The Components of an Effective Test Dose Prior to Epidural Block

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Following placement of the needle or plastic tubing for an epidural block via either a lumbar interspace or the sacral hiatus, the standard procedure is to aspirate for blood or spinal fluid. When neither is obtained, other tests to determine whether an intravascular or a subarachnoid injection could result have become optional.1-3

The purpose of this study was to determine the components of a single test dose of a local anesthetic solution which, within two minutes from its injection, produces clinical evidence that the needle has penetrated either a blood vessel or the dura.

METHODO

This study was approved by our local Committee on Human Research, and each patient's written consent was obtained. Of the 225 adults in the study, 10 were volunteers who received no premedication. Each volunteer was given 3 ml saline with 1:200,000 epinephrine (0.015 mg) intravenously at the rate of 1 ml/s. Every 15 s for three minutes after the injection, heart rate was monitored by palpation of the radial artery and arterial blood pressure by sphygmomanometry. Then the volunteer was observed for another 10 min.

The remaining 215 patients received 3 ml of one of the following solutions intravenously at the rate of 1 ml/s prior to an epidural block for surgery. The solutions were 0.75 per cent bupivacaine (22.5 mg), 1.5 per cent mepivacaine (45 mg), 1.5 per cent lidocaine (45 mg), and 3.0 per cent chloroprocaine (90 mg). These dosages when injected subarachnoidally produce evidence of spinal anesthesia in two minutes.4-5 Forty of these patients were not medicated, and they received local anesthetic solutions with no epinephrine (N = 10 for each drug). The remaining 175 patients received epinephrine 1:200,000 (0.015 mg) along with the local anesthetic solution. Fifteen minutes prior to the injection, they were medicated with 0.1 mg intravenous fentanyl and 10–15 mg diazepam. They were divided into groups of 25. Each group was given a different preparation of the local anesthetic drug so as to simulate as closely as possible the solutions.
employed for epidural blocks not only by us, but by other anesthesiologists (table 1).

The 3 ml of local anesthetic solution injected intravenously was that which was to be used for the therapeutic dose (the dose needed to establish the required analgesia). The injection was made, unknown to the patient, into the port in the unclamped intravenous tubing closest to the plastic catheter needle. The 40 patients who received no epinephrine were monitored the same as the volunteers. The 175 patients who received epinephrine were monitored by an electrocardioscope with a heart rate indicator, and questioned when their heart rates increased. An electrocardiogram was obtained prior to the intravenous injection. Upon completion of the injection, the electrocardiograph was immediately started, and was continued until the increased heart rate slowed and approximated the control rate.

In some of the 175 patients the electrocardioscope indicated arrhythmias other than tachycardia of short duration following the intravenous injection, which terminated when the heart rate returned to the control rate. However, because our attention was focused on the heart rates, these arrhythmias could not be evaluated in a controlled manner. Therefore, in an attempt to determine whether the epinephrine, the local anesthetic drug, or both was the etiology, 30 additional surgical patients, premedicated and monitored as the 175 surgical patients, were given 3 ml intravenously of one of the following: 1) epinephrine 1:200,000 in saline (n = 10), 2) lidocaine 1.5 per cent (n = 10), or 3) bupivacaine 0.75 per cent (n = 10).

After completing the intravenous injections and allowing the heart rates of the 185 receiving epinephrine to return to control levels, we inserted a needle into the epidural space and injected a 3-ml test dose of the local anesthetic solution with 1:200,000 epinephrine. If, after two minutes, there was no change in heart rate or no perception or evidence of anesthesia as indicated by response to Allis forceps pinch of the buttocks as compared to the neck, 16 to 25 ml of the local anesthetic solution was injected into the epidural space.

The data were analyzed using the t test for unpaired data.

### RESULTS

All 10 volunteers had a typical epinephrine response, which included circumoral pallor, palpitation, and nervousness. The maximum mean increase in heart rate and systolic blood pressure was 31.6 ± 7 beats/min (P < 0.0001) and 21.8 ± 14 torr (P < 0.0001), respectively. These signs and symptoms occurred within 20–40 s and lasted about 3 min, except for nervousness, which lasted up to 10 min.

