**CASE REPORT**

**Abdominal muscle action during expiration can impair pressure controlled ventilation**

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**Summary**

Pressure controlled ventilation, and pressure support for spontaneous breathing are often used in intensive care because coordination of the ventilator with patient efforts can improve comfort and possibly reduce sedation. However we report a series of 10 patients whose efforts did not synchronise with pressure controlled ventilation. This was incorrectly diagnosed as inadequate sedation, and treated with increased sedation or muscle paralysis. Better recognition of this condition showed that slow respiratory rates and increased abdominal muscle action during expiration can affect pressure-controlled ventilation and pressure assisted breathing. If the condition is not recognised, treatment for poor synchronisation may delay weaning or be inappropriate.

**Keywords** Abdominal muscles. Intermittent positive-pressure ventilation. Analgesics; opioid.

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Sedation practice in intensive care varies. Data used to formulate guidelines have been drawn from heterogenous sources [1]. Current guidelines suggest that analgesia should be with an opioid and then sedation provided with either benzodiazepines or propofol [2]. These guidelines list potential adverse effects of opioid therapy such as respiratory depression, hypotension, sedation, hallucinations, and constipation, but do not mention difficulty in synchronisation with the ventilator. The guidelines advise that neuromuscular blocking agents should remain a ‘last resort’ to aid synchronisation [1].

Muscle rigidity is a recognised side effect of opioids, often noted after large doses at induction of anaesthesia [3,4], and also after surgery [5]. This effect can be troublesome when pressure controlled ventilation is used during intensive care. We experienced problems in patients recovering from residual neuromuscular block after surgery, and in patients whose level of sedation was decreasing after use of larger doses of sedative agents at intubation.

Pressure controlled ventilation (bilevel positive airway pressure [BiPAP]) can be set to deliver time-cycled alternation between two positive airway pressure values, and can be used along with pressure support for spontaneous breathing (assisted spontaneous breathing [ASB]). Pressure control limits airway pressure so that possible lung damage may be reduced. When this mode of ventilation was used for patients receiving opioids, we noted that some of the increases in airway pressure did not generate a tidal volume. At first we did not appreciate the link between this poor synchronisation and abdominal contraction during expiration, and inappropriate management resulted.

**Methods**

One of the authors (VP) noticed a series of 10 patients who failed to synchronise with the ventilator. The patients were receiving opioid infusions (morphine, fentanyl, or alfentanil) and were ventilated with either Draeger Evita (Dräger Ltd, Hemel Hempstead, Herts, UK) or Puritan Bennet (Mallinckrodt UK Limited, Bicester, Oxfordshire, UK) ventilators set to deliver...
either time-cycled pressure control ventilation (BiPAP) or patient triggered inspiratory support (ASB). Patient consent was not sought for formal further investigations.

Results

Index patient

A patient developed septic shock requiring mechanical ventilation. After rapid sequence induction of anaesthesia and tracheal intubation, sedation and analgesia were provided with propofol 150 mg h⁻¹ and fentanyl 250 μg h⁻¹. Ventilation was with a Dräger Evita 4 ventilator set for BiPAP and ASB using pressure support values of 5 and 20 cmH₂O, with a respiratory rate set at 12 breaths min⁻¹ and an inspiratory:expiratory ratio of 1:2. With these settings, the measured tidal volume was about 700 ml. A capnograph (Datex, Datex Ohmeda Ltd, Hatfield, Herts, UK) was used to estimate end-tidal carbon dioxide and ventilatory rate measured from thoracic impedance changes from the electrocardiograph monitor.

About one hour after intubation, the respiratory rate indicated by the capnograph and impedance monitor decreased progressively to between 5 and 9 breaths min⁻¹, and tidal volume increased to 1000–1200 ml (Fig. 1). End tidal carbon dioxide fraction also increased. However, the ventilator display showed pressure cycles applied regularly at 12 breaths min⁻¹ and the ventilator indicated a respiratory rate of 12. When the ventilator rate setting was reduced, or the ventilator was set to trigger mode alone, the rate observed on the capnograph and impedance monitors (which was the clinically observed rate) was not affected. A fault with the ventilator was suspected, but exactly the same conditions occurred with a replacement ventilator. After increasing the sedation, increasing the ventilator rate to 15 breaths min⁻¹ and reducing the end-tidal carbon dioxide fraction to about 4.2%, a stable respiratory rate was obtained.

Patient 2

After major surgery, the lungs of a 70-year-old 90-kg patient were ventilated with a Dräger Evita 4 ventilator, using BiPAP with ASB, set to deliver pressures of 5 and 20 cmH₂O, respiratory rate 10 breaths min⁻¹, and an inspiratory:expiratory ratio of 1:2. With these settings, tidal volume was 700–800 ml. Sedation was with propofol 50 mg h⁻¹, and morphine 1 mg h⁻¹. On the day after surgery, these infusions were stopped in preparation for weaning from mechanical ventilation. The respiratory rate indicated by the capnometer decreased progressively to 5 breaths min⁻¹ and tidal volume increased to 1200 ml. The ventilator displayed a rate of 10 breaths min⁻¹. The ventilator display showed that an increase in airway pressure was being applied, but no flow occurred into the patient. With these events the pressure profile was different, with a constant greater airway pressure throughout. These events were counted as ‘breaths’ by the ventilator, but were not clinically evident and did not register on the impedance or capnometer monitors. During expiration, the abdomen was rigid to palpation and central venous pressure increased considerably.

