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Cardiac Arrests during Spinal Anesthesia: Unexpected?

The recently published analysis of 14 cases of cardiac arrest in healthy patients during spinal anesthesia[1] suggests a relationship between the occurrence of cardiac arrest and administration of opiates and sedatives. In approximately one-half of the cases, the administration of two or more of these agents early in the course of spinal anesthesia was followed by sleep, then cyanosis and circulatory arrest, all within a 5–25-min period. Although spinal anesthesia, by its effects on the circulation, could have played a facilitatory role in the genesis of these arrests, the temporal sequence of events implied that sedation-induced respiratory failure was the critical precipitating factor.

Why did sedation apparently have such a devastating effect on respiration in these previously healthy patients? Both the authors of the report and the writer of an accompanying editorial speculate on this crucial question,[2] but neither provide an explicit explanation.

Although the details of individual cases are not available in the report, it appears that most patients who displayed evidence of respiratory depression received the following opiate/sedative drugs: 1) morphine as a premedicant (dose and route not given), 2) fentanyl 25–200 μg iv during the first 30 min of spinal anesthesia, and 3) one or more of diazepam 2–10 mg, droperidol 1.25–7.5 mg, and thiopental 50–200 mg iv during the same 30-min period. Not surprisingly, several patients then fell asleep.

How would this combination of agents, together with sleep, affect resting ventilation? It is well known that opiate analgesics reduce ventilation in a dose-related manner, especially in the absence of noxious stimulation—as with successful spinal anesthesia. It is also known that, as opiates depress ventilation and impair chemical mechanisms of ventilatory control, they render ventilation more dependent upon the state of wakefulness. Thus, sleep in the presence of opiates potentiates ventilatory depression.[3] What may not be so widely appreciated is the magnitude of this potentiating effect. In the occasional individual who is free of pain and receives a typical analgesic dose of opiate, physiological sleep destabilizes ventilation markedly and produces severe hypoventilation (fig. 1). Thus, the agents and conditions that were apparently present in these patients (*i.e.*, two opiates without pain and one or more sedatives sufficient to induce sleep) would be expected to occasionally precipitate profound respiratory depression.

If one employs opiates to provide sedation during regional anesthesia, two facts should be kept in mind. First, opiates are more potent depressants of ventilation in the absence of noxious stimulation, and especially so if sleep supervenes (whether that sleep is spontaneous or induced by drugs). Second, the occasional apparently normal patient can be exquisitely sensitive to opiate in these conditions (fig. 1).

R. L. KNILL, M.D., F.R.C.P.(C)
Professor
Department of Anesthesia
University of Western Ontario
London, Ontario, Canada, N6A 5A5

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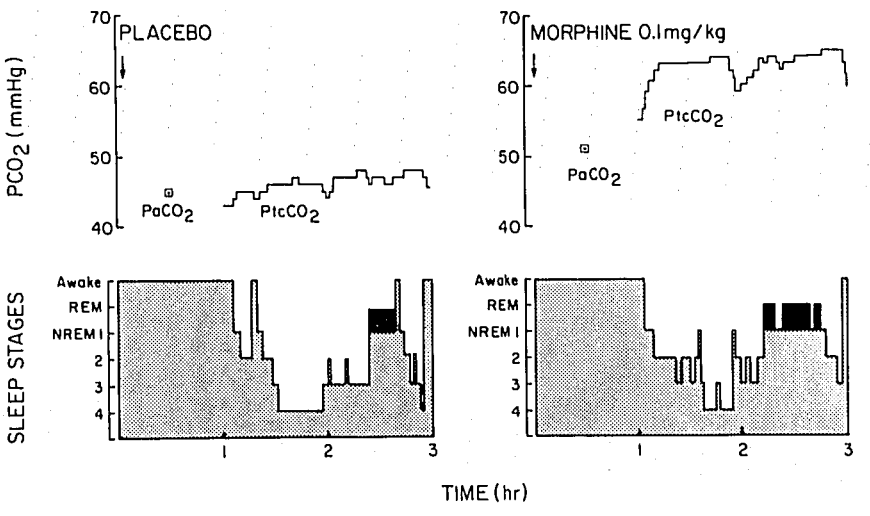

FIG. 1. The effect of physiological sleep on morphine-induced ventilatory depression in an unusually sensitive but healthy pain-free male (age 35 yr; height 182 cm; weight 78.4 kg; ventilatory response to CO_2 1.75 $l \cdot min^{-1} \cdot mmHg^{-1}$). Graphs depict observations of arterial P_{CO_2} (Pa_{CO_2}) during wakefulness and transcutaneous P_{CO_2} (Ptc_{CO_2}) during a period of sleep following administration of placebo (left) and morphine 0.1 $mg \cdot kg^{-1}$ im (right). Each set of observations was obtained on a separate night. Transcutaneous CO_2 was measured by a Radiometer TCM 20 system, calibrated and corrected for drift. (After placebo, the measured $Pa_{CO_2} - Ptc_{CO_2}$ difference was 1 mmHg and after morphine, −1 mmHg). Ventilatory pattern was monitored using an inductive plethysmograph. States of wakefulness and sleep were determined by EEG, EMG, and EOG criteria. (The study was approved by the Health Sciences Standing Committee on Human Research at the University of Western Ontario.) During wakefulness, this modest dose of morphine caused only moderate hypoventilation, as indicated by the small increase of Pa_{CO_2} 30 min following administration. During the subsequent period of sleep, however, its effects on ventilation were profound. The ventilatory pattern became unstable with many episodes of non-obstructive hypopnea and/or apnea and the Ptc_{CO_2} climbed to 65 mmHg.