Although lidocaine is commonly accepted as an anti-arrhythmic drug, it can also be used intravenously as an anesthetic agent. The author focuses on how lidocaine can be used as an integral part of the anesthetic itself for patients with cardiac arrhythmias, particularly premature ventricular contractions.

Lidocaine has been used for many years as an adjunct to general anesthesia. In earlier years, it was used intravenously to suppress the cough reflex in light planes of anesthesia. In recent years, it has been used extensively in conscious patients for the treatment of ventricular arrhythmias. We believe that intravenous lidocaine can be used advantageously for the patient requiring general anesthesia who has or has had premature ventricular contractions (PVCs).

In instances where the patient is known to have PVCs, we plan the use of intravenous lidocaine as an essential part of the anesthetic and not as a treatment for the PVCs after the anesthetic has begun. While the uses of lidocaine as an anti-arrhythmic and a local anesthetic are widely known, it is less known that the central nervous system depression of lidocaine can be used as an adjunct in general anesthesia.

Effects of lidocaine on the central nervous system

When lidocaine is given intravenously, it has an analgesic effect mediated through the central nervous system (CNS). It also may cause a blockade of sensory endings in the skin and viscera. The reason that one does not commonly see this depressant effect upon the CNS when lidocaine is used for local infiltration, regional anesthesia, and treatment of arrhythmias is that the blood levels are not high enough to produce them.

The effects of local anesthetics are dose related. The larger the dose, the greater the response. Lidocaine, when given in a dose that reaches a toxic blood level, will cause convulsant activity. However, in less than toxic levels, it is an anti-convulsant and will cause CNS depression.1

Lidocaine has this effect as demonstrated by a slowing of the brain waves. The level of pCO₂ plays an important role as to how much lidocaine is needed to cause a toxic reaction. The higher the pCO₂ (hypercarbic state), the lower the dose of lidocaine needed to cause convulsions. The converse of this is also true. The lower the pCO₂ (hypocarbic state), the greater the dose of lidocaine needed to cause convulsions.2

Effects of lidocaine on the cardiovascular system

The effects of lidocaine on the cardiovascular system are also dose related. The slow injection of intravenous lidocaine of up to 5mg/kg will cause an increase in blood pressure in 50% of patients. This is believed to be due to an increase in the stroke volume of the heart and an increase in the heart rate. However, in 6% of patients, hypotension can develop which can be severe and rapid in onset.
Lidocaine's antiarrhythmic properties are due to a decrease in membrane excitability. Lidocaine also causes a slowing of the electrical conduction in the S-A node, A-V node and the Bundle of His. Therefore, in patients with complete heart block, the use of lidocaine as an anesthetic should be avoided.

Lidocaine passes through the blood-organ barrier easily. A dose of intravenous lidocaine will reach a maximum blood level within seconds, but this quickly falls. Because of this redistribution, the body can apparently tolerate these transient high blood levels of lidocaine without ill effect. Lidocaine enters into the vessel rich organs (brain, heart, liver, lung, kidney, and spleen) first and then into the less perfused tissues (muscle and fat).

As time passes and redistribution occurs, the percentage of lidocaine falls in the vessel rich organs and rises in the muscle and fat. Lidocaine has a high lipid solubility and diffuses into the fat easily. This redistribution curve of lidocaine is very similar to that of thiopental. In both cases, repeat injections of these drugs are needed at short intervals or by continuous infusion to maintain a stable blood level.

While the redistribution is rapid, the metabolism is much slower. At the end of 24 hours, it is nearly complete; only 10% of lidocaine is left unchanged in the body. This metabolism is accomplished in the liver by enzymatic hydrolysis or an oxidation process. Lidocaine is not detoxified in either the kidney or brain.

