IV vs. LTA Lidocaine: Does It Make Any Difference?

To the Editor—The recent article by Hamill et al.\textsuperscript{1} makes a seemingly invalid comparison between intravenous (iv) and laryngotraceal (LTA) administered lidocaine. A serious flaw in the protocol design relates to the fact that the LTA group was laryngoscoped twice while the iv group underwent a single laryngoscopy. The authors might have considered transtracheal administration of lidocaine as a means of avoiding a second laryngoscopy in the LTA group.

A further analysis of their data leads me to conclude that there may be very little, if any, difference between the two modes of administration. Utilizing data from figure 1 of their article, it appears that the rise in ICP following the initial laryngoscopy equalled 11 mmHg in the “untreated” LTA group (laryngoscopy only) vs. 8 mmHg in the group which had received lidocaine 1.5 mg/kg, iv (laryngoscopy-intubation). Furthermore, the ΔICP after laryngoscopy and intubation was 9 mmHg (from 17 to 26 mmHg) in the group pretreated with endotracheal lidocaine vs. 7 mmHg (from 9 to 16 mmHg) in the iv lidocaine group. The per cent increase in MAP was 36 per cent (−12 to 24) in the iv lidocaine group vs. “only” 48 per cent (−4 to 44) in the LTA group, even though the latter group was laryngoscoped twice! Finally, although the authors measured the intracranial compliances, the values were not reported. It would be interesting to know whether these patients were on the relatively flat vs. steep portions of the intracranial pressure-volume curve.

The authors noted no difference in peak ICP, MAP, and HR values after laryngoscopy and intubation between the LTA group and an historical control group\textsuperscript{2} who received “a similar anesthetic technique except that no lidocaine was given.” Intravenous lidocaine unquestionably lowers the baseline ICP\textsuperscript{3}; however, I am not convinced, on the basis of this study, that intravenous lidocaine is superior to endotracheal lidocaine in blocking the increases in ICP and MAP associated with laryngoscopy and intubation. Although the study of Hamill et al.\textsuperscript{1} does not permit a direct comparison (as there was no control group), one might question the efficacy of both LTA and iv lidocaine in blunting the hypertensive responses to laryngoscopy and intubation.

Paul F. White, Ph.D., M.D.
Assistant Professor of Anesthesiology
Stanford University Medical Center
Stanford, California 94305

References

(Accepted for publication December 11, 1981)
In reply—Since both intravenous (iv) and laryngotraacheally administered (LTA) lidocaine had previously been shown to blunt the cardiovascular responses to endotracheal intubation,1,2 we did not feel justified in exposing a third group of patients to the risks of a “no lidocaine” control protocol.

Dr. White’s concern over the LTA lidocaine technique is well-intentioned, yet it ignores the realities of clinical care. A survey of clinicians both at our institution and elsewhere indicated that most adhere to the technique as originally described3 and do, in fact, laryngoscope patients twice when using the LTA® kit or a facsimile thereof. It was this specific clinical technique which we were interested in comparing with lidocaine given as a bolus iv. We could have chosen ultrasonic inhalation of lidocaine solution as a less noxious method for administering laryngotraacheal lidocaine but felt it lacked clinical relevance because it is not widely used. We ruled out the use of transtracheal lidocaine instillation, not only because of the vigorous coughing it causes but also because of the well-known hazards associated with transtracheal puncture.3

Intracranial compliance data were deleted at the suggestion of ANESTHESIOLOGY’s reviewers, although we, too, believe these are pertinent whenever changes in ICP are examined. Actual values were: LTA lidocaine group, 3.9 mmHg/ml ± 0.6 SE; and iv lidocaine group, 3.5 mmHg/ml ± 0.4 SE.

Increases in intracranial pressure or cardiovascular variables may be either benign or harmful depending on the baseline values from which they are derived, even though the changes are similar in magnitude. Dr. White focuses on the absolute changes with intubation and concludes that these responses are similar in both groups of patients. If, however, LTA lidocaine administration has raised ICP or myocardial oxygen demand near the threshold for either brain herniation or myocardial ischemia, then an additional increase caused by endotracheal intubation becomes potentially deleterious to the patient. Accordingly, we believe that the fashion in which the data are presented and the clinical relevance of these findings are self-evident.

JAMES F. HAMILL, M.D.
ROBERT F. BEDFORD, M.D.
DAVID C. WEAVER, M.D.
AUSTIN R. COLOHAN, M.D.
University of Virginia Medical Center
Charlottesville, Virginia 22908

REFERENCES

1. Abou-Madi MN, Keszler H, Yacoub JM: Cardiovascular reactions to laryngoscopy and tracheal intubation following small and large intravenous doses of lidocaine. Can Anaesth Soc J 24:12–19, 1977

(Accepted for publication December 11, 1981.)