Midazolam as Intravenous Sedative for Electrocadioversion*

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Of the various agents which have been employed for sedation in patients undergoing electrocardioversion, diazepam has had the most extensive use. However, this agent possesses several disadvantages including pain and venous complications at the site of injection and a lower incidence of amnesia. Midazolam, a benzodiazepine derivative, is being increasingly used in general and local anesthesia as well as for procedures requiring conscious sedation, eg, endoscopy. We used intravenous midazolam for conscious sedation in 12 patients undergoing a total of 17 cardioversions. All of the patients experienced amnesia for the procedure and manifested no significant adverse effects. However, serious respiratory failure may occur when intravenous midazolam is used in patients with COPD, debilitated patients, or when the drug is injected rapidly. The use of midazolam should therefore be confined to areas that are able to deal with cardiorespiratory complications. Using guidelines and precautions described here, we encountered no major complications. We conclude that midazolam offers a safe and effective alternative to other agents for conscious sedation in patients undergoing electrocardioversion.

(Electrical cardioversion is commonly employed for the management of supraventricular tachyarrhythmias as well as ventricular tachycardia. Diazepam has been successfully used for conscious sedation to facilitate cardioversion.1-3 Midazolam is another benzodiazepine which is being increasingly preferred over diazepam for procedures requiring conscious sedation such as endoscopy, dental procedures, and regional anesthesia.4-8 Advantages of midazolam over diazepam include a rapid onset of action,5,8 lack of pain and venous complications at injection site5,8 and a greater amnestic effect.5,6,9 We describe the use of intravenous midazolam in the setting of transthoracic electrical cardioversion.

METHOD

Twelve patients underwent elective electrocardioversion in accordance with standard protocol at The Memorial Hospital of Rhode Island. This protocol consists of obtaining informed consent from the patient, discontinuation of digoxin therapy for at least 48 hours, ensuring normal values of electrolytes and ensuring adequate anticoagulation for at least two weeks prior to cardioversion (in those without contraindications to anticoagulation). Deviation from the protocol is allowed if the situation is emergent or semiemergent, eg, ventricular tachycardia, hemodynamically unstable supraventricular tachyarrhythmias. Cardioversion is carried out in the coronary care unit by or under the supervision of a cardiologist and an anesthesiologist. Patients routinely receive low flow (2 L/min) oxygen by nasal canula prior to and following cardioversion. For the patients reported here, midazolam, 2 mg, was initially administered intravenously over a period of two to four minutes. Subsequently, it was administered intravenously at intervals of two to four minutes in 1 mg increments until the patient developed slurred speech and was not easily arousable by verbal and physical stimuli. Vital signs were monitored constantly before and following cardioversion until they were stable and the patient was fully alert. Blood pressure was monitored with an automated recorder every two minutes until stable. Indications for cardioversion included atrial fibrillation, atrial flutter, and hemodynamically stable ventricular tachycardia. Table 1 lists the characteristics of these patients. The 12 patients underwent a total of 17 cardioversions, with some patients requiring more than one cardioversion on separate occasions because of recurrence of arrhythmia. At each cardioversion attempt, serial shocks using higher energy levels were utilized, if lower energies failed to convert the arrhythmia. Details are shown in Table 2. Statistical analysis was performed by the paired Student's t-test. Results were considered significant when p values were less than 0.05.

Table 1—Patient Characteristics and Diagnoses

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Arrhythmia*</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65/M</td>
<td>A Flutt</td>
<td>Hypertensive heart disease; congestive heart failure</td>
</tr>
<tr>
<td>2</td>
<td>73/M</td>
<td>A Fib</td>
<td>Abdominal aortic aneurysm; congestive heart failure</td>
</tr>
<tr>
<td>3</td>
<td>57/F</td>
<td>V Tach</td>
<td>Idiopathic congestive cardiomyopathy</td>
</tr>
<tr>
<td>4</td>
<td>61/F</td>
<td>A Fib</td>
<td>Hypertension; superior vena cava syndrome</td>
</tr>
<tr>
<td>5</td>
<td>70/M</td>
<td>A Fib</td>
<td>Cerebral vascular accident</td>
</tr>
<tr>
<td>6</td>
<td>74/F</td>
<td>A Flutt</td>
<td>Coronary artery disease; diabetes mellitus</td>
</tr>
<tr>
<td>7</td>
<td>64/F</td>
<td>A Flutt</td>
<td>Hypertensive heart disease; chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>8</td>
<td>55/M</td>
<td>A Fib</td>
<td>Hypertensive heart disease</td>
</tr>
<tr>
<td>9</td>
<td>52/M</td>
<td>A Fib</td>
<td>Congenital aortic incompetence</td>
</tr>
<tr>
<td>10</td>
<td>61/M</td>
<td>A Fib</td>
<td>Hypertension; diabetes mellitus</td>
</tr>
<tr>
<td>11</td>
<td>67/F</td>
<td>V Tach</td>
<td>Chronic renal failure</td>
</tr>
<tr>
<td>12</td>
<td>55/M</td>
<td>A Fib</td>
<td>Coronary artery disease</td>
</tr>
</tbody>
</table>

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### Results

Cardioversion with return to normal sinus rhythm was achieved in 12 out of 17 procedures. Patients who remained in atrial fibrillation or flutter were maintained on medical regimen aimed at controlling the ventricular rate. Table 2 lists parameters recorded prior to and following cardioversion, dosages of midazolam used, and details of cardioversion.

#### Amnesia

Upon regaining full consciousness, patients were asked if they had any recollection of having undergone cardioversion. Except for patient 9, all had total amnesia for the procedure. Patient 9, who had undergone cardioversion using diazepam sedation on two previous occasions, had full memory of severe discomfort from prior cardioversions.

#### Blood Pressure

As shown in Figure 1 and Table 2, a transient mild drop in systolic blood pressure was noted in some patients but none required a vasopressor or intravenous fluid volume support. A few patients manifested transient rise in blood pressure following cardioversion possibly related to enhanced adrenergic activity following cardioversion. As noted in Figure 1 and Table 2,
2, values for systolic blood pressures did not change significantly before and after midazolam administration.

**Respirations**

Respiratory status of each patient was monitored clinically until the patient had regained consciousness. There was a decline in the respiratory rate following midazolam treatment (postcardioversion) ranging from 0 to 8 from the baseline value prior to midazolam administration. Although this decline in respiratory rate was significant (p<0.0039), none of the patients, including the one with the lowest recorded rate of 12/min, manifested any clinical evidence of respiratory distress requiring assisted ventilation. All patients received low flow oxygen, as noted earlier, as part of the protocol for cardioversion.

**Recovery**

The majority of patients regained full consciousness spontaneously by one hour and all by two hours. With the exception of one patient who experienced transient diplopia, no neurologic deficit was evident when patients became fully alert.

**Dose of Midazolam**

The intravenous dose of midazolam for the 17 procedures ranged from 2.5 to 16 mg (average 6.6 mg).

**DISCUSSION**

Diazepam has been used successfully in the past for conscious sedation and antegrade amnesia in patients undergoing electrical cardioversion. However, diazepam appears to have certain drawbacks. It possesses active metabolites with a long half-life which may cause prolonged sedation, an undesirable feature for brief procedures such as cardioversion. Additionally, the incidence of nonamnesia with diazepam in cardioversion has been reported to be up to 37 percent. Midazolam possesses a shorter half-life of 2.5 hours and its metabolites are pharmacologically inactive. In addition, midazolam has a more rapid onset of action, a property attributed to its enhanced lipophilicity at a physiologic pH of 7.4. Rapid onset of action and short duration makes midazolam an attractive choice for brief procedures such as cardioversion. Unlike diazepam, parenteral formulation of midazolam excludes propylene glycol which renders it less irritant and therefore less liable to cause pain at the site of injection and lesser chance of subsequent venous sequelae compared to diazepam. Our 12 patients who underwent a total of 17 cardioversions showed a favorable response to midazolam with no significant adverse effects.

None of our patients, with one exception, had any recollection of undergoing cardioversion. The sole exception was a patient who remembered undergoing cardioversion but had no recollection of pain or discomfort. In contrast, the same patient had a vivid recollection of pain and discomfort associated with at least two cardioversions carried out under diazepam sedation. Although our study was not meant to compare midazolam with diazepam, previous studies have indicated that midazolam is more potent in producing antegrade amnesia compared to diazepam.

Some of the major concerns of intravenous sedation relate to its effects on respiration and blood pressure. As shown in Table 2, almost an equal number of cardioversion attempts was associated with transient hypotension or hypertension. Patients who experienced a transient drop in blood pressure did not require support in the form of vasopressors or volume expansion, as they had an adequate palpable pulse and the hypotension was very brief, ie, minutes. Transient hypertension in patients given midazolam could be related to enhanced adrenergic activity in response to pain of local injection, or in our cases, following delivery of electrical therapy. Another possible reason or contributing factor for increase in blood pressure could be related to improvement in cardiac hemodynamics from normalization of rhythm.

As mentioned earlier and as indicated in Table 2, some of our patients had a decline in respiratory rate, although none required pharmacologic or mechanical ventilatory support. Although significant respiratory depression was not encountered in healthy volunteers given intravenous midazolam for conscious sedation, clinically significant respiratory depression may occur in patients with COPD, in those receiving concomitant CNS depressant medication, and those who are older and debilitated. In addition, rapid injection has been associated with severe respiratory depression and should therefore be avoided. In view of the remote but real danger of severe respiratory depression, the use of midazolam should be confined to areas equipped with cardiopulmonary resuscitation facilities and under the supervision of an anesthesiologist or a physician adequately trained in handling cardiopulmonary emergencies. In addition, it may be prudent to monitor respiratory status more objectively using, for example, ear oximetry.

We exercised the aforementioned precautions and guidelines and did not encounter clinically significant cardiorespiratory complications in our patients with various diagnoses (Table 1), even in those requiring larger than the recommended 0.1 mg/kg dose. We therefore favor the use of midazolam for conscious sedation in patients undergoing cardioversion. However, appropriate care should be exercised in using this agent in order to prevent the remote but real danger of side effects, especially respiratory complications.
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