tight fit and a soft plastic coating prevents the stylet from being removed easily from the endotracheal tube. When the endotracheal tube is held firmly, pressure on the wall of the tube traps the stylet and makes it difficult to remove. The natural response is to hold the endotracheal tube more firmly to prevent its dislodgment, which makes the stylet even more difficult to remove. A vicious cycle results. In our case, the “pinching” of the endotracheal tube by the pediatric resident and the vigorous tugging by the assistant caused the pliable, nonbonded coating to be sheared from the tip of the stylet. It was fortunate that the tip remained in the endotracheal tube, where it was quickly discovered, and did not become lodged in the newborn’s trachea or bronchi, where greater harm could have resulted.

Use of the 6-French Satin Slip® intubating stylet with a 2.5-mm endotracheal tube can be hazardous. In addition to the problem we encountered, accidental extubations have resulted from the manipulations required to remove this stylet. Thin, pliable metal stylets with beaded tips are available and work well with very small endotracheal tubes. If stylets with unbonded coatings are used, a loose fit should be assured and the risk of shearing must be kept in mind.

REFERENCES


Comparison of High-dose Epinephrine and Phenylephrine in Spinal Anesthesia with Tetracaine

CRAIG CALDWELL, M.D.,* CARL NIELSEN, M.D.,† TIMOTHY BALTZ, M.D.,†
PETER TAYLOR, M.D.,† BETTY HELTON, M.D.,† PATRICK BUTLER, M.D.†

Epinephrine and phenylephrine have been added to local anesthetics to prolong the duration of spinal anesthesia for many years. In general, most authors have reported that phenylephrine prolongs sensory anesthesia and motor blockade to a greater degree than epinephrine. Unfortunately, the absence of controlled studies and variations in vasoconstrictor doses has resulted in the reporting of wide variations in the efficacy of both drugs.1-5 Recently, Concepcion et al.5 carried out a comparison of the effects of “equipotent,” low doses of epinephrine and phenylephrine on tetracaine spinal anesthesia. The results of that study demonstrated that there was no statistical or clinically significant difference between the vasoconstrictors in the regression of sensory or motor block. In light of this observation, we decided to determine whether the effects of epinephrine and phenylephrine differed in higher equipotent doses. We felt this was particularly relevant in view of recent evidence suggesting that α-adrenergic agonists have direct antinociceptive effects on the spinal cord and that vasoconstriction may not completely explain the mechanism by which α-agonists prolong spinal anesthesia.6,7

METHODS

Thirty ASA class I or II patients scheduled for lower extremity surgery, prostate resection, vaginal hysterectomy, or inguinal hernia repair consented to participate in a study that was approved by our institutional review committee. The patients were randomly assigned to one of three groups. All patients received 2 ml of a 5% dextrose solution containing 10 mg of tetracaine. Patients in Group 1 did not have vasoconstrictor added to their anesthetic. Patients in Groups 2 and 3 had 0.5 mg epinephrine or 5 mg phenylephrine added to their local anesthetic, respectively. Premedication consisted of diazepam 10 mg orally or morphine 0.1 mg/kg im. Lactated Ringer’s solution was infused during induction of anesthesia to minimize the occurrence of hypotension.
Blood pressure was measured by auscultation or with an oscillometric device (Dinamap®) and lead 2 of the ECG was monitored to determine pulse rate.

After obtaining baseline measurements of blood pressure and pulse rate, the patients were placed in the lateral position. Lumbar puncture was performed at the L3-4 interspace with a 22-gauge needle. Aspiration of clear cerebrospinal fluid (CSF) confirmed needle placement in the subarachnoid space. The anesthetic solution was injected over a 30-s period by an anesthesiologist—observer who was unaware of the composition of the solution. Immediately after injection, the patient was turned into the supine position.

The level of sensory anesthesia was tested by the anesthesiologist–observer by the patient’s response to pinprick. Motor block was assessed using a 0–3+ scale described by Rocco et al. The level of sensory anesthesia and motor block were determined every 2 min after injection for the first 30 min, and at 15-min intervals thereafter until the level of sensory anesthesia had regressed to L1 and all signs of motor block had disappeared. The time required to obtain the maximal segmental level of anesthesia was recorded. Onset of anesthesia was measured by recording the time required to obtain a T10 sensory level. Duration of anesthesia was determined by recording the time required for regression to T10 and L1 sensory levels. The time required to achieve complete motor blockade as well as the time required for complete recovery of motor function were recorded. Blood pressure and heart rate were recorded every 2 min for the first 30 min, and then at 5-min intervals intraoperatively and at 15-min intervals postoperatively.

Episodes of hypotension, characterized by more than a 30% decrease in systolic pressure below baseline values, were treated with incremental doses of 5–10 mg of ephedrine if the patient became symptomatic. Bradycardia, characterized by a heart rate of less than 60 beats/min, was treated with atropine if the patient was hypotensive.

