Propofol in the emergency department: another interpretation of the evidence

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Propofol, a short-acting alkyl phenol used for the induction and maintenance of anesthesia as well as for procedural sedation (PS), has been studied extensively for more than a decade. It has gained increasing acceptance in emergency departments (EDs) and in outpatient surgery, mainly due to its ease of titration and short duration of action. In this issue, Wilbur and Zed have reviewed the evidence supporting its use in the emergency setting, concluding that more ED-based randomized trials are required.1

In some instances it may be important for emergency physicians to repeat studies performed elsewhere to determine applicability and safety in the ED. The studies reviewed by Wilbur and Zed deal primarily with stable and preoperative patients. Propofol induction doses that are safe in stable elective anesthesia cases may cause unacceptable hypotension in critically ill ED patients, and other unknown adverse effects may become evident only in unstable patients. One cannot generalize information derived from stable patients to unstable patients, without verification studies in the hemodynamically unstable population of interest — whether they are in the intensive care unit, the operating room or the ED. Note that the setting of the study is not critical, as long as the patients studied have similar attributes to those treated in EDs.

When used for PS, there is no reason to believe that propofol will have different efficacy or adverse effects in the ED than it does in other settings. Patients undergoing PS are by definition stable: ASA (American Society of Anesthesiologists) Category I or II. Therefore, procedural sedation data from non-ED settings should be included in any review of this topic, and are relevant to ED practice. When such data are included, 2 important facts become apparent: 1) bolus sedation and infusions are not the only way to use propofol; titrated doses have been studied in detail, and 2) propofol infusions and titrated dosing are extraordinarily safe.

Smith and colleagues2 studied patient-controlled sedation using doses of 16 and 25 mg/min. In neither group were patients able to get deeper than “eyes closed, responds to speech.” Thorpe and coworkers found that a dose of 33 mg/min, when self-administered by the patients using a lock-out pump, resulted in roughly 10% being over sedated and unresponsive to shaking.3 It should be noted that no manufacturer presently makes a pump that will infuse more than 25 mg/min for patient-controlled sedation. (I have tried to find one for a patient-controlled sedation study.) Studies reporting significant apnea rates would therefore seem to reflect a lack of physician knowledge or experience rather than an inherent danger of the medication. Intravenous opioids will produce apnea if the dose is too large, but this does not prevent anyone from titrating opioids many times daily for analgesia, procedural pain or in the OR. Contrary to the cautious position of Wilbur and Zed, there are ample data demonstrating the efficacy and safety of propofol to justify our using it; it’s just that the studies weren’t done in the ED.

The literature cited in their article suggests that apnea is common when propofol is used for cardioversion. But apnea is directly related to the dose used and the rate of administration. In the cardiology studies quoted, total doses often exceeded recommended induction doses (2.0–2.5 mg/kg). In our hospital, anesthetists use similar doses and often patients have had to receive assisted ventilation for a minute or two. Conversely, for the last 3 years our emergency physicians have used mini-dose titration (20 mg every 45–60 seconds) and have had no cases of apnea, nor...
any serious oxygen desaturations. Total ED doses vary from 40–160 mg, but titration is always to the same end-point: verbal reaction if shaken. Careful titration also avoids hypotensive episodes.

Wilbur and Zed state that midazolam cannot cause apnea and is inherently a safer medication. Midazolam does produce apnea when given in induction doses, just as propofol did in the studies where large doses were used. In addition, it is difficult if not impossible to titrate midazolam rapidly, and recovery takes 45–60 minutes.

The advantages of propofol — easy and rapid titration to a precise endpoint, amnesia, and rapid recovery — make it an ideal agent for procedural sedation. This is not a medication to be restricted, but it is an agent that requires knowledge, understanding and careful administration. We do not need more studies. As with many other areas of medicine, we need to read and correctly interpret the literature that is already available — literature that has demonstrated propofol’s excellent track record when used properly. Propofol should be a standard part of our medical armamentarium for procedural sedation.

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References

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