Toxic reactions

Toxic reactions to lidocaine are not produced by an allergy to the drug. The systemic toxic effects of lidocaine are dose related. The most common and obvious toxic effect of lidocaine is generalized seizure activity. The most important treatment of these seizures is the maintenance of ventilation. The seizures are self-limited: this means that the seizures will stop when the serum lidocaine level falls below that which triggers the seizure activity. It has been shown that epileptics do not have a greater susceptibility to the toxic effects of lidocaine.2

The increased muscle activity during a seizure causes a rise in oxygen consumption. This along with impaired respirations and a depressed myocardium (also due to a lidocaine toxicity), makes it imperative that the anesthetist's first concern is to ventilate with oxygen. A small dose of barbiturate (thiopental) will usually stop the convulsions, but caution must be used since too much barbiturate will further depress respirations and the myocardium.

Allergic reactions to lidocaine are exceedingly rare. Most allergic reactions to local anesthetics involve the ester-linked group, of which procaine is a member.

Lidocaine usage

Due to the advances in all fields of medicine, we now see an increase in the number of patients with heart disease and arrhythmias coming to surgery for elective operations. They often have a history of PVCs or come to the operating room with PVCs present. The anesthetist is faced with the dilemma of how to manage these patients and what type of anesthesia to administer.

Twenty years ago, Steinhaus and Howland studied a series of 135 patients to whom they administered approximately equal doses of lidocaine and thiopental with 70% nitrous oxide. There was only one incidence of a convulsion, which was treated successfully with a successive dose of thiopental. At the time of the convulsion, the dosage of lidocaine was more than twice that of the thiopental dosage. The duration of anesthesia was from 30 minutes to 5 hours and 20 minutes and the dosage of lidocaine was from 250-1000 mg.

Steinhaus and Howland concluded that when thiopental and lidocaine are given in equal amounts, there is no evidence that seizure activity is produced. When ECGs were performed on patients having a thiopental, lidocaine and nitrous oxide anesthetic, a slowing of the brain waves was noted. After a dosage of 350 mg of lidocaine, the brain waves slowed from 10/sec to 6/sec.

In 1977, Himes, DiFazio and Burney determined in a clinical study to what degree lidocaine lessens the amount of thiopental needed to achieve an anesthetic level. They found that with a plasma lidocaine concentration of 3.2 micrograms/ml, a MAC fraction equivalency of .15-.28 will be obtained. This means that if a patient's blood level of lidocaine is 3.2 micrograms/ml, he or she will need approximately 1/5 to 1/4 less inhalation anesthesia.

Contraindications for use of lidocaine for intravenous anesthesia are: (1) history of allergy to lidocaine, and (2) complete heart block.

Clinical study

In our institution, we first followed the technique described by Steinhaus and Howland. The patients were premedicated with an appropriate amount of a sedative and a narcotic. The induction in patients, who were not having arrhythmias at that moment, was accomplished with approximately equal doses of thiopental and lidocaine in divided doses to effect.
In patients who had dysrhythmias at the time of induction, a loading dose of lidocaine was given in 50 mg boluses until the desired results were obtained. Then we proceeded with the induction in the manner above. (In this second instance, the dosage of lidocaine will be higher than the dosage of thiopental.) Intubation, if needed, was accomplished with a bolus of succinylcholine or a succinylcholine drip.

Maintenance of the anesthetic was with 70% nitrous oxide, 30% oxygen, and intermittent doses of lidocaine and thiopental. These were given each five minutes. Equal doses of 20 mg (lidocaine and thiopental) were administered. After one hour, these dosages were cut in half.

In the last several months, we have been using a continuous lidocaine drip technique for maintenance of anesthesia. Two grams of lidocaine are mixed with 500 ml of solution (4mg/ml). This is administered with an intravenous infusion pump at the rate of 5 mg/min for the first hour. After the first hour, the dosage is halved. As with any type of anesthetic, the rates of administration are variable. These dosages have been cited as a guideline and not as an absolute standard.