No epinephrine response occurred in those 40 patients who received 3 ml of local anesthetic drug with no epi-

<table>
<thead>
<tr>
<th>Drugs (N = 25 for each drug)</th>
<th>Heart Rate (Beats/Min)</th>
<th>Time from Injection to Start of Increase in Heart Rate (s)*</th>
<th>Duration of Maximum Increase in Heart Rate (s)*</th>
<th>Time Elapsed from Slowing of Maximum Heart Rate Until Control Rate Approached (s)*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control (Prior to Injection of Epinephrine)*</td>
<td>After Epinephrine*</td>
<td></td>
<td></td>
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<tr>
<td>Bupivacaine (Marcaine®)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>0.75 per cent</td>
<td>76 ± 13</td>
<td>109 ± 13*</td>
<td>24 ± 7</td>
<td>25 ± 8</td>
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<tr>
<td>commercially prepared†</td>
<td>81 ± 18</td>
<td>111 ± 17*</td>
<td>24 ± 7</td>
<td>37 ± 19</td>
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<tr>
<td>mixed by anesthesiologist‡</td>
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<tr>
<td>Chloroprocaine (Nesacaine-CE®) 3.0 percent</td>
<td>82 ± 14</td>
<td>121 ± 17*</td>
<td>22 ± 5</td>
<td>43 ± 19</td>
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<tr>
<td>mixed by anesthesiologist‡</td>
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<tr>
<td>Lidocaine (Xylocaine®) 1.5 per cent</td>
<td>78 ± 13</td>
<td>109 ± 14*</td>
<td>23 ± 4</td>
<td>38 ± 20</td>
</tr>
<tr>
<td>commercially prepared†</td>
<td>80 ± 14</td>
<td>108 ± 16*</td>
<td>22 ± 6</td>
<td>20 ± 10</td>
</tr>
<tr>
<td>commercially prepared§</td>
<td></td>
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</tr>
<tr>
<td>mixed by anesthesiologist‡</td>
<td>81 ± 11</td>
<td>111 ± 11*</td>
<td>24 ± 5</td>
<td>27 ± 7</td>
</tr>
<tr>
<td>Mepivacaine (Carbocaine®) 1.5 per cent</td>
<td>77 ± 14</td>
<td>109 ± 19*</td>
<td>22 ± 6</td>
<td>25 ± 15</td>
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<tr>
<td>mixed by anesthesiologist‡</td>
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</tr>
<tr>
<td>ALL DRUGS (175 Patients)</td>
<td>79 ± 14</td>
<td>111 ± 15*</td>
<td>23 ± 6</td>
<td>31 ± 17</td>
</tr>
</tbody>
</table>

* Means ± SD.
† Not autoclaved prior to use.
‡ Both drugs autoclaved prior to mixing by anesthesiologist. Autoclaving—114°C (270°F), under 13.5 kg (30 lbs) pressure for 28 min
§ Autoclaved prior to use.
¶ P < 0.0001.
neprhin intravenously. Heart rates did not change. When questioned, 27 of the patients (68 per cent) reported changes such as feeling relaxed, drowsiness, or buzzing in their ears. The others had no symptoms from the injection.

The heart rates of the 175 patients who received the local anesthetic plus epinephrine increased from a mean of 79 ± 14 to 111 ± 15 beats/min \((P < 0.0001)\), starting within 23 ± 6 seconds after injection. The rate returned to approximately the control rate in 32 ± 33 seconds (table 1). When these patients were aroused and questioned during the increased heart rate, only 56 (32 per cent) reported a response, namely, palpitation. The others simply said they were sleepy.

Scrutiny of the electrocardiograms revealed a slowing of the heart rate prior to the subsequent increase in 34 patients (19 per cent). And, during its increase in 88 patients (50 per cent), one or more of the following occurred: junctional rhythm \((N = 7)\); premature ventricular contractions \((N = 3)\); atrial tachycardia \((N = 2)\); slowing \((N = 1)\); T wave flattening \((N = 39)\); T wave inversion \((N = 38)\); ST depression \((N = 4)\); and bigeminy \((N = 1)\). These irregularities reverted to the control pattern as the heart rate returned to control rate. In the investigation of the 30 additional patients, only those receiving 0.015 mg epinephrine had electrocardiographic abnormalities similar to those observed in some of the 175 surgical patients.

In none of the 245 surgical patients did the 3-ml test dose injected into the epidural space prior to administering the therapeutic dose raise the heart rate, nor did evidence of a spinal block result. Therapeutic doses of the local anesthetic solution also did not result in systemic toxicity or spinal anesthesia.

During the study, three of the heavily medicated patients had been taking propranolol, which had been discontinued two days prior to surgery. The 0.015 mg epinephrine intravenously evoked no electrocardiographic changes. Therefore, after 15 minutes had elapsed they were challenged with another 0.015 mg and their blood pressures were also monitored. Again, no electrocardiographic changes occurred, but their systolic and diastolic pressures rose 50 to 70, and 20 to 25 torr, respectively, 50–60 seconds following the injection. No subjective evidence of the injection resulted. These patients were excluded from the study and three others were added.

