ACTA MEDICA IRANICA
Vol. 31, FALL 1993

LIDOCAINE'S CONTROVERSY

Zahid Hussain Khan*, MD

* *Assistant Professor, Tehran University of Medical Sciences, Faculty of Medicine, Imam Khomeini Hospital: Department of Anesthesiology.*

SUMMARY

In a cross section of the patients scheduled for various operations, the plausability of lidocaine as an induction agent is studied and described. The results were dismaying ,suggesting, henceforth, that as an induction agent, lidocaine altogether fails to accomplish the assignment assigned to this drug.

KEY WORDS: *Hypovolemia; Induction agent; Lidocaine; Thiopental sodium.*

INTRODUCTION

Through these columns, I would like to draw the attention of the worthy readers to the effects of lidocaine which either remain unresolved or as an enigma. I read the parts pertaining to lidocaine in professor Miller's masterpiece on anesthesia wherein he assiduously tackles lidocaine and its effects in considerable detail. The part that the author advocates it as an induction agent evoked my inquisitiveness and as a result I was lured to employ it on our patients.

MATERIALS AND METHODS

The age of the patients studied for the effects of lidocaine ranged from 10-48 years and the opera--tions varied from elective ones to the emergency operations such as craniectomies for extradural haematomas, splenectomies, and emergency thora--cotomies. For emergency operations, the case selection was based on the criteria of hypovolemia. Lidocaine (1.5 mg/kg) may be superior to thiopental in hypovolemic patients, as it tends to be less of a cardiovascular depressant. However, lidocaine provides less sedation than induction doses (3-4 mg/kg) of thiopental (1). Using the recommended dose, we failed to notice sedation and attempts at intubation were futile as the patients developed marked resistance. Likewise, attempts at intubation were totally dismaying in another group of patients in whom lidocaine was preceded by premedication. For premedication we employed 0.5 mg/kg pethidine and 0.1 mg/kg diazepam injected intravenously before lidocaine. An eleven-year-old boy, who was not premedicated, developed nystagmus and blurred vision, but the attempts at intubation were fruitless and it could be only accomplished after the patient received a sleep dose of thiopental and a paralysing dose of succinylcholine.

RESULTS

Lidocaine had been used as an induction agent in the Balkan States and some African countries in the 1980s, but it never gained widespread popularity in the other parts of the planet. Even in the afore-mentioned places, lidocaine eventually lost its ephemeral fervor and finally went into disrepute for the reasons not traceable in literature.

The fact that professor Miller introduced once again as an induction agent in his book was tempting. I tried it and found it unworkable. Our exercise misfired and signally failed. Professor Miller

is requested to either provide substantial evidence, in this regard, or delete this part from his voluminous book on anesthesia as by propagation of the efficacy of lidocaine as an induction agent would not only tarnish the widespread popularity that his book has gained but also it might create confusion amongst its readers. I have set the ball rolling and would welcome others to continue scientific debate which focuses on an issue that requires not only much urgent thought and attention but also compromises the canonical morality of the medical profession.

DISCUSSION

The toxic symptoms of lidocaine are crystal clear and depicted well in Table 29.6 (1). Toxic symptoms ensue as light headedness and tinnitus when serum concentration reaches 5 mg/ml (1). Again, in the same vein it is stated that symptoms such as mild drowsiness or agitation may occur at plasma concentrations of 5 mg/ml (2). It is also mentioned that toxic levels of local anesthetics probably lead initially to depression of cortical inhibitory pathways; thereby allowing unopposed activity of an excitatory nature (1). Thus, it can safely be deduced that it is the agitation or excitation that appears first and thus, lidocaine, under no circumstances,is suitable for the induction. It has also been mentioned that following the absorption, all nitrogenous local anesthetics may cause the stimulation of the CNS, producing restlessness and tremor that may proceed to clonic convulsions (3). Thus, if we employ lidocaine intravenously to achieve sedation so as to facilitate intubation, there may be every probability that we achieve agitation instead. Moreover, Table 29.6 (1) makes it much more clear that the predominant toxic effects of lidocaine are only excitatory at serum concentrations of up to 10mg/ml and the depressive toxic effects would ensue only when the serum levels of lidocaine exceed 10 ml. Sedation, as far as I can comprehend from the table cataloguing the effects of lidocaine, would fall somewhere between convulsions and unconsciousness and aiming at that stage could conceivably be an impending disaster. If it is reckoned that sedation customarily or logically follows convulsion, then it can be deduced that larger doses would be required to achieve such a state and that would mean we are heading for a catastrophe.

Again it is mentioned in the aforementioned book that 6.4 mg/kg is the maximum dose for lidocaine, and CNS toxicity would appear when this dose exceeds. But 6.4 mg/kg of lidocaine employed for intercostal block leads to higher plasma levels than the same dose employed for epidural of brachial plexus blocks. Whether to accept 6.4 mg/kg as the maximum safest dose for all the blocks or not, remains unresolved and nowhere in the book is this aspect featured prominently. It is pertinent to state here that toxicity undoubtedly depends upon the administered dose; nevertheless, it depends more upon the serum levels of lidocaine which in turn is largely governed by the rapidity of injection. To put it simply, fast and rapid injections would elevate the plasma levels more quickly.

ACKNOWLEDGEMENT

I am grateful to Dr. Mehran Khoei, a resident of anesthesiology for his assistance in this study.

REFERENCES

1) Miller, RD. (1986). Anesthesia (in 3 volumes), (2nd ed.). (pp. 514-515, 1006-1008, 1091-1092).

2) Hurst, JW, & Schlant RC.(1985). The Heart, (7th ed.). (pp. 1683, 1691-1693). McGraw-Hill Information Services Company.

3) Gilman, AG, Rall, TW, Nies, AS, & Taylor, P. (1991). The pharmacological basis of therapeutics. Goodman and Gilman's (8th ed.). (pp. 859-861, 1688). Maxwell Macmillan Pergamon Publishing Corporation.