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Avoiding the Perils of Obstructive Sleep Apnea

Lewis S Coleman*

Chair of the Science and Education Board of the American Institute of Stress, 220 Adams Dr. suite 280-#224, Weatherford, Texas 76086, USA

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***Corresponding author:** Lewis S Coleman, Chair of the Science and Education Board of the American Institute of Stress, 220 Adams Dr. suite 280-#224, Weatherford, Texas 76086, USA Email: coleman006@gmail.com

Abstract

Obstructive Sleep Apnea (OSA) has emerged as a serious anesthesia safety dilemma because it often causes postoperative respiratory arrest and death and cannot be reliably detected by pre-operative history and physical examination [1-3]. This essay will explain how and why the harmful habit of hyperventilation that pervades anesthetic practice is the underlying cause of this "dead in bed" problem, and how it can be prevented by supplementing general anesthesia with opioids and hypercarbia.

Keywords: OSA Obstructive Sleep Apnea; Surgery; Hypercarbia; REM: Rapid Eye Movement; Heart Attacks; Strokes; Drowning; Smoke Inhalation

Introduction

Abnormal anatomy of the hypopharynx causes Obstructive sleep apnea. During REM (rapid eye movement) sleep the nervous system automatically inhibits voluntary motor activity to prevent dangerous dream-related movements [4-9]. This paralyzes pharyngeal muscles, causing soft tissues to collapse into the abnormal hypopharynx and obstruct airflow. The resulting hypoxia partially restores consciousness and relieves the obstruction, whereupon the cycle repeats itself, causing progressive hypoxic brain damage and leaving the victim sleepy the following day. The problem is most common in obese, middle-aged patients, who are often aware of their problem, but it can be exceedingly deceptive in pediatric patients, who, unlike adult victims, are often thin and active, and exhibit normal growth and development, so that parents and caregivers may be unaware of the condition, and practitioners cannot detect it via physical examination. The condition may cause secondary tonsillar hypertrophy, and tonsillectomy doesn't necessarily eliminate the OSA problem.

OSA has become particularly problematic for practitioners since 1995, when medical insurance companies suddenly refused reimbursement unless surgical patients are discharged immediately after surgery in the hands of medically ignorant friends and family [10,11]. This thrusts responsibility for detecting OSA before surgery into the laps of anesthetists, which is an impossible task, especially in pediatric victims. To make matters worse, the inherently dangerous and nearly universal habit of hyperventilation, which is incompatible with opioids, has caused practitioners to abandon therapeutic opioids in favor of toxic and questionably effective NSAID analgesics to meet the demands of fast tracking. The sudden abandonment of opioid supplementation during stressful cardiac bypass procedures to facilitate "fast tracking" caused an epidemic of lethal postoperative "SIRS" (systemic inflammatory response syndrome) [12-17]. Abandoning opioids mitigates but fails to eliminate the problem of unexpected postoperative respiratory arrest caused by CO_2 depletion, especially in patients with OSA [18,19].

Hyperventilation is inherently harmful and confers no benefits [20]. It dangerously depletes body reserves of carbon dioxide that are essential for both the "primary" respiratory drive of consciousness and the "secondary" respiratory drive that sustains breathing during sleep and general anesthesia when consciousness is extinguished. OSA becomes exceedingly dangerous during general anesthesia, when intraoperative OSA airway obstruction is prevented by either elective endotracheal intubation or other airway devices. Under these circumstances spontaneous or iatrogenic hyperventilation can readily deplete CO_2 body reserves. As a result, patients appear to breathe normally when they regain consciousness after surgery, because consciousness sustains breathing despite CO_2 depletion.

However, if CO_2 depleted patients fall asleep and lose consciousness, they may unexpectedly suffer lethal postoperative respiratory depression. This problem has been understood since the dawn of modern anesthesia, and it is most common in geriatric patients, whose low metabolism may not replenish CO_2 reserves for hours after surgery [21,22]. For example, it explains the mysterious death of Andy Warhol, who died in his private hospital room several hours after uncomplicated cholecystectomy, when his private nurse administered sedatives and withdrew to avoid disturbing him [23]. OSA drastically exaggerates this danger, especially in the context of outpatient surgery, where patients are sent home immediately after surgery in the care of medically ignorant friends and family, whereupon they are discovered to be "dead in bed".

