In conclusion, we as anesthesiologists should be aware of the potential dangers of drug interactions and that lidocaine containing epinephrine produces hypokalemia. Therefore, we must closely observe not only the patient, but also the ECG during operation even with regional nerve block.

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REFERENCES


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The Needle Tilt Test: An Aid to Epidural Needle Insertion

To the Editor—Many techniques and devices have been recommended to help identify the loss of resistance that signals that a needle has entered the epidural space. But uncertainty often arises earlier when an advancing needle is passing through compliant tissue spaces.
fig. 1. Tilting the needle during insertion compresses soft tissue, increasing resistance to injection. 

or planes that offer little resistance to injection. We need to distinguish these spaces from the epidural space. The needle tilt test dispels this uncertainty. It tells us reliably when a needle has not yet entered the epidural space.

Resistance to injection is tested before and during a firm sideways tilting of the needle, in a direction opposite to the bevel (fig. 1). Tilting compresses the surrounding soft tissue against the side-facing bevel, and creates an unmistakable increase in resistance to injection. This artificially increased resistance can be produced with the bevel facing in any direction, but most noticeably when it is facing the unyielding interosseous ligament. If the test is positive, i.e., when tilting increases resistance, the needle can be confidently advanced further. After the bevel has entered the epidural space, tilting does not affect resistance to injection.

This test for needle position is laughably simple. It requires no devices and is performed in a moment. When there is doubt, it tells unfailingly what we need to know: whether to advance the needle further. In my hands, it has surpassed all other tests for verifying needle position.

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Blood Flow Limits and Signal Detection Comparing Five Different Models of Pulse Oximeters

To the Editor—Lawson et al.1 recently described a protocol that compared relative laser Doppler blood flow and the percent of systolic blood pressure at which a pulse oximetry (Nellcor Inc., Hayward, CA) fails to detect a signal during tourniquet occlusion of a limb. We utilized a similar protocol, to compare five different pulse oximeters during periods of increasing and decreasing tourniquet occlusion using a mercury manometer blood pressure cuff (Nellcor model 200, Hayward, CA; Novametrix 500 and 505, Wallingford, CT; Ohmeda Box 3700, Boulder, CO; and Criticare Poet, Milwaukee, WI). As in Lawson's study, we used a laser Doppler flow probe (Laserflo, TSI, St. Paul, MN) to measure relative changes in blood flow during different occlusion pressures. We were interested in comparing different models of pulse oximeters, which utilize different pulse detection algorithms, to determine the percent of systolic blood pressure and relative blood flow at which they fail to detect a pulse and report saturation data. The occlusion pressures and Doppler blood flows at which the pulse oximeters lost and regained a signal in our study are presented in tables 1 and 2.

Lawson et al.1 found that the Nellcor pulse oximeter failed to sense at a mean occlusion pressure which was 96% of control systolic pressure and a relative flow that was 8.6 ± 5.9% of baseline. We found that the five pulse oximeters studied showed failure signals at slightly higher systolic pressures (100–108% of baseline). Furthermore, the pulse oximeters studied were nearly identical in performance, although they have different pulse detection and saturation algorithms. We found no significant difference with Nellcor’s EKG lockout feature or Novametrix 505’s “super bright” LED. The pulse oximeters in this study continued to display saturation data very close to baseline values (98–100%) during progressive tourniquet occlusion until just prior to signal failure. The last saturation obtained before loss of signal detection was 95% or greater in 73% of our trials. We found that when the saturation did decrease below this level, the decrease was rapid and the pulse was inaccurate compared with that from the simultaneously monitored EKG. Furthermore, the oximeters displayed “low signal strength” or other alarm messages at this time.

In contrast to Lawson’s findings, we recorded higher percent of baseline flows at the time of signal failure (35 ± 18%). We also observed that the Doppler flows were quite variable at any given blood pressure, although the percent flow at which each pulse oximeter failed to sense was remarkably similar (22–28% of baseline). Unlike Lawson’s protocol,

### Table 1. Cuff Inflation Phase (12 Subjects)

<table>
<thead>
<tr>
<th>Pulse Oximeter</th>
<th>Occlusion Pressure at Signal Failure</th>
<th>% Systolic BP at Signal Failure</th>
<th>% Blood Flow at Signal Failure</th>
<th>Saturation Prior to Signal Failure</th>
<th>Baseline Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nellcor 200</td>
<td>132 ± 10</td>
<td>106 ± 5</td>
<td>24 ± 17</td>
<td>96.6 ± 2.5</td>
<td>100</td>
</tr>
<tr>
<td>CSL-Poet</td>
<td>135 ± 11</td>
<td>107 ± 4</td>
<td>23 ± 21</td>
<td>95 ± 2.1</td>
<td>98</td>
</tr>
<tr>
<td>Novametrix 505</td>
<td>127 ± 10</td>
<td>101 ± 4</td>
<td>28 ± 18</td>
<td>96.7 ± 2.6</td>
<td>98</td>
</tr>
<tr>
<td>Novametrix 500</td>
<td>126 ± 12</td>
<td>108 ± 6</td>
<td>22 ± 16</td>
<td>97 ± 1.4</td>
<td>98</td>
</tr>
<tr>
<td>Blox 3700</td>
<td>135 ± 12</td>
<td>108 ± 6</td>
<td>22 ± 16</td>
<td>95 ± 2.3</td>
<td>98</td>
</tr>
</tbody>
</table>

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