dac enzyme determinations, suggests that the myocardium may have been protected during coronary artery clamping and cardiac standstill.

Finally, Dr. Lennon misinterpreted our rationale for using adenosine in this patient. We did not give adenosine for the purpose of preconditioning. After we were unable to effectively decrease the patient's heart rate with esmolol, we gave adenosine for the sole purpose of providing intermittent, brief periods of cardiac standstill so that the surgeons could complete the anastomosis. As we stated in our discussion, there are no reports that suggest that intermittent cardiac standstill produced by the administration of adenosine mimic ischemic preconditioning.

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To the Editor.—We read with great interest the study by Gautier et al.1 in the March issue of Anesthesiology. Clinical trials to evaluate the interactions of analgesics at the spinal cord level are very important. Gautier et al. present only the maximum pain relief score, the time at which this occurred, and the duration of adequate analgesia (i.e., time to first analgesic request after intrathecal injection). However, two agents that share these three parameters may not be equally effective (as illustrated in figure 1 using hypothetical data). A similar comprehensive description of side effects may be useful in analyzing their incidence and severity. Furthermore, such analysis may provide useful information on possible synergistic or additive interactions between sufentanil and clonidine in this particular clinical setting.

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Reference


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