Correspondence

95% CI: 37.78-59.20 (Figure). Press Ganey “Overall ED Patient Satisfaction” scores showed a clinically and statistically significant improvement (78.1 versus 82.1; mean difference: 4.0, 95% CI: 3.59-4.41.). The absolute number of patients who left without being seen decreased from 281 to 190, although this did not reach statistical significance (0.8%, 95% CI = −1.5 to 3.0%), despite an increase in ED census of 10.2%.

Although our interventions were specifically targeted toward admitted patients, it is not surprising that turnaround times for all patient categories improved. Clearly, acute care patients who were ultimately discharged could be seen more expeditiously as turnaround times for admitted patients decreased. We believe the turnaround times for fast track patients declined because the acute and fast track areas are not physically separate in our department, allowing for flexibility in both bed assignments and staffing between the 2 areas. As admitted acute patients moved through the department more quickly, there were more beds and staff available for the fast track area of the department. In hospitals where the acute and fast track areas are completely separate and staffing is not flexible between the 2, similar improvements in fast track patient turnaround times may not be seen.

Although much has been written about ED crowding, there are few studies which suggest solutions to the problem. In 2003 JCAHO called on hospitals to incorporate solutions to improve quality and reduce cost. Although forces beyond the department impact operations, the solution may lie in system improvements. The Institute of Medicine states that although forces beyond the department impact operations, the solution may lie in system improvements. The American College of Emergency Physicians has called for further research on ambulance diversion and ED crowding. Available at: http://www.acep.org/webportal/PatientsConsumers/critissues/overcrowding/FactSheetAmbulanceDiversionandEDOvercrowding. Accessed January 1, 2007.

Heroin: What’s In the Mix?

To the Editor:

Heroin abuse is a public health problem within the United States. Heroin intoxication has a well-recognized toxicity syndrome involving central nervous system depression, respiratory depression, and pupillary constriction. However, over the past decade, our poison control center has encountered several heroin adulterants that changed the toxicity syndrome observed after overdose.

In the late 1990s, contamination of heroin with the anticholinergic drug scopolamine led to heroin overdose victims presenting with unusual manifestations of hallucination, mydriasis, tachycardia, and dry mucous membranes. More recently, a heroin-laced acetaminophen and diphenhydramine mixture known as “cheese” has become a popularized heroin source for inexperienced users, and may also produce notable anticholinergic features.

An epidemic of naloxone-resistant heroin overdoses due to fentanyl adulteration has led to significant morbidity and mortality throughout the central and eastern United States. According to records of the Philadelphia County Medical Examiner’s office, at least 250 overdose deaths have been associated with fentanyl between April 1, 2006, and March 1, 2007. At our poison control center, xylazine, an alpha-2 adrenergic agonist which may produce pupil constriction and somnolence mimicking heroin effects, has also been found as an occasional contaminant of heroin.

Most recently, clenbuterol, a long-acting beta-2 adrenergic agonist, has again surfaced in an epidemic of unusual heroin overdoses with symptoms and signs including tachycardia, tremor, diaphoresis, and laboratory findings of hyperglycemia, hypokalemia, and lactic acidosis. Additionally, quinine has been detected in the urine of heroin abusers presenting with tinnitus.

Many agents can be added to heroin to bulk its volume or to change or enhance its pharmacology. In the latter case, the heroin may be marketed on the street by attractive “brand” names such as Polo, Homicide, etc. Health care professionals confronted with atypical heroin overdoses are cautioned to consider the effects of potential adulterant drugs and chemicals,

and to utilize regional poison control centers in epidemic surveillance.

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Additional Thoughts on the Controversy of Lidocaine Administration Before Rapid Sequence Intubation in Patients with Traumatic Brain Injuries

To the Editor:

I enjoyed the clinical controversies review on lidocaine in traumatic brain injury intubations by Vaillancourt et al.1 However, there are a few key points, that were not mentioned on either side of this debate.

Both sides of the controversy examined the issue of lidocaine as a potential blunter of the sympathetic response to laryngoscopy. While I agree with Vaillancourt et al that the literature support for this role is scarce, I believe there is an additional reason to administer lidocaine during rapid sequence intubation in traumatic brain injury patients: its ability to blunt the fasicaltions induced by succinylcholine.

Succinylcholine is the most commonly used neuromuscular blocker in emergency medicine. It is the agent recommended for traumatic brain injury intubations by many of the experts in emergency airway management.2 Succinylcholine causes patients to fasicaltate; these fasicaltions may raise intracranial pressure by afferent spinal column stimulation. Often, it is recommended to use a small dose of a non-depolarizing neuromuscular blocker, e.g., 0.01 mg/kg of vecuronium, as a premedication prior to rapid sequence intubation to blunt these fasicaltions.3 However, in a meta-analysis by Schreiber et al, lidocaine was shown effective for the prevention of fasicaltions; the number needed to treat compares favorably to that of the commonly used paralytic agents.3 Since lidocaine is available in preloaded syringes throughout emergency departments, it is oftentimes more readily available during intubations than the competitive neuromuscular blockers.

Another issue raised by Vaillancourt et al was the possibility of lidocaine dropping the mean arterial pressure. This was discussed in the context of patients receiving thiopental as an induction medication, an agent known to drop mean arterial pressures. If lidocaine blunts sympathetic response to intubation, then it is expected that patients who received lidocaine would have lower mean arterial pressures than the control group, which did not have this counterbalancing sympathetic response attenuated. This raises a crucial point regarding traumatic brain injury intubation: there are 2 distinct types of pharmacologic rapid sequence intubation strategies in these patients.

In traumatic brain injury patients with high pre-intubation mean arterial pressures, premedications are used to prevent any further increase in mean arterial pressure or ICP during intubation. This regimen might include etomidate, a cardiac stable induction agent; succinylcholine as a neuromuscular blocker; fentanyl, a sympathetic-blocking opioid; a small dose of vecuronium, a non-competitive neuromuscular blocker to blunt fasicaltions; and lidocaine, both for its potential effects to block sympathetic response and its ability to further blunt fasicaltions.

In hypotensive traumatic brain injury patients, the strategy will be quite different. In this situation, the risk of intubation is further dropping the mean arterial pressure and adversely affecting cerebral perfusion. The induction agent should be one that would increase or maintain mean arterial pressure such as ketamine. Ketamine is often maligned in traumatic brain injury patients, because of its associated induction of sympathetic activity. However, in a patient with low mean arterial pressure, this sympathetic activity is potentially beneficial. Succinylcholine would be given as neuromuscular blocker along with a small dose of a non-competitive neuromuscular blocker, like vecuronium. Fentanyl and lidocaine should be withheld as blocking of the sympathetic response would be deleterious.

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