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Near-fatal misuse of medical tape around an endotracheal tube connector during inhalation anesthesia in a horse

Rachael Gregson, R. Eddie Clutton

Abstract — A 7-year-old gelded Irish sports horse weighing 650 kg was anesthetized on 2 consecutive days for lavage of a septic right radio-carpal joint. On both occasions the endotracheal tube connector, which had been bound in medical tape to produce an airtight seal, functioned as a unidirectional valve during mechanical ventilation, retarding expiration, imposing positive end expiratory pressure (PEEP), and probably continuous positive airway pressure (CPAP). The equipment dysfunction was not identified on either occasion despite close inspection prompted by progressive increases in airway pressure and thoracic distension. Whilst the PEEP and CPAP exerted unexpectedly modest cardiovascular effects and the horse recovered uneventfully on both occasions, the improvisation may have proven fatal in a higher-risk subject.

Résumé — Mauvais usage quasi-mortel de ruban adhésif médical autour d’un raccord de tube endotrachéal durant l’anesthésie par inhalation chez un cheval. Un hongre de sport Irlandais âgé de 7 ans pesant 650 kg a été anesthésié deux journées consécutives pour le lavage d’une articulation radio-carpienne droite septique. Lors des deux instances, le raccord du tube endotrachéal, qui avait été attaché à l’aide de ruban adhésif médical pour produire une fermeture hermétique, a servi de valve unidirectionnelle durant la ventilation mécanique, retardant l’expiration, imposant une pression positive en fin d’expiration et probablement une ventilation spontanée en pression positive continue. La défaillance de l’équipement n’a pas été identifiée dans l’une ou l’autre de ces instances, malgré une inspection minutieuse suscitée par des hausses progressives de la pression des voies aériennes et un gonflement thoracique. Même si la pression positive en fin d’expiration et la ventilation spontanée en pression positive continue ont eu des effets cardiovasculaires modestes inattendus et que le cheval s’est rétabli sans incident lors des deux instances, l’improvisation aurait pu s’avérer mortelle chez un sujet présentant des risques plus élevés.

(Traduit par Isabelle Vallières)

Blockage of endotracheal tubes and their connectors by foreign bodies (1), blood or secretions (2), or resulting from manufacturing faults (3) has caused problems in human anesthesia and at least 1 porcine fatality has been reported after a flexometallic tube was crushed (bitten) and subsequently occluded in-situ (4). In the current report, a 7-year-old Irish sport horse was anesthetized for lavage and arthroscopy of a septic radio-carpal joint on 2 occasions. The endotracheal tube (ETT) connector used within the 28-mm OD ETT was too small and fitted loosely within the tube. Consequently, the outside surface of the connector was bound with medical tape to increase its outer diameter and create a gas-tight seal when fitted within the ETT. After the horse’s trachea had been intubated and mechanical ventilation imposed, the tape shifted within the tube and assumed a conical profile, creating a crude unidirectional valve. This allowed relatively normal inspiration but impeded expiratory gas flow resulting in varying degrees of continuous positive airway pressures (CPAP) and positive end expiratory pressure (PEEP). Whilst the latter has been used in anesthetized horses in attempts to increase arterial oxygen tensions (PaO₂), increased work of breathing (5,6) and reduced cardiac output (7,8) have also been reported.

Case description

A 7-year-old Irish sport horse weighing 650 kg was anesthetized for the arthroscopic examination and lavage of a penetrating left radiocarpal joint wound. Phenylbutazone (Equipalazone 1 g oral powder; Dechra Veterinary Products, Shrewsbury, UK), 1.5 mg/kg body weight (BW) had been administered to the horse orally twice daily for the 8 d following its injury, and an intravenous (IV) dose (Equipalazone 200 mg/mL Solution for Injection; Dechra), 3.5 mg/kg BW, was injected immediately before anesthesia. Benzyl penicillin sodium (Crystapen
5 Mega Units 3 g; MSD Animal Health, Milton Keynes, UK), 10 mg/kg BW, and gentamicin (Genta Equine 10% Solution for Injection; Dechra), 6.6 mg/kg BW, were also administered IV at this time. Food had not been withheld before surgery. In line with standard practice at this hospital, a range of ETTs (26, 28, and 30 mm OD) were prepared before pre-anesthetic medication was administered. Each tube had been rinsed with hot water and its lumen cleaned with a bottle-brush after previous use, and each had been stored with their connectors in-situ.