Data were analyzed using one-way analysis of variance and Student’s t test with Bonferroni’s correction for multiple comparisons. A value of $P < 0.05$ was taken as indicating statistical significance.

RESULTS

Patient characteristics are presented in table 1. There were no significant differences with regard to patient age, weight, and height between the three groups.

Onset. No significant difference was found in the time of onset of anesthesia between the three groups. No significant difference was found in the time required to attain the maximum level of sensory anesthesia, and no difference was found in the total number of spinal segments blocked in the three groups. No significant difference was observed in the time of onset of complete motor blockade between the three groups (table 2).

Regression. The duration of sensory anesthesia to the T10 and L1 dermatomes was significantly shorter in Group 1 compared with Group 2 ($P < 0.01$) and Group 3 ($P < 0.01$). The duration of sensory anesthesia to the L1 dermatome was significantly shorter in Group 2 compared with Group 3 ($P < 0.025$). No significant difference was observed between Groups 2 and 3 in the duration of anesthesia to the T10 dermatome. Addition of epinephrine to the block solution prolonged sensory anesthesia to T10 and L1 by 47% and 43%, respectively. Addition of phenylephrine prolonged anesthesia by 72% and 77%, respectively (table 3).

The duration of motor blockade was significantly shorter in Group 1 compared with Group 2 ($P < 0.01$) and Group 3 ($P < 0.01$). No difference was observed between Groups 2 and 3. Motor blockade was prolonged 67% and 72% by the addition of epinephrine and phenylephrine, respectively (table 3).

DISCUSSION

Current anesthesia texts suggest that in optimal doses, epinephrine and phenylephrine prolong spinal anesthesia by approximately 50% and 100%, respectively. The optimal doses recommended are 0.2 mg for epinephrine and 2–5 mg for phenylephrine. However, the absence of controlled studies and the great variation in local

<table>
<thead>
<tr>
<th>Table 1. Patient Characteristics</th>
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<tr>
<td><strong>Group</strong></td>
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<tr>
<td>Tetracaine</td>
</tr>
<tr>
<td>Tetracaine plus epinephrine</td>
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<td>Tetracaine plus phenylephrine</td>
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Mean ± SD.

<table>
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<th>Table 2. Onset Times</th>
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<td><strong>Group</strong></td>
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<tr>
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<td>Tetracaine plus phenylephrine</td>
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Mean ± SD.
anesthetic and vasoconstrictor doses found in the literature makes it difficult to confirm the validity of these recommendations with certainty.

Recently, Concepcion et al.5 reported the results of a double-blind comparison of the effects of epinephrine (0.2 and 0.3 mg) and phenylephrine (1 and 2 mg) on the duration of tetracaine spinal anesthesia. While both drugs significantly prolonged the duration of sensory and motor block compared with the effect of plain tetracaine, no significant difference was found between the degree of prolongation caused by the vasoconstrictors. Unlike others who have studied these drugs,2,4,6 the authors concluded that in “equivalent” or “equi-potent” doses (1 mg epinephrine = 10 mg phenylephrine) the effects of epinephrine and phenylephrine are not different. Thus, according to this view, dose, and not the choice of a particular drug, may be the critical factor in determining the degree of prolongation.

In this study we have shown that the use of 0.5 mg epinephrine or 5 mg phenylephrine with tetracaine spinal anesthesia results in a significant prolongation of sensory anesthesia and motor blockade. These findings are in agreement with previous studies.1–8 In addition, we have shown that phenylephrine causes a significantly greater prolongation of the time of regression of sensory anesthesia to L1 than epinephrine under the conditions of this study. Given these results, we must take issue with the conclusions of Concepcion et al.5 Our data indicate that phenylephrine prolongs the duration of sensory anesthesia to a greater extent than epinephrine in higher, clinically recommended doses with tetracaine spinal anesthesia. The results also suggest that it is the interaction of the particular vasoconstrictor and the dose administered that determines the extent of prolongation.

We believe the apparent discrepancies between the conclusions drawn from this study and those of Concepcion et al. can be explained. The classic explanation for the prolongation of spinal anesthesia by epinephrine and phenylephrine is that α-adrenergic mediated vasoconstriction decreases the vascular uptake of local anesthetic. However, this mechanism may only partially explain how α-adrenergic agonists prolong spinal anesthesia. Early work by Converse et al.7 demonstrated that epinephrine does not inhibit the washout of tetracaine from cerebrospinal fluid. Recent work by Collins et al.,8 using epinephrine doses comparable to those used in human patients, and Yaksh and Reddy9 suggest that α-adrenergic agonists have direct, dose-dependent effects on the spinal cord that modulate the central transmission of pain information. Concepcion et al.5 imply that equipotent vasoconstrictor doses should produce indistinguishable effects. However, if direct α-adrenergic effects play a role in prolonging spinal anesthesia, the relative vasoconstricting potencies of epinephrine and phenylephrine may not correlate with their spinal cord effects. Therefore, while the doses used by Concepcion et al.5 may represent roughly equivalent anesthetic doses, our data suggest that the 1:10 potency ratio does not extend to higher doses.

