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Title: A call for standards on perioperative CO2 regulation

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Dear Dr. Miller:

I have reviewed your recommendations and the comments of your reviewer, which I appreciated. I have changed the title and accepted the other changes you suggested, all of which were excellent and improved the document.

I wholeheartedly agree with the comments of the reviewer that it is safe and even advantageous to employ spontaneous breathing with mild hypercarbia in healthy anesthetized patients. I utilize this technique every day in my present pediatric dental practice. However, I believe that adding a discussion of this to my letter might distract the attention of readers from its central message. The intention of the letter is to call attention to the fact that iatrogenic hyperventilation and hypocarbia can be harmful, and that there should be published standards that clarify the safe management of carbon dioxide during anesthesia. Therefore, with your permission, I would like to leave the body of the letter essentially unchanged. However, I did modify the last sentence to call attention to spontaneous ventilation under anesthesia and soda lime in anesthesia machines. I believe this latter issue deserves scrutiny because soda lime confers no benefit in modern anesthesia machines, and it produces toxic by-products. I will leave these modifications to your judgment and discretion. My hope is that this letter will inspire convocation of experts who can discuss all these matters in detail and formulate appropriate standards.

Respectfully,
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Competing interests: None declared

A call for standards on perioperative CO2 regulation

To the Editor:

The Journal recently published the “2010 Guidelines and international standards to the practice of anesthesia.” These standards mandate the use of capnography, but they fail to provide guidelines for carbon dioxide management. Carbon dioxide is inert, odorless, tasteless, invisible, and remarkably benign and beneficial except under rare circumstances. It is heavier than other atmospheric gases, so that it accumulates in dependent locations such as mines where air circulation is lacking. Under these conditions it displaces oxygen and causes death by drowning. Because this phenomenon was attributed to toxicity in earlier times, and also because early gas research confused CO2 effects with carbon

monoxide toxicity, carbon dioxide remains widely but mistakenly feared as toxic and narcotic.

All vertebrate cells produce CO₂ continuously. It saturates body tissues and fluids, and 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. Its traditional use to counteract brain swelling is nowadays discouraged. Mechanical hyperventilation rapidly depletes CO₂ 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, instead of 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 CO₂ reserves necessary for normal respiratory chemoreceptor activity. Nor was it understood that mild hypoventilation beneficially reduces blood viscosity, increases cardiac output, promotes tissue perfusion and oxygenation, protects lung tissues, preserves tissue reserves of carbon dioxide, offsets the respiratory depressant effects of

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4 opioids, and prevents opioid “trapping” in brain tissues. During that past era, evidence was
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6 often misinterpreted or overlooked in favor of pre-existing beliefs about carbon dioxide
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8 toxicity. For example, Boniface and Brown mistakenly concluded that CO₂ causes toxic
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10 depression of cardiac contractility even though their study(1) documented beneficial
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12 decreases in systemic vascular resistance that offered a simpler explanation for decreased
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14 cardiac work.
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21 Unfortunately, anesthesia hyperventilation remains entrenched even though critical care
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23 experts have embraced the safety of permissive hypercapnia. The practice is reinforced by
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25 the routine observation that hyperventilated patients usually breathe adequately upon
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27 anesthetic emergence, provided that opioid dosage has been carefully constrained. This is
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29 because conscious awareness sustains breathing despite the absence of chemoreceptor
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31 activity, particularly in the presence of pain.(2,3) However, this unstable form of
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33 respiratory drive renders patients vulnerable to opioid treatment that mitigates pain and
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35 thereby facilitates the onset of sleep, whereupon patients may unexpectedly lose
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37 consciousness, stop breathing, and suffer brain damage and death in quick succession.(4)
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43 Geriatric patients are especially endangered, because they have low metabolic rates and
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45 cannot readily replenish CO₂ tissue reserves. This phenomenon can persist for hours, and
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47 it may explain serious adverse events that are variously attributed to “opioid
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49 hypersensitivity,” “re-narcotization,” inadequate monitoring, inept opioid management by
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51 surgeons and nurses, or the patients themselves. Meanwhile, anesthetic mismanagement
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53 invariably escapes scrutiny.(5)
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Times have changed. A modern re-assessment of perioperative carbon dioxide management to provide anesthesia guidelines and standards consistent with scientific principles, including the issues of iatrogenic mechanical hyperventilation, permissive hypercarbia, spontaneous breathing under anesthesia, and the continued presence of soda lime in anesthesia machines, is long overdue.