Before use, the cuffs were inflated to check for leaks (which were not apparent). The large animal anesthetic machine (Tafonius; Vetronic Services, Devon, UK) which had been used without problem in the previous 48 h, was inspected, leak-tested to find to be in good working order by the first author (RG).

The horse was pre-medicated with IV romifidine (Sedivet; Boehringer Ingelheim, Bracknell, Berkshire, UK), 80 μg/kg BW. Five minutes later when marked sedation was present, anesthesia was induced with ketamine (Vetalar V; Pharmacia & Upjohn Animal Health, Sandwich, UK), 2.2 mg/kg BW, and diazepam (Diazepam Injection BP; Hameln Pharmaceuticals, Nexus, Gloucester, UK), 0.05 mg/kg BW, mixed in the same syringe immediately before injection. Once the horse was recumbent, its trachea was intubated blindly with a 28-mm endotracheal tube (ETT) and the animal was hoisted onto the table where it was positioned in dorsal recumbency. The ETT was connected to the anesthetic machine which was pre-filled with oxygen and 5% sevoflurane (Sevofo; Abbot Animal Health, Maidenhead, Berkshire, UK). Initial vaporizer settings were 5% sevoflurane carried in 5 L/min oxygen and the animal breathed spontaneously with a tidal volume of approximately 8 L at 12 breaths/min; chest excursions appeared to be normal and the brisk respiratory rate was attributed to a light plane of anesthesia. Vaporizer setting was adjusted to maintain end-tidal sevoflurane concentrations at approximately 2.2% throughout anesthesia. During surgical preparation, the right facial artery was cannulated for the direct measurement of arterial pressure and the collection of blood samples. The transducer for blood pressure measurement was positioned level with the thoracic inlet and zeroed to atmospheric pressure before beginning measurement. The arterial blood pressure (aBP) was monitored along with respiratory rate and muscle tone and caused the nystagmus to disappear. Heart rate remained constant at 35 beats/min during this period, although blood pressure monitoring had not yet begun. Morphine (Morphine Sulphate injection BP; Martindale Pharmaceuticals, Brentwood, Essex, UK) 90 mg was administered IV at this time.

A blood gas sample taken 30 min after induction revealed a modest respiratory acidosis [pHa was 7.38 while PaCO₂ was 7.73 kPa (58 mmHg)]. The arterial O₂ tension was 11.01 kPa (83 mmHg). At the onset of surgery (40 min after induction) systolic (SAP), mean (MAP), and diastolic (DAP) arterial pressures were 90, 74, and 60 mmHg, respectively. For the first hour of surgery the horse continued to breathe spontaneously at approximately 6 breaths/min; PéCO₂ remained at approximately 6.67 kPa (50 mmHg). Inadequate anesthesia — based on an unexpected increase in blood pressure (SAP: 100 mmHg; MAP: 75 mmHg; DAP: 60 mmHg) whilst other variables remained constant — was controlled adequately after 65 min of surgery with another ketamine/midazolam injection at the previous doses. A second arterial blood sample taken at this time revealed deterioration in PaO₂ to 7.01 kPa (53 mmHg) without change in pHa and PaCO₂. Consequently, positive pressure ventilation was imposed. A tidal volume of 8 L was delivered at 8/min reflecting a minute volume increase 10% greater than the previous value recorded during spontaneous breathing.

