conclusion of neurologic injury when changes in upper-extremity SEPs are caused by subdural gas. Intraoperative skull roentgenograms obtained in response to unexplained changes in the upper-extremity SEPs affirmed the presence of subdural gas. This simple technology is readily available. Since the upper- and lower-extremity SEPs are transmitted in the medial lemniscus of the brainstem, it is unlikely that one portion of the lemniscus would be affected profoundly by brainstem retraction and the adjacent lamellae spared.

Whether SEP changes associated with subdural gas are due to an insulator effect caused by the gas interface or whether they are a result of injury from tension pneumocephalus is difficult to ascertain. We have previously shown that patients who become symptomatic from tension pneumocephalus do so a variable time after surgery. Since SEP amplitude changed prior to dural closure in this setting, injury from pneumocephalus is less likely to account for the SEP change.

In conclusion, we believe that subdural gas can accumulate and alter upper-extremity SEPs in patients in the sitting position. Preservation of lower-extremity SEPs in the presence of such subdural air affirms the benignity of this condition. Intraoperative skull radiography can demonstrate subdural gas under the parietal electrodes and obviate a potentially dangerous alteration in standard surgical technique.

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Prevention of Electroconvulsive Therapy-Induced Dysrhythmias with Atropine and Propranolol

STEPHEN W. LONDON, M.D.,* AND D. DAVID GLASS, M.D.†

Prophylactic antidysrhythmia treatment for electroconvulsive therapy (ECT) should inhibit adverse alterations in cardiac rhythm while not interfering with the therapeutic efficacy of the seizure. While atropine sulfate fulfills these requirements and is routinely used as a premedication for ECT, ventricular dysrhythmias frequently develop in patients at risk despite atropine pretreatment. Although lidocaine inhibits ventricular ectopy associated with ECT, the seizure duration is reduced on a dose-related basis, thereby decreasing the therapeutic efficacy of ECT. Intravenously administered propranolol suppressed ventricular dysrhythmias during ECT in a patient who had previous seizure inhibition associated with lidocaine pretreatment, but in our case described below, it failed to prevent severe bradycardia with ventricular premature contractions (VPCs), bigeminy, and coupling of ectopic beats. The combined pretreatment regimen of atropine with propranolol appears to be a logical and effective approach.

REPORT OF A CASE

A 61-year-old man was scheduled to receive a series of ECT treatments for major depression resistant to maximal medical therapy.

REFERENCES


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Medical history was significant for chronic hypertension that was controlled with diuretic therapy. ECG revealed normal sinus rhythm with first-degree atrioventricular block as well as a nonspecific intraventricular conduction defect. Chest roentgenogram was without any evidence of acute disease. Thyroid function tests were within normal limits. Serum electrolytes were normal at the time of initiation of ECT.

The patient received general anesthesia for each of the ECT treatments with continuous ECG, EEG, and arterial blood pressure monitoring. Ventilation was assisted or controlled via a face mask and 100% oxygen during apnea, seizure, and in the postictal period, using an oral airway as needed. For the first five treatments, atropine 0.4 mg was given iv, followed by mexibesital 80 mg iv and succinylcholine 60–80 mg iv. Seizures with a duration of 55 to 70 s were elicited by a unilateral stimulus in the 16–45-watt-s range. During the second and third treatments, an occasional VPC was noted in the postictal period. In the immediate poststimulus period of the sixth treatment, a series of multiple unifocal VPCs occurred that were successfully treated with 100 mg lidocaine iv. To avoid the emergence of VPCs during the subsequent three treatments, 100 mg lidocaine was administered iv several minutes prior to induction of anesthesia. However, the attempts at ECT failed to elicit significant seizure activity, and no cardiac dysrhythmias were noted.

For the tenth treatment, pretreatment with both lidocaine and atropine were withheld, but the patient received propranolol 0.5 mg iv prior to induction of anesthesia. The electrical stimulation was followed immediately by a brief run of bigeminy, which converted spontaneously to the typical postshock sinus tachycardia. Near the end of the 1 min of seizure activity, however, degeneration to a sinus bradycardia of 36 bpm with ventricular bigeminy and coupled VPCs was observed. With the administration of atropine 0.5 mg iv as well as continued oxygenation via face mask, the bradydysrhythmia improved and the ectopic beats became less frequent, clearing completely over the next 2 min.

