detailed analyses. As we have shown in a letter to Critical Care Medicine,[3] the meta-analysis of Kellum and Decker has minor flaws in data selection and presentation and several major flaws in data analysis that make its results questionable. There was no attempt by Kellum and Decker to reject our criticism, so we can assume that most of our points are valid.

The abstract of Kellum and Decker's publication in the Canadian Journal of Anesthesia contains a table with the main outcome data, which states that "values have been re-calculated", although it is unclear why, how and by whom. This re-calculation includes two wrong numbers. For one, the risk of death is 12/258 = 4.65% in the dopamine group and 14/250 = 5.60% in the placebo group, giving a relative risk ratio of 0.83, not 0.86. Secondly, the risk for onset of renal failure is 38/253 = 15.0% with dopamine and 59/270 = 21.9% with placebo, resulting in a relative risk of 0.69, not 0.72.

Finally, although the re-calculated risk ratios are not significant at the usual 0.05 level, it should be mentioned that the trend for reduction of acute renal failure with dopamine is quite strong, with $P < 0.06$ in Fisher's exact test. Consequently, we think that Bracco's conclusion that "there are no data from prospective, well-controlled, randomized clinical trials that support the use of dopamine in critically ill patients..." is premature. Rather, we would conclude with Parlow that a "further large-scale investigation into effective means of prevention is warranted".

This should include a state-of-the-art meta-analysis that replaces the values of Kellum and Decker with reliable (and updated) data. Such a meta-analysis should preferably be conducted by a group who is not biased by its own prior publications on dopamine. Alternatively, it could be a joint effort of scientists who have published pro and contra dopamine in the past.

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REPLY

I thank Dr. Maleck and his colleagues for their interest in the summary[1] on the meta-analysis of Kellum and Decker.[2] As the editor for the Best Evidence in Anesthetic Practice, my policy is to recalculate the effect estimates of a featured study if sufficient data has been published. This policy is consistent with that of secondary journals such as the ACP Journal Club.

Kellum and Decker identified 24 studies that reported at least one of their outcomes of interest; 18 were randomized controlled trials. The authors state that, "because a sufficient number of randomized trials were identified, the remainder of the analysis was restricted to these studies."[2] Cumulative risk ratios (RR) were calculated using the Mantel-Haenszel fixed effects model. The recalculated values featured in Best Evidence[1] are based on data published in Table II of the original report[2] using the same model and Meta-Analyst version 0.988 (© Joseph Lau, Boston, MA, USA). The RR of 0.83 and 0.69 for death and onset of renal failure suggested by Maleck et al. appear to be based on an equal effects model, which assumes identical within-study and between-study variances for all pooled studies.[3] This is analogous to assuming that all the results are from one single study. Such an assumption is not valid for this meta-analysis.

Like all other types of studies, the validity of a systematic review can be threatened by various confounders and biases. However, Kellum and Decker's meta-analysis provided the most up-to-date summary of the relevant literature at the time of its publication. Two recent studies have reported similar results.[4,5] An updated meta-analysis would be welcomed and eagerly appraised.

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Accidental intravascular injection of levobupivacaine and lidocaine during the transarterial approach to the axillary brachial plexus

To the Editor:

Racemic bupivacaine, when injected intravascularly, is associated with serious cardiac complications[1] such as ventricular fibrillation resistant to successful resuscitation. No such serious outcome was reported hitherto with levobupivacaine. The present case reports the accidental intravascular injection of a combination of levobupivacaine and lidocaine used for axillary brachial plexus blockade.

A 35-yr-old patient was admitted to the hospital for orthopedic surgery. Following premedication with midazolam (4 mg) and placement of all monitors, he received an axillary plexus block by the transarterial approach using a mixture of lidocaine 2% (20 mL) and levobupivacaine 0.75% (20 mL). Twenty-five millilitres of the local anesthetic mixture was deposited posterior and 15 mL anterior to the axillary artery. Briefly after deposition and without showing signs of light central nervous system (CNS) toxicity (lightheadedness, tinnitus, metallic taste), the patient exhibited three interrupted episodes of tonic-clonic seizures, each lasting for about three seconds and eventually resulting in unconsciousness. The patient's heart rate (HR) showed a sinus tachycardia of 160 beats·min^{-1}, the blood pressure (BP) increased to 180/120 mmHg and the SPO_2 decreased to 40% within one minute. For seizure control, the patient was given 5 mg of midazolam and 100 mg of propofol *iv*. Following mask ventilation with 100% oxygen, he was intubated and brought to the postanesthesia care unit. His vital signs stabilized within 30 min (BP 103/74, HR 84, SPO_2 97%) without further pharmacologic support and he was extubated. Two hours after extubation, he was alert and oriented and discharged to home. The patient did not show signs of sensory and motor blockade.

A recent case report of an accidental intravascular injection following epidural anesthesia with 19 mL of levobupivacaine 0.75% resulted in only minor CNS side effects (drowsiness, slurred speech) and, most importantly, no cardiac sequelae.[2] Plasma levels were not taken until 14 min after epidural injection, still they revealed a toxic range of levobupivaciane that, most likely, was substantially higher immediately after its intravascular administration. Though the severity of side effects remains unknown had racemic bupivacaine been administered in this patient, previous reports hint at a more serious outcome after racemic bupivacaine 0.75%.[1] Similarly, no cardiac effects other than a sinus tachycardia occurred in this young and otherwise healthy patient.

It appears that levobupivacaine is a safer drug than racemic bupivacaine, still vigilance and the laws of regional anesthesia (slow and intermittent injections, frequent aspirations) need to be practiced to take advantage of levobupivacaine's wider margin of safety.

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Skin analgesia with lidocaine tape prior to epidural blockade

To the Editor:

Lidocaine tape (Penles®, Japan Lederle, Tokyo, Japan) is a self-adhesive poultice for local anesthesia containing 18 mg of lidocaine at a concentration of 60% in a 30.5 × 50.0 mm polyester film. It has been reported that lidocaine tape provides effective skin analgesia, minimizing the pain caused by percutaneous cannulation, stellate ganglion block, and propofol injection.[1–3] Eutectic mixture of local analgesics has also been used to alleviate cutaneous pain in children and adults.[4] However, for optimal analgesic effects, the correct amount of the drug must be applied and the skin should be properly dressed for an effective absorption.[4] In this regard, lidocaine tape has advantages and is frequently used because of easier application. However, although the tape is clinically useful, elevation of the pain threshold as measured by depth of needle insertion and the optimal duration of application remain unclear.