated in another patient, a 70-year-old male patient who was transported to the EICU eight days following a car accident. He suffered severe head and chest trauma requiring mechanical ventilation. There was no history of heart disease. On the morning of the fifth day during shift turnover, an episode of SVT (heart rate 174 beats/minutes) developed, as evidenced by electrocardiograph. Fortunately, his BP and SpO_2 were not significantly affected. A blood biochemical test revealed hypokalaemia (3.3 mmol/l). He received intravenous pre-prepared potassium to correct the hypokalaemia. Similarly, bilateral alternate carotid sinus massage was performed to reverse the SVT but was futile. A high PEEP strategy was then applied based on previous experience (Figure 2 A and B). It needs to be emphasised that this case experienced hypotension (BP 80/43 mmHg) when adjusting PEEP to 12 cm H_2O, as shown in Figure 2A. For safety considerations, PEEP was reduced to 10 cm H_2O, and BP was increased to 87/47 mmHg, but SVT was not reversed. PEEP was then increased to 12 cm H_2O again, and the SVT was quickly reversed to sinus rhythm within seconds.

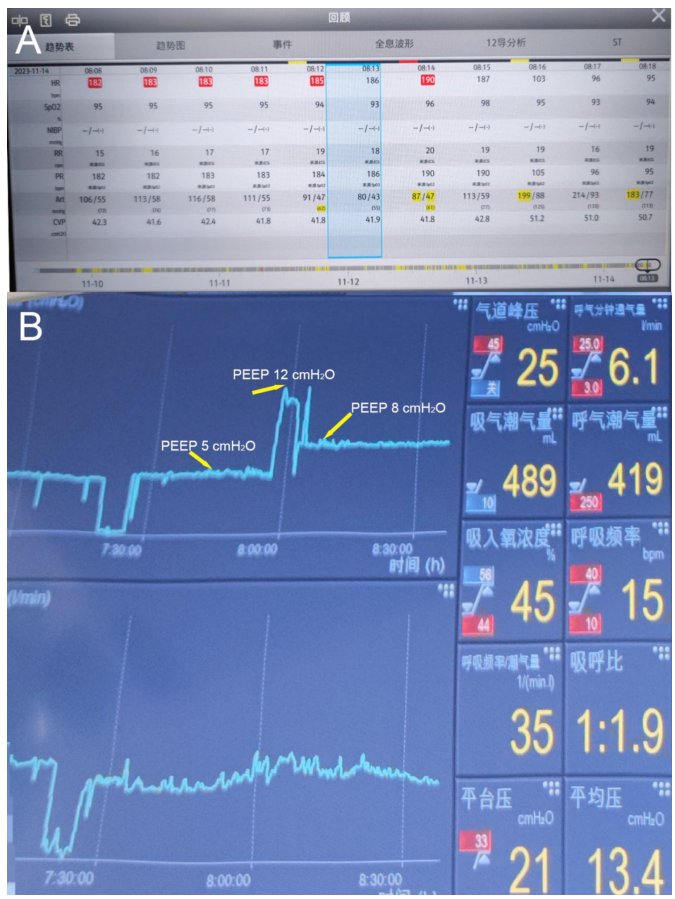

Figure 2: The second case of reversion of SVT through high positive end-expiratory pressure (PEEP) during mechanical ventilation. (A) displays the monitoring of the SVT episode, which persisted for about 20 minutes before reverting to sinus rhythm at 8:16. (B) following a gradual increase in PEEP to 12 cm H_2O. It should be noted that the ventilator's time reading was not calibrated to the actual time, resulting in a 15-minute delay.

This is the first attempt worldwide to reverse SVT using high PEEP during mechanical ventilation. The exact mechanism remains unclear but might be similar to the Valsalva manoeuver, suggesting that increased intrathoracic pressure from high PEEP could enhance parasympathetic nerve excitability. It should be noted that high PEEP could negatively affect cardiac output and cerebral perfusion, which requires close monitoring.[5] However, a moderate PEEP (5-10 cm H_2O) has been shown to have no adverse influence on arterial blood pressure in

mechanically-ventilated trauma patients.[6] It seems unlikely that the reversal of SVT can be attributed to spontaneous remission in both the patients, especially since it lasted at least for 15 minutes and carotid sinus massage proved ineffective. Nonetheless, further research is necessary to validate this approach and determine the optimal ventilator settings, including the PEEP threshold, for such cases.

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