brinogen concentrations can be used to predict coronary artery disease with predictive power as high as more generally accepted risk factors.

Marmot et al. have shown that mortality from coronary artery disease is inversely related to employment grade with a relative risk more than three times greater in the lowest grade of employment compared with the highest grade. Markowe et al. reported significant differences in plasma fibrinogen concentrations between men in the lowest grades of employment and those in the highest classifications. They also demonstrated a positive correlation between the workers’ fibrinogen concentrations and the stress of their respective jobs as determined by a questionnaire composed of items that had been shown to identify stress related to increased risk of coronary artery disease.

Combining the findings of Rosenfield et al. with the epidemiologic data prompts the speculation that lower social class and employment grade produce stress leading to an increase in circulating epinephrine, which results in increased plasma fibrinogen concentration, which in turn is predictive of coronary artery disease and perhaps causally related.

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Local Anesthetic Test Dose to Predict Effective Epidural Opioid Analgesia: I

To the Editor.—Weitz and Drasner1 address a clinically important subject, because epidural analgesia is used frequently to provide postoperative analgesia in patients undergoing extensive and potentially painful operations, which may require general anesthesia due to length of surgery or position of patient during surgery. Some anesthesiologists will not give a preoperative epidural dose of local anesthetic adequate to produce motor or sensory block for fear of intraoperative hypotension. Thus, the patient may arrive in the recovery room with no proof of the proper epidural location of the catheter. We agree with the authors’ major conclusion that demonstrable sensory anesthesia is a predictor of good epidural morphine analgesia, because the epidural catheter must be located within the epidural space for epidural analgesia to be effective.

Data by Weitz and Drasner show that, on the operative day, patients with little or no demonstrable sensory block (0–7 points) after testing the epidural catheter with 150 mg lidocaine had mean ± SEM visual analog (VAS) pain scores of 5.5 ± 0.5. These VAS scores were significantly higher than the VAS scores (1.0 ± 0.25) of patients whose catheters were clearly demonstrated to be located in the epidural space (16–24 points), judging by extent of sensory anesthesia after lidocaine injection. Ranges of the VAS scores were not given, but one can surmise maximum VAS score in the former group of patients were about 7 or 8. In our practice, we would consider VAS pain scores higher than 5 to be an indication that epidural analgesia is not effective. In light of absence of expected sensory block after epidural lidocaine occlusion, we would assume the epidural catheter

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Local Anesthetic Test Dose to Predict Effective Epidural Opioid Analgesia: II

To the Editor—Weitz and Drasner studied a possible correlation between dermatomal spread of anesthesia from epidural injection of 10 ml 1% lidocaine to predict subsequent epidural morphine postoperative pain efficacy (2.5 mg plus continuous infusion)1 Patients were studied in three groups (minimal, moderate, and extensive local anesthesia from the lidocaine dose) including seven (14% of studied) patients, where no clinical evidence of epidural block developed. The type of catheter (single vs multiple-orifice), statistical evaluation of demographic factors, ASA status, and the absolute numbers, means, and standard deviations of dermatomes developing anesthesia were not reported for any group. While a 5-ml pre- and postoperative test dose containing epinephrine was used to “exclude intravenous or intrathecal injection of local anesthetic,” in the face of the continuing debate regarding the usefulness of the epinephrine test dose, no exclusion of patients with factors known to mask epinephrine effects (i.e., β-blockade, pacemakers) nor monitors or methodology used to evaluate intravascular effects is mentioned.2

I would question: (1) the validity in assuming catheter localization to the epidural space in the absence of effects from 10 ml lidocaine; (2) why the 10 ml lidocaine test was not done preoperatively (as 3 ml were injected then anyway) before the effects of pain, general/local anesthesia, and epidural morphine compromised the evaluation of either lidocaine’s effects or the opioid analgesia in the postanesthesia care unit;3 and (3) whether any of the group 2 patients had unilateral effects, indicating possibly paravertebral instead of epidural injection.

The authors noted effective analgesia (VAS < 4) only in patients who developed a block to 16 or greater (maximal effect group). Pain relief was, however, “found” in all seven patients who experienced no lidocaine effects “despite relatively high VAS scores” (indicating significant pain?), and the groups exhibiting minimal and moderate lidocaine effects had similar pain scores. Does this study attest to the frequent ineffectiveness of epidural opioids in general (or this particular dosing regimen), patient tolerance of postoperative pain (and the needlessness of this epidural opioid regimen) or merely a very high (>14%) rate of (resident) nonepidural catheter placement?

Preoperative dosing and maintenance of epidurals (with local anesthetic and then morphine) during combined epidural/general anesthesia provide reduced intraoperative general anesthetic requirements and a pain-free emergence in the recovery room.4 When morphine is placed in the epidural space in sufficient amounts causally, effective opioid analgesia results to manage even thoracic pain, which raises the question as to why local anesthetic effect above L1 from 10 ml would be a predictor of morphine effect.5 What is the utility of this method, which only misdiagnoses nonfunctional catheters as epidural catheters and requires patients to unnecessarily experience postoperative pain on emergence.

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