



Correspondence

Airway management in a prehospital combat setting



To the Editor,

In a retrospective review of US Army medical evacuation patient care records, Hardy et al. [1] compared outcomes of US military injured that received prehospital advanced airway interventions. The authors conclude that patients who received a supraglottic airway devices (SAD) had higher morbidity demonstrated by fewer ventilator, hospital, and ICU free days than those receiving cricothyrotomy or mask ventilation.

We would like to add several appreciations. The authors do not specify the SADs used. This data is essential to make an accurate analysis of the data. It is well known that second-generation SADs, although they do not completely protect the airway from aspiration, provides better efficacy and safety compared with first-generation devices [2]. The first-generation SADs (e.g. the classic laryngeal mask airway) have several limitations, fundamentally providing only a moderate pharyngeal seal that may be associated with inadequate ventilation, gastric inflation, regurgitation and pulmonary aspiration. The design of second-generation supraglottic airways allows for greater pharyngeal seal pressures and they contain an oesophageal port which provides functional separation of the respiratory and gastrointestinal tracts and allows for the draining or aspiration of gastric contents. Second-generation SGAs are also more likely to enable oxygenation and ventilation [3]. Thus, only second-generation SADs are recommended in the recent guidelines [3–5]. Likewise, each second-generation SAD has specific attributes as the time of placement, seal pressure, type of separation of gastrointestinal and respiratory tracts and use as a conduit for endotracheal intubation (blind or fibre-optically guided tracheal intubation).

The competence and experience of the operator with the device also play a relevant role since they influence the success of insertion and correct placement. Different studies indicate a low failure rate in the clinical use, although the consequences of failure included an increase in hospital admission and ICU admission [6].

All of this justifies the need to specify the kind of SAD since they constitute a heterogeneous group of non-equated devices. Therefore, it is necessary to take all these data into account. Otherwise, the conclusions of this interesting work could be misleading.

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Impact of prehospital airway management on combat mortality



Response to Letter to Editor,

We appreciate the inquiry regarding our publication and thank you for the letter. We were unable to determine the type or generation of supraglottic airway devices used in the prehospital combat setting from the MEDEVAC documentation. An additional consideration affecting patient outcomes regarding supraglottic airway devices in the prehospital combat setting is the difference in the combat environment versus the hospital environment. As mentioned in our discussion section, combat medics do not carry paralytics, the patients are not fasting, and the environment is considerably different than that of the hospital. Furthermore, the primary causes of injury in our patient population are improvised explosive devices and high velocity rifles which result in injuries significantly different from those in the US civilian setting. This is in addition to the differing types of supraglottic airway and varying competence of the operator that you mentioned. Regardless, further research comparing the different supraglottic airway devices is necessary to draw more definitive conclusions.

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Other aspects of nitrous oxide-related neuromyelopathy



The point is well made that the “work-up” of suspected nitrous oxide (NO₂) myelopathy should include, not only serum vitamin B12 (i.e., cobalamin) measurement, but also documentation of serum homocysteine, methylmalonic acid (MMA), and folate levels [1]. The rationale is that some patients in whom cobalamin derangements are implicated in the aetiopathogenesis of NO₂-related neurotoxicity may, nevertheless, have serum cobalamin levels within the normal range [2]. In one such patient (case 1) NO₂-related neuropathy was initially mistaken for Guillain-Barre syndrome, arguably because there was no concurrent spinal cord demyelination and, hence, no abnormality detected on magnetic resonance imaging (MRI) of the entire spinal cord. On the basis of the provisional diagnosis of Guillain-Barre syndrome that patient was prescribed a 5 days course of intravenous immunoglobulin. The correct diagnosis was made only after eliciting a history of NO₂ recreational use, and also after documentation of a serum MMA level of 29,653 mmol/l (normal < 280 mmol/l). He gradually improved after he was prescribed vitamin B12 injections [2]. In the same series there was a patient (case 3), with NO₂-related myelopathy, in whom a normal serum vitamin B12 level coexisted with an elevated serum homocysteine level [2]. That patient experienced a complete recovery after a course of vitamin B12 injections [2].

NO₂-related neuropathy in the absence of abnormalities on MRI imaging of the spinal cord has been documented in anecdotal reports [2–4] and also in a retrospective study of 33 patients evaluated in one institution over a period of 10 years [5]. In the latter study the coexistence of myelopathy was confirmed using clinical manifestations or T2 hyperintensity on spinal cord MRI. Fifteen patients were evaluated by spinal cord MRI, among whom seven had T2 hyperintensity of the posterior columns. Overall 20

patients exhibited clinical features or MRI abnormalities suggestive of myelopathy [5]. A pseudo Guillain-Barre presentation similar to the one in case 2 [2] was documented in one anecdotal case report of NO₂ neurotoxicity [6]. This was a patient who initially experienced flaccid bilateral foot drop and sensory deficits in a stocking distribution. MRI of the lumbar spine was normal. The cerebrospinal fluid showed a protein concentration of 72 mg/dl and no pleocytosis. Within 24–48 h paresthesiae ascended to the nipple line, and he experienced clumsiness of fine finger movements. MRI of the cervical spine was then performed, and this showed focal nonenhancing posterior T2 lesion at C2–C6. He experienced significant improvement after a course of vitamin B12 injections [6].

The normal levels of serum vitamin B12 documented in cases 1 and 3 [2], and in other anecdotal reports [3,4] exemplify so-called “functional” cobalamin deficiency, the latter characterised by coexistence of normal serum cobalamin levels and accumulation of substrates of the reactions catalysed by cobalamin, namely, MMA and homocysteine [2–4]. In all subtypes of cobalamin deficiency the toxicity of NO₂ is mediated by the inactivation of vitamin B12 as a result of oxidation by NO₂. This leads to a reduction in the conversion of homocysteine to methionine [3], the latter a precursor of S-adenosylmethionine, which is necessary for myelin production in the central and in the peripheral nervous system [7,8].

Patients at risk of NO₂-related myelopathy are, reportedly, also those in whom NO₂ is administered as a general anaesthetic in the presence of predisposing factors for cobalamin deficiency, such as old age, inflammatory bowel disease, and vegetarianism [1]. Previous bariatric surgery should also be included in that list, given the fact that it, too, is a risk factor for cobalamin deficiency [9].

When NO₂ is administered as a general anaesthetic NO₂-related neurotoxicity may also occur in the presence of 5,10-methylenetetrahydrofolate reductase (MTHFR) deficiency [10]. This occurred in a 3 month old child who had good physical status prior to administration of NO₂ as a general anaesthetic, the latter for the purpose of enabling resection of a mass in the left leg. On the 25th postoperative day he experienced seizures, and was found to be hypotonic and arreflexive. The plasma homocysteine level was elevated at 0.6 mg/decilitre (normal value < 0.01) and the vitamin B12 level was normal. Genomic DNA-sequence analysis revealed that MTHFR deficiency was caused by a novel MTHFR mutation which changes the conserved methionine at position 581 of the enzyme to isoleucine. The consequence was that NO₂-related inactivation of methionine synthetase was superimposed on MTHFR deficiency, the combined derangements giving rise to an excessive accumulation of homocysteine [10], which, in turn, has well documented neurotoxic potential in its own right [11,12].

Misgivings that anesthesiologists have about using NO₂ as an anaesthetic agent have been articulated in a recent review which concluded that it was incumbent upon anesthesiologists to evaluate their patients for risk factors that might predispose them to NO₂-related myelopathy, and to avoid NO₂ if such risk factors are present [13].

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