For the eleventh ECT treatment, the patient was pretreated with propranolol 0.5 mg and atropine 0.4 mg iv. Ninety seconds after administration of succinylcholine, unifocal VPCs developed with some regularity, which resolved after an additional 0.4 mg atropine. The seizure stimulus was followed by sinus tachycardia without

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**Fig. 1.** Simultaneous ECG and EEG recordings from ECT treatments no. 7, 10, and 11, respectively. Chart speed: 10 mm/s.
ventricular ectopy. For the twelfth treatment the patient was pretreated with propranolol 0.5 mg and atropine 0.8 mg iv. Rare unifocal VPCs were observed, but significant bradycardia and bigeminy were avoided. Duration of the seizure for the two treatments in which propranolol and atropine were administered was in the therapeutic range. Figure 1 depicts representative tracings from the seventh, tenth, and eleventh treatments. A summary of the antidyssrhythmic pretreatment drug and dose, seizure stimulus and duration, and subsequent cardiac response for each ECT treatment is presented in Table 1.

### Table 1. Summary of the 12 ECT Treatments Described in the Text

<table>
<thead>
<tr>
<th>ECT</th>
<th>Pretreatment</th>
<th>Stimulus</th>
<th>Seizure Duration (s)</th>
<th>Cardiac Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 5</td>
<td>Atropine 0.4 mg</td>
<td>U</td>
<td>55 to 70</td>
<td>Sinus tachycardia with few unifocal VPCs</td>
</tr>
<tr>
<td>6</td>
<td>Atropine 0.4 mg</td>
<td>U</td>
<td>72</td>
<td>Sinus tachycardia with multiple VPCs</td>
</tr>
<tr>
<td>7</td>
<td>Atropine 0.4 mg</td>
<td>U (2)</td>
<td>No seizure</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>8</td>
<td>Lidocaine 100 mg</td>
<td>U (2)</td>
<td>12</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>9</td>
<td>Atropine 0.5 mg</td>
<td>B</td>
<td>10</td>
<td>Mild sinus bradycardia</td>
</tr>
<tr>
<td>10</td>
<td>Lidocaine 100 mg</td>
<td>B</td>
<td>60</td>
<td>Bigeminy, sinus tachycardia, severe sinus bradycardia with bigeminy and coupled VPCs</td>
</tr>
<tr>
<td></td>
<td>Propranolol 0.5 mg</td>
<td>B</td>
<td>75</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>11</td>
<td>Atropine 0.8 mg</td>
<td>B</td>
<td>55</td>
<td>Sinus tachycardia with few unifocal VPCs</td>
</tr>
<tr>
<td>12</td>
<td>Propranolol 0.5 mg</td>
<td>B</td>
<td>75</td>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td></td>
<td>Atropine 0.8 mg</td>
<td>B</td>
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<td>Sinus tachycardia</td>
</tr>
<tr>
<td></td>
<td>Propranolol 0.5 mg</td>
<td>B</td>
<td>75</td>
<td>Sinus tachycardia</td>
</tr>
</tbody>
</table>

### Discussion

Cardiac dysrhythmias in association with ECT are common, occurring with especially great frequency in patients with organic heart disease. Although dysrhythmias are usually self-limited after ECT, they have been associated with serious ventricular dysrhythmias and death. The etiology of these dysrhythmias is multifactorial but centers around the complex interplay of parasympathetic and sympathetic dysrhythmias associated with the tonic and clonic phases of seizure activity, respectively, as well as their variable effects in the postictal period. Hypoxia, hypercapnia, medications administered prior to ECT, and anesthetic induction agents play a role as well. Methods of dysrhythmia prevention have included preoxygenation followed by controlled ventilation during the seizure, the use of methohexital as the anesthetic induction agent, and pretreatment with antidyssrhythmic agents.

