known. While it would have been valuable to include at least one group not treated with an oral airway, our Human Research Committee felt that such a group would be at greater risk and would not approve their inclusion in the study. However, the low incidence and severity of postoperative sore throat in the unlubricated double contour cuffed group suggests that the contribution of the oral pharyngeal airway, whatever it is, is not great.

Another criticism of the study could be that the method utilized to determine whether postoperative sore throat was present, namely evaluating for hoarseness and asking specific questions with regard to postoperative sensation in the patient's throat, may have contributed to an apparently higher incidence than really exists. While it is difficult to critically evaluate this question, it is our impression, after conducting numerous studies of this kind, that many patients will not volunteer information about their throat and pharynx unless specifically questioned. Consequently, although the apparent incidence of postoperative sore throat in this study may be higher than the incidence found by most clinicians, the real incidence is probably higher than is appreciated by most clinicians.

In conclusion, our results demonstrate that both endotracheal tube cuff design and lubrication are factors in the development of postoperative sore throat. We believe improved design and better lubricants could further minimize postoperative sore throat.

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Failure to Initiate Electroconvulsive Seizures in a Patient Pretreated with Lidocaine

DAVID D. HOOD, CAPT., USAF, MC,* AND ROGER S. MECCA, MAJ., USAF, MC†

Ventricular arrhythmias and hypertension are serious complications of electroconvulsive therapy (ECT) and are associated with much of the morbidity and mortality.[1–3] Lidocaine, administered iv, often is used to treat these arrhythmias.[2,4] In this case report, iv administered lidocaine interfered with the initiation of seizure activity during ECT. We describe an alternative method of prophylaxis against ventricular arrhythmias.

Report of a Case

A 58-year-old woman was scheduled to receive a series of 15 ECT treatments for major depression refractory to conventional drug therapy. History revealed the diagnosis of a 1-cm, left internal carotid artery aneurysm near the cavernous sinus, but no other evidence of cardiovascular or respiratory disease. The electrocardiogram and serum electrolyte values were within normal limits.

The patient received general anesthesia for all of her ECT treatments. Monitoring was accomplished with continuous ECG, EEG, precordial stethoscope, blood pressure cuff, and a second blood pressure cuff inflated to a pressure greater than the patient's systolic blood pressure. This second blood pressure cuff was inflated prior to the administration of iv succinylcholine. General anesthesia for the initial two ECT treatments was administered uneventfully with the inhalation of 100% oxygen via a mask for five min and 0.4 mg atropine, iv, followed by 1 mg/kg methohexital, iv, 1 mg/kg succinylcholine, iv, and assisted or controlled ventilation via a mask with 100% oxygen

* Currently Fellow, Department of Anesthesiology, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina.

† Staff Anesthesiologist, Department of Anesthesiology, Wilford Hall USAF Medical Center, San Antonio, Texas.

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Address reprint requests to Dr. Hood: Department of Anesthesia, Bowman Gray School of Medicine, Wake Forest University, 300 South Hawthorne Road, Winston-Salem, North Carolina 27103.

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during apnea, seizure, and the post-seizure period. Seizure activity was monitored with continuous EEG (1-channel) and observation of the somatic seizure activity in the unparalyzed arm. Prolonged seizure activity (1–2 min) was easily elicited with a 30 watt-s electroshock. After induction of anesthesia with methohexital before the third treatment, hypertension (160/100 mmHg), multifocal premature ventricular contractions (PVCs), and 3-mm ST depression in the monitored lead II occurred. This patient was felt to be receiving adequate ventilation with 100% oxygen via a mask, as evidenced by good color, clear and unchanged breath sounds, reasonable chest wall excursion, and no obvious change in pulmonary compliance. These ECG changes resolved with hyperventilation and 60 mg (1 mg/kg) lidocaine iv. The procedure was cancelled, and the patient was admitted to the coronary care unit where the diagnosis of myocardial infarction was ruled out.

Anesthesia for subsequent treatments was identical to that described above except atropine was omitted. During the fourth treatment, hypertension (180/110 mmHg), multifocal PVCs, and 2-mm ST depression developed during the clonic phase of the seizure, despite supplemental methohexital and clinically adequate ventilation with 100% oxygen via a mask. The PVCs and ST changes resolved with 60 mg lidocaine, iv, although the hypertension persisted. In order to avoid the emergence of PVCs during the clonic phase of the fifth treatment, 100 mg (1.6 mg/kg) of lidocaine was administered iv approximately 3 min prior to the induction of anesthesia. With this regimen, seizure activity could not be elicited with the previously effective energy level. With the fourth shock, a brief 30-s seizure was initiated at an energy level four times greater (120 watt-s) than previously required. During the clonic phase of this seizure, moderate hypertension (160/100 mmHg) and occasional multifocal PVCs resulted.