During the past 10 months, all patients anesthetized in this manner at our institution were male between the ages of 60 and 90 years. They all had a history of dysrhythmias and, in most cases, came to the preanesthesia room with frequent PVCs. The duration of the anesthetics was between 30 minutes and 3 hours. The surgical procedures performed included a wide variety (cystoscopy, direct laryngoscopy, oral surgery, inguinal hernia repair, and so on). The dosage of lidocaine was from 100-460 mg. The dosage of thiopental was from 100-520 mg.

Using this technique, we have not seen any evidence of seizure activity during the anesthetic or in the immediate post anesthetic period. In the past month, we have been able to obtain serum lidocaine levels, which ranged from 3.0 micro-
grams/ml - 3.7 micrograms/ml. These samples were drawn at the conclusion of the anesthetic. A wider range of values will likely occur as we add to the number of the cases done in which the serum lidocaine level is measured.

Case report

A 70-year-old male was brought to the operating room for a ventricular-atrial shunt. He had suffered a head injury after falling at home three months previously. He was admitted to another hospital and underwent a craniotomy for evacuation of a subdural hematoma. Three weeks postoperative he was transferred to Omaha Veterans Administration Medical Center. His physical status at the time was poor.

He was dehydrated and debilitated due to severe rheumatoid arthritis and the effects of two cerebral vascular accidents in the past two years. He was bedridden and had decubiti on both buttocks. He was also confused, combative and agitated. He was anemic and his electrolytes showed signs of decreased renal function. His preoperative chest x-ray indicated a resolving pneumonia and his electrocardiogram noted several unifocal PVCs.

Upon arriving in the operating room, it was discovered that the patient had a bigeminal rhythm (Figure 1). We were unable to start an intravenous infusion on the patient due to his poor peripheral vasculature and his lack of cooperation. The decision was made to induce anesthesia with enflurane, start an intravenous infusion as soon as the patient was relaxed, and then change to a lidocaine-thiopental technique with 70% nitrous oxide and 30% oxygen. Following induction, the patient continued to have frequent PVCs and runs of bigeminal contractions (Figure 2). We then began to administer 50 mg boluses of lidocaine.

After 10 minutes in which a total of 200 mg of lidocaine had been given, the heart was no longer in bigeminal rhythm and the frequency of the PVCs was steadily decreasing (Figure 3).
Twenty minutes after the lidocaine administration had begun, the patient had received 270 mg and a continuous drip of 4 mg/min had been started. The heart was in a regular sinus rhythm with no PVCs noted (Figure 4). A serum lidocaine level done at this time was 2.9 micrograms/ml. Continuous infusion of lidocaine at 4 mg/min was administered for one hour and then the rate was adjusted to 2 mg/min. A serum lidocaine level was done and was 2.4 micrograms/ml. The operation was concluded 20 minutes later and the lidocaine infusion was not restarted.

During the last 1 hour and 20 minutes of the anesthetic, the cardiac status indicated a regular sinus rhythm with only an infrequent PVC. In the recovery room, the patient continued to have infrequent PVCs and the lidocaine infusion was not restarted.

Summary

We have been able to greatly decrease the frequency of PVCs and other arrhythmias during the course of anesthesia by using lidocaine as an integral part of the anesthetic. All the patients in our series had fewer PVCs under anesthesia compared to the immediate preoperative period. In many instances, no PVCs were seen during anesthesia. One patient was having 15-20 PVCs/min just prior to the administration of anesthesia. While the anesthesia was being given, no PVCs were noted; however, after one hour in the recovery room, the patient was again exhibiting frequent PVCs.

We believe that intravenous lidocaine, used as a part of the anesthetic itself, provides a comparatively safe and useful alternative to other agents in the high risk patients who have PVCs or a history of PVCs. At this hospital, we have a high percentage of geriatric patients with multiple system diseases, including atherosclerotic heart disease. We have confidently gone forward with lidocaine as an anesthetic component in patients with frequent PVCs when we would have been reticent to proceed using other agents.

