Spinal narcotics: What should be the standard for monitoring?

To the Editor:

Many years ago the most frequent reason for a needle to trespass the dura mater barrier was to obtain cerebrospinal fluid for analysis. Today, the most common reason for such trespass is to deposit a drug (i.e., local anesthetic or narcotic) into the fluid.

Narcotics deposited directly into the cerebrospinal fluid have a profound effect on counteracting the reception of painful stimuli. In an occasional patient, the narcotic can cause severe respiratory depression and, if not recognized, may result in death.

Traditionally, intermittent intramuscular narcotics for pain control have always been monitored by observing the level of sedation and/or the effect on respiratory rate. Thus, it was only reasonable that when intrathecal narcotics were introduced, that they too would be monitored in the same fashion. I suspect the majority of hospitals use some form of hourly monitoring of respiratory rate and sedation level to follow the course of spinal narcotics. It did not take long to discover that narcotics in the cerebrospinal fluid do not behave in the same manner as narcotics absorbed from a muscular site into the blood. The higher the dose of intrathecal narcotic, the more cases of respiratory depression/arrest were noted. Keeping the dose of the narcotic to lower levels has certainly improved the safety of use, but there is still the rare patient who has a respiratory arrest.

In 1993, Bailey and associates found that the accepted norm of monitoring sedation and respiratory rate was really unsatisfactory for monitoring patients given intrathecal narcotics. In 20 healthy volunteers, they administered various doses of intrathecal morphine and monitored the volunteers by pulse oximetry, respiratory rate, arterial blood gas analysis, slope of the ventilatory response to carbon dioxide, pressure algometry at the tibia (measurement of analgesia), heart rate, blood pressure, sedation level, pupil size, and incidence of adverse effects of morphine. The investigators found a dose-related:

1. Incidence of hemoglobin oxygen saturation.
2. Increase in arterial carbon dioxide.
3. Decrease in ventilatory response to inhaled carbon dioxide.
4. Increase in the analgesic effect.

The respiratory depression caused by intrathecal morphine could be detected for as long as 19 hours. No consistent correlation was found between the level of respiratory depression and the levels of sedation, respiratory rate, or pupil size.

It must be remembered that volunteers do not have the consistent pain of surgery which tends to stimulate respiration. They also do not have the concomitant depressive effects of anesthetic drugs, sedative drugs, and medical disease, such as chronic obstructive pulmonary disease. However, the study by Bailey and associates is good evidence that the higher the dose of intrathecal narcotics, the greater the pain relief and the higher the risk of respiratory depression.

Will pulse oximetry monitoring of patients given spinal narcotics become a standard of care? Pulse oximetry monitoring has certainly gained the concomitant depressive effects of anesthetic drugs, sedative drugs, and medical disease, such as chronic obstructive pulmonary disease. However, the study by Bailey and associates is good evidence that the higher the dose of intrathecal narcotics, the greater the pain relief and the higher the risk of respiratory depression.

REFERENCES


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Congestive heart failure associated with endoscopic cholecystectomy

To the Editor:


"Decreased venous return" would tend to ameliorate CHF, not cause it. "Increased intrapleural pressure" does not cause "transudation or ultrafiltration of fluid and the resultant pulmonary edema." Fluid movement into or out of the pulmonary vascular bed is governed by the Starling relationship. In the acute setting, the only possible derangements which might lead to transudation would be an increase in pulmonary capillary endothelial permeability or increased pulmonary capillary pressure. Increased intrapleural pressure has no effect on either of these variables. "Increased intrathoracic pressure" does not necessarily cause "a decrease in cardiac output similar to that exhibited in cardiac tamponade." Robotham and associates have pointed out that when intrathoracic pressure increases, pleural pressure is transmitted to the left ventricle and intrathoracic aorta but not to the extrathoracic aorta. The tension that the left ventricle must generate to eject blood is therefore lower. Cardiac function actually improves.2

I submit that the most likely cause of CHF in a patient "with a history of CHF and coronary artery disease" is decreased inotropy secondary to myocardial ischemia. Mr. Spain does not mention whether the patient's CHF antedated his coronary artery bypass grafting. Certainly if the patient had had an episode of CHF since his bypass, invasive hemodynamic monitoring should have been instituted preoperatively.

REFERENCES

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