During the sixth treatment, lidocaine therapy was withheld until electrical evidence of seizure activity had ceased, even though multifocal PVCs and hypertension (160–180/110–120 mmHg) again developed during the clonic phase. Analysis of arterial blood gases during the period of ventricular ectopy revealed a Pa_{O_2} of 300 mmHg, with normal pH and Pa_{CO_2}. Serum electrolytes were also normal. Lidocaine (1.6 mg/kg) iv was administered immediately upon cessation of electrical seizure activity. Venous blood levels of lidocaine, drawn at 5 and 8 minutes after injection, were 3.5 μg/ml and 3.3 μg/ml, respectively. (These time intervals closely approximated the period between injection of lidocaine and attempted seizure initiations during the previous treatment.)

For the remaining 10 ECT treatments, 0.5 mg iv propranolol was administered two minutes prior to initiation of the seizure. Lidocaine, 1.6 mg/kg, was administered only after electrical evidence of seizure activity had ceased. Neither bradycardia nor PVCs developed during any of the ten treatments. The rise in systemic blood pressure during the clonic phase was markedly attenuated (140/90 mmHg maximum recorded level). The patient was well-oriented and communicative on discharge, and was judged to have a beneficial result from the ECT therapy.

Discussion

Preexisting cardiac disease, acid-base status, oxygenation, premedication, and the degree of hypertension accompanying the seizure influence the severity and incidence of arrhythmias during ECT (8–75% of all patients).[5,6] Rhythm disturbances probably are triggered by uncontrolled autonomic nervous system activity caused by diffuse electrical stimulation in the brain. During the initial 10- to 15-s tonic phase of the seizure, arrhythmias attributable to parasympathetic nervous system predominance are common (bradycardia, asystole, ventricular escape beats), in conjunction with other signs of increased vagal tone (increased salivation, hypotension). During the subsequent clonic phase, rhythm disturbances are more characteristic of sympathetic nervous system predominance (sinus tachycardia, multifocal PVCs) in conjunction with hypertension.[4] Hypoxemia, acidosis, and myocardial ischemia also may promote or exacerbate these arrhythmias.[2,6]

The biphasic autonomic nervous system response makes treatment of arrhythmias during ECG difficult. Although pretreatment with atropine sometimes eliminates or attenuates the initial parasympathetic nervous system induced arrhythmias, this therapy is often ineffective and may exacerbate the subsequent sympathetic nervous system induced clonic phase arrhythmias.[3,6] Lidocaine iv alone or with atropine pretreatment often proves effective in treating ventricular arrhythmias during the clonic phase.[4] However, lidocaine pretreatment also may suppress or shorten the seizure activity in a dose-related manner, interfering with the primary therapeutic goal of ECT.[1,7,8]

Seizure activity is a well-documented manifestation of toxic blood levels of local anesthetics, including lidocaine (approximately 6 μg/ml CNS threshold level).[9,10] Seizures, however, are probably a result of the depressant effects of local anesthetics on inhibiting neurons in the central nervous system.[8] Lidocaine is an effective anticonvulsant at low to moderate blood levels, both for spontaneous seizures refractory to standard anticonvulsants[11,12] and for electrically induced seizures during ECT.[7,8,13]

The total seizure time probably correlates with ECT treatment efficacy.[13,14] Barbiturates increase seizure threshold in a dose-related manner, shortening the duration of the seizure or preventing seizure initiation.[14,15] Increasing the barbiturate induction dose might have attenuated the hypertension and ventricular irritability in our patient. However, inadequate ECT treatment resulting from shortened seizure duration or failure to induce a seizure increases the patient's risk by requiring further ECT treatments to attain the same therapeutic goal.

The anticonvulsive effects of barbiturates and lidocaine when combined have been shown to be additive.[14,15] Seizure duration is reduced significantly with the addition of 1 mg/kg iv lidocaine to a standardized treatment protocol using hexabarbital.[14]

In this case, lidocaine was administered before the seizure to prevent the emergence of the serious ventricular arrhythmias which occurred during previous treatments. The resulting lidocaine blood level appar-

ently prevented the initiation of seizures, even with a stimulus level three times that previously required. Increasing the stimulus by a factor of four elicited a short seizure judged suboptimal for therapy by the attending psychiatrist. In spite of the lidocaine prophylaxis, ventricular arrhythmias occurred during this seizure. These PVCs probably were not caused by hypoxemia, acidosis, or electrolyte imbalance, since values for these variables were found to be within normal limits on analysis of the arterial blood gases drawn during the seizure and arrhythmias in the subsequent ECT.

Since PVCs during the clonic phase probably resulted from excessive sympathetic nervous system stimulation at the $beta_1$ receptor, pretreatment with intravenous propranolol should eliminate these ventricular arrhythmias and yet not interfere with seizure activity. Oral pretreatment with propranolol has been reported effective in suppressing clonic phase ventricular arrhythmias in one patient[16] without exacerbation of bradycardia during the tonic phase. In both the previously reported case and in our case, propranolol pretreatment also attenuated the hypertensive response characteristic of the clonic phase, diminishing the risk of myocardial ischemia and ventricular ectopy as well as suppressing the emergence of ectopic impulses from stretched ventricular fibers at high left ventricular end-diastolic pressures.

In summary, this case illustrates the undesirable anticonvulsant effect of iv lidocaine when used for prophylaxis against ventricular arrhythmias during ECT. Our case illustrates the efficacy of intravenous propranolol pretreatment as alternative therapy.

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