The anesthetist should remember that the use of intravenous lidocaine will reduce the amount of other anesthetic agents needed. To avoid symptoms of lidocaine toxicity (such as seizure activity) the dosage of lidocaine should be approximately equal to that of thiopeptal.

REFERENCES


ADDITIONAL REFERENCES

For general information, these references will serve as a useful adjunct.

ACKNOWLEDGEMENT

The author would like to thank Cloid D. Green, MD, head of the Anesthesia Department at the Omaha Veterans Administration Medical Center, for his assistance and guidance.

AUTHOR

James F. Sorrell, CRNA, was a physiology major at the University of Nebraska in Lincoln, Nebraska. He received his education in nursing at Lincoln General School of Nursing in Lincoln, Nebraska. He underwent his anesthesia education at the Minneapolis Veterans Administration School of Anesthesia, Minneapolis, Minnesota. He is employed at the Omaha Veterans Administration Medical Center in Omaha, Nebraska, where he is the chief nurse anesthetist.
You can’t buy a more efficient, easier to use, more economical hyper-hypothermia system than BLANKETROL®

We want the chance to prove it to you.

First, start with the BLANKETROL unit. Patient temperature control accuracy and heating and cooling rates are unsurpassed. Primary and secondary thermostats assure patient safety. The BLANKETROL is simple to operate, mobile and completely field serviceable. Add our blanket to the unit. The BLANKETROL Herculite® Staph-Chek® long-life blanket is light-weight, easy to fold and store. It heats or cools more efficiently because distilled water circulates through it 4 to 6 times faster than competitive blankets.

If punctured, the latex internal tubing can be replaced in minutes. Thus, it lasts an average of 7 to 10 years. Then, protect blanket and patient for greater efficiency. DISPOSA-COVERS™ are inexpensive, easy to use covers for hyper-hypothermia blankets. They help any blanket last longer and protect patients from moisture and cross-contamination. DISPOSA-COVERS, used with Herculite® long-life blankets, eliminate the need for expensive disposable blankets.

Challenge us to prove what we say. Call your BLANKETROL dealer or call us. We’ll demonstrate the superiority of BLANKETROL.

Cincinnati: Sub-Zero Products, Inc.
2612 Gilbert Avenue
Cincinnati, Ohio 45206
(513) 751-8810

M/S Skyward leaving Miami
Ports of Call
* Cap Haitien - Haiti
* San Juan - Puerto Rico
* St. Thomas - Virgin Islands
* Puerto Plata - Dominican Republic

March 7-14, 1981

CARIBBEAN

C. E. CREDITS

The M/S Skyward is the flagship of the Norwegian Caribbean lines® which takes more people to the Caribbean than any other line. Experience the fabulous Meals, Entertainment, and Ports of Call with the “First Fleet of the Caribbean.”

Meeting Registration $225.00 per participant
M/S Skyward rates:
Category 6 - $1010.00 inside room, double bed
Category 8 - $1040.00 outside room, 2 beds
Category 9 - $1010.00 outside room, double bed
Category 10 - $970.00 inside room, double bed
Category 15 - $930.00 inside room, double bed

All rates are per person double occupancy. Rates include all Port taxes, Meals and Entertainment on board ship. Single and family rates available on request. All rooms have private shower and toilet.

Reservations: Send $200 (refundable) deposit per person. State category of room desired - full details by return mail.

For information and reservations, contact:
MTSA Faculty, P.O. Box 1414, Madison, Tennessee 37115
(615) 865-2373 ext. 4530

M.T.S.A. Faculty
Middle Tennessee School of Anesthesiology

WITH guest speakers:

Ronald L. Katz, M.D.
Professor & Chairman, Dept. of Anesthesia UCLA

Leah E. Katz, CRNA, MA
Director, Program of Nurse Anesthesia, UCLA