Lewis S. Coleman, MD

References

1. Boniface KJ, Brown JM. Effect of carbon dioxide excess on contractile force of heart, in situ. Am J Physiol 1953;172:752-6.
2. Fink BR. Influence of cerebral activity in wakefulness on regulation of breathing. J Appl Physiol 1961;16:15-20.
3. Bellville JW, Howland WS, Seed JC, Houde RW. The effect of sleep on the respiratory response to carbon dioxide. Anesthesiology 1959;20:628-34.
4. Forrest WH, Jr., Bellville JW. The Effect of Sleep Plus Morphine on the Respiratory Response to Carbon Dioxide. Anesthesiology 1964;25:137-41.
5. Overdyk FJ. Postoperative opioids remain a serious patient safety threat. Anesthesiology 2010;113:259-60; author reply 60-1.

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Competing interests: None declared

November 22, 2010

Mechanical Hyperventilation and Opioid Hypersensitivity

To the Editor~~Dear Dr. Miller:~~

~~The Journal~~ Last month your journal recently published the “2010 Guidelines and 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.

Carbon dioxide is inert, odorless, tasteless, invisible, and remarkably benign and beneficial except under rare circumstances. It is heavier than other atmospheric gases, so that it accumulates in dependent locations such as mines where air circulation is lacking.

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Under these conditions it displaces oxygen and causes death by drowning. Because this phenomenon was attributed to toxicity in earlier times, and also because early gas research confused CO₂ effects with carbon monoxide toxicity, carbon dioxide remains widely but mistakenly feared as toxic and narcotic.

All vertebrate cells produce CO₂ continuously. It saturates body tissues and fluids, and 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, considerable adverse events or occult mischief, including “shallow water blackout syndrome,” brain damage in mountain climbers, and increased morbidity and mortality in otherwise healthy polio victims. Its traditional use to counteract brain swelling is nowadays discouraged. Mechanical hyperventilation rapidly depletes CO₂ 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, instead of 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 CO₂ reserves necessary for normal respiratory chemoreceptor activity. Nor was it understood that mild hypoventilation beneficially reduces blood viscosity, 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 favor of pre-existing beliefs about carbon dioxide toxicity. For example, Boniface and Brown mistakenly concluded that CO₂ causes toxic depression of cardiac contractility even though their study [\[reference please\]](#) 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.(2,3) However, this ~~treacherously~~ 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.(4) Geriatric patients are especially endangered, because they have low metabolic rates and cannot readily replenish CO₂ tissue reserves. This phenomenon can persist for hours, and it [may](#) explain ~~serious adverse numerous lethal misadventures events~~ that are variously attributed to “opioid hypersensitivity,” “re-narcotization,” inadequate monitoring, inept

opioid management by surgeons and nurses, or the patients themselves. Meanwhile, anesthetic mismanagement invariably escapes scrutiny.(5)

Times have changed. A modern re-assessment of perioperative carbon dioxide management to provide anesthesia guidelines and standards consistent with scientific principles, is long overdue.

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References

1. Merry AF, Copper JB, Soyannwo O, Wilson I, Eichorn J. Miller DR. 2010
Guidelines and International standards for a safe to the practice of anesthesia.
Can J Anaesth 2010;57: 1027-34~~957-60~~.
2. Fink BR. Influence of cerebral activity in wakefulness on regulation of breathing.
J Appl Physiol 1961;16:15-20.
3. Bellville JW, Howland WS, Seed JC, Houde RW. The effect of sleep on the
respiratory response to carbon dioxide. Anesthesiology 1959;20:628-34.
4. Forrest WH, Jr., Bellville JW. The Effect of Sleep Plus Morphine on the
Respiratory Response to Carbon Dioxide. Anesthesiology 1964;25:137-41.
5. Overdyk FJ. Postoperative opioids remain a serious patient safety threat.
Anesthesiology 2010;113:259-60; author reply 60-1.

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