Lidocaine anesthesia for attenuation of stress responses in cardiovascular surgery: An old technique revisited

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Lidocaine anesthesia in cardiovascular surgery has the potential for decreasing anesthesia mortality and morbidity by attenuating the cardiovascular stress responses to induction, intubation and surgical incision in those patients who could not otherwise tolerate these responses. The author reviews the hazards of anesthesia for cardiovascular surgery and one technique to prevent complications from these responses.

The challenge of cardiovascular anesthesia requires that the patient be protected from the noxious stimuli of laryngoscopy with tracheal intubation and surgical stimulation and, at the same time, prevent cardiovascular depression from the anesthetic. Myocardial ischemia must be avoided. The use of lidocaine as an anesthetic of choice to meet this challenge is discussed in this article.

Case study

A 54-year-old, 100-kg male with left carotid stenosis presented for anesthesia. The patient had severe peripheral vascular disease, severe pulmonary disease and a history of myocardial infarction complicated by congestive heart failure. His hematocrit was 46.3% and hemoglobin was 14.2 g%. Triglycerides were elevated while other laboratory studies were normal. Pulmonary function studies were consistent with obstructive disease.

The patient was premedicated with diphenhydramine hydrochloride 50 mg orally. Upon arrival in the operating room, the patient had a radial artery catheter and Swan-Ganz™ catheter inserted under local anesthesia.

Anesthesia was induced with diazepam 30 mg and lidocaine 400 mg intravenously in incremental doses. This was followed by thiopental 100 mg IV. Laryngoscopy and tracheal intubation was facilitated by succinylcholine 100 mg IV. Anesthesia was maintained with 50% nitrous oxide in oxygen and a lidocaine drip 8 mg/min. Isoflurane was used to titrate the blood pressure to desired levels. All hemodynamic parameters remained stable for preoperative levels, including systemic vascular resistance and cardiac output. Pancuronium was used to facilitate mechanical ventilation.

The surgical procedure, a left carotid endarterectomy, was accomplished without complications. The patient was easily extubated. He was taken to the intensive care unit awake and alert at the end of the procedure, having been off the lidocaine drip 15 minutes prior to emergence. The patient received a total dose of 1000 mg lidocaine and 30 mg diazepam.

Materials and methods

When a lidocaine technique is to be em-
ployed, a sedative may be given as a premedicant. A tranquilizer, such as diazepam, is best suited for this purpose. It should be kept in mind, however, that respiratory depression must be avoided, as an elevated PaCO₂ will increase the toxic potential of lidocaine.

Anesthesia was induced slowly over the course of 15 minutes with the patient initially breathing 100% oxygen. Diazepam 0.3 mg/kg and lidocaine 4 mg/kg were given incrementally until the patient failed to respond to verbal stimuli. At this time, thiopental 1-2 mg/kg was able to be administered. When the lid reflex was lost, succinylcholine 1.5 mg/kg was given to facilitate laryngoscopy and tracheal intubation. When adequate ventilation was established, a lidocaine drip was started at a rate of 8 mg/min. All patients utilizing this technique were mechanically ventilated to maintain a PaCO₂ between 25-35 torr during the course of anesthesia and surgery.

The lidocaine drip was continued at 8 mg/min for the first 60 minutes, at which time the dosage was halved to 4 mg/min. Each hour the dosage was successively halved until 15 minutes prior to emergence, when the drip was discontinued.

Upon emergence, the patient’s muscle relaxant was adequately reversed and the patient was allowed to breathe 100% oxygen.

Though any inhalation agent may be used in conjunction with nitrous oxide to titrate the blood pressure, we have found that this is often not necessary at these dosage levels. This method has been used successfully in short procedures as well as lengthy operations with the same results: an awake, alert and oriented patient. Patients frequently experience euphoria on emergence and in recovery, and analgesia is usually present many hours postoperatively without the respiratory depression often seen with narcotics.

This technique must not be employed in those patients with hypersensitivity to lidocaine or in those with atrioventricular block.

**Discussion**

Inadequate attenuation of cardiovascular responses to anesthesia (induction, laryngoscopy, intubation) and surgical (incision) stress is often observed with other anesthetic techniques. These responses, occurring in patients with cardiovascular disease, can create a situation of increased oxygen demand which may lead to myocardial ischemia and death. Significant increases in heart rate, mean arterial blood pressure, rate-pressure product and systemic vascular resistance are frequently encountered at induction and incision. Many techniques of anesthesia have been employed to attenuate these responses including the administration of “industrial doses” of narcotics. These techniques are not without their hazards (such as hypotension at induction, hypertension at incision and postoperative respiratory depression) and are also to be avoided.