**DISCUSSION**

Although the 3-ml doses of the local anesthetic drugs used in this study should produce evidence of spinal anesthesia within two minutes, they did not provide definitive evidence of an intravascular injection. We found that adding 0.015 mg epinephrine to the 3 ml of the local anesthetic drug (the amount in a 1:200,000 epinephrine concentration) compensated for this inadequacy by resulting in a recognizable epinephrine reaction of short duration. An excellent method of detecting an epinephrine reaction, especially in the medicated patient, while performing an epidural block is to observe the electrocardioscope. Although blood pressure readings were determined in the volunteers, the 40 unmedicated patients, and the first 10 premedicated patients, they were not taken in the remaining patients because doing so when executing an epidural block using a sterile technique (anesthesiologist gloved) requires another person, and frequently one is not available. However, since beta-blockers negate the increase in heart rate from epinephrine but not the blood pressure rise, the latter must be monitored when a test dose is used in a patient taking such a drug if an intravascular injection is to be detected.

The electrocardiographic abnormalities produced by 0.015 mg epinephrine were transient and therefore apparently constituted no contraindication to its intravenous injection.

We conclude that for a single test dose of a local anesthetic solution to be of value in signalling in all patients the possibility of an intravascular or subarachnoid injection while performing epidural block, it must contain 0.015 mg epinephrine and a milligram dose of the local anesthetic drug which rapidly results in evidence of spinal anesthesia. Furthermore, it must be monitored meticulously, preferably with an electrocardioscope. If the effects of a beta-blocker are present, then the blood pressure must also be monitored.

This investigation validates that when a test dose containing a small quantity of epinephrine is injected intravascularly, an epinephrine response, namely, an increase in heart rate, results in all patients unless the effects of a beta-adrenergic blocker are present. Furthermore, it confirms that beta-blockers negate the heart rate increase of such a response but not the increase in blood pressure.

If the local anesthetic solution to be used for the therapeutic dose in epidural block does not contain the necessary components, then one that does should be employed as a test dose. In an intermittent (continuous) regional anesthetic technique, not only should the initial therapeutic dose be preceded by an effective test dose, but so should reinforcing (refill, “top-up”) doses, because the plastic tubing may penetrate the dura or the wall of a blood vessel between doses. The authors thank Robert M. Paine, M.D., cardiologist, and Linda L. Riley, Assistant Supervisor, Cardiac Laboratory, of The Mason Clinic, for interpretation of the electrocardiograms.

The Use of Compressed Air Made Easy—A Table

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Nitrous oxide, oxygen, and muscle relaxants are often used to anesthetize preterm and full-term newborn infants. Nitrous oxide is used to dilute the oxygen to prevent retrolental fibroplasia1,2 and to provide analgesia. Occasionally, these infants are unable to tolerate the cardiovascular effects of nitrous oxide.3,4 If so, the nitrous oxide concentration can be lowered by using compressed air and oxygen. This trimix technique can be supplemented with intravenous narcotic analgesia as necessary. The immediate change from a nitrous oxide–oxygen to a nitrous oxide–compressed air–oxygen technique requires determining the component gas liter flows required for a three-gas mixture of nitrous oxide, oxygen, and air for a specific total liter flow.

A table (table 1) is provided that gives the liter flows of nitrous oxide, oxygen, and air necessary for a 3 l/min total flow (three-gas mixture). The desired FiO₂ and the FNO₂ can be correlated with the flow settings on the table. Using a trimix with compressed air allows a decrease in nitrous oxide (FNO₂) while maintaining simultaneously a low FiO₂.

The anesthesiologist can select the desired concentration of nitrous oxide and oxygen required for the clinical situation and match the vertical and horizontal columns to find the needed settings for the anesthesia machine. If a change is required, reselecting the columns as desired will provide the desired mixture. Ideally, the maintenance of proper oxygen levels could best be accomplished by the monitoring of the PaO₂ of the infant during the procedure. Arterial blood gases would provide the most accurate monitoring tool. However, if an arterial line is not available, intermittent arterial sticks could be done, or a transcutaneous oxygen monitor could be used.

The table’s figures should be used in conjunction with an oxygen monitor in the circuit to provide a check on both the selection of the correct settings of the liter flows of the three gases, and on the actual delivered oxygen concentration. The settings given in the table are correct to the ±0.1 FiO₂ and FNO₂ values. If a total liter flow other than the three liters on the table is desired, flexibility can be achieved in two ways: 1) The ratio obtained by dividing the new desired total liter flow by three, multiplied by the contents of any selected square will give the liter flow settings required for the new total liter flow, while maintaining the same FiO₂ and FNO₂. 2) A similar computer-generated set of tables for total liter flows equal to 2.0, 2.5, 3.0, 3.5, 4.0, 4.5, and 5.0 are available by mail on request. Finally, it could be argued that since an oximeter is used routinely during an anesthetic, one could adjust the flows of the three gases until the desired mixture is achieved. This is a time-

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Key words: Oxygen: blood levels; delivery systems. Monitoring: oxygen. Anesthetics, gases: nitrous oxide.