Patient 3

A 69-year-old lady with community-acquired pneumonia required mechanical ventilation. She was sedated with propofol 100 mg h⁻¹ and fentanyl 250 μg h⁻¹. A Dräger Evita 4 ventilator was set using pressure support values of 5 and 20 cmH₂O, with a respiratory rate of 12 breaths min⁻¹. After 6 h after intubation, the ventilator alarm indicated – ‘low tidal volume’ and ‘tube block’. Manual ventilation with a resuscitation bag was easy with no evidence of obstruction. When mechanical ventilation was re-started, the alarm still indicated ‘tube blocked’. After senior advice had been obtained, the patient was given neuromuscular blocking agents and the tracheal tube was changed. Mechanical ventilation was easy for 3 h, and then the same features recurred. The patient was given more neuromuscular blocking agents and bronchoscopy was performed to exclude possible obstruction. No abnormality was found. After a further 4 h, the problem recurred for a third time. On examination, the abdomen was felt to be rigid during expiration. The central venous pressure was 35 cmH₂O. Intra-abdominal pressure, estimated from the bladder pressure [6], was also 35 cmH₂O. The ventilator indicated a respiratory rate of 12 breaths min⁻¹, but the respiratory

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**Figure 1** Plot of patient measurements noted during management of the index patient. The patient was intubated 90 min before the chart starts. The arrow indicates when sedation was given and manual ventilation started. Shortly after this, mechanical ventilation was re-started at 12 breaths min⁻¹.
rate clinically, and by capnograph, was 5 breaths.min\(^{-1}\). When a ventilator pressure wave synchronised with the patient’s inspiration, tidal volume was about 1200 ml, but when a mandatory pressure cycle was applied during patient expiration, then the tidal volume was less than 20 ml. Opioid induced expiratory muscle activity was suspected. Naloxone 40 µg intravenously abolished the effect, and the respiratory rate became 12–15 breaths. min\(^{-1}\). The effect lasted 15 min, and then respiratory rate and oxygen saturation decreased, and end-tidal carbon dioxide increased. Neuromuscular blocking agents were given. Tidal volume synchronised with the ventilator, and central venous and bladder pressure decreased to about 15 cmH\(_2\)O. When neuromuscular blocking agents were stopped to allow weaning, the same clinical scenario occurred. The patient was allowed to breathe spontaneously with pressure support, with a low respiratory rate. Three days later she was weaned successfully.

**Further patients**

After the problem had occurred in these three patients, the clinical pattern became clear and management became simpler. A further seven patients with similar features were noted, and in all sedation was with propofol infusions from 50 to 200 mg.h\(^{-1}\) and an opioid (either morphine 1–3 mg.h\(^{-1}\), fentanyl 250 µg.h\(^{-1}\) or alfentanil 1.5–3 mg.h\(^{-1}\)). A tracing of airway pressure and flow is shown from one of these patients (Fig. 2). All patients had a low intrinsic respiratory rate. The abdomen was rigid during expiration, even after expiratory flow had ceased. Central venous pressure and intra-abdominal pressure were high and changed in phase with the intrinsic respiratory rate of the patient. In each patient, either naloxone or neuromuscular blocking agents abolished the condition.

**Discussion**

Abdominal rigidity is well described in anaesthetised patients [7,8]. Fentanyl can reduce functional residual capacity by about 500 ml, equivalent to about 10 cmH\(_2\)O expiratory pressure, even without clinically evident muscle activity [9]. Opioids increase muscle tone by a complex pathway involving several transmitter systems, including central noradrenergic and glutaminergic pathways via the locus coeruleus [10,11]. In patients after abdominal surgery, phasic expiratory abdominal muscle action is often prominent. This activity is low during inspiration, increases rapidly with the onset of expiration, persists during expiration and decreases rapidly with the onset of the next inspiration. This activity can increase abdominal pressure by up to 20 cmH\(_2\)O [5]. Similar features have been reported during anaesthesia after 25 µg fentanyl [12]. In the present report, the effect of naloxone in the third patient showed that the phenomenon was probably mediated by opioid actions.

The patients in this report had a low intrinsic respiratory rate with prolonged expiration. During expiration, the abdominal muscles were actively contracting so that respiratory system compliance was reduced. When the ventilator applied higher airway pressures during patient expiration, no inspiratory flow was generated. Hypercapnia developed and intrinsic drive was increased so that the spontaneously inspired tidal volume increased. Increased carbon dioxide levels also stimulate abdominal contraction, since the drive to these muscles is increased in the same way as to the inspiratory muscles [13]. If further sedatives are given to ‘treat’ the dys-synchrony, this could delay weaning. If the condition is not recognised, inappropriate therapy, as in our third patient, may be given.

Inspection of chest wall movement and abdominal palpation are generally sufficient to confirm the presence of marked abdominal muscle activity. Naloxone can reduce excessive expiratory abdominal muscle activity, but it may cause inadequate analgesia. Abdominal contraction is reduced, by lowering CO\(_2\) levels, and this has support from animal studies [14]. However early recognition should avoid ineffective treatment and we hope that these descriptions will alert clinicians to this clinical problem so allowing prompt and appropriate management.
References