Of these agents, anticholinergics can prevent the profound bradycardia or occasional cardiac standstill associated with the strong surge of vagal tone produced by electrical stimulation. Large doses of atropine (i.e., 2.4 mg iv) prior to the ECT treatment have been recommended, yet such therapy subsequently may enhance the sympathetic-related dysrhythmias seen in the clonic phase of the seizure. Lidocaine has proved effective in preventing and treating the ventricular dysrhythmias associated with ECT that are unaffected by atropine therapy. However, its anticonvulsant activity shorts the electrically induced seizure activity without raising seizure threshold. Although improvement per total seconds of seizure discharge is not altered, improvement per treatment is reduced. Topical nitroglycerine ointment attenuates the hypertension and tachycardia seen with multiple single-session ECT, but its effect on prevention of ventricular dysrhythmias is unstudied. Thus, effective alternative pretreatment would allow seizure activity of therapeutic duration to occur concomitant with protection against dysrhythmias.

In this regard, pretreatment with propranolol 0.5 mg iv is an effective ventricular antidyssrhythmic associated with ECT presumably by attenuating sympathetic stimulation seen in the clonic phase of seizure activity. The development of bradycardia was not observed in any of the 10 ECT treatments described in that report in which propranolol alone was administered prophylactically. In our case, however, the ECT treatment in which propranolol alone was given as pretreatment was followed by significant sinus bradycardia with coupled VPCs and bigeminy. Presumably, the etiology of this rhythm was the parasympathetic stimulation that was left uninhibited by omission of atropine pretreatment. In fact, the dysrhythmia cleared with administration of iv atropine. For the final two treatments the combination of propranolol with atropine was used prophylactically in order to dampen the effects of the simultaneous sympathetic and parasympathetic activation associated with ECT. This regimen effectively prevented the vagal bradycardia and subsequent ventricular dysrhythmias seen with propranolol pretreatment alone and allowed seizures of therapeutic duration to occur.

In summary, this case illustrated the undesired effects of both lidocaine (diminished seizure duration) and propranolol (intense bradycardia) when given individually for the prevention of ventricular dysrhythmias during ECT. The combination of intravenous propranolol and
atropine is shown to be an effective pretreatment therapy in this setting.

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Transient Decreases in Respiratory Rate Following Epidural Injections

DAVID GISEN, M.D.,* AND DAVID E. LEITH, M.D.†

Pressure-volume relationships in the spinal space and the interactions between the intracranial and spinal compartments rarely have been studied. While performing epidural anesthesia for routine orthopedic procedures, we were recording epidural pressure. In one patient we noted a decrease in respiratory rate following epidural injection of local anesthetic. We describe our observations on three patients and discuss possible mechanisms for this respiratory response to an epidural injection.

REPORT OF A CASE

Three patients, ASA physical status III, with severe rheumatoid arthritis, were given epidural anesthesia for elective lower-extremity total joint replacement. All were older than 60 years of age and had multiple joint disease. Oral diazepam and morphine sulfate were given as premedication. In the operating suite, a central venous line was inserted through the right internal jugular vein and diazepam and morphine sulfate given iv until each patient was comfortably sedated. The patients then were placed in the lateral position and a lumbar epidural catheter inserted using a 17-gauge Tuohy needle and the hanging drop technique. The catheter was advanced 2 cm beyond the tip of the needle. Lidocaine 1.5% with 1:200,000 epinephrine was then given through the catheter in divided doses. All patients received 3 ml at a time for a total of 12 ml as an induction dose. The patients were positioned and surgery begun. Ninety minutes after the initial dose and at 60-min intervals thereafter, each patient received two additional 3-ml doses of 0.5% bupivacaine. Each 3-ml dose was injected over 9 s and repeated when epidural catheter pressure returned to within 30% of preinjection pressure. This happened within 1.5 min.

The anesthetics were uneventful. All patients had adequate levels of anesthesia and were awake and breathing spontaneously throughout the procedure. After a short stay in the recovery room, each patient was returned to the ward.

Epidural and central venous catheter pressures were measured continuously using Gould® transducers (P23) and a recorder. The epidural catheter was connected to a 10-ml syringe and transducer via a four-way stopcock (fig. 1). The epidural catheter was connected to the needle; during injection the stopcock was opened to include the syringe in the system. We analyzed changes in epidural and central venous pressure using the recorded traces. Respiratory rate was noted as periodic decrements in the central venous pressure of about 3 cmH₂O.

RESULTS

Patient 1 showed no measurable decrement in respiratory rate with the first additional 3-ml dose. However, with the second 3-ml dose, respiratory rate changed from 16.5 breaths/min to 10.25 breaths/min, a decrease

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