The patient presenting for carotid endarterectomy frequently exhibits other cardiovascular abnormalities attributed to the atherosclerotic process as do other vascular patients presenting for surgery including embolism, ischemia and arterial obstruction. This emphasizes the need for a stress-free anesthetic induction and incision in these patients.

Explained here was an acceptable technique to avoid life-threatening cardiovascular responses to induction and incision, as well as emergence from anesthesia with extubation. To exact the therapeutic effects of this technique in these patients it is necessary to understand the central nervous system and cardiovascular system effects of lidocaine.

**Central nervous system effects of lidocaine**

As with other local anesthetic agents, lidocaine interrupts the conduction of nerve impulses in peripheral nerves. Lidocaine passes easily through the blood-brain barrier. It also affects the more sensitive nerve cells of the central nervous system. Eriksson has reported a decrease in the alpha activity and an increase in lower frequencies of the EEG in dosages of local anesthetics below the convulsive level. Bartlett has reported on the analgesic actions of lidocaine but that the dosage required for this centrally mediated action is close to the convulsive level.

In conscious patients, convulsive phenomena are rarely seen with administration of lidocaine in doses of 1-3 mg/kg and the rate of injection is limited to 0.5 mg/kg/min. Knight reported the use of lidocaine in doses of 21 mg/kg in nitrous oxide-oxygen-diazepam anesthesia for cardiac surgery. Electroencephalographic evidence of seizure activity was absent.

Lidocaine does not significantly interact with the autonomic nervous system. De Jong has reported that hypercarbia enhances the convulsant activity of local anesthetics emphasizing the need to support ventilation.

**Cardiovascular effects of lidocaine**

Local anesthetics have long been recognized as cardiovascular depressants. We are all familiar with the anti-dysrhythmic effects of lidocaine.
Lidocaine causes depression of myocardial contractility and arterial blood pressure to a lesser extent than does procaine amide.7 Lidocaine given to anesthetized patients tends to cause a slight blood pressure elevation.

Kao6-8 reports this drug action is mediated by central stimulation probably through the sympathetic nervous system. Although high blood levels of local anesthetics tend to cause dilation of blood vessels, the release of epinephrine or increase of sympathetic activity may block this action.

Falk has shown a decrease in cardiac index, systolic arterial blood pressure, and left ventricular stroke work index in patients given lidocaine following induction of anesthesia similar to that observed in Knight's study.8

Lidocaine can, indeed, cause acute and severe hypotension when used as a supplement to nitrous oxide-thiobarbiturate anesthesia. This is infrequent and the reported cases have not differentiated the cause, for example, additional drug administration. This lack of vasomotor depression with the administration of lidocaine is advantageous in patients who could not tolerate an increase in oxygen demand in the face of decreased perfusion.

The major determinants of increased myocardial oxygen demand include heart rate, myocardial wall tension and contractility. The ability of lidocaine to attenuate the stress response to noxious stimuli (that is, hypertension and tachycardia during induction and surgery) has been shown in numerous studies.9-11 Thus, increased oxygen demand may not occur when this technique is employed. If one extrapolates these data to therapeutic blood levels after intravenous administration of lidocaine it is easily seen how advantageous this agent can be as a general anesthetic.

The intravenous administration of succinylcholine has been shown to increase the serum potassium concentration. This increase may be associated with serious dysrhythmias and even cardiac arrest.12 Administration of lidocaine may prevent this phenomena and its accompanying dysrhythmias.18

Conclusion

Lidocaine has been administered intravenously as well as in bolus form for (1) the control of cardiac dysrhythmias, (2) to provide tracheal tranquility during laryngoscopy and intubation; and (3) for the control of extubation stress at anesthesia emergence. The technique reported here has the advantage of standardizing a dosage regimen and controlling the therapeutic blood level of lidocaine. This technique can be utilized in many procedures regardless of the length of surgery. It has added advantages in use in the elderly where hemodynamically stable inductions are desired as well as when early extubation is necessary to prevent post-emergence stress.

Intravenous lidocaine, utilized as an adjunct to diazepam-nitrous oxide anesthesia, provides another regimen which can be part of the anesthetists' armamentarium in caring for the patient with atherosclerotic cardiovascular disease.

REFERENCES


AUTHOR

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