Cardiovascular collapse has occurred following the inadvertent intravascular injection of local anesthetics during regional anesthesia. Clinical experience suggests that death or prolonged resuscitation may be more common following bupivacaine than lidocaine, and a number of possible explanations for this phenomenon exist. Direct myocardial depression by these agents probably plays a role in the outcome, but the direct effect of the anesthetic on the heart is obscured by concomitant systemic and central nervous system events such as seizures, hypoxia, and acidosis. The present study has used direct intracoronary injection of small bolus doses of lidocaine and bupivacaine to circumvent these problems. This approach allows determination of the duration of depression as well since little recirculation occurs.

Six dogs were anesthetized with alphachloralose and morphine. Each dog was ventilated and kept warm during a left thoracotomy. Piezoelectric crystals were implanted in the myocardium supplied by the circumflex coronary artery to measure regional myocardial contraction. The circumflex artery was cannulated and perfused with blood from a femoral artery. Small bolus doses of local anesthetics were injected directly into the tubing supplying blood to the circumflex artery and the effect on contraction was noted. Mean arterial pressure (80 mmHg) and heart rate (100 b/min) were unaffected by intracoronary injections and were quite constant throughout the experiment.

Following intracoronary boluses of local anesthetics, regional contraction decreased within about 45 seconds to a nadir and then returned to control over a period that was roughly proportional to the dose injected. Values of systolic thickening at the nadir were normalized by dividing by thickening observed just prior to injection, and the duration of the effect was taken as the time necessary for thickening to return to 95% of the pre-injection value.

Figure 1 demonstrates that the magnitude of myocardial depression caused by lidocaine and bupivacaine was dose-related. Similar degrees of depression were obtained with 4-5 times as much lidocaine as bupivacaine, a response quite in proportion to the anesthetic potencies of these agents. Figure 2 demonstrates that the duration of depression was prolonged about 25% at equivalent degrees of depression when bupivacaine was given compared to the results with lidocaine.

These findings suggest that differences in the magnitude or duration of direct myocardial depression cannot explain the clinical perception that the cardiovascular toxicity of bupivacaine is greater than that of lidocaine.