OSA can best be appreciated in the context of anesthesia history. The original anesthetic safety problem was healthy patients who regained consciousness after uneventful surgery and appeared to breathe normally, but then unexpectedly fell asleep, stopped breathing, and died. Dr. Yandell Henderson correctly determined that surgery stimulates spontaneous hyperventilation during anesthesia, which depletes CO_2 tissue reserves and causes unexpected respiratory arrests. He recommended that patients breathe 5% CO_2 during anesthesia to prevent CO_2 depletion. This eliminated postoperative respiratory depression. It also reduced unexplained deaths during surgery and prevented postoperative asthma, atelectasis, pneumonia, laryngospasm, ether explosions, nausea, and vomiting. Henderson subsequently proved that CO_2 has powerful therapeutic properties for heart attacks, strokes, drowning, smoke inhalation, carbon monoxide poisoning, and newborn babies with breathing problems [24-31].

During the same era, Dr. George Washington Crile proved that intramuscular morphine supplementation improves surgical outcome and cures sepsis and peritonitis and by controlling harmful nervous activity [32]. With the help of modern research, we can understand that morphine improves tissue perfusion and oxygenation by reducing microvascular flow resistance and elevating body reserves of CO_2, which enhances the release of oxygen from blood into tissues. The enhanced tissue perfusion and elevated tissue oxygenation poisons the offending microbes and cures the infections. These principles would be even more effective with the addition of antibiotics. Meanwhile, Dr. Donald Jackson introduced the first "closed circuit" anesthesia machines that were designed to conserve expensive anesthetic gases, and Dr. John Lundy demonstrated that CO_2 supplementation speeds anesthetic induction and emergence [33-35].

After WWI Crile established a school of Nurse anesthesia that trained its students to supplement ether anesthesia with CO_2 and morphine using Jackson's machines to prevent ether explosions and optimize surgical outcome. Their success inspired physicians to embrace Carbogen (therapeutic mixtures of oxygen and carbon dioxide) to treat heart attacks, strokes, asthma, atelectasis, pneumonia, inebriation, drowning, carbon monoxide poisoning, smoke inhalation, and newborn babies with breathing problems. This threatened to revolutionize medical care during the 1930's [36].

Unfortunately, overenthusiastic CO_2 supplementation during anesthesia with Jackson's "closed circuit" machines sometimes caused CO_2 asphyxiation that manifested as alarming "ether fits." Dr. Ralph Waters, the first chairman of a university anesthesiology department, vilified CO_2 as "toxic waste, like urine" that must be "rid from the body" using mechanical hyperventilation during anesthesia [37]. His specious animal research and fabricated clinical accounts entrenched the harmful habit of mechanical hyperventilation [38]. Soon thereafter, halothane supplanted explosive ether, and "open circuit" anesthesia machines eliminated CO_2 asphyxiation. Ironically, the "open circuit" machines re-introduced the forgotten danger of CO_2 depletion that persists to the present and causes unreasoned fear of narcotics [39-41]. Meanwhile misinterpreted research and misleading publications have obscured the problem [42-48].

Crile's influence established the principle of discharge a day after surgery to enable trained hospital staff to detect postoperative problems. Thus, postoperative respiratory arrests were of little concern to anesthesiologists until 1995, when medical insurance companies-imposed reimbursement requirements for immediate discharge of surgical patients in the hands of medically incompetent friends and family. This transferred liability for postoperative respiratory arrests from hospitals into the laps of anesthesiologists. The resulting postoperative respiratory arrests were blamed on narcotics, but the underlying cause of CO_2 depletion was ignored [49-52]. Instead, anesthesiologists abandoned nontoxic opioids in favor of toxic NSAIDs and problematic analgesic blocks [53-72]. This mitigated the postoperative respiratory arrests but unmasked the problem of the "dead in bed" phenomenon caused by OSA [73,74].