After about 10 min of mechanical ventilation a hissing sound was heard emanating from the vicinity of the “Y-piece” and the endotracheal tube connector, but a leak could not be localized to the breathing system despite an attempt being made to seal potential sites. There was no smell of sevoflurane. The airway pressure was noted to have risen from <2 cmH₂O to 40 cmH₂O and arterial pressure had risen: DAP: 80; MAP: 95; SAP: 120 mmHg. The plane of anesthesia was still judged to be adequate, as there was satisfactory muscle relaxation and no nystagmus. Mechanical ventilation was suspended, and increasingly negative (~5 cmH₂O) airway pressures were recorded at each inspiratory effort; negative pressure during the previous period of spontaneous ventilation was in the region of ~2 cmH₂O. The blood pressure remained elevated. Eight actuations of salbutamol (Ventolin Evohaler; GlaxoSmithKline, Middlesex, UK; 100 μg/delivery), 120 μg/kg BW were administered via the ETT using a proprietary connector as described by Robertson and Bailey (9). During this process, when the ETT connector was temporarily disconnected from the “Y piece,” gas was heard and felt emerging from the ETT. The expired gas analyzer was disconnected for 10 min after salbutamol administration during which time blood pressure remained elevated at DAP:110; MAP:130; SAP:148 mmHg; and inadequate anesthesia, based on increased muscle tone, a brisk blink reflex and nystagmus, was treated with a further and identical dose of ketamine/midazolam. When the expired gas analyzer was re-connected it registered an Fe’CO₂ of 10.39 kPa (78 mmHg) and there was significant re-breathing: FiCO₂ was 1.33 kPa (10 mmHg). Surgery ended and when the drapes were removed, the animal’s abdomen and thorax were seen to be notably distended.

Intravenous xylazine (Chanazine, Chanelle Animal Health, Galway, Ireland), 0.2 mg/kg BW was administered and the horse was transferred to the recovery box where it was placed in right lateral recumbency. Oxygen was insufflated into the airway at 15 L/min. A marked expiratory sound which was present throughout recovery disappeared on tracheal extubation. At
this time the thoraco-abdominal distension was not apparent. The horse recovered from anesthesia briskly and uneventfully. A note was left on the anesthetic machine indicating the possibility of a mechanical problem; no obvious problem could be identified by the first author on a post-anesthetic machine check and cleaning.

The horse was re-anesthetized the following day by the second author (REC) for the same procedure. The same anesthetic machine was leak-tested and the ventilator performance tested before use; no problems were identified. The same ETT had been cleaned by rinsing with hot water and brushing the lumen without removing the connector; the cuff was checked immediately before use. The anesthetic technique used was similar, except that morphine was included and given by constant rate infusion (0.1 mg/kg BW per hour) and end-tidal sevoflurane (Fe\textsuperscript{9} sevo) was maintained at a lower level (approximately 2.0%). Physiological variables were similar (HR: 36 beats/min, DAP: 45 mmHg, MAP: 60 mmHg, SAP: 85 mmHg at the start of the anesthetic), and the same problems were encountered. The horse appeared to be “light” on 3 occasions: first, during the initial 5 min of anesthesia; second, after approximately 60 min of anesthesia; and third, approximately 30 min after surgery had commenced. As before, anesthesia was deepened with midazolam (0.02 mg/kg BW) — ketamine (0.2 mg/kg BW) injections on each occasion. The first arterial blood gas sample taken 30 min after the induction of anesthesia when FiO\textsubscript{2} was 0.81 revealed a mild respiratory acidosis [PaCO\textsubscript{2}: 6.67 kPa (50 mmHg)] and a low PaO\textsubscript{2} [11.7 kPa (88 mmHg)]. The MAP was 70 mmHg. Positive pressure ventilation was begun with a tidal volume of 7.5 L and a rate of 8 breaths/min. This caused unexpectedly high end-expiratory pressures (40 to 50 cm H\textsubscript{2}O). The MWPL (maximum working pressure limit) alarm sounded continuously and progressive thoracic distension was observed. The “Y-connector” was disconnected on 3 occasions, and on each, gas was felt and heard flowing from the endotracheal tube; this coincided with a reduction in thoracic distension. A tentative diagnosis of equipment failure was made but surgery ended before its precise cause could be identified. During the transfer of the horse from the theater to recovery, dyspnea was apparent, and inspection of the ETT lumen revealed a “rosette” of pink-colored material within the lumen, opening and closing with the animal’s respiratory efforts. Immediate tracheal extubation restored eupnea and was associated with a prolonged “expiration” which coincided with thoracic deflation. Nasal O\textsubscript{2} was insufflated during recovery and the horse recovered uneventfully.

Examination of the endotracheal tube revealed that the connector had been wrapped with at least 10 layers of Leukoplast Sleek (Smith and Nephew UK, Willerby, Hull, UK) medical tape (Figure 1). This had been done because the connector was too small for the ETT and had required modification to increase its external diameter, improve its fit, and restore an airtight seal within the proximal end of the ETT. Over an unknown period, the outermost layers of the tape had slipped towards the distal end of the ETT forming an elongated cone of tape. Being relatively unsupported by underlying tape layers and the connector, this tape “rosette” began moving readily in response to pressures changes across the constriction. The “rosette” allowed ingress of gas, although the narrowing to the tube diameter demanded higher airway pressures during IPPV and increased effort during spontaneous breathing. The egress of gas was more severely restricted, with the escape of excess gas only being obvious when the ETT was intermittently disconnected during anesthesia, when the horse’s thorax and abdomen had already become distended. The slipped tape had become a functional one-way valve, opening partially during inspiration yet restricting gas egress on expiration. The restriction of expiratory gas flow and raised airway pressures meant that the tape “rosette” was also producing an undefined level of PEEP and CPAP, with accompanying physiological effects.

Discussion

In human beings, PEEP prevents small airway closure, increases FRC (10,11) and prevents alveolar collapse (12). It improves arterial oxygenation in situations where FRC is reduced, for example, in abdominal surgery (11) and in 1 lung ventilation during thoracotomy (13). Positive end expiratory pressure has also been used to improve arterial oxygenation in cases of acute respiratory disease characterized by low lung compliance because

Figure 1. Two views of the endotracheal tube connector and medical tape adaptation which functioned as a crude unidirectional (expiratory) valve in an anesthetized horse.
of reduced surfactant, or pulmonary edema (12,14). However, its effects on arterial oxygenation may be variable, and undesirable physiological effects include reductions in minute alveolar ventilation (12), arterial pressure (14), and cardiac output when combined with CPAP (13).

The effects of PEEP and CPAP on the derangements commonly encountered in anesthetized horses, for example, large alveolar/arterial oxygen tension differences (15–17), appear to be highly variable — as are their associated cardiovascular effects. In 1 study of spontaneously breathing horses (5) PEEP had no significant effect on arterial oxygen tension, diaphragmatic position, or FRC, and rather than opening collapsed airways, it appeared to overinflate already well-ventilated lung. In another study, PEEP caused consistent increases in PaCO2 and high pressures (20 cmH2O) increased cardiac output, possibly by increasing the work required to exhale against the PEEP valve (6). In contrast, Wilson and Soma (8) found marked reductions in cardiac output when PEEP was applied to the mechanically ventilated lungs of ponies in dorsal recumbency, a change they attributed to a rise in intrathoracic pressure. However, in that study, PEEP increased FRC and PaO2 despite increasing dead space ventilation. Swanson and Muir (7) found that PEEP (up to 10 cmH2O) with dobutamine did not significantly decrease P(α-a)O2 in healthy, anesthetized horses during mechanical lung ventilation. However, when used as a treatment for hypoxemia in horses undergoing exploratory laparotomy, 10 to 15 cmH2O PEEP improved PaO2, but the effects were variable and most improvement occurred in animals with the lowest PaO2 (18).

Furthermore, a number of horses required cardiovascular support (fluids and dobutamine) for hypotension arising after PEEP was imposed.

The application of PEEP to selected lung lobes has a more predictable effect on arterial O2 tensions in horses, although the technique is not straightforward. Significant increases were observed when PEEP was applied only to the dependent diaphragmatic lung lobes in anesthetized horses in dorsal recumbency (19). Moens et al (20) used a tube-in-tube technique that allowed the application of PEEP to the dependent lung in anesthetized horses in lateral recumbency, and found that while this improved PaO2 through gas redistribution and increased FRC, it did so at levels of PEEP (20 cmH2O) associated with reduced cardiac output.

Continuous positive airway pressure (CPAP) increases FRC in the same way as PEEP and is recommended in non-anesthetized humans for the treatment of moderate to severe sleep apnea (21). It is commonly employed in non-invasive ventilation (NIV) (22) — the provision of respiratory support without endotracheal tubes. Used initially in intensive care units to treat chronic obstructive respiratory disease and acute respiratory failure, NIV/CPAP is currently used prophylactically to improve gas exchange and reduce atelectasis in patients with compromised respiratory function (22). Pre-oxygenation with CPAP has been used in obese female human patients in an attempt to delay desaturation during induction, but did not increase the time to desaturation compared to standard pre-oxygenation techniques (23). In anesthetized humans undergoing one-lung ventilation, 10 cmH2O CPAP is as effective as 10 cmH2O PEEP at improving arterial oxygenation (13). To the authors’ knowledge, there is no information on the use of CPAP in anesthetized horses.

During the first anesthetic in the case described here, the lungs were ventilated after approximately 1 h of anesthesia because PaO2 values were declining. This coincided with an increase in MAP (from 74 mmHg to 95 mmHg), which is difficult to explain, because PEEP in mechanically ventilated horses normally reduces MAP by increasing intrathoracic pressures and so reducing stroke volume and cardiac output (8). Raised airway pressures at this time confirm that intrathoracic pressure was increased. The unexpectedly high MAP could have been attributed to an increase in sympathetic tone caused by surgical stimulation, or the ketamine used to deepen anesthesia (24,25). However, MAP remained elevated (130 mmHg) once IPPV was suspended. As PEEP was probably still present at this stage, it is possible that cardiac output may have increased in an attempt to compensate for an increased expiratory effort (5,6). The high values for Fi’CO2 and Fi CO2 present at this time support the possibility that both cardiac output and alveolar dead space ventilation were increased. The increased CO2 tension could also have contributed to the continued high MAP, due to its effect on sympathetic tone (26). Problems were resolved once the trachea was extubated, confirming the adverse effects of the defective endotracheal tube connector.

During the second anesthetic, in which IPPV was imposed almost from the outset, arterial blood pressure remained adequate despite high airway and intrathoracic pressures. When IPPV was suspended towards the end of surgery, MAP rose from approximately 70 to 100 mmHg which suggests that IPPV and PEEP may have been exerting a greater hypotensive effect than spontaneous ventilation and PEEP. The modest increase of PaO2 from 11.7 kPa (88 mmHg) to 12.66 kPa (95 mmHg) may have been attributable to PEEP but other factors may also have been important. Wilson and McFeely (18) found that the most significant increases in PaO2 resulting from PEEP occur when hypoxemia (7.99 kPa, < 60 mmHg) is present. The animal in the current report was not markedly hypoxic.

The maximum level of PEEP imposed in this horse was unknown. However, high levels (50 to 60 cmH2O) caused by a malfunctioning PEEP valve have caused volutrauma and tension pneumothoraces in human subjects (27).

In the current case, the “rosette” valve did not occlude the airway completely because the tape’s inner surface had lost its adhesive properties (presumably during the process of elongation) which stopped the valve remaining closed on inspiration. Such additional complications cannot be dismissed as improbable. Had this occurred while the horse had been breathing spontaneously, the resulting obstruction would have lowered the intrathoracic pressure during inspiration and favoured alveolar transudation. If severe and, or prolonged, this may have resulted in pulmonary edema (28).

The clinical effects of PEEP observed in the current case are at odds with the literature, although differences may have arisen because the horse described here was undergoing surgery. The effects observed were also less severe than those reported elsewhere, being modest and short-lived. However, the inadvertent imposition of PEEP, or other features of ventilatory
management, resulting from the imprudent modification of anesthetic equipment may have had a less satisfactory outcome in horses which are hypovolemic and, or hypotensive. As a consequence of this case, the use of medical tape on ETT and anesthetic machines has been prohibited, and there is a requirement that patency of endotracheal tubes be established visually before their use.

References