In summary, we have demonstrated that phenylephrine significantly prolongs sensory anesthesia to a greater extent than epinephrine in higher, clinically useful1,2,6 doses with tetracaine spinal anesthesia. This information may be useful in deciding whether to choose epinephrine or phenylephrine when planning spinal anesthesia for various surgical procedures on the lower extremities.

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The following table shows the regression times for different groups:

<table>
<thead>
<tr>
<th>Group</th>
<th>T10 (min)</th>
<th>L1 (min)</th>
<th>Motor Block (min)</th>
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</thead>
<tbody>
<tr>
<td>Tetracaine</td>
<td>159.0 ± 41.3</td>
<td>229.5 ± 54.8</td>
<td>234.0 ± 67.9</td>
</tr>
<tr>
<td>Tetracaine plus epinephrine</td>
<td>234.0 ± 59.2*</td>
<td>327.0 ± 71.7*</td>
<td>391.5 ± 75.3*</td>
</tr>
<tr>
<td>Tetracaine plus phenylephrine</td>
<td>275.0 ± 89.7*</td>
<td>406.5 ± 62.0*†</td>
<td>403.5 ± 66.1*†</td>
</tr>
</tbody>
</table>

Mean ± SD.
* Significantly different from Group 1; P < 0.01.
† Group 3 significantly different from Group 2; P < 0.025.
Transitory Global Amnesia Following General Anesthesia

THOMAS WOOD, D.O.,* AND JUDITH DONEGAN, M.D., PH.D.†

Transient global amnesia (TGA) is characterized by sudden, transient loss of memory for recent events with preservation of personal identity, immediate recall, and remote memory.1,2 TGA usually occurs in middle-aged or older patients3 and can last for minutes to hours but no longer than 1 day.3 Other neurologic deficits are absent,2 the electroencephalogram (EEG) usually although not always remains unchanged,4 and no systemic abnormalities are detected. The cause of the disorder is believed to be episodic vascular insufficiency of the medial temporal lobes.5 TGA has been associated with migraine headaches,5,6 intracerebral tumor,7 diazepam overdose,5 and cardiac dysrythmias,8 and has been described after cerebral angiography9 and spinal anesthesia.10 Following is the description of a patient who exhibited the characteristic signs and symptoms of TGA for several hours following a general anesthetic for a neurosurgical procedure.

REPORT OF A CASE

The patient is a 60-year-old man with trigeminal neuralgia involving primarily the distribution of the maxillary portion of the left trigeminal nerve. He had received medical therapy and underwent a microvascular decompression of the fifth cranial nerve 2 years previously, but neither provided permanent pain relief. He received general anesthesia for his previous surgery, and his postoperative course was reportedly uncomplicated. A repeat microvascular decompression was planned for this hospitalization.

His history included hypertension well controlled by diuretic therapy, asymptomatic peptic ulcer disease treated with cimetidine, and a 20 pack year smoking history. An old inferior wall myocardial infarction and first degree atrioventricular block were present. Complete blood count, serum electrolytes, and urinalysis were within normal limits.

The patient received diazepam 10 mg po with his usual morning medications including carbamazepine, Dyazide®, and cimetidine 1.5 h prior to transport to the operating room. A peripheral intravenous catheter, a radial arterial line, and a central venous line were inserted prior to induction of anesthesia; correct placement of the tip of the central venous catheter was confirmed by chest roentgenogram.

Anesthesia was induced with thiopental 275 mg and fentanyl 150 µg iv with an Fio2 of 1.0. Intubation of the trachea was performed following atracurium 0.5 mg/kg iv. The patient’s legs were wrapped with elastic bandages, and the head of the table gradually elevated to 80 degrees. The patient’s neck was slightly flexed, care being taken to ensure that the airway as well as the cerebral venous pathways remained unobstructed. After the patient was placed in the seated position, a precordial Doppler transducer was applied and the accuracy of position checked by injecting 5 ml of saline through the central venous catheter. A left subclavian craniectomy and microvascular decompression of the left trigeminal nerve were performed.

Anesthetic maintenance for the 4.5 h case included 50% nitrous oxide and 1% inspired isoflurane. End-tidal carbon dioxide was maintained at 23–26 mmHg until closure of the dura was begun; this corresponded to a Petco2 of 29–32 mmHg. Repeated intraoperative analysis of arterial blood gases revealed adequate oxygenation. Vital signs remained stable with normotension and sinus rhythm throughout surgery, and urine output was approximately 1 ml·kg⁻¹·hr⁻¹. No surgical complications were encountered, and blood loss was minimal. At the conclusion of surgery, adequate reversal of neuromuscular...