OSA is a nightmare for outpatient anesthesia because it cannot be reliably detected by history and physical examination, and eliminating narcotics cannot abolish the "dead in bed" problem [75]. Many patients are not aware that they suffer from OSA. Obesity, snoring, male sex, and a thick neck are clues, but OSA can occur in their absence. Enlarged tonsils are typically the result of OSA rather than its cause, and OSA patients have been found "dead in bed" after uncomplicated adenotonsillectomy, which is a dangerous procedure unto itself. OSA is especially treacherous in children, who, unlike adults, can be thin, active, and otherwise seemingly healthy, and their caregivers may be unaware of the problem. The only way OSA can be confirmed is by professional evaluation in a sleep clinic, which entails overnight monitoring. Obviously, this is not a practical means of anesthesia screening. Failure to detect the syndrome can easily result in tragedy when the OSA patient is sent home immediately after surgery in the care of medically ignorant friends and family, and subsequently discovered to be "dead in bed".

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There is an effective means to avoid OSA disasters: "permissive" hypercarbia [76-80]. I have safely anesthetized thousands of patients with the "Crile/Henderson" technique over the course of fifteen years using modern machines, medications, and monitors. I used pulse oximetry to assure effective oxygenation of arterial blood emerging from the lungs, and I used capnography to monitor and measure therapeutic metabolic CO_2 accumulation that reflected adequate opioid supplementation to prevent spontaneous hyperventilation and dangerous CO_2 depletion. With this approach, there is no need for hazardous CO_2 supplementation from tanks of Carbogen or carbon dioxide that might threaten asphyxiation by disrupting oxygen transport and delivery [81-94]. Patients were treated with fentanyl to control surgical nociception, prevent hyperventilation, and maintain end-tidal CO_2 in the safe range of 50-100 torr with spontaneous breathing. They were sent home with instructions to take a nap, with excellent results. Copies of my computerized anesthetic records that illustrate how hypercarbia protects respiratory drive and elevates tissue oxygenation are available on my website [95].

During this time there was 5 you. male with deceptively normal weight and apparent good health whose father deliberately concealed his knowledge of his son's severe OSA from the dentist, the nurse, and me, and insisted that he was healthy and normal. The boy was anesthetized with the "Crile/Henderson" technique and underwent uneventful extraction of an undescended bicuspid tooth using a total of 12mcg/kg fentanyl (total 350 mcg). He was breathing normally at 17 breaths/min. at the conclusion of surgery and in the recovery room, but he exhibited prolonged emergence that was due to his OSA condition. This was mistakenly attributed to Versed pre-medication after he was treated with Roma icon and promptly regained consciousness. With this technique I routinely sent patients home with instructions for them to take a nap to optimize stress control. Healthy patients consistently woke up from their naps, had lunch, and went off to play as if they never had surgery.

This patient was sent home, where he exhibited prolonged emergence from his nap that caused consternation, but he did not suffer respiratory arrest because his respiratory drive remained intact. His severe OSA condition (apnea-hypopnea index 20 episodes per hour) was subsequently confirmed in a sleep clinic. Had this patient been managed with either the "open airway" technique or the "Leake/Waters" technique, he would likely have wound up "dead in bed" [96]. This case illustrates the inherent safety of the "Crile/Henderson" technique. I subsequently performed a study in which I monitored 1500 patients in an outpatient dental clinic using capnography and transcutaneous $O2/CO_2$ monitoring to demonstrate how the Crile/Henderson technique elevates tissue oxygenation and CO_2 levels, preserves respiratory drive, and prevents postoperative respiratory depression [97]. The details are described in my book.

Conclusion

Hyperventilation is dangerous and bereft of benefits. The same applies to inadequate analgesia during and after surgery [98]. I have done my best to draw attention to the danger of CO_2 depletion and the need for professional guidelines for CO_2 management during anesthesia, but thus far without result. The anesthesiology profession is ideally poised to realize priceless public prestige by correcting defective dogma, publishing standards for CO_2 management, confirming the "mammalian stress mechanism" with clinical studies, re-revolutionizing surgery, and restoring medical advance [99].

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