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CORRESPONDENCE

A call for standards on perioperative CO_2 regulation

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To The Editor,

The *Journal* recently published the *International Standards to the Practice of Anesthesia*.[1] These *Standards* mandate the use of capnography, but they fail to provide guidelines for carbon dioxide management. Except under rare circumstances, carbon dioxide is inert, odourless, tasteless, invisible, and remarkably benign and beneficial. It is heavier than other atmospheric gases, so it accumulates in dependent locations, such as mines, where air circulation is lacking. Under these conditions, it displaces oxygen and causes death by drowning. Since this phenomenon was attributed to toxicity in earlier times and early gas research confused CO_2 effects with carbon monoxide toxicity, carbon dioxide remains widely but mistakenly feared as toxic and narcotic.

All vertebrate cells produce CO_2 continuously. It saturates body tissues and fluids, and it equilibrates with the external environment. Carotid and aortic respiratory chemoreceptors gradually adapt to and maintain this equilibrium. Synergistic combinations of hypercarbia and hypoxemia exponentially increase chemoreceptor activity and respiratory drive. Hyperventilation is unnatural and abnormal in all circumstances. It confers no tangible benefits, and it may cause serious adverse events, including "shallow water blackout syndrome", brain damage in mountain climbers, and increased morbidity and mortality in otherwise healthy polio victims. Nowadays, its traditional use to counteract brain swelling is discouraged. Mechanical hyperventilation rapidly depletes CO_2 tissue reserves, which obtunds chemoreceptors and undermines respiratory drive.

Mechanical hyperventilation during anesthesia originated before pulse oximetry and capnography were available. In that bygone era, carbon dioxide was assumed to be a "toxic waste gas" that must be rid from the body rather than an essential element of normal physiology that is rapidly depleted by mechanical hyperventilation and requires careful conservation. It was not understood that hyperventilation damages lung tissues, impairs tissue perfusion and oxygenation, inhibits opioid clearance, traps opioids in brain tissues, and depletes CO_2 reserves necessary for normal respiratory chemoreceptor activity. Moreover, it was not understood that mild hypoventilation reduces blood viscosity beneficially, increases cardiac output, promotes tissue perfusion and oxygenation, protects lung tissues, preserves tissue reserves of carbon dioxide, offsets the respiratory depressant effects of opioids, and prevents opioid "trapping" in brain tissues. During that past era, evidence was often misinterpreted or overlooked in favour of pre-existing beliefs about carbon dioxide toxicity. For example, Boniface and Brown mistakenly concluded that CO_2 causes toxic depression of cardiac contractility, even though their study[2] documented beneficial decreases in systemic vascular resistance that offered a simpler explanation for decreased cardiac work.

Unfortunately, anesthesia hyperventilation remains entrenched, even though critical care experts have embraced the safety of permissive hypercapnia. The practice is reinforced by the routine observation that hyperventilated patients usually breathe adequately upon anesthetic emergence, provided that opioid dosage has been carefully constrained. This is because conscious awareness sustains breathing despite the absence of chemoreceptor activity, particularly in the presence of pain.[3,4]

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However, this unstable form of respiratory drive renders patients vulnerable to opioid treatment that mitigates pain and thereby facilitates the onset of sleep, whereupon patients may unexpectedly lose consciousness, stop breathing, and suffer brain damage and death in quick succession.[5] Geriatric patients are especially endangered because they have low metabolic rates and cannot readily replenish CO_2 tissue reserves. This phenomenon can persist for hours, and it may explain serious adverse events that are variously attributed to "opioid hypersensitivity", "re-narcotization", inadequate monitoring, and inept opioid management by surgeons and nurses or the patients themselves. Meanwhile, anesthetic mismanagement invariably escapes scrutiny.[6]

Times have changed. A modern reassessment of perioperative carbon dioxide management to provide anesthesia guidelines and standards consistent with scientific principles is long overdue. Particular issues that should be addressed include iatrogenic mechanical hyperventilation, permissive hypercarbia, spontaneous breathing under anesthesia, and the continued presence of soda lime in anesthesia machines.

Competing interests None declared.

References

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Reply

Thank you for the opportunity to respond to Dr. Coleman. We think his letter adds a useful perspective to the *International Standards for a Safe Practice of Anesthesia*.[1]

The *Standards* have been developed by an iterative process of literature review and expert consultation.[2] The publication of the *Standards* in a peer-reviewed journal with provision of open access was undertaken, amongst other reasons, to promote feedback and commentary through the *Journal's* correspondence columns. The intention is to revise the *Standards* regularly, with the next iteration planned to coincide with the World Congress of Anaesthesiologists in Buenos Aeries in 2012. Revision will be informed by such feedback and commentary.

The *Standards* do not mandate the use of capnography; in the carefully crafted language of the *Standards*, capnography is "Recommended". In contrast, pulse oximetry is "Highly Recommended" (the first and highest level of importance). The primary reason for monitoring carbon dioxide during anesthesia is to confirm correct placement of an endotracheal tube and adequacy of ventilation; in this context, an undetected disconnection of the circuit or failure to switch on a ventilator are potentially hazardous.

Nevertheless, the presence of capnography does facilitate greater accuracy in management of ventilation, and the points made by Dr. Coleman are well taken. We think there are significant advantages to spontaneous ventilation when muscle relaxation is not needed and a patient is otherwise suitable for this approach to general anesthesia. In essence, there is inherent safety in keeping things simple and avoiding the risks of muscle relaxation when reasonably possible. Dr. Coleman's letter should reassure practitioners who might otherwise feel obliged to institute mechanical or manual (in some parts of the world) ventilation simply to avoid or respond to moderate hypercapnia. It is not entirely clear whether our views and those of Dr. Coleman warrant adoption in the next revision of the *Standards*. This will depend on how consequential these views are thought to be and on the extent to which they are considered adequately grounded in evidence and/or supported by expert consensus. It may be that subsequent correspondence will be enlightening in this regard.

In the meantime, our response to Dr. Coleman is to say, "We agree".

Competing interests None declared.

References

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