◇ This Month in

ANESTHESIOLOGY

■ Does Intrathecal Fentanyl Reduce Requirements for Bupivacaine during Labor? Stocks *et al.* (page 593)

Stocks *et al.* designed a study to assess the ability of different doses of intrathecal fentanyl to reduce requirements for intrathecal bupivacaine administered to women during the first stages of labor. The 124 women recruited for the study were all in labor and dilated between 2–6 cm. They were volume-loaded with saline, and then an intrathecal injection was administered as part of a combined spinal–epidural technique. (The subsequently inserted epidural catheter was not used for this study.) In the first phase of the study, women were randomized (30 per group) to administration of either bupivacaine alone or bupivacaine in combination with 25 μg fentanyl. In the second phase of the study, bupivacaine, in combination with either 5 or 15 μg fentanyl, was administered to the women.

Bupivacaine was administered in either higher or lower doses according to an up–down sequential allocation, with testing intervals set at 0.25 mg bupivacaine. Efficacy of the study drug was assessed using a 100-mm visual analog scale, and *effective dose* was defined as the amount of drug resulting in a visual analog pain score of 10 mm or less within 15 min after injection. An effective dose directed a decrement of 0.25 bupivacaine for the next patient randomized to that particular group. An *ineffective dose* (visual analog pain score of more than 10 mm, 15 min after administration) directed an increase of 0.25 bupivacaine in the next person in the group. Rescue boluses of 2 μg/ml fentanyl were offered to women who experienced ineffective analgesia.

The researchers recorded statistics, such as weight, height, and parity, for each patient, as well as use of oxytocin and prostaglandin for induction of labor. Sensory levels were assessed by reaction to ethyl chloride spray and to pin prick. The minimum local analgesic dose of intrathecal bupivacaine was calculated at 1.99 mg using the Dixon and Massey formula. There were significant reductions in minimum local analgesic dose for all bupivacaine–fentanyl groups compared with the bupivacaine control group. The addition of 5 μg intrathecal fentanyl showed the same bupivacaine dose-sparing effect as 15 and 25 μg. Increasing fentanyl doses resulted in increased pruritus and duration of spinal analgesia. These findings suggest that analgesia in the first stage of labor can be achieved with lower doses of fentanyl than those commonly used, with a resulting shorter duration of action.

■ Use of Transesophageal Echocardiography and Compound Tomography in Diagnosing Aortic Injuries Resulting from Blunt Trauma. Vignon *et al.* (page 615)

Although multiplane transesophageal echocardiography (TEE) and helical compound tomography (CT) of the chest have been evaluated in comparison with aortography for their sensitivity in detecting acute traumatic aortic injuries, the two techniques have not been compared with each other. Accordingly, Vignon *et al.* conducted a prospective study to determine the diagnostic accuracy of the two methods for detecting traumatic cardiovascular injuries. During a 3-yr period, the authors examined 285 patients admitted for blunt trauma to their institution. After initial examination, 110 (88 men and 22 women) were considered to be at high risk of having sustained a traumatic cardiovascular injury. A total of 95 patients underwent both TEE and CT, and a traumatic arterial injury was diagnosed in 17 patients (15.5%). All acute traumatic aortic injuries involved the aortic isthmus, except for one subadventitial disruption of the ascending aorta. Both TEE and CT identified all subadventitial disruptions of the aortic isthmus or ascending aorta that required surgical repair. Two patients died before surgery, but the remaining patients all underwent successful surgical repair. CT only depicted one disruption of the innominate artery. TEE was more sensitive than CT for the identification of intimal or medial lesions of the thoracic aorta. In cases in which hemodynamically unstable patients cannot be transported safely to the radiology department, TEE may offer a safe alternative for ruling out acute traumatic aortic injuries at the bedside.

■ Effects of Ketamine and Its Isomers on Ischemic Preconditioning. Molojavyi *et al.* (page 623) and Mullenheim *et al.* (page 630)

Ischemic preconditioning, in which brief periods of myocardial ischemia followed by reperfusion provide protection from subsequent ischemic injury, has been shown to occur in animals as well as humans. Current

evidence suggests that opening of the adenosine triphosphate–sensitive potassium channel is a key mechanism. Based on this evidence and the finding of a concentration-dependent inhibitory effect of ketamine on adenosine triphosphate–sensitive potassium channel activity, two research teams from the same institution investigated the effects of ketamine and its stereoisomers on preconditioning in animal models.

In the first study, Molojavyi *et al.* subjected 80 isolated rat hearts to 30 min of no-flow ischemia followed by 60 min of reperfusion. The hearts were assigned randomly to one of 10 groups: two groups of eight hearts received no preconditioning stimulus before undergoing the study protocol; in another group of eight hearts, preconditioning was elicited by two 5-min periods of ischemia before the 30-min ischemic period. In six treatment groups, ketamine, *R*(−)-ketamine, or *S*(+)-ketamine were administered at concentrations of 2 or 20 μg/ml before the preconditioning stimulus. A final group of eight hearts received 20 μg/ml *R*(−)-ketamine before ischemia. Left ventricular (LV) end-diastole pressure, LV developed pressure, and maximum and minimum dP/dt were obtained from digitized signals and later processed on a personal computer. LV end-diastole was determined as the point at which dP/dt started its rapid upstroke after crossing the zero line.

Baseline LV developed pressure was similar in all groups of hearts. Those hearts in the control groups showed the poorest recovery of LV developed pressure, as well as a high creatine kinase release (evidence of cellular injury) after ischemia. Preconditioning improved recovery of LV developed pressure and reduced creatine kinase release. Administration of 2 μg/ml ketamine or 2 or 20 μg/ml *S*(+)-ketamine did not influence recovery of LV developed pressure. After 20 μg/ml ketamine or 2 or 20 μg/ml *R*(−) ketamine, the protective effects of preconditioning were abolished.

In vivo, the pathophysiology of preconditioning and ischemia–reperfusion injury is more complex. Accordingly, Mullenheim *et al.* investigated the effects of ketamine and *S*(+)-ketamine on preconditioning in rabbit hearts *in vivo*. Forty-eight male New Zealand white rabbits were anesthetized with α-chloralose and instrumented for measurement of left ventricular pressure, cardiac output, and myocardial infarct size. Rabbits were assigned to one of five groups: a control group of 10 rabbits that received no pretreatment or conditioning before ischemia; a preconditioning group (n = 10) that underwent 30 min of coronary artery occlusion followed by 60 min of reperfusion; a group to which either 10 mg/kg ketamine (n = 9) or 10 mg/kg *S*(+)-ketamine (n = 8) was administered before preconditioning; and a final group of nine rabbits to which only 10 mg/kg^{-2} ketamine was administered before ischemia. Two rabbits died during coronary artery occlusion, so the researchers were able to evaluate a total of 46 rabbits for analysis.

All rabbits underwent 30 min of coronary occlusion, during which left ventricular pressure was reduced to 83 ± 14% of baseline values, and cardiac output was reduced to 84 ± 19% of baseline values. Functional recovery after 2 h of reperfusion did not differ significantly among groups. Infarct size (assessed by triphenyltetrazolium staining) was reduced from 45 ± 16% of the area at risk in controls to 24 ± 17% in the preconditioning group. Administration of ketamine had no effect on infarct size in animals without preconditioning but abolished the cardioprotective effects of preconditioning. *S*(+)-ketamine, however, had no effect, so the researchers conclude that the influence of ketamine on ischemic preconditioning is most likely